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CONTAGION EXPOSURE AND PROTECTION TECHNOLOGY

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Abstract

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1 Introduction

An undesired side-effect of the agglomeration of human population over the last centuries has been the ease of propagation of infectious diseases. The bubonic plague repeatedly decimated London's population in the 16th and 17th centuries.[1] The Spanish influenza epidemic of 1918-1920, with an estimated global toll of 50 million lives, featured higher death rates in more densely populated areas.[2] High population density contributed to the rapid spread of the 2009 influenza pandemic in Mexico, where the pandemic originated.[3] The potential propagation of infectious diseases due to greater exposure to contagion will almost certainly continue to be a challenge in the future, as the fraction of people living in cities is set to increase both in developed and in developing nations.[4] As economies and individuals become increasingly reliant on digital networks, these concerns also extend to cybersecurity.[5] The general prospect of increasing exposure in physical and online environments raises both positive and normative questions. Should we expect more intense exposures to inevitably lead to higher prevalence of infections? To what extent will policy interventions be desirable?

The changes in exposure associated with urbanization and digitalization may be beyond an individual's control, but we are not entirely passive subjects in the propagation of infection. In fact, we regularly invest in protection against contagion during our interactions. I propose an extension of the *susceptible-infected-susceptible* (SIS) model to study the equilibrium and welfare implications of the protection technology at different levels of exposure.[6]

Investments in protection typically depreciate, but when and how they do so very much depends on the context. In many cases the protection technology is essentially interaction-specific (e.g. the use of hand sanitizer or a face mask, or avoiding social encounters). Alternatively, some technologies have the form of a fixed cost that, once

[1] See e.g. Sutherland [28] and Appleby [2].

[2] The global death toll estimate is from Johnson and Mueller [23]. Using data from six censuses for 199 districts in India, the country with the highest number of deaths, Chandra et al. [4] find significant differences in death rates between low and high population density areas.

[3] Zepeda-Lopez et al. [31].

[4] The fraction of the population living in cities is projected to increase from 77% in 2011 to 86% in 2050 in developed countries, and from 47% to 64% in developing countries (United Nations [30]). Glaeser [18] postulates that advances in communication technology are likely to increase the need for offline encounters, as online and offline interactions very often complement each other.

[5] Ensuring a smooth functioning of digital networks has become a primary concern for policy makers. In the US, the 2012-2016 Infrastructure Protection Strategic Plan states that "Our Nation's critical infrastructure - both physical and cyber - is crucial to the functioning of the American economy and our way of life. [...] Our critical infrastructure is increasingly connected and interdependent and protecting it and enhancing its resilience is an economic and national security imperative" (Department of Homeland Security [9]).

[6] For an introduction to the SIS model, see e.g. Anderson and May [1].

paid, allows the individual to enjoy the benefits of protection in multiple interactions. Vaccination is a canonical example. In the context of cybersecurity, anti-virus softwares have a number of distinctive features. Since they take up processing power, they partly involve variable costs that scale up with usage. Like some vaccines, they require fixed updating costs. However, a distinctive feature of anti-virus softwares is that they may need to be re-installed upon infection.[7]

Motivated by these examples, I consider a classification that distinguishes between two "pure" types of technologies. On the one hand, those investments in protection whose expiration takes place *exogenously*, with a per-period probability that I will denote as η. Parameterized by η we encompass a wide spectrum of specific protective measures, ranging from interaction-specific protection ($\eta = 1$) to universal vaccines ($\eta = 0$).[8] On the other hand, I consider the implications of protection investments that fully depreciate (i.e. expire) upon infection. As the probability of infection is affected by others' investment on protection, in this case the expiration of protection is essentially *endogenous*.

I find that inefficiencies associated with decentralized protection decisions, and thus the desirability of interventions, critically depend on the characteristics of the available protection technology. Individual incentives to protect under exogenous expiration will increase hand in hand with exposure only if expiration takes place with low enough probability. The first main result of the paper (Theorem 1) shows that there exists a unique threshold for the durability of protection (as measured through η) such that if durability is below it (i.e. η is *above* the threshold), low-degree individuals find protection most attractive.[9] Since meetings are relatively rare, a one-off payment for protection buys low-degree individuals a relatively long (expected) stream of health premium. Together with the fact that investments in protection are substitutes, the implication is that the desirability of interventions will generally be non-monotonic in population density. Because protection decisions are substitutes, when population density is low so that prevalence is low even without protection, individuals will not protect. If population density is high, there will be reasons not to protect because protection is expensive. In between there will generally be some level of protection. While the prevalence of infections increases monotonically with population density, decentralization costs will be higher for societies that are either relatively sparse or very dense.

[7] E.g., Corrigan-Gibbs and Chen [20] and Brewer et al. [10] report the difficulties in maintaining computers protected due to reinstallation requirements in Ghana, and India and Cambodia, respectively.

[8] The term *universal* refers to vaccines that are robust to viral mutations, and so the protection provided by them never expires.

[9] Following several papers in the literature, I will denote the level of exposure to interactions as *degree*, and refer to exposure and degree interchangeably. I will use 'population density' when referring to the average degree in the population.

Individual incentives, and thus aggregate results, are tipped over if the durability of protection is on the other side of the threshold. In particular, results become a mirror image of the ones just described: with durable protection, it is the extent of protection that has a monotonic relation with population density, while steady state prevalence is non-monotonic in the society's degree. In sparse societies, individuals do not actively prevent the infection from spreading, but as societies become denser, protection becomes more attractive and prevalence may decrease as a result.

The second main result (Theorem 2) shows that there is a fundamental difference in the strategic incentives to protect if the expiration of protection is related to the infectious status: protection decisions are in this case strategic complements. If protection expires upon infection, then protection investments yield a higher bang for the buck (in terms of the expected duration of the investment) if others are investing in protection as well. This has two important implications. The first one concerns the conditions for an infection to become endemic, i.e. to remain in the population in the long run. If protection decisions are substitutes, then, regardless of the cost and effectiveness of protection, even arbitrarily low initial infection rates will spread and the disease will never be eradicated without policy interventions. In fact, infections will spread up to the point in which they are bounded either by spontaneous recovery or because protection becomes optimal. The situation is different when protection decisions are strategic complements. Protection is particularly attractive at low prevalence rates, and thus active efforts from the population may result in the disease dying out even if from a purely physiological perspective (i.e. without protection) it could become endemic.

The downside, however, is that multiple positive-infection steady states are possible, and which one is chosen is entirely up to the individuals' coordination (Theorem 3). If everyone protects, then the infection rate is low, and therefore protecting is profitable. If nobody protects, then the opposite is true. Multiple steady states exhibit a "tipping point" feature: any infection rate below some threshold will prompt full protection, while any infection rate above this threshold will unravel protection. For a policy-maker who intends to shift the society from a steady state of no protection to a steady state with full protection, I find that the effort needed to reach this tipping point will be larger the denser the society.

Table 1 summarizes the paper's main findings, showing how inefficiencies in decentralized protection at varying levels of exposure critically depend on the protection technology.

The paper lies at the intersection of two different strands of the economics literature on epidemics. A comparatively small set of papers has studied the effects of the structure of the society, as characterized by the links between individuals through which

Table 1: When can intervention be desirable?
Inefficiencies in protection

	Interaction-specific	Durable vaccine	Expiration upon infection
Dense society	High	Low	High
Intermediate	Low	Medium	Low or High*
Sparse society	High	High	Low

* Depending on individuals' coordination.

contagion can occur, on the prevalence of infections. This literature was originally initiated by researchers in other fields (mainly in physics, in the work by Pastor-Satorras and Vespignani [27]), and subsequently developed further in economics (Jackson and Rogers [22], López-Pintado [25]).[10] While these papers offer rich insights on the relevance of the social structure on disease diffusion, they abstract from the effects that individual behaviour may have on prevalence. Individual incentives in epidemiological contexts have been studied extensively by economists in the last two decades. Contributions include papers that study individual incentives to vaccinate (Francis [12] and [13], Geoffard and Philipson [17], Chen and Toxvaerd [5]), to engage in interaction-specific protection (Kremer [24], Geoffard and Philipson [16], Toxvaerd [29]), or both (Goyal and Vigier [19]). By incorporating incentives in a model where the structure of interactions matters, this paper is closest to the work by Galeotti and Rogers [15]. Galeotti and Rogers [15] propose a model where agents strategically choose whether to vaccinate in a society divided into two groups, and analyze the implications of different assortativity patterns. In the present paper I abstract from the possibility of the society being segmented and instead focus on how incentives to protect relate to the frequency of interactions, and how this relation is affected by the protection technology.

The rest of the paper is organized as follows. Section 2 presents the model. Section 3 characterizes optimal protection decisions by the individual. Section 4 presents the steady state analysis. Section 5 studies the first best solution and discusses the welfare properties of decentralized equilibria. Section 6 concludes by discussing policy implications. Appendix A contains all proofs.

[10] Further references can be found in Jackson [21], chapter 7.

2 The Model

Social encounters. There is a population of mass one of individuals. In every period t, $t = 1, \ldots$, each individual i in the population is called for an interaction with some probability $\delta_i \in [\varepsilon, 1]$, where $\varepsilon \in (0, 1]$. I assume that the society is regular, and so $\delta_i = \delta$ for all i.[11] Individuals who are called to interact are randomly matched in pairs.

Contagion and protection. Individuals may be *susceptible* or *infected*. An infected individual recovers spontaneously with per-period probability $\alpha \in [0, 1]$. Susceptible individuals may become infected when interacting with an infected individual. Specifically, a susceptible individual who interacts with an infected individual gets infected with probability $p \in [p_L, p_H]$, with $0 \leq p_L < p_H \leq 1$. The protection level p is chosen by the individual, and entails a cost

$$\frac{p_H - p}{p_H - p_L} c,$$

where $c > 0$ is the constant marginal cost of protection. Protection is temporary, in the sense that it may expire. Two different expiration processes are considered:

(a) *Exogenous expiration*. Protection expires with per-period probability $\eta \in [0, 1]$.

(b) *Endogenous expiration*. Protection expires if the individual becomes infected.

Preferences. In periods in which the individual is susceptible, she earns a gross payoff of π_S. When she is infected, the gross payoff is of $\pi_I < \pi_S$. The per-period net payoff u_t at time t is equal to the gross payoff minus protection spending in that period. I assume that individuals are infinitely patient and thus seek to maximize their expected per-period payoffs (see e.g. Fudenberg and Tirole [14], pp. 148-9):[12]

$$\lim_{T \to \infty} \mathbb{E} \frac{1}{T} \sum_{t=0}^{T} u_t. \tag{1}$$

[11] In Appendix B I extend the steady state analysis to societies with arbitrary degree distributions. The theoretical results and numerical simulations show that the findings of the main text do not depend on the degree distribution being regular.

[12] As shown in the Appendix, this formulation of individual payoffs is obtained if agents discount the future at a rate $\beta \to 1$. The model could be written with discounting, making the problem less tractable without changing the qualitative results. Since the benefits of protection partly accrue in the future, with discounting the policy functions would reflect weaker incentives to protect. As the discount factor approaches one, the policy functions would get arbitrarily close to the ones presented below.

Steady state. Let $\theta_t(\delta)$ denote the probability that, in a society of degree δ, a meeting is with an infected individual. Since the society is regular, $\theta_t(\delta)$ is also the fraction of individuals who are infected at time t. The dynamics of $\theta_t(\delta)$ are given by

$$\theta_{t+1}(\delta) - \theta_t(\delta) = (1 - \theta_t(\delta))\ \delta\ \theta_t(\delta)\ p_t - \alpha\ \theta_t(\delta), \tag{2}$$

where $p_t \in [p_L, p_H]$ is the average protection level of susceptible individuals. Equation (2) describes the standard dynamics of the SIS model for regular societies. The intuition is as follows. Consider the first term on the right-hand side of (2). A fraction δ of the fraction $(1 - \theta_t(\delta))$ of susceptible individuals are called for an interaction. Of those individuals, a fraction $\theta_t(\delta)p_t$ is matched with infected individuals and becomes infected. The second term on the right-hand side of (2) represents the fraction of infected individuals of degree δ who spontaneously recover. Netting out the recovering individuals from the newly infected ones yields the change in the infection rate, i.e. the left-hand side of (2).

In a steady state, $\theta_{t+1}(\delta) = \theta_t(\delta) = \theta(\delta)$. Using (2) it follows that a steady state infection rate in a degree-δ society must satisfy

$$\alpha\ \theta(\delta) = (1 - \theta(\delta))\ \delta\ \theta(\delta)\ p_t = (1 - \theta(\delta))\ \delta\ \theta(\delta)\ p, \tag{3}$$

where the second equality follows from the fact that, if $\theta_t(\delta)$ is time independent, then p_t must be as well. Let $p^*(\theta)$ denote an optimal protection level that is chosen by degree-δ individuals when the steady state probability of meeting an infected individual equals θ.

Definition 1. *A steady state is a pair* $(\theta^{ss}(\delta), p^*(\theta^{ss}))$ *such that*

$$\alpha\ \theta^{ss}(\delta) = (1 - \theta^{ss}(\delta))\ \delta\ \theta^{ss}(\delta)\ p,$$

where $p = p^*(\theta^{ss})$.

Notice the fixed-point nature of a steady state. An infection rate corresponds to a steady state if and only if it prompts an individual behaviour that results in that particular infection rate.

It is worth noting that, for any degree distribution and set of parameters, there is always a steady state with no infection and no protection. The interest will thus be in understanding the circumstances under which a positive infection rate can be sustained. When a steady state with positive infection exists, then the infection rate is given by

$$\theta^{ss}(\delta) = 1 - \frac{\alpha}{\delta\ p^*(\theta^{ss})}, \tag{4}$$

where $\alpha < \delta p^*(\theta^{ss})$.

3 Optimal Protection Decisions

This section studies the individual's incentives to protect taking the aggregate as given. The problem is greatly simplified by the fact that, due to homogeneous mixing, there is a single aggregate state variable, θ_t, that summarizes all that the individual is interested in with regards to the society. Thus, the solution to the individual problem does not depend directly on the society's degree distribution. Furthermore, since individuals are infinitely patient, they care about long-run probabilities. This implies that the relevant probability of meeting an infected individual is the steady state level θ^{ss}.

3.1 Optimal protection under exogenous expiration

The following result provides a general characterization of individual protection decisions under exogenous expiration.

Theorem 1. *Suppose that protection expires with per-period probability $\eta > 0$, and that the economy converges to a steady state $\theta^{ss} > 0$ in the long run. For sufficiently small θ^{ss} protection is never optimal. Furthermore, there exists a unique $\bar{\eta} \in (0,1)$ such that:*

(a) If $\eta > \bar{\eta}$ and protection is profitable for some degree, then a degree-δ individual protects if and only if $\delta \in [0, \bar{\delta}]$ for some $\bar{\delta} \in [0,1]$.

(b) If $\eta < \bar{\eta}$ and protection is profitable for some degree, then a degree-δ individual protects if and only if $\delta \in [\underline{\delta}, \bar{\delta}]$ for some $\underline{\delta}, \bar{\delta} \in [0,1]$. For sufficiently effective protection (sufficiently low p_L), $\bar{\delta} = 1$.

A few observations are in order with respect to this result. First, protecion is never optimal from the individual's perspective if aggregate infection is sufficiently small. This is a manifestation of the substitutability of investment decisions at low infection rates. The thought exercise is if a sufficiently large fraction of the population decided to protect, causing aggregate infection to drop so much as to make protection unattractive from the individual's perspective.

Secondly, it is clear from Theorem 1 that whether more intense exposure leads to more protection depends on the durability of the latter. In particular, low-degree individuals find protection more attractive if protection expires frequently. The intuition is straightforward: since meetings are relatively rare, a one-off payment for protection buys a relatively long (expected) stream of health premium. If, however, durability is above

the threshold ($\eta < \bar{\eta}$), then incentives are tipped over and protection may be profitable for individuals facing relatively more intense exposure.

Theorem 1 leaves open the possibility that, if protection is long-lasting but not sufficiently effective (i.e. $p_L > 0$), individuals with high exposure will prefer to remain unprotected. This feature is closely related to the concept of *rational fatalism* (Kremer [24]), whereby agents facing high enough probability of becoming infected may give up on any costly preventive measure.

While the probability of becoming infected is a function both of the individual's degree δ and the aggregate infection rate θ, the fatalistic behavior just described is in relation to the former only. Whether incentives to protect increase monotonically with the aggregate steady-state infection rate (so that protection decisions are *global* substitutes) depends not only on the effectiveness of protection (p_L), but also on the extent to which it can be used in multiple interactions (η). The following two corollaries provide an explicit characterization of optimal protection decisions in two prominent instances: when protection is interaction-specific ($\eta = 1$), and when protection is long-lasting and perfect (small η and $p_L = 0$). In both cases we find that protection incentives increase monotonically with aggregate infection. In other words, if an individual finds protection optimal under a steady-state with infection θ, it will also find it optimal under steady-state infection $\theta' > \theta$.

Corollary 1. *(Interaction-specific protection) Suppose protection is interaction-specific ($\eta = 1$), and that the economy converges to a steady state $\theta^{ss} > 0$ in the long run. An individual of degree δ optimally chooses:*

(1) full protection if $\delta < \delta_X^*(\theta^{ss})$,

(2) any protection level if $\delta = \delta_X^*(\theta^{ss})$,

(3) no protection if $\delta > \delta_X^*(\theta^{ss})$,

where

$$\delta_X^*(\theta) := \frac{\theta(p_H - p_L)(\pi_S - \pi_I) - c\alpha}{c\theta p_H}. \tag{5}$$

Corollary 2. *(Durable and perfect protection) Suppose that protection is perfect and expires with per-period probability $\eta < \frac{1}{1+c/(\pi_S - \pi_I)}$, that individuals either fully protect or do not protect, and that the economy converges to a steady state $\theta^{ss} > 0$ in the long run. An individual of degree δ optimally chooses:*

(1) full protection if $\delta > \delta_X^{**}(\theta^{ss})$,

(2) full protection or no protection if $\delta = \delta_X^{**}(\theta^{ss})$,

(3) no protection if $\delta > \delta_X^{**}(\theta^{ss})$,

where

$$\delta_X^{**}(\theta) := \frac{\eta\left[\alpha c - \theta p_H(\pi_S - \pi_I)\right]}{\theta p_H\left[(1-\eta)(\pi_S - \pi_S) - \eta c\right]}. \tag{6}$$

These corollaries offer a clear contrast on the qualitatively different incentives that may emerge depending on the durability of protection.[13] For illustration, consider the two panels in Figure 1. If protection is interaction-specific, then low-degree individuals find protection most attractive. For a fixed degree, incentives to protect are increasing in the probability of meeting an infected individual (strategic substitutes). This case is shown in panel (a). If protection is not interaction-specific but expires with a high-enough exogenous probability (relative to the cost-benefit ratio $c/(\pi_S - \pi_I)$), then incentives are also as shown in this panel. If, on the other hand, protection expires with low-enough exogenous probability, then high-degree individuals find protection most attractive. For a fixed degree, incentives to protect decrease in the probability of meeting an infected individual (substitutes). This case is depicted in panel (b). It follows that, in the context of contagious diseases among humans, the development of a new vaccine will produce qualitatively different results only if the protection it provides is sufficiently long-lasting. Section 4 will expand upon this point.

3.2 Optimal protection under endogenous expiration

Protection decisions have a number of distinctive features if protection expires upon infection, as shown by the next result.

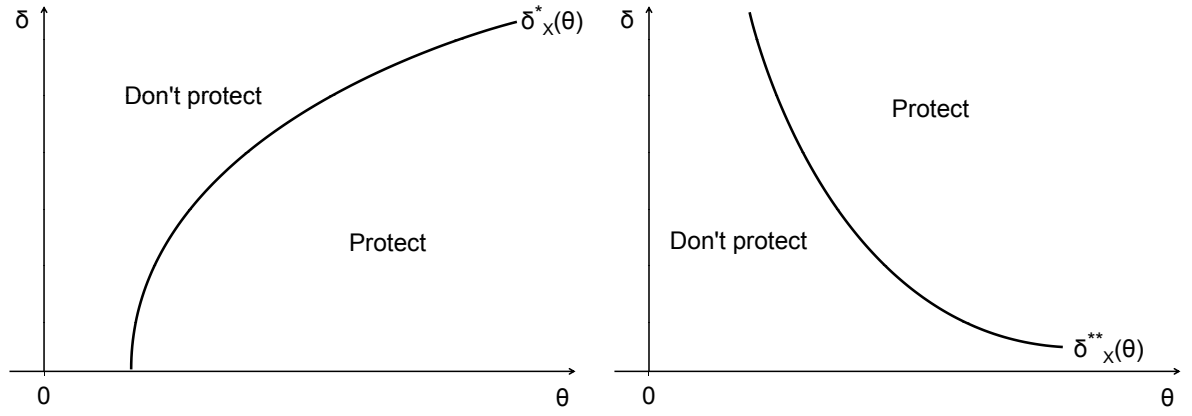
Theorem 2. *Suppose that protection expires if the individual becomes infected, and that the economy converges to a steady state* $\theta^{ss} > 0$ *in the long run. An individual of degree* δ *optimally chooses:*

(1) full protection if $\delta < \delta_N(\theta^{ss})$,

(2) any protection level if $\delta = \delta_N(\theta^{ss})$,

(3) no protection if $\delta > \delta_N(\theta^{ss})$,

[13] The assumptions on protection being perfect and individuals choosing either full or no protection ensure that in the long run an individual who protects will spend positive fractions of time only in healthy states, where the set of such states is the unique closed communicating class of the Markov chain describing the individual's transitions. This allows obtaining the invariant distribution from which the individual's policy function is derived.



(a) (b)

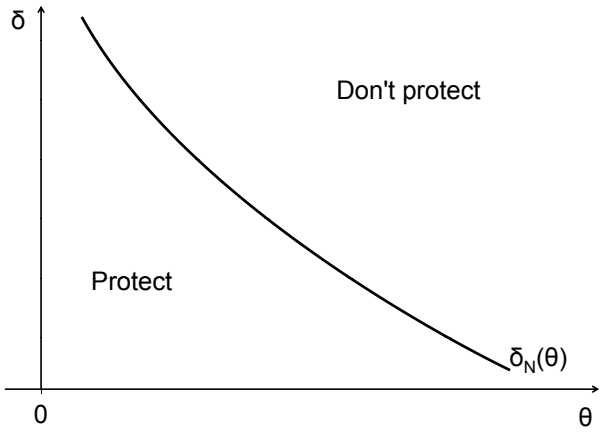
Figure 1: Optimal protection decisions. Left panel: interaction-specific protection. Right panel: sufficiently durable vaccination.

where

$$\delta_N(\theta) = \frac{(p_H - p_L)(\pi_S - \pi_I)}{c\theta p_H p_L} - \frac{\alpha}{\theta p_H}. \tag{7}$$

Clearly, if protection guarantees that no infection will ever take place ($p_L = 0$), then an individual will choose to protect regardless of her degree. This results from the assumption that individuals are infinitely patient: a one-off payment of c achieves protection forever. Since future payoffs are not discounted, this one-off payment is negligible and protection is always attractive. More surprisingly, we find that the individuals who have lower degree are the ones with the strongest incentives to protect. One would have thought that individuals with many interactions should benefit most from protection, as the fixed cost of protection implies a lower average cost of protection per interaction. On the other hand, individuals with more frequent interactions become infected more often, and are therefore forced to frequently spend resources if they wish to remain protected. Theorem 2 shows that this second effect dominates, and low degree individuals have relatively stronger incentives to protect.

Finally, consider the strategic incentives to protect. Note that if $\delta_X(\theta)$ is positive (so that there might be individuals who protect), then it is decreasing in θ. Thus, for a fixed degree δ, we find that the individual will choose to protect if θ is low enough, and may switch to no protection as θ increases. It is worth paying the cost of protecting in each interaction only if the individual is relatively unlikely to meet an infected person. Since a low level of θ in the population is associated with people protecting, it follows that



(a)

Figure 2: Optimal protection decisions when protection expires upon infection.

protection decisions are in this case strategic complements.

It is well-known that strong complementarities can result in multiple equilibrium outcomes (Cooper and John [6]). Understanding how the strength of complementarities relates to the parameters of the model is therefore important. The strength of the effect of others' protection decisions is captured by

$$\left|\frac{d\delta_N(\theta)}{d\theta}\right| = \left|-\frac{1}{\theta^2}\ \frac{(p_H - p_L)(\pi_S - \pi_I) - \alpha c p_L}{c p_H p_L}\right|.$$

Note that the interesting case is when $(p_H - p_L)(\pi_S - \pi_I) - \alpha c p_L > 0$, or otherwise $\delta_N(\theta) \leq 0$ and there can never be protection. Under this condition, it is easy to see that complementarities are stronger: the larger the health premium, the more infectious the disease (higher p_H), the more effective the protection technology (lower p_L), the lower the cost of protection, and the lower the recovery rate.

For comparability with the previous section. Figure 2 presents a stylized characterization of how protection decisions depend on individual degree and the (steady state) probability of meeting an infected individual when protection expires upon infection. While low-degree individuals find protection most appealing, incentives to protect are decreasing in the probability of meeting an infected individual (complements).

4 Decentralized Equilibria

What are the aggregate implications of individual behaviour, and how do these depend on the available protection technology? This section addresses these questions. For the results that follow, it will be useful to define

$$\begin{aligned} \theta_l^{ss}(\delta) &= 1 - \frac{\alpha}{\delta p_L}, \\ \theta_h^{ss}(\delta) &= 1 - \frac{\alpha}{\delta p_H}. \end{aligned}$$

These quantities give, respectively, lower and upper bounds for steady state infection rate θ^{ss}.

4.1 Exogenous expiration

As seen in Section 3, closed-form solutions for indivudals' optimal protection policies are cumbersome to characterize for general parameter values in the case of exogenous expiration of protection. However, two particularly relevant cases have simple characterizations, and in this section we focus on the steady-state properties of these cases.

For any set of real numbers $\{a_1, \ldots, a_n\}$, let the *median operator* $M\{a_1, \ldots, a_n\}$ return the median value of the set. We can now give the characterization of the steady state when protection lasts for a single interaction.

Proposition 1. *(Interaction-specific protection) Consider a regular society with degree δ, and suppose that protection is interaction-specific ($\eta = 1$). A steady state with positive infection rate exists if and only if $p_H > \alpha/\delta$. The unique such steady-state infection rate is characterized by:*

$$\theta^{ss}(\delta) = M\Big\{\theta_l^{ss}(\delta), {\delta_X^*}^{-1}(\delta), \theta_h^{ss}(\delta)\Big\}.$$

If a positive steady-state infection rate exists, then it is increasing in δ.

Notice that the condition for existence of a positive-infection steady state depends on the physiological characteristics of the disease (as captured by p_H and α) and the society's density (as measured by δ). Remarkably, the condition is unrelated to the effectiveness of protection and its cost. Protection can even be perfect and arbitrarily cheap and yet the disease will remain in the population as long as $p_H > \alpha/\delta$. The result follows from the fact that protection decisions are strategic substitutes: when prevalence is small, there are no incentives to protect. As a result, any small initial infection rate will spread in

the population until it is bounded either because people spontaneously recover or because protection becomes optimal.

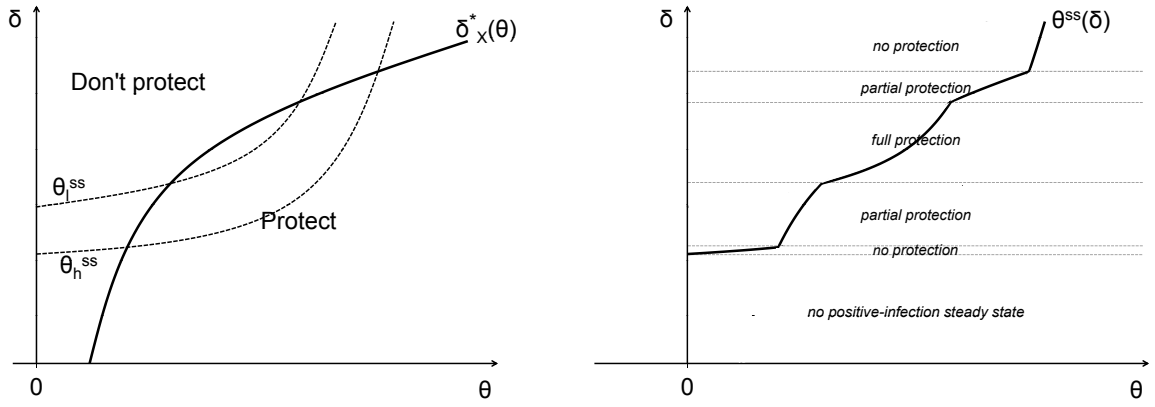
The intuition for the expression of the unique positive steady state neighbour infection rate is as follows. While $\delta_X^*(\theta)$ is the degree that is indifferent between any protection level given a probability θ of meeting an infected individual, the inverse function ${\delta_X^*}^{-1}(\delta)$ gives the infection rate at which a degree-δ individual is indifferent between any protection level. Moreover, since protection decisions are substitutes, for any infection rate below (above) ${\delta_X^*}^{-1}(\delta)$ no protection (full protection) is strictly preferred. Thus, suppose for example that $0 < {\delta_X^*}^{-1}(\delta) < \theta_l^{ss}(\delta) < \theta_l^{ss}(\delta)$. It is easy to see that no infection rate strictly larger than $\theta_l^{ss}(\delta)$ can be a steady state: facing this probability of meeting an infected person, all susceptible individuals would protect, which is not consistent with the infection rate being higher than $\theta_l^{ss}(\delta)$. The only possible positive steady-state infection rate is $\theta_l^{ss}(\delta)$, and in this steady state all susceptible individuals protect.

The last statement of the proposition states that steady state infection is increasing in the density of the society. This does not mean, however, that people protect less as the society becomes denser. The relation between population density and protection is in fact non-monotonic. Figure 3 illustrates how these features can be reconciled. Panel (a) shows the functions $\theta_l^{ss}(\delta)$ and $\theta_l^{ss}(\delta)$ alongside the threshold $\delta_X^*(\theta)$ of the policy function. It is easy to identify, for a given degree δ, the unique positive steady-state infection rate (if it exists). For example, points on the curve $\theta_l^{ss}(\delta)$ can represent steady-state levels of infection only if they lie below $\delta_X^*(\theta)$, indicating that it is in fact optimal for individuals to fully protect. Following this intuition, panel (b) shows how prevalence is increasing in the degree of the society. For very low degrees, any initial infection dies out even if individuals do not protect. As degree increases, there can be positive infection in steady state, and this level of prevalence is low enough that individuals do not find protection attractive enough. For somewhat higher degrees, however, protection starts to become appealing: either all individuals fully protect, or there is a steady state with partial protection.[14] Eventually, however, protection becomes too costly (because protection costs are scale up with exposure), and people do not protect.

Let us now consider the case of sufficiently-durable and perfect protection. To preserve the analogy with Proposition 1, let us assume here that $\theta_l^{ss}(\delta) = -\infty$.[15] The characterization of the steady state is as follows.

[14] In a steady state with partial protection, a fraction of the population fully protects and the rest remain unprotected, or all individuals use a level of protection $p \in (p_L, p_H)$.

[15] Since protection is perfect (i.e. $p_L = 0$), $\theta_l^{ss}(\delta)$ is in fact undefined. We are taking $\theta_l^{ss}(\delta)$ to be $\lim_{p \to +0} 1 - \frac{\alpha}{\delta p}$.



(a) (b)

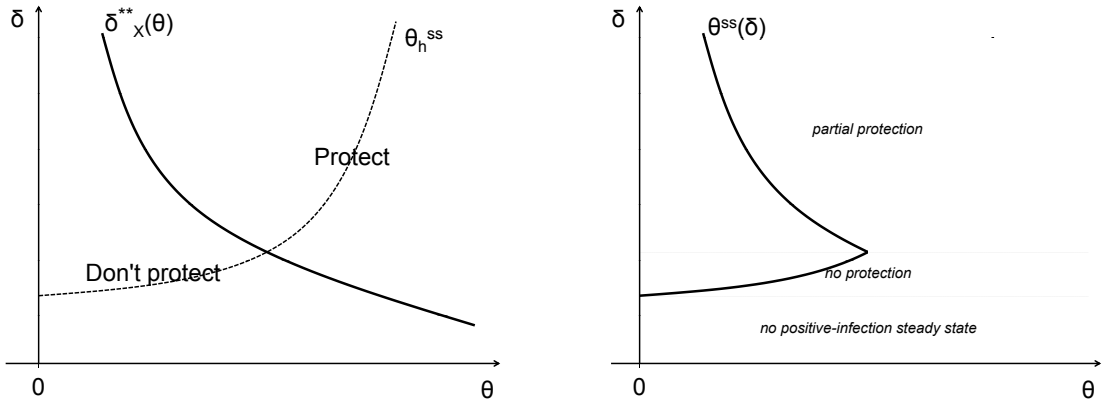
Figure 3: Steady state infection rate in regular societies if protection is interaction-specific. If a positive-infection steady state exists, then it is increasing in the society's degree. Protection spending will in general be non-monotonic in degree.

Proposition 2. *(Durable and perfect protection) Consider a regular society with degree δ. Suppose that protection is perfect and expires with per-period probability $\eta < \frac{1}{1+c/(\pi_S-\pi_I)}$, and that the individual can choose either full protection or no protection. A steady state with positive infection rate exists if and only if $p_H > \alpha/\delta$. The unique such steady state infection rate is characterized by:*

$$\theta^{ss}(\delta) = M\Big\{\theta_l^{ss}(\delta), \delta_X^{**-1}(\delta), \theta_h^{ss}(\delta)\Big\}.$$

If a positive-infection steady state exists, then it is increasing in δ if $\theta^{ss}(\delta) = \theta_h^{ss}(\delta)$, and decreasing in δ otherwise, while protection spending is weakly increasing in δ.

Notice how the results in this case mirror those under interaction-specific protection: with durable protection, it is the extent of protection that has a monotonic relation with population density, while steady state prevalence is non-monotonic in the society's degree. The intuition is simple. In sparse societies, individuals do not actively prevent the infection from spreading. As societies become denser, protection becomes attractive. So much so that prevalence may be lower in dense societies than in sparse ones. The result is illustrated in Figure 4. For sufficiently low degrees, any infection dies out even if nobody protects. For intermediate degrees, the infection remains in the population, but is not high enough to prompt protection. When interactions are very frequent, protection becomes attractive and the infection is actively contained by individual's increasing spending on protection.



(a) (b)

Figure 4: Steady state infection rate in regular societies if protection expires with exogenous probability. If a positive-infection steady state exists, then it is non-monotonic in degree. Protection spending is (weakly) increasing in degree.

4.2 Endogenous expiration

As will be shown in this section, complementarities in protection decisions can give rise to multiple steady states with positive infection rates. In the present model, agents have perfect foresight and which steady state arises is up to the individuals' coordination. As a refinement, however, one could ask how individuals respond to slight perturbations of the steady state probability of meeting an infected individual. If the individual response to a perturbation is consistent with further departures from the steady state infection rate, then there is a sense in which the steady state is fragile or unstable. In this spirit, I will say that a steady-state infection rate is unstable if a small negative (positive) perturbation of the long-run infection rate prompts more (less) protection by individuals.

Definition 2. *In a regular society of degree δ, the basin of attraction of a steady state with infection rate $\theta^{ss}(\delta) = 1 - \frac{\alpha}{\delta p^*(\delta)}$ is $[\theta^{ss}(\delta) - \epsilon_1, \theta^{ss}(\delta) + \epsilon_2]$, where*

$$\epsilon_1 = \sup\left\{\epsilon \in \mathbb{R} : \textit{ for any } \theta \in [\theta^{ss}(\delta) - \epsilon_1, \theta^{ss}(\delta)] \textit{ there exists } p' \geq p^*(\delta) \textit{ s.t. } p' \textit{ is optimal}\right\},$$
$$\epsilon_2 = \sup\left\{\epsilon \in \mathbb{R} : \textit{ for any } \theta \in [\theta^{ss}(\delta), \theta^{ss}(\delta) + \epsilon_2] \textit{ there exists } p' \leq p^*(\delta) \textit{ s.t. } p' \textit{ is optimal}\right\}.$$

$\epsilon_1 + \epsilon_2$ is the size of the basin of attraction.[16] *A steady state $\theta^{ss}(\delta)$ is said to be unstable if its basin of attraction is of size zero.*

[16]The reason that the definition of ϵ_1 is based on the supremum is that the maximum of the set may not exist. In particular, if $\theta_l^{ss} > 0$ is a steady state infection rate, then individuals find full protection optimal for any $\theta \in (0, \theta_l^{ss}]$ but (obviously) not for $\theta = 0$. For the case of ϵ_2, suprema and maxima always coincide.

Steady states with positive infection rates are characterized by the following result.[17]

Theorem 3. *Consider a regular society with degree δ. Suppose that protection expires upon infection and that $0 < p_L < p_H \frac{1}{1+\frac{\alpha c}{\pi_S - \pi_I}}$. A steady state with positive infection rate exists if and only if (a) $\delta > \frac{\alpha}{p_L}$, or (b) $\delta \geq \frac{(p_H - p_L)(\pi_S - \pi_I)}{c p_L p_H}$. There are three possible non-zero steady-state infection rates:*

(1) $\theta_h^{ss}(\delta)$ is a steady-state infection rate if and only if $\delta_N^{-1}(\delta) \leq \theta_h^{ss}(\delta)$.

(2) $\theta_l^{ss}(\delta)$ is a steady-state infection rate if and only if $0 < \theta_l^{ss}(\delta) \leq \delta_N^{-1}(\delta)$.

(3) $\delta_N^{-1}(\delta)$ is a steady-state infection rate if and only if $\theta_l^{ss}(\delta) \leq \delta_N^{-1}(\delta) \leq \theta_h^{ss}(\delta)$.

A positive-infection steady state is unstable if and only if its infection rate is strictly between $\theta_l^{ss}(\delta)$ and $\theta_h^{ss}(\delta)$. If multiple steady states exist for degrees δ and $\delta' > \delta$, then the basin of attraction of the steady state with full protection (no protection) is strictly larger (strictly smaller) under δ than under δ'.

There are a number of features in this case that are evidently different from the ones considered previously. When the expiration of protection is tied up to the infectious status, the condition $p_H > \alpha/\delta$ is necessary but not sufficient for the disease to be endemic. The conditions for existence now concern not only the "physiological" parameters of the disease (p_H and α) but also the cost and effectiveness of the protection technology. Protection is particularly attractive at low prevalence rates, and thus active efforts from the population may result in the disease dying out even if without protection it would become endemic.

The second new feature is that existence does not imply uniqueness. For some degrees, different prevalence levels can be self-fulfilling. If nobody else protects, then the disease will be so widespread in the long run that it is in fact optimal not to protect. If, however, everyone protects, then protection is optimal: as the individual rarely becomes infected (thereby having to repurchase protection), investing in protection is attractive. When multiple positive-infection steady states exist, there will also be a steady state with partial protection. In its own right, this steady state is uninteresting due to its instability; a small departure from the steady state prevalence will induce an individual behaviour that will magnify the original discrepancy. The interesting feature of this infection rate is that it provides the boundary between the basins of attraction of the full protection and no protection steady states. This threshold can be interpreted as a "tipping point": any

[17] In the following result it is assumed that $0 < p_L < p_H \frac{1}{1+\alpha c/(\pi_S - \pi_I)}$. If $p_L = 0$, then infinitely patient individuals will always protect. If $p_L \geq p_H \frac{1}{1+\alpha c/(\pi_S - \pi_I)}$ then individuals will never protect. Hence the assumption is made so that the protection problem is interesting.

infection rate below this threshold will prompt full protection, while any infection rate above this threshold will unravel protection. For a policy-maker who intends to shift the society from a steady state of no protection to a steady state with full protection, the effort needed to reach this tipping point will be larger the larger the degree of the society. Figure 5 illustrates the results of Proposition 3.

Finally, let us turn to the question of whether people tend to protect more as aggregate exposure increases. It follows from the conditions of Theorem 3 that people will not protect if population density is sufficiently high, and may fully protect or not protect for intermediate densities. Full protection will be observed in sparser societies only if the cost of protection is sufficiently small, relative to the gains it provides.[18]

5 Exposure, Protection Technology, and Inefficiencies

How does the protection technology affect inefficiencies in protection? Naturally, when an individual protects, other individuals in the population benefit through an expected lower future exposure to contagion. As a necessary benchmark to understand the inefficiencies associated to this externality, the next result characterizes first-best protection.[19] The following quantities will be used in the next result:

$$\delta_X^{fb} := \text{the unique positive real root of } f(\delta), \tag{8}$$

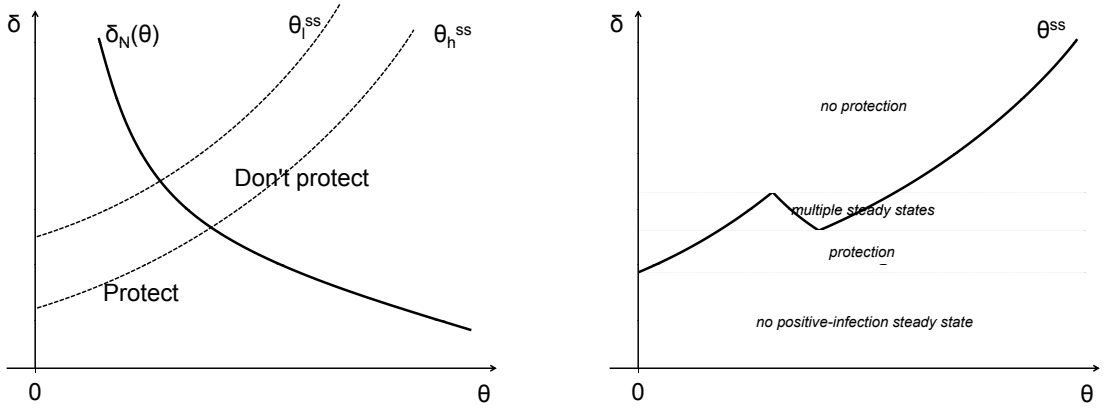
$$\delta_N^{fb} := \frac{(p_H - p_L)(\pi_S - \pi_I)}{p_L p_H c} + \frac{\alpha}{p_L}, \tag{9}$$

where

$$\begin{aligned} f(\delta) &= [p_L p_H \eta(1-\eta)c]\ \delta^2 \\ &+ \left[p_H \eta^2 c - (1-\eta)(\eta + (1-\eta)\alpha)(p_H - p_L)(\pi_S - \pi_I)\right]\ \delta \\ &- [\eta(\eta + (1-\eta)\alpha)(p_H - p_L)(\pi_S - \pi_I)]\,. \end{aligned} \tag{10}$$

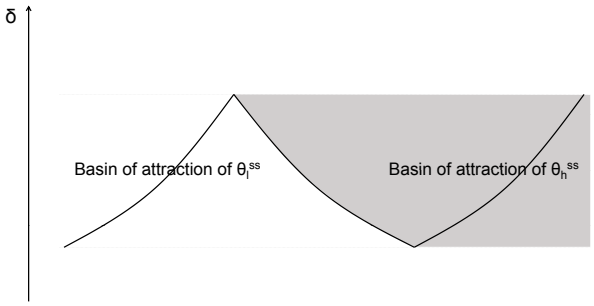
[18] Specifically, sparse societies will exhibit full protection if $c < (1 - p_L/p_H)(\pi_S - \pi_I)/\alpha$, and no protection if $c > (1 - p_L/p_H)(\pi_S - \pi_I)/\alpha$.

[19] In regular societies all individuals enjoy the same steady-state payoffs, and it is thus natural to measure welfare as the individual's expected per-period utility.



(a)

(b)



(c)

Figure 5: Steady state infection rates in regular societies if protection expires upon infection. If multiple positive-infection steady states exist, then only those with full or no protection are stable. The size of the basin of attraction of the steady state with no protection (full protection) is increasing (decreasing) in the society's degree.

Proposition 3. *Consider a regular society with degree δ. If $p_L \leq \alpha/\delta$, then full protection is the unique first best protection level. Otherwise, for protection technology $j = X, N$, first best protection is*

(1) Full protection if $\delta < \delta_j^{fb}$.

(2) Any protection level if $\delta = \delta_j^{fb}$.

(3) No protection if $\delta > \delta_j^{fb}$.

Note that, under the assumptions of the paper, the decision of the central planner is non-trivial only if $p_L > \alpha/\delta$. Since individuals are infinitely patient, whenever $p_L \leq \alpha/\delta$, it is optimal to protect everyone until the disease dies out. Thus, for example, any steady state featuring positive infection when protection is perfect ($p_L = 0$) is necessarily inefficient.

Social incentives to protect decrease with the intensity of interactions, regardless of the protection technology. It is perhaps surprising that this feature is present even if protection can be used in multiple interactions, as is the case e.g. under vaccination ($j = X$, with $\eta << 1$). Even though in this case protection costs are seemingly unrelated to exposure, first best protection depends on population density for two reasons. First, once protection expires, it is re-purchased on the first interaction. Hence, while protection expiration is orthogonal to degree, protection spending is not. Second, the *benefits* of protection are not independent of degree. Namely, the drop in prevalence resulting from protection is less dramatic the higher the degree of the society.[20]

Inefficiencies in the decentralized solution are perhaps most evident when protection is interaction-specific. For a fixed θ, the wedge between individual incentives and social objectives is given by $\delta_{X,\eta=1}^{fb} - \delta_X^*(\theta) = \frac{\alpha}{\theta p_H}$. If θ^{ss} is a steady state infection rate, then the decentralized solution is inefficient whenever $\delta_X^*(\theta^{ss}) < \delta < \delta_X^{fb}$. By this measure, inefficiencies are less likely to emerge the worse the physiological profile of the disease, i.e. the less likely recovery and the more infectious the disease. However, conditional on inefficiencies arising, their welfare implications (i.e. the discrepancy between first best and decentralized welfare) will be more severe if recovery is less likely and the disease is more infectious.

[20] To see this, recall that $\theta_l^{ss}(\delta) = 1 - \frac{\alpha}{\delta p_L}$ and $\theta_h^{ss}(\delta) = 1 - \frac{\alpha}{\delta p_H}$ denote the steady state prevalence levels with and without protection (assuming $\delta p_L > \alpha$ so that $\theta_l^{ss}(\delta) > 0$). All else equal, one can measure the benefits from protection for regular societies of different degrees by the ratio of $\theta_h^{ss}(\delta)$ to $\theta_l^{ss}(\delta)$, which (for $\delta > \frac{\alpha}{p_L}$) is decreasing in δ.

Corollary 2 in the Appendix gives a a necessary and sufficient condition for full protection to be first best for *any* degree when protection expires exogenously.

In the case where protection expires upon infection, the difference between the protection thresholds in first best δ_N^{fb} and the decentralized case $\delta_N(\theta)$ is not always positive. The reason is that for arbitrarily low values of prevalence θ, protection is infinitely attractive for the individual. This notwithstanding, it is possible to show that there can only be *under*-investment in protection in a steady state. This is established by the following corollary, that encompasses all cases studied above.

Corollary 3. *Consider a regular society of degree δ. For the classes of protection technologies considered in Propositions 1-2 and Theorem 3, if there exists a steady state with some positive level of protection, then full protection is the unique first best protection level.*

It follows from Corollary 1 that any steady state with partial protection is inefficient. Moreover, if there are multiple steady states then these can be Pareto-ranked: welfare is higher the more is spent on protection, and thus the smaller the prevalence of the disease.

We can now return to one of the questions posed in the introduction: How does the protection technology affect inefficiencies in protection? To make the question interesting, suppose that the infection does not die out when no individual protects (i.e. $p_H > \alpha/\delta$), and that the first best involves full protection even in the densest of societies. When protection is interaction-specific, inefficiencies will be non-monotonic in population density. In sparse societies, individuals fail to protect because the benefits of doing so are not sufficiently large at low prevalence rates. In very dense societies, the difference between steady state and first best protection levels arises because, since protection scales up with exposure, the individual benefits of protection are outweighed by its costs. In between it is possible to have no differences between first best and decentralized protection levels. If protection expires independently of the infectious status, then the wedge between decentralized and first best protection will narrow as the society becomes denser; individuals will find this type of protection increasingly attractive. Finally, consider the case where protection expires upon infection. In sparse societies, there may or may not be inefficiencies in protection. It is possible for individuals to find protection attractive, to the point where active protection efforts imply that no positive-infection steady state can exist even if from a purely physiological perspective the infection would become endemic. For societies of intermediate density, multiple steady states are possible, and so whether there are inefficiencies depends on individuals' coordination. In sufficiently dense societies, a person will become infected very frequently even if everyone protects. In such circumstances, protection will be very taxing for the individual, and there will be a positive cost of decentralization.

6 Concluding Remarks

Depending on context and availability, individuals protect from the possibility of contagion using diverse technologies. This paper studied the implications of the technology on disease prevalence and welfare at different levels of population density. I conclude by drawing attention to the policy implications that follow from the analysis of the paper.

The eradication challenge. The strategic substitutes nature of protection decisions implies that, in the context of infectious diseases, the eradication of infections will generally require external interventions. This point was raised by Geoffard and Philipson [17] in the context of vaccination. This paper thus extends this to diseases for which no vaccine currently exists. By showing that protection investments in cybersecurity may feature complementarities, the paper also shows that this result need not hold in online environments.

The value of vaccines and the equilibrium consequences of antigenic drift. As societies become denser, the social value of developing new vaccines increases. The reason is that as individuals become more exposed, their incentives to vaccinate increase, whereas the incentives to use non-vaccine protection may decrease. The development of new vaccines is therefore a way of exploiting the narrowing wedge between first best and decentralized protection in dense societies.

The statement in the previous paragraph comes with an important qualification. The protection provided by the vaccine must be long-lasting for inefficiencies in protection to decrease with population density. In the case of influenza, existing vaccines result in selection pressures to avoid the immune system, leading to genetic variations in the virus. This process is known as antigenic drift, and constitutes a major research theme in immunology. The evolution into new strains takes place on average every 2-8 years, and implies that individuals who were immunized become susceptible within a few years of infection (Carrat and Flahault [11]). The goal of developing a universal influenza vaccine (i.e. a vaccine that is consistent across strains) has so far proven elusive. By characterizing the equilibrium consequences of antigenic drift, this paper establishes the social value of universal vaccines.

Interventions in the absence of vaccines. In the absence of vaccines, people will rely on interaction-specific protection measures to avoid contagion. In most circumstances it is not possible to waive/subsidize variable protection costs, such as the discomfort associated with using hand sanitizer or wearing a face mask. The main policy tool consists in closing public meeting places (e.g. schools, railway stations, theaters), which can be thought

of as a prohibitively expensive tax on using certain venues. This paper shows that the desirability of these measures does not increase monotonically with population density. That said, understanding whether the conditions for decentralized protection to be first best actually hold in practice can be hard. Exposure has to be large enough for individuals to face a nontrivial probability of meeting an infected person, but not too high or otherwise protection will be too costly.

Preventing coordination failures. By pointing out that protection in online environments may expire upon infection, the paper uncovered possible complementarities in cybersecurity. While in sparse societies infection may be contained without the need for external intervention, multiple steady states may exist in denser societies. The strength of complementarities in protection can give an indication of the likelihood of multiplicity. Multiple steady states are thus more likely the larger the health premium, the more infectious the disease, the more effective the protection technology, the lower the cost of protection, and the slower the rate of recovery from infection. In such circumstances, there is a role for policies that ensure coordination in the efficient steady state. Because of the tipping-point aspects associated with multiple steady states, a policy-maker who intends to shift the society from a steady state of no protection to a steady state with full protection will find the task more difficult the higher the population density.

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A Proofs

A.1 The effect of patience

As stated in the formulation of the model, the individual payoffs given by (1) are the limit payoffs if agents discount the future at rate $\beta \to 1$. Formally, suppose that agents discount future payoffs at rate $\beta < 1$. If $\{\mathbb{E}u_t\}$ is a sequence of expected per-period payoffs, then an agent's payoff can be written as

$$(1-\beta)\sum_{t=0}^{\infty}\beta^t\mathbb{E}u_t,$$

where the factor $(1-\beta)$ is the customary normalization of discounted payoffs so that a constant utility of u in every period yields a discounted value of u.

In the present model, transitions across states are Markovian. Fix the transition matrix $\mathbf{T}$, and let $\mathbf{u}$ be the vector with elements corresponding to the current payoffs in each state. We can thus write the vector of values in the different states recursively as $\mathbf{V} = (1-\beta)\mathbf{u} + \beta\mathbb{E}\mathbf{V} = (1-\beta)\mathbf{u} + \beta\mathbf{T}\mathbf{V}$, or, equivalently,

$$(\mathbf{I} - \beta\mathbf{T})\mathbf{V} = (1-\beta)\mathbf{u}.$$

For $\beta < 1$ the matrix $(\mathbf{I} - \beta\mathbf{T})$ is non-singular (in general; i.e. provided β is not in the spectrum of $\mathbf{T}$). For $\beta < 1$ we can thus write

$$\mathbf{V} = (1-\beta)(\mathbf{I} - \beta\mathbf{T})^{-1}\mathbf{u} = \frac{(1-\beta)}{\det(\mathbf{I} - \beta\mathbf{T})}Co(\mathbf{I} - \beta\mathbf{T})\mathbf{u},$$

where $Co(\cdot)$ denotes the transpose of the matrix of cofactors. Note that $\det(\mathbf{I} - \beta\mathbf{T})$ is nothing but the characteristic polynomial of $\mathbf{T}$. It is therefore easy to see that the limit $\lim_{\beta\uparrow 1}\mathbf{V}$ exists if and only if the unit eigenvalue of $\mathbf{T}$ has multiplicity one (i.e. the characteristic polynomial has a unique unit root). This condition amounts to $\mathbf{T}$ featuring a unique invariant distribution, and holds throughout the paper. If the limit exists, then, by Theorem 1 in Marinacci [26], it is given by (1).[21]

A.2 Proofs of Section 3

Theorem 1. For conciseness, let A ($\neg A$) denote the case where the individual does not need (needs) to purchase protection, I denote being infected, S denote being susceptible,

[21] Theorem 1 in Marinacci can also be found as Theorem 3.9.10 in Corbae, Stinchcombe and Zeman [7] (p. 104).

and m ($\neg m$) having a meeting (not having a meeting). If the individual is infected, then her payoff is independent of whether or not a meeting takes place. The relevant partition of the set of individual states is thus: $(I, \neg A)$, (I, A), $(S, \neg A, m)$, $(S, \neg A, \neg m)$, (S, A, m), and $(S, A, \neg m)$. For an individual who has a degree δ, transition probabilities between these individual states are characterized by the following matrix:

$$\mathbf{T}_X = \begin{bmatrix} (1-\alpha) & 0 & \alpha\delta & \alpha(1-\delta) & 0 & 0 \\ (1-\alpha)\eta & (1-\alpha)(1-\eta) & \alpha\eta\delta & \alpha\eta(1-\delta) & \alpha(1-\eta)\delta & \alpha(1-\eta)(1-\delta) \\ \theta p_L \eta & \theta p_L(1-\eta) & (1-\theta p_L)\eta\delta & (1-\theta p_L)\eta(1-\delta) & (1-\theta p_L)(1-\eta)\delta & (1-\theta p_L)(1-\eta)(1-\delta) \\ 0 & 0 & \delta & (1-\delta) & 0 & 0 \\ \theta p_L \eta & \theta p_L(1-\eta) & (1-\theta p_L)\eta\delta & (1-\theta p_L)\eta(1-\delta) & (1-\theta p_L)(1-\eta)\delta & (1-\theta p_L)(1-\eta)(1-\delta) \\ 0 & 0 & \eta\delta & \eta(1-\delta) & (1-\eta)\delta & (1-\eta)(1-\delta) \end{bmatrix}.$$

The invariant distribution, given by the left eigenvector of $\mathbf{T}_X$ associated with its unit eigenvalue, is

$$\mathbf{v}_X = \begin{bmatrix} \frac{\delta\eta\theta p_L}{(\delta\theta p_L+\alpha)(\eta+(1-\eta)\alpha)} \\ \\ \frac{\alpha\delta(1-\eta)\theta p_L}{(\delta\theta p_L+\alpha)(\eta+(1-\eta)\alpha)} \\ \\ \frac{\alpha\delta\eta\ [\eta+(1-\eta)(\delta\theta p_L+\alpha)]}{(\delta\theta p_L+\alpha)(\eta+(1-\eta)\alpha)(\eta+(1-\eta)\delta)} \\ \\ \frac{\alpha(1-\delta)\eta\ [\eta+(1-\eta)(\delta\theta p_L+\alpha)]}{(\delta\theta p_L+\alpha)(\eta+(1-\eta)\alpha)(\eta+(1-\eta)\delta)} \\ \\ \frac{\alpha\delta^2(1-\eta)\ [\eta(1-\theta p_L)+(1-\eta)\alpha]}{(\delta\theta p_L+\alpha)(\eta+(1-\eta)\alpha)(\eta+(1-\eta)\delta)} \\ \\ \frac{\alpha\delta(1-\delta)(1-\eta)\ [\eta(1-\theta p_L)+(1-\eta)\alpha]}{(\delta\theta p_L+\alpha)(\eta+(1-\eta)\alpha)(\eta+(1-\eta)\delta)} \end{bmatrix}.$$

We can therefore write the value a degree-δ individual as[22]

$$V(\delta) = \max_{p\in[p_L,p_H]} \frac{\delta\theta p}{\delta\theta p+\alpha}\pi_I + \frac{\alpha}{\delta\theta p+\alpha}\left(\pi_S - \frac{\delta\eta\left[\eta+(1-\eta)(\delta\theta p_L+\alpha)\right]}{(\eta+(1-\eta)\alpha)(\eta+(1-\eta)\delta)}\,\frac{p_H-p}{p_H-p_L}\,c\right). \quad (11)$$

[22]Note that the assumption of infinite patience implies that the individual chooses protection so as to maximize her expected per-period payoff. I will be using the term value to denote the expected per-period payoff of the individual.

Therefore, protection is a best response if and only if

$$F(\delta,\theta) \;:=\; A\;\theta^2\;\delta^2 \;+\; B\;\theta\;\delta \;+\; C\;\theta \;+D \;\leq 0, \tag{12}$$

where

$$\begin{aligned}
A &:= \left[p_H p_L \eta(1-\eta)c\right],\\
B &:= \left[\eta c\Big(p_H\Big(\eta+(1-\eta)\alpha\Big)+p_L(1-\eta)\alpha\Big)-(1-\eta)\Big(\eta+(1-\eta)\alpha\Big)(p_H-p_L)(\pi_S-\pi_I)\right],\\
C &:= -\eta\Big(\eta+(1-\eta)\alpha\Big)(p_H-p_L)(\pi_S-\pi_I),\\
D &:= \eta\Big(\eta+(1-\eta)\alpha\Big)\alpha c.
\end{aligned}$$

Let us define the set of degrees that find protection profitable under a steady-state infection rate θ, $\Delta(\theta)=\{\delta\in[0,1]: F(\delta,\theta)\leq 0\}\equiv[0,1]\cap[r_1,r_2]$, where r_1, r_2 are the roots of the polynomial in δ defined by $F(\delta,\theta)$.

Note that, since $D>0$, for any δ there exists small enough θ such that (12) does not hold, and so $\Delta(\theta)=\varnothing$. That is, no individual finds protection profitable. This establishes the first claim of the Proposition.

In general, note that if r_1, r_2 are not real, then $\Delta(\theta)=\varnothing$, protection is not profitable for any degree, and all claims in the Proposition are trivially true. Let us assume henceforth that r_1, r_2 are real, and, without loss of generality, that $r_1\leq r_2$.

Note that B is a degree-2 polynomial in η. Moreover, it is easy to see that $B<0$ if $\eta=0$ and $B>0$ if $\eta=1$. From these two facts, it follows that there exists $\bar{\eta}\in(0,1)$ such that $B<0$ for any $\eta<\bar{\eta}$ and $B>0$ for any $\eta>\bar{\eta}$. Let us define $\theta^0:=\frac{\alpha c}{(p_H-p_L)(\pi_S-\pi_I)}$.

Case (a): $\eta>\bar{\eta}$. It is easy to see (by Vieta's formula) that both r_1 and r_2 are negative if $\theta<\theta^0$ (so that $C\theta+D>0$), in which case $\Delta(\theta)=\varnothing$ and no degree finds protection profitable. If $\theta\geq\theta^0$, then $r_1<0$ and $r_2\geq 0$, so that $\Delta(\theta)=[0,\bar{\delta}]$, where $\bar{\delta}=\min\{r_2,1\}$.

Case (b): $\eta<\bar{\eta}$. If $\theta<\theta^0$, then (by Vieta's formula) r_1, r_2 are both positive. If, moreover, $r_1>1$, then $\Delta(\theta)=\varnothing$ and the claim in the Proposition is trivially true. If $r_1\leq 1$, then $\Delta(\theta)=[\underline{\delta},\bar{\delta}]$, where $\underline{\delta}=r_1$ and $\bar{\delta}=\min\{r_2,1\}$. Finally, if $\theta\geq\theta^0$, then $r_1\leq 0$ and $r_2>0$. Thus, $\Delta(\theta)=[0,\bar{\delta}]$, where $\bar{\delta}=\min\{r_2,1\}$.

The last claim in point (b) of the Proposition follows from the fact that $\lim_{p_L\to 0} r_2=+\infty$.

□

Corollary 1 and Corollary 2. The results follow from solving (11) under the conditions provided. □

Theorem 2. On the first meeting since her last recovery, the individual must decide how much to invest in protection.[23] For conciseness, let $msr = 1$ ($msr = 0$) indicate that, before the start of the current period, the individual has (not) had a meeting since her last recovery. Following the same terminology as in the proof of Theorem 1, the relevant partition of the set of individual states is therefore: I, $(S, msr = 0, m)$, $(S, msr = 0, \neg m)$, $(S, msr = 1, m)$, $(S, msr = 1, \neg m)$. For a fixed θ, the transition probabilities between individual states are described by the transition matrix

$$\mathbf{T}_N = \begin{bmatrix} 1-\alpha & \alpha\delta & \alpha(1-\delta) & 0 & 0 \\ \theta p & 0 & 0 & (1-\theta p)\delta & (1-\theta p)(1-\delta) \\ 0 & \delta & (1-\delta) & 0 & 0 \\ \theta p & 0 & 0 & (1-\theta p)\delta_i & (1-\theta p)(1-\delta) \\ 0 & 0 & 0 & \delta & (1-\delta) \end{bmatrix},$$

so that the fraction of time that an individual of degree δ spends in each state in the long run is given by

$$\mathbf{v}'_N = \left(\frac{\delta\theta p}{\delta\theta p + \alpha}, \frac{\alpha\delta\theta p}{\delta\theta p + \alpha}, \frac{\alpha(1-\delta)\theta p}{\delta\theta p + \alpha}, \frac{\alpha\delta(1-\theta p)}{\delta\theta p + \alpha}, \frac{\alpha(1-\delta)(1-\theta p)}{\delta\theta p + \alpha} \right).$$

The value of an individual of degree δ is therefore

$$V(\delta) = \max_{p \in [p_L, p_H]} \frac{\theta p \delta}{\theta p \delta + \alpha} \pi_I + \frac{\alpha}{\theta p \delta + \alpha} \left(\pi_S - \delta\theta p \left(\frac{p_H - p}{p_H - p_L} \right) c \right). \tag{13}$$

Solving (13) yields the desired result. □

A.3 Proofs of Section 4

Proposition 1. First note that, since $p \in [p_L, p_H]$, any steady state with positive infection rate must be in $\mathcal{S} = [\theta_l^{ss}, \theta_h^{ss}] \cap \mathbb{R}_{++}$, where $\mathbb{R}_{++} \equiv (0, +\infty)$. Consider then the statement that a steady state with positive infection rate exists if and only if $p_H > \alpha/\delta$.

[23] In principle, the individual could delay the protection decision and purchase protection only after having had $x > 0$ meetings. This can be relevant if θ_t is not constant over time. In a steady state (where $\theta_t = \theta_{t+1}$ for all t) postponement can never be optimal.

For $\Rightarrow$, suppose that $p_H \leq \alpha/\delta$. It follows that $\mathcal{S} = \varnothing$. The unique steady state infection rate is $\theta^{ss} = 0$.

Consider next $\Leftarrow$. To that end, suppose now that $p_H > \alpha/\delta$, which implies that $\theta_h^{ss} > 0$. It follows that $\mathcal{S} \neq \varnothing$. Note that $\delta_X^*(\theta)$ is strictly increasing in θ (see (5)). Thus, $\theta_l^{ss} < \theta_h^{ss}$ implies $\delta_X^*(\theta_l^{ss}) < \delta_X^*(\theta_h^{ss})$. The following cases are then possible:

Case 1: $\delta \geq \delta_X^*(\theta_h^{ss}) > \delta_X^*(\theta_l^{ss})$. In the unique steady state no individual chooses protection ($p^* = p_H$) and $\theta^{ss} = \theta_h^{ss}$. To see that this is indeed a steady state, note that if $p^* = p_H$, then the aggregate steady state infection must be θ_h^{ss}. Under this infection rate, it is optimal for individuals to protect. To see that this steady state is unique, suppose, for a contradiction, that there is a steady state where individuals choose some protection, resulting in an aggregate infection rate $\theta^{ss} < \theta_h^{ss}$. Since $\delta_X^*(\theta)$ is strictly increasing in θ, $\delta > \delta_X^*(\theta^{ss})$. Individuals would then strictly prefer to protect, a contradiction.

Case 2: $\delta_X^*(\theta_l^{ss}) < \delta < \delta_X^*(\theta_h^{ss})$. The unique steady state is given by $\theta^{ss} = {\delta_X^*}^{-1}(\delta)$, with individuals choosing a level of protection

$$p^* \quad = \quad \frac{\alpha}{\delta(1 - {\delta_X^*}^{-1}(\delta))},$$

To see that this is a steady state, note that if individuals choose p^*, then the steady state infection rate is ${\delta_X^*}^{-1}(\delta)$. Under this infection rate, individuals find it optimal to choose p^*. For uniqueness, suppose for a contradiction that individuals choose higher (lower) protection. Then the steady state aggregate infection rate θ^{ss} would be lower (higher) than ${\delta_X^*}^{-1}(\delta)$. Since $\delta_X^*(\theta)$ is increasing, individuals would prefer not to protect at all (to fully protect), in which case θ^{ss} cannot be a steady state.

Case 3: $\delta \leq \delta_X^*(\theta_l^{ss})$. In the unique steady state all individuals choose full protection ($\gamma^* = 1$, so that $p^* = p_L$) and $\theta^{ss} = \theta_l^{ss}$. The proof of this claim is analogous to the one for Case 1 and therefore omitted.

With regards to the last statement of the proposition, this simply follows from the fact that all three functions $\theta_l^{ss}(\delta)$, ${\delta_X^*}^{-1}(\delta)$, and $\theta_h^{ss}(\delta)$ are strictly increasing in δ. $\square$

Proposition 2. The proof follows similar steps to those described for the proof of Proposition 1. Since $p \in [p_L, p_H]$, any steady state with positive infection rate must be in $\mathcal{S} = [\theta_l^{ss}, \theta_h^{ss}] \cap \mathbb{R}_{++}$, where $\mathbb{R}_{++} \equiv (0, +\infty)$. Consider then the statement regarding existence. For $\Rightarrow$, suppose that $p_H \leq \alpha/\delta$. It follows that $\mathcal{S} = \varnothing$. The unique steady state is $\theta^{ss} = 0$.

Consider next $\Leftarrow$. Suppose that $p_H > \alpha/\delta$, which implies that $\theta_h^{ss} > 0$. It follows that

$\mathcal{S} \neq \varnothing$. Since $\theta_l^{ss} = -\infty$, $M\left\{\theta_l^{ss}(\delta), \delta_X^{**-1}(\delta), \theta_h^{ss}(\delta)\right\} \equiv \min\left\{\delta_X^{**-1}(\delta), \theta_h^{ss}(\delta)\right\}$. The following cases are then possible:

Case 1: $\theta_h^{ss}(\delta) \leq \delta_X^{**-1}(\delta)$. Since $\delta_X^{**}(\cdot)$ is a decreasing function, this is equivalent to $\delta \leq \delta_X^{**}(\theta_h^{ss})$. It is easy to see that θ_h^{ss} is a steady-state infection rate: by Proposition 2, $\delta \leq \delta_X^{**}(\theta_h^{ss})$ implies that under this aggregate infection rate, every individual finds no protection optimal. For uniqueness, suppose for a contradiction that there is a steady state with positive infection with some protection in the population. The infection rate would be $\theta^{ss} < \theta_h^{ss}$. Since $\delta_X^{**}(\theta)$ is strictly decreasing in θ, $\delta < \delta_X^{**}(\theta^{ss})$. Individuals would then strictly prefer not to protect, a contradiction.

Case 2: $\theta_h^{ss}(\delta) > \delta_X^{**-1}(\delta)$, or $\delta > \delta_X^{**}(\theta_h^{ss})$. The unique steady state is characterized by $\theta^{ss} = \delta_X^{**-1}(\delta)$, with the average level of protection being

$$p^*(\delta) = \frac{\alpha}{\delta(1 - \delta_X^{**-1}(\delta))}. \tag{14}$$

To see that this is a steady state, note that if p^* is the average level of protection, then the infection rate is $\delta_X^{**-1}(\delta)$. Under this infection rate, individuals are indifferent between protecting and not protecting. For uniqueness, suppose for a contradiction that individuals choose higher (lower) protection. Then the steady state aggregate infection rate θ^{ss} would be lower (higher) than $\delta_X^{**-1}(\delta)$. Since $\delta_X^{**}(\theta)$ is decreasing, individuals would prefer not to protect at all (to fully protect), in which case θ^{ss} cannot be a steady state.

If $\theta_h^{ss}(\delta) < \delta_X^{**-1}(\delta)$ then the fact that $\theta_h^{ss}(\delta)$ implies that $\theta^{ss}(\delta)$ is increasing in δ, and no protection takes place for any δ in this range. If $\theta_h^{ss}(\delta) > \delta_X^{**-1}(\delta)$, then $\delta_X^{**-1}(\delta)$ being decreasing in δ implies that $\theta^{ss}(\delta)$ is decreasing in δ.

For any δ such that $\theta_h^{ss}(\delta) < \delta_X^{**-1}(\delta)$, there is no steady state with positive protection spending. Consider now degrees δ' and δ'' such that $\theta_h^{ss}(\delta') > \delta_X^{**-1}(\delta')$ and $\theta_h^{ss}(\delta'') > \delta_X^{**-1}(\delta'')$, and suppose without loss of generality that $\delta'' > \delta'$. Since in this range $\theta_h^{ss}(\delta)$ is decreasing in δ, $\theta_h^{ss}(\delta'') < \theta_h^{ss}(\delta')$. For any δ, the level of protection $p^*(\delta)$ is given by (14), and $\theta^{ss}(\delta) = 1 - \frac{\alpha}{\delta p^*(\delta)}$. Since $\delta'' > \delta'$, for $\theta_h^{ss}(\delta'') < \theta_h^{ss}(\delta')$ to hold it is necessary that $p^*(\delta'') > p^*(\delta')$. That is, protection spending is increasing in δ. □

Theorem 3. The proof is organized as follows: points (1)-(3) are shown first; secondly, I show the statements regarding stability; finally, I show the statement regarding existence.

Consider (1). For the direction $\Rightarrow$, suppose that θ_h^{ss} is a steady state with positive infection rate. Individuals must then find it optimal not to protect. By Theorem 2, it must be

that $\delta \leq \delta_N(\theta_h^{ss})$, or $\theta_h^{ss} \geq {\delta_N}^{-1}(\delta)$. For $\Leftarrow$, by Theorem 2, $\theta_h^{ss} \geq {\delta_N}^{-1}(\delta)$ implies that at $\theta = \theta_h^{ss}$ individuals will find it optimal not to protect. Further note that ${\delta_N}^{-1}(\delta) > 0$ for any $\delta > 0$. It follows that θ_h^{ss} is a steady state with positive infection.

The proof of (2) is similar to that of (1), and therefore omitted. Consider then (3). By Theorem 2, at long-run infection rate ${\delta_N}^{-1}(\delta)$ any level of protection $p \in [p_L, p_H]$ is optimal. Consider then

$$p^*(\delta) = \frac{\alpha}{\delta(1 - {\delta_N}^{-1}(\delta))} \in [p_L, p_H].$$

At this level of protection, the steady state infection rate is in fact ${\delta_N}^{-1}(\delta)$.

There are two statements regarding stability. Consider the first one. For $\Leftarrow$, suppose that $\theta^{ss} = {\delta_N}^{-1}(\delta) \in (\theta_l^{ss}(\delta), \theta_h^{ss}(\delta))$ (i.e. ${\delta_N}^{-1}(\delta)$ is *strictly* between $\theta_l^{ss}(\delta)$ and $\theta_h^{ss}(\delta)$). By Theorem 2, for any $\epsilon > 0$, the unique optimal protection strategy for long-run infection ${\delta_N}^{-1}(\delta) - \epsilon$ is $p_L < p^*(\delta)$, whereas the unique optimal protection strategy for long-run infection ${\delta_N}^{-1}(\delta) + \epsilon$ is $p_H > p^*(\delta)$. The basin of attraction of this steady state is of size zero, i.e. the steady state is unstable. For $\Rightarrow$, if $\theta_l^{ss} > 0$ is a steady state, then since $\delta_N(\theta)$ is decreasing in θ, p_L is optimal for any $\theta \in (0, \theta_l^{ss}]$. That is, θ_l^{ss} is not unstable. Supose next that $\theta_h^{ss} > 0$ is a steady-state infection rate. $\alpha > 0$ implies that $\theta_h^{ss} < 1$. Since $\delta_N(\theta)$ is decreasing in θ, p_H is optimal for any $\theta \in [\theta_h^{ss}, 1]$. That is, θ_h^{ss} is not unstable.

For the second statement regarding stability, it is easy to see that the basin of attraction of the full protection (no protection) steady state is $[0, \delta_N^{-1}(\delta)]$ ($[\delta_N^{-1}(\delta), 1]$). The result then follows by noting that $\delta_N^{-1}(\delta)$ is decreasing in δ.

Consider finally the statement regarding existence. Let us first show the direction $\Leftarrow$. We will show that if either one of (a) or (b) hold, then one of the three cases (1)-(3) holds. Suppose (a) $\delta > \alpha/p_L$ holds, which implies that $\theta_h^{ss}(\delta) > \theta_l^{ss}(\delta) > 0$. Since $(p_H - p_L)(\pi_S - \pi_I) > \alpha c p_L$ by assumption, we have that $\delta_N^{-1}(\delta) > 0$ for any δ. Hence three cases are possible. (i) If $0 < \delta_N^{-1}(\delta) < \theta_l^{ss}(\delta) < \theta_h^{ss}(\delta)$, then (1) holds. (ii) If $0 < \theta_l^{ss}(\delta) \leq \delta_N^{-1}(\delta) \leq \theta_h^{ss}(\delta)$, then (1)-(3) hold. (iii) If $0 < \theta_l^{ss}(\delta) < \theta_h^{ss}(\delta) < \delta_N^{-1}(\delta)$, then (2) holds. Suppose next that (b) $\delta \geq \frac{(p_H - p_L)(\pi_S - \pi_I)}{c p_L p_H}$ holds. This implies that $\delta_N^{-1}(\delta) \leq \theta_h^{ss}(\delta)$. Hence (1) holds.

For the direction $\Rightarrow$, note that $\delta \leq \alpha/p_L$ implies $\theta_l^{ss}(\delta) \leq 0$. Thus, for there to be a positive infection rate in steady state, individuals must not fully protect. But $\delta < \frac{(p_H - p_L)(\pi_S - \pi_I)}{c p_L p_H}$ implies that $\delta_N^{-1}(\delta) > \theta_h^{ss}(\delta)$, so that for any possible long-run infection rate individuals strictly prefer to fully protect. □

A.4 Proofs of Section 5

Proposition 3. If $p_L \leq \alpha/\delta$, then the infection dies out in the long-run under full protection. Since agents are infinitely patient, protection is first best regardless of its cost. Suppose then that $p_L > \alpha/\delta$.

Case (i): $j = X$. Welfare under full protection is

$$\pi_I + \frac{\alpha}{\delta p_L}\left[(\pi_S - \pi_I) - \frac{\delta\eta\,(\eta + (1-\eta)\delta p_L)}{(\eta + (1-\eta)\alpha)(\eta + (1-\eta)\delta)}\, b\right],$$

whereas welfare under no protection is of

$$\pi_I + \frac{\alpha}{\delta p_H}(\pi_S - \pi_I).$$

Thus, first best protection is

$$\begin{aligned} p_X^{fb} &= \arg\max_{p\in\{p_L,p_H\}} \left\{ \pi_I + \frac{\alpha}{\delta p_L}\left[(\pi_S - \pi_I) - \frac{\delta\eta\,(\eta + (1-\eta)\delta p_L)}{(\eta + (1-\eta)\alpha)(\eta + (1-\eta)\delta)}\, b\right], \right. \\ &\qquad \left. \pi_I + \frac{\alpha}{\delta p_H}(\pi_S - \pi_I) \right\} \\ &= \begin{cases} p_L & \text{if } \delta < \delta_X^{fb} \\ p \in \{p_L, p_H\} & \text{if } \delta = \delta_X^{fb} \\ p_H & \text{otherwise} \end{cases}, \end{aligned}$$

where δ_X^{fb} is the unique positive real root of the polynomial (10). That $f(\delta)$ has a unique positive real root follows from $f(\cdot)$ being strictly convex and $f(0) < 0$.

Case (ii): $j = N$. It is easy to verify that welfare under full protection is

$$\pi_I + \frac{\alpha}{\delta p_L}\left[(\pi_S - \pi_I) - (\delta p_L - \alpha)b\right],$$

whereas welfare under no protection is of

$$\pi_I + \frac{\alpha}{\delta p_H}(\pi_S - \pi_I).$$

Thus, first best protection is

$$\begin{aligned} p_N^{fb} &= \arg\max_{p\in\{p_L,p_H\}} \left\{ \pi_I + \frac{\alpha}{\delta p_L}\left[(\pi_S-\pi_I) - (\delta p_L - \alpha)b\right], \pi_I + \frac{\alpha}{\delta p_H}(\pi_S-\pi_I) \right\} \\ &= \begin{cases} p_L & \text{if } \delta < \delta_N^{fb} \\ p \in \{p_L, p_H\} & \text{if } \delta = \delta_N^{fb} \\ p_H & \text{otherwise} \end{cases}, \end{aligned}$$

where $\delta_N^{fb} := \frac{(p_H-p_L)(\pi_S-\pi_I)}{p_L p_H b} + \frac{\alpha}{p_L}$. □

The following corollary gives a necessary and sufficient condition for full protection to be first best for *any* degree when protection expires exogenously.

Corollary 4. *Consider a regular society of degree δ, and suppose that protection expires with exogenous probability η and that $\alpha < p_L$. Full protection is the unique first best for any $\delta > \frac{\alpha}{p_L}$ if and only if*

$$\eta c < \left(\frac{\eta + (1-\eta)\alpha}{\eta + (1-\eta)p_L}\right)\left(1 - \frac{p_L}{p_H}\right)(\pi_S - \pi_I). \tag{15}$$

For high enough durability (i.e. low enough η), full protection is first best for any degree. Similarly, condition (15) is more likely to hold the more effective protection is (i.e. the lower p_L is). More suprisingly, the right-hand side of (15) is increasing in α. Protection is more attractive for the social planner if recovery takes place rather quickly.[24] All else equal, when recovery takes long periods of time, protection is likely to expire while the individual is infected. If, on the other hand, the individual recovers quickly, then a single investment in protection will be useful in many interactions where the individual is susceptible from getting the disease, even if she is occasionally infected.

Corollary 2. Note that the polynomial (10) evaluated at $\delta = 1$ is $f(1) < 0$ if and only if (15) holds. Since $f(\cdot)$ is convex, this implies that $\delta_X^{fb} > 1$ if and only if (15) holds. By Proposition 3, this is necessary and sufficient for full protection to be the unique first best for any $\delta \in \left(\frac{\alpha}{p_L}, 1\right]$. □

Corollary 1. We divide the analysis into the three possible cost structures.

[24] That this is true in general can be seen from the fact that an increase in α shifts the polynomial (10) downwards.

Interaction-specific protection. By Proposition 1, in any steady state with positive protection spending, individuals weakly prefer to protect, i.e. $\delta \leq \delta_X^*(\theta^{ss})$. Since $\delta_X^*(\theta^{ss}) < \delta_X^{fb}$ for any θ^{ss}, we have that $\delta < \delta_X^{fb}$. Therefore, by Proposition 3 full protection is the unique first best.

Durable and perfect protection. In any steady state with positive protection spending, individuals weakly prefer to protect. Thus, condition (12) is satisfied. It is easy to see that then $f(\delta) < 0$, which by Proposition 3 implies that full protection is the unique first best protection level.

Protection that expires upon infection. By Theorem 3, if there is a steady state with some positive level of protection, then there is a steady state with full protection. The following two facts will be useful.

Fact 1.

$$\delta_N^{fb} \begin{cases} < \delta_N(\theta) & \text{if } \theta < \bar{\theta} \\ = \delta_N(\theta) & \text{if } \theta = \bar{\theta} \\ > \delta_N(\theta) & \text{otherwise} \end{cases},$$

where $\bar{\theta} := \frac{(p_H - p_L)(\pi_S - \pi_I) - \alpha p_L c}{(p_H - p_L)(\pi_S - \pi_I) + \alpha p_H c}$.

Fact 2.

$$\begin{aligned} \bar{\theta} &= 1 - \frac{\alpha c(p_L + p_H)}{(p_H - p_L)(\pi_S - \pi_I) + \alpha p_H c} \\ &< 1 - \frac{\alpha c p_H}{(p_H - p_L)(\pi_S - \pi_I) + \alpha p_H c} \\ &= 1 - \frac{\alpha}{\delta_N^{fb} p_L}. \end{aligned}$$

Let us divide the analysis into two cases.

Case 1: $\theta^{ss} \leq \bar{\theta}$. Let p^* denote the protection level chosen by individuals in steady state. Note that

$$\theta^{ss} = 1 - \frac{\alpha}{\delta p^*} \leq \bar{\theta} < 1 - \frac{\alpha}{\delta_N^{fb} p_L},$$

where the second inequality follows from Fact 2. It follows that $\delta p^* < \delta_N^{fb} p_L$, or

$$\delta < \frac{p_L}{p^*} \delta_N^{fb} \leq \delta_N^{fb} \leq \delta_N(\theta^{ss}),$$

where the last inequality follows from $\theta^{ss} \leq \bar{\theta}$ and Fact 1. $\delta < \delta_N(\theta^{ss})$ implies that

individuals must fully protect, i.e. $p^* = p_L$. $\delta < \delta_N^{fb}$ implies that full protection is first best.

Case 2: $\theta^{ss} > \bar{\theta}$. By Fact 1, $\delta_N^{fb} > \delta_N(\theta^{ss})$. For θ^{ss} to be a steady state with full protection, $\delta \leq \delta_N(\theta^{ss})$. It follows that $\delta < \delta_N^{fb}$. That is, full protection is the unique first best protection level. □

B Arbitrary degree distributions

To study how the results of the steady state analysis extend to non-regular societies, here I consider the case of arbitrary degree distributions. This appendix is organized as follows. First, Section B.1 states how the setup needs to be adjusted for this extension. Sections B.2-B.4 consider the steady state analyses for the cases of interaction-specific protection, durable and perfect protection, and protection that expires upon infection, respectively. The proofs of the results stated in those sections are contained in Section B.5.

B.1 Extended model

Degree distribution. The mass of individuals with a probability of at most δ is equal to cumulative distribution function $F(\delta)$, with density $f(\delta)$, mean $\langle\delta\rangle$, and variance σ^2. I will refer to δ as the *degree*, and to $F(\delta)$ as the *degree distribution*. It follows that, from the perspective of an individual who is called to interact, the likelihood that the interaction is with an individual of degree δ is given by $f(\delta)\delta/\langle\delta\rangle$.

Steady state. Let $\rho_t(\delta)$ denote the fraction of degree-δ individuals who are infected at time t. It follows that, conditional on having a meeting at time t, the probability that the meeting is with an infected individual is

$$\theta_t = \int_{\varepsilon}^{1} \frac{f(\delta)\delta}{\langle\delta\rangle}\rho_t(\delta)\ d\delta. \tag{16}$$

I will refer to θ as the *neighbour infection rate*, which in general differs from the average infection rate in the population,

$$\rho_t = \int_{\varepsilon}^{1} f(\delta)\rho_t(\delta)\ d\delta.$$

The dynamics of $\rho_t(\delta)$ are given by

$$\rho_{t+1}(\delta) - \rho_t(\delta) = (1 - \rho_t(\delta))\ \delta\ \theta_t\ p_t(\delta) - \alpha\ \rho_t(\delta), \tag{17}$$

where $p_t(\delta) \in [p_L, p_H]$ is the average protection level of degree-δ susceptible individuals. Consider the first term on the right-hand side of (17). A fraction δ of the fraction $(1-\rho_t(\delta))$ of degree-δ susceptible individuals are called for an interaction. Of those individuals, a fraction $\theta_t p_t(\delta)$ is matched with infected individuals and becomes infected. The second term on the right-hand side of (17) represents the fraction of infected individuals of degree δ who spontaneously recover.

In a steady state, $\rho_{t+1}(\delta) = \rho_t(\delta) = \rho^{ss}(\delta)$ for every $\delta \in [\varepsilon, 1]$. From (16) this implies that, in a steady state, $\theta_t = \theta^{ss}$. Therefore, using (17) it follows that

$$\rho^{ss}(\delta) = \frac{\delta\ p_t(\delta)\ \theta^{ss}}{\delta\ p_t(\delta)\ \theta^{ss} + \alpha} = \frac{\delta\ p(\delta)\ \theta^{ss}}{\delta\ p(\delta)\ \theta^{ss} + \alpha}, \tag{18}$$

where the second equality follows from the fact that, if θ_t and $\rho_t(\delta)$ are time independent, then $p_t(\delta)$ must be as well.

Let $p^*(\delta, \theta)$ denote an optimal protection level that is chosen by degree-δ individuals when the steady state probability of meeting an infected individual equals θ, and let $P^*(p, \theta)$ denote the set of degrees who find protection level p optimal under θ. That is, $P^*(p, \theta) = \{\delta \in [\varepsilon, 1] : p^*(\delta, \theta) = p\}$.

Definition 3. *A steady state is a pair* $\left(\theta^{ss}, \{P^*(p, \theta^{ss})\}_{p \in [p_L, p_H]}\right)$ *such that*

$$\theta^{ss} \ = \ \int_{p \in [p_L, p_H]} \int_{\delta \in P^*(p, \theta^{ss})} \frac{f(\delta)\delta}{\langle \delta \rangle}\ \frac{\delta p \theta^{ss}}{\alpha + \delta p \theta^{ss}}\ d\delta\ dp. \tag{19}$$

To avoid unnecessary complications in the statements of the results, I will restrict attention to degree distributions with cumulative density functions that are continuously differentiable. The c.d.f. of the degree distribution being continuous means that any given degree has zero mass. Thus, the actual choice of individuals who are indifferent between full protection and no protection is irrelevant.

It is important to note that the optimal behaviour characterized by Propositions 1-2 holds regardless of the degree distribution, and will therefore remain valid in the analysis of this section. However, as noted earlier, it is not possible in general to obtain closed-form solutions of steady-state infection rates. In the spirit of Jackson and Rogers [22], however, we can ask how steady state infection rates relate to how dense the society is. One way to think about whether a society is denser than another is in terms of first-order

stochastic dominance of their degree distributions.

Definition 4. *A degree distribution F' first-order stochastically dominates (FOSD) a degree distribution F if $F'(\delta) \leq F(\delta)$ for all $\delta \in [\varepsilon, 1]$, with strict inequality for some $\delta \in [\varepsilon, 1]$.*

Before proceeding, note that for every distribution of degrees $F(\delta)$, there is an associated distribution of neighbours' degrees $\tilde{F}(\delta)$ defined as

$$\begin{aligned} \tilde{F}(\delta) &:= \int_{\varepsilon}^{\delta} \tilde{f}(x)\, dx, \text{ where} \\ \tilde{f}(x) &:= \frac{f(x)x}{\langle x \rangle}. \end{aligned}$$

B.2 Interaction-specific protection

The following result characterizes existence and uniqueness of a positive-infection steady state for any degree distribution if individuals face interaction-specific protection costs.

Proposition 4. *Consider a society with degree distribution $F(\delta)$, and suppose that protection is interaction-specific ($\eta = 1$). A steady state $\theta^{ss} > 0$ exists if and only if*

$$\langle \delta \rangle \left(\frac{\alpha}{p_H} - \langle \delta \rangle \right) < \sigma^2. \tag{20}$$

If such a steady state neighbour infection rate exists, it is unique.

Suppose a steady state $\theta^{ss} > 0$ exists under F, and let F' be another degree distribution with maximum strady-state neihbour infection rate $\theta^{ss'}$. If $\tilde{F}'$ FOSD $\tilde{F}$, then $\theta^{ss'} > \theta^{ss}$.

Note that condition (20) depends only on the society's degree distribution and the physiological characteristics of the disease, α and p_H. Therefore, for any degree distribution the existence of a positive infection rate in the population is independent on the cost and effectiveness of the protection technology. Secondly, it is evident from (20) that if a steady state with positive infection exists in a society with a given degree distribution $F(\delta)$, then a steady state with positive infection also exists in a society with a degree distribution which is a mean-preserving spread of $F(\delta)$. However, since contagion probabilities are endogenous, it is not possible to rank infection rates across mean-preserving spreads as in Jackson and Rogers [22].

With regards to the effect of population density, first order dominance shifts in the

neighbour degree distribution imply higher steady state neighbour infection rates.[25] I have not been able to show, however, that first order dominance shifts also imply higher infection rates, i.e. that $\rho^{ss'} > \rho^{ss}$ if $F'(\delta)$ FOSD $F(\delta)$ and $\tilde{F}'(\delta)$ FOSD $\tilde{F}(\delta)$. By Proposition 4, such a dominance shift will increase the steady state neighbor infection rate. It follows that those individuals who choose the same level of protection in both steady states will become infected more often. That is, if for a degree δ the same level of protection is chosen under θ^{ss} than under $\theta^{ss'} > \theta^{ss}$, then $\rho^{ss'}(\delta) > \rho^{ss}(\delta)$. However, a higher neighbour infection rate will prompt some degrees to protect. The more sensitive incentives are to changes in the probability of meeting an infected individual, the more contained the spread of the disease will be.[26]

I conclude this section with an example with power-law degree distributions.

Example 2. Suppose that the degree distribution follows a power-law. That is, $f(\delta) \propto \delta^{-\lambda}$, with $\lambda \geq 0$. Power-law degree distributions have been observed in the epidemiology and computer network domains that motivate this paper.[27] Power-law degree distributions are easily ordered according to first-order stochastic dominance. Let F_λ and $F_{\lambda'}$ denote two power-law degree distributions with exponents λ and λ', respectively. $\lambda' < \lambda$ implies that $F_{\lambda'}$ FOSD F_λ.[28]

Suppose $p_H = .8$, $p_L = .01$, $\alpha = .01$, $\varepsilon = .01$, $(\pi_S - \pi_I) = 1$, $c = 1$. Figure 6 illustrates the steady state analysis for different values of the exponent λ. Each negative-slope curve indicates, for the corresponding degree distribution, the resulting neighbour infection rate θ if individuals whose degree is smaller than or equal to a given δ fully protect and individuals with degree greater than δ do not protect. If no protection technology was available (or if it was prohibitively expensive) then steady states would be on the intersection of these curves with the horizontal line crossing the vertical axis at $\delta = \varepsilon$. In the present example with $c = 1$, steady states correspond to the intersection of these curves with $\delta_X^*(\theta)$. For example, for $\lambda = 1$ we observe that if degrees up to 0.902 protect, then the neighbour infection rate is of 14.6%. And, at this infection rate, and individual

[25] As noted by Jackson [21], for many of the distributions usually studied (including the power law, which will be used in examples below) a FOSD shift in the neighbour degree distribution $\tilde{F}$ necessarily implies a FOSD shoft in the degree distribution F.

[26] Formally, this refers to the derivative of $\delta_X^*(\theta)$ evaluated at the steady state θ^{ss}, which is $\frac{\alpha}{p_H \theta^{ss}}$. If $c/(\pi_s - \pi_I)$ is small, then θ^{ss} will be small. Hence this is particularly relevant for low marginal protection costs.

[27] Danon et al. [8] estimate the network of social encounters using data from a large survey and find that the degree distribution has a log-normal body and a power-law tail with exponent 2.45. Power-law degree distributions, typically with exponents between 2 and 3, have also been found in computer networks (see e.g. Barabási and Albert [3] and Pastor-Satorras and Vespignani [27], and the literature cited therein).

[28] The converse is true as well. Suppose $\lambda \neq 1$. Then $F_\lambda(\delta) = \frac{\delta^{1-\lambda} - \varepsilon^{1-\lambda}}{1-\varepsilon^{1-\lambda}}$, which is increasing in λ. The statements regarding first-order stochastic dominance follow.

protects if and only if her degree is between ε and 0.902.

It is easy to see that, as the society becomes sparser (λ increases), the steady-state neighbour infection rate decreases. The decrease in the neighbour infection rate results in protection being chosen by a smaller set of degrees. However, since a larger fraction of the population has low degrees, the fraction of the population protecting, also known as *coverage*, may actually increase. Table 2 provides a summary. Note that coverage initially increases as the society becomes sparser, and then it drops (from 99.7% if $\lambda = 2$ to 99.6% if $\lambda = 3$). This is the same feature that was observed for regular societies: the infection rate becomes so small that protecting is not worth the cost. Note that the prevalence of the disease falls from 15.2% if $\lambda = 0$ to 0.1% if $\lambda = 3$.

B.3 Durable and perfect protection

The next result presents the conditions for existence in the case where protection expires with exogenous probability.

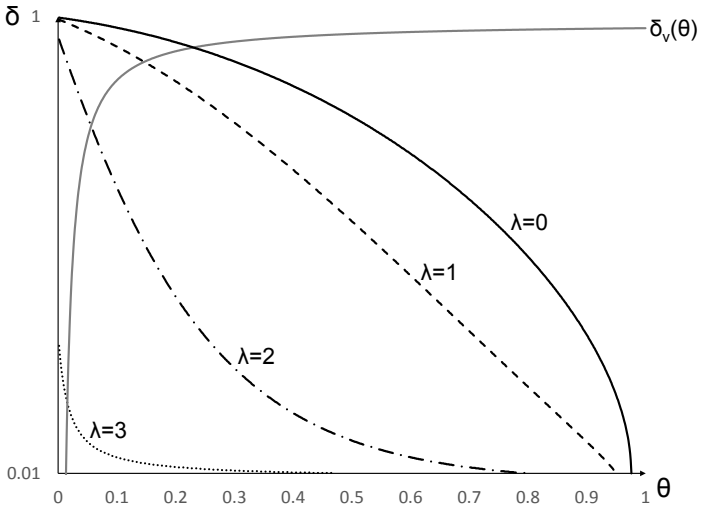
Proposition 5. *Consider a society with degree distribution $F(\delta)$. Suppose that protection is perfect and expires with per-period probability η, that the individual can choose either full protection or no protection, and that $\eta < \frac{1}{1+c/(\pi_S - \pi_I)}$. A steady state $\theta^{ss} > 0$ exists if and only if*

$$\langle\delta\rangle \left(\frac{\alpha}{p_H} - \langle\delta\rangle \right) < \sigma^2.$$

If such a steady state neighbour infection rate exists, it is unique.

When studying regular societies, we found that, once the level of protection is endogenized, denser societies need not feature higher infection rates. The following example illustrates this for non-regular societies.

Example 3. Suppose that the degree distribution follows a power-law, and that $p_H = 0.8$, $p_L = .001$, $\alpha = .01$, $\varepsilon = .01$, $(\pi_S - \pi_I) = 1$, $c = 50$, and $\eta = 0.01$. Figure 7 illustrates the steady state analysis for different values of the exponent λ. Each positive-slope curve indicates, for the corresponding degree distribution, the resulting neighbour infection rate θ if individuals whose degree is greater than or equal to a given δ fully protect and individuals with degree smaller than δ do not protect. If no protection technology was available then steady states would be on the intersection of these curves with the horizontal line crossing the vertical axis at $\delta = 1$. Thus, the result by Jackson and Rogers [22] regarding first order stochastic dominance arises as the special case where protection



(a)

Figure 6: Steady states with interaction-specific protection under power-law degree distributions: $p_H = .8$, $p_L = .01$, $\alpha = .01$, $\varepsilon = .01$, $(\pi_S - \pi_I) = 1$, $c = 1$. Each negative-slope curve indicates, for the corresponding degree distribution, the resulting neighbour infection rate θ if individuals whose degree is smaller than or equal to a given δ fully protect and individuals with degree greater than δ do not protect. If no protection technology was available then steady states would be on the intersection of these curves with the horizontal line crossing the vertical axis at $\delta = \varepsilon$. In the present case, steady states correspond to the intersection of these curves with $\delta^*_X(\theta)$.

Table 2: Interaction-specific protection: Power-law degree distribution with exponent λ.*

	$\lambda = 0$	$\lambda = 1$	$\lambda = 2$	$\lambda = 3$
θ^{ss}	22.8%	14.6%	5.6%	1.5%
ρ^{ss}	15.2%	4.7%	0.5%	0.1%
$\delta_{\max}$	.933	.902	.762	.164
Coverage	93.2%	97.8%	99.7%	99.6%

* $p_H = .8$, $p_L = .01$, $\alpha = .01$, $\varepsilon = .01$, $(\pi_S - \pi_I) = 1$, $c = 1$.

$\delta_{\max}$ is maximum degree that protects.

Coverage is % of population that protects, i.e. $F(\delta_{\max})$.

is too expensive. In the case illustrated in the figure, steady states correspond to the intersection of these curves with $\delta_X^{**}(\theta)$. For example, for $\lambda = 1$, if degrees $[0.193, 1]$ protect, then the neighbour infection rate is of 6.0%. And, at this infection rate, an individual protects if and only if her degree is in $[0.193, 1]$.

Note from Figure 7 how, as the set of degrees that protect becomes larger, the associated infection rate drops faster in denser societies. The reason is simple: these societies have more individuals with high degrees. If protection is sufficiently affordable, it is then possible for denser societies to feature lower infection rates. In this example, the steady-state prevalence of the disease is largest in the sparsest society. This is entirely due to the endogenous vaccination efforts: Table 3 shows that the fraction of the population vaccinated is 69.7% in the case of $\lambda = 0$, and merely 3.6% if $\lambda = 3$.

B.4 Endogenous expiration

To present the results for the case of protection that expires upon infection, it will be useful to generalize the notion of stability.

Definition 5. *For a given degree distribution F, let $\theta^*(\Delta)$ denote the maximum possible neighbour infection rate if individuals whose degree is in $\Delta \subseteq [\varepsilon, 1]$ fully protect and all other individuals do not protect. A steady state $\theta^{ss} > 0$ is stable if there exist $\epsilon_1 > 0$ and $\epsilon_2 > 0$ such that*

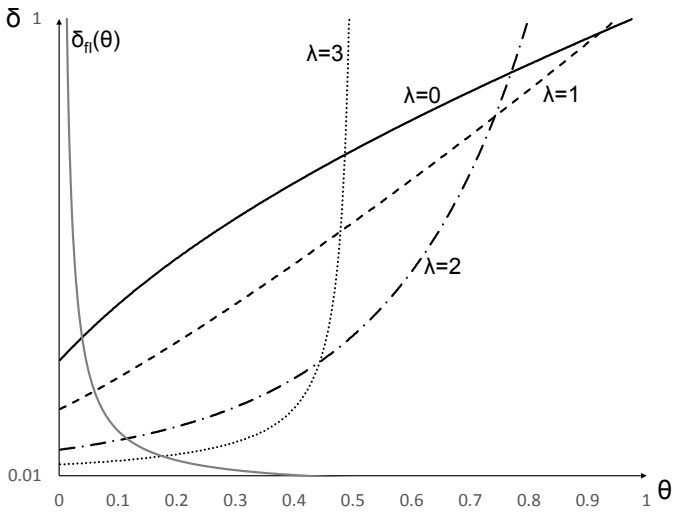
- *for any $\theta \in [\theta^{ss} - \epsilon_1, \theta^{ss})$, $\theta < \theta^*([\varepsilon, \delta_N(\theta)])$.*
- *for any $\theta \in (\theta^{ss}, \theta^{ss} + \epsilon_2]$, $\theta > \theta^*([\varepsilon, \delta_N(\theta)])$.*

A steady state is said to be a saddle (unstable) if only one (none) of these conditions holds.

Note that a steady state neighbour infection rate θ^{ss} satisfies $\theta^{ss} = \theta^*([\varepsilon, \delta_N(\theta^{ss})])$. Thus, in a stable steady state small decreases (increases) in the neighbour infection rate do not make protection so attractive (unattractive) as to cause further decreases (increases) in the infection rate. Let us further define:

$$\begin{aligned} \hat{\theta} &:= \frac{(p_H - p_L)(\pi_S - \pi_I) - cp_L\alpha}{cp_Lp_H}, \\ \hat{H}_F(\theta) &:= \int_\varepsilon^1 \frac{f(\delta)\delta}{\langle\delta\rangle} \frac{\delta p_L \theta}{\alpha + \delta p_L \theta} d\delta. \end{aligned}$$

The quantity $\hat{\theta}$ represents the maximum neighbour infection rate such that protection is profitable for all possible degrees. For any neighbour infection rate greater than $\hat{\theta}$,



(a)

Figure 7: Steady states with durable and perfect protection under power-law degree distributions: $p_H = .8$, $p_L = .01$, $\alpha = .01$, $\varepsilon = .001$, $(\pi_S - \pi_I) = 1$, $c = 50$, $\eta = 0.01$. Each positive-slope curve indicates, for the corresponding degree distribution, the resulting neighbour infection rate θ if individuals whose degree is greater than or equal to a given δ fully protect and individuals with degree smaller than δ do not protect. If no protection technology was available then steady states would be on the intersection of these curves with the horizontal line crossing the vertical axis at $\delta = 1$. In the case illustrated in the figure, steady states correspond to the intersection of these curves with $\delta_X^{**}(\theta)$.

Table 3: Durable and perfect protection: Power-law degree distribution with exponent λ.*

	$\lambda = 0$	$\lambda = 1$	$\lambda = 2$	$\lambda = 3$
θ^{ss}	3.9%	6.0%	11.4%	17.5%
ρ^{ss}	9.5%	13.3%	15.6%	17.8%
$\delta_{\min}$	.310	.193	.091	.053
Coverage	69.7%	35.7%	10.1%	3.6%

* $p_H = .8$, $p_L = .001$, $\alpha = .01$, $\varepsilon = .01$, $(\pi_S - \pi_I) = 1$, $c = 50$, $\eta = .01$. $\delta_{\min}$ is minimum degree that protects.

Coverage is % of population that protects, i.e. $1 - F(\delta_{\min})$.

individuals of degree one strictly prefer not to protect. Fixed points of the function $\hat{H}_F(\theta)$ correspond to steady state neighbour-infection rates where all individuals in the population protect. Thus, for example, if the unique fixed point is at zero, then the disease would be eradicated if everyone in the population protected.

Proposition 6. *Consider a society with degree distribution $F(\delta)$. Suppose that protection expires upon infection and that $0 < p_L < p_H \frac{1}{1+\frac{\alpha c}{\pi_S - \pi_I}}$. If a steady state $\theta^{ss} > 0$ exists, then*

$$\langle \delta \rangle \left(\frac{\alpha}{p_H} - \langle \delta \rangle \right) < \sigma^2.$$

If $\hat{\theta} \leq \hat{H}_F(\hat{\theta})$, then a steady state $\theta^{ss} > 0$ exists. If $\hat{\theta} > \hat{H}_F(\hat{\theta})$ and a steady state $\theta^{ss} > 0$ exists then the smallest such steady state where not all agents protect is not stable.

As already seen in the case of regular societies, for there to be a positive-infection steady state it is necessary but not sufficient for the disease to become endemic in the absence of protection. The conditions for existence are related to the cost and effectiveness of the protection technology if protection expiration is related to the infectious status. The condition $\hat{\theta} \leq \hat{H}_F(\hat{\theta})$ means that the resulting neighbour infection rate if everyone protected is *higher* than the one required to incentivize protection by the entire population. As a result, not to protect is a dominant strategy for high-degree individuals and a steady state with positive infection must exist.[29] Conversely, under the condition $\hat{\theta} > \hat{H}_F(\hat{\theta})$, if everyone else protects and there is still a positive probability of meeting someone infected, then protection is a strict best response even for the highest-degree individuals.

The following result characterizes the comparative statics of having a denser society in the sense of first order stochastic dominance.

Proposition 7. *Suppose protection expires upon infection and a positive-infection steady state exists under F. Let $\tilde{F}'$ FOSD $\tilde{F}$. Then there exists a steady state under F' with a neighbour infection rate that is strictly larger than any steady state neighbour infection rate under F. If, in addition, $\hat{\theta} > \hat{H}_{F'}(\hat{\theta})$ then the smallest steady state neighbour infection where not all agents protect is strictly smaller under F' than under F.*

The first statement of this result is simple to understand: denser societies feature larger steady-state infection rates. The second statement also offers an interesting insight. The

[29] In generic cases, a stable steady state will exist under this condition. But this cannot be established in general. Consider, for example, a degree distribution such that, for any θ, $\theta = \theta^*([\varepsilon, \delta_N(\theta)])$. That is, if the degrees that find protection profitable under θ protect, then the resulting infection rate is θ. Then any θ is an unstable steady state infection rate.

smallest steady state such that not all individuals in the population protect can be interpreted as a "tipping point" infection rate. Initial infection rates that are below this level will die out: some individuals find protection attractive, thereby reducing infection, which in turn makes protection attractive for a larger fraction of the population, and so on. Beyond this level, the disease will spread further and protection will unravel. The result thus states that the denser the society the lower the *neighbour* initial infection required in order to reach this tipping point. I conclude the section with the following illustrative example.

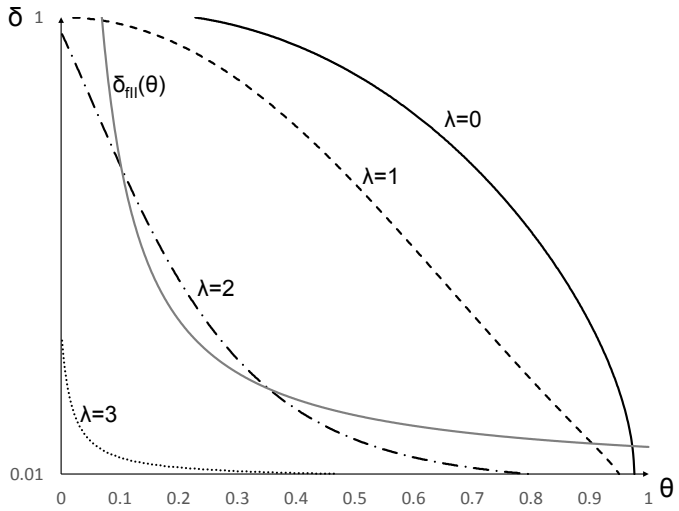
Example 4. Suppose that the degree distribution follows a power-law, and that $p_H = 0.8$, $p_L = .02$, $\alpha = .01$, $\varepsilon = .01$, $(\pi_S - \pi_I) = 1$, and $c = 600$. Figure 8 illustrates the steady state analysis for different values of the exponent λ, while Table 4 presents a summary of the results. As in the example with interaction-specific protection, the figure shows, for each degree distribution, the resulting neighbour infection rate θ if individuals whose degree is smaller than or equal to a given δ fully protect and individuals with degree greater than δ do not protect.

In the very sparse society with $\lambda = 3$, there is no steady state with positive infection; for any neighbour infection rate that is possible given the society's density, protection is a strictly dominant strategy for every individual. And if all individuals protect, then the disease cannot be sustained in the population. For power-laws with exponents one and two, two positive-infection steady states are possible. The denser society features the highest *stable* steady state $\lambda = 1$ (with 55.0% of infected individuals as opposed to only 5.8% for $\lambda = 2$). The denser society also features the smallest *unstable* steady state neighbour infection rate (6.9% vs. 10.2% for $\lambda = 2$). Note, however, that the fraction of infected individuals (ρ^{ss}) in the unstable steady state is in fact lower in the case of $\lambda = 2$ (1.3%) than under $\lambda = 3$ (2.9%). Finally, in the densest of societies considered ($\lambda = 0$), there is only one (stable) steady state, where only part of the population protects and the infection rate is highest.

B.5 Proofs

An important step in the analysis lies in understanding the conditions for existence of positive-infection steady states. The following simple but powerful result sheds light on this issue. The question posed is the following: if we fix the behaviour of each individual in the population, under what conditions will this behaviour imply a positive infection rate in the aggregate?

Lemma 1. *Consider a society with degree distribution $F(\delta)$. Fix the sets $\{P(p)\}_{p\in[p_L,p_H]}$*



(a)

Figure 8: Steady states with protection that expires upon infection under power-law degree distributions: $p_H = 0.8$, $p_L = .02$, $\alpha = .01$, $\varepsilon = .01$, $(\pi_S - \pi_I) = 1$, and $c = 600$. No positive-infection steady state exists if in the sparse society with $\lambda = 3$. Multiple steady states exist for $\lambda = 1$ and $\lambda = 2$. Finally, in the densest of societies considered ($\lambda = 0$), there is only one (stable) steady state, where only part of the population protects and the infection rate is highest.

Table 4: Protection that expires upon infection: Power-law degree distribution with exponent λ.*

	$\lambda = 0$	$\lambda = 1$	$\lambda = 2$	$\lambda = 3$
Positive θ^{ss} exists	Yes	Yes	Yes	No
Low (unstable) steady state				
θ^{ss}	-	6.9%	10.2%	-
ρ^{ss}	-	2.9%	1.3%	-
$\delta_{\max}$	-	.993	.674	-
Coverage	-	99.8%	99.5%	-
High (stable) steady state				
θ^{ss}	97.2%	90.4%	35.2%	-
ρ^{ss}	90.8%	55.0%	5.8%	-
$\delta_{\max}$	.074	.076	.195	-
Coverage	6.5%	44.2%	95.8%	-

* $p_H = .8$, $p_L = .02$, $\alpha = .01$, $\varepsilon = .01$, $(\pi_S - \pi_I) = 1$, $c = 600$.

$\delta_{\max}$ is maximum degree that protects.

Coverage is % of population that protects, i.e. $F(\delta_{\max})$.

such that an individual of degree δ chooses protection level $p \in [p_L, p_H]$ if and only if $\delta \in P(p) \subseteq [\varepsilon, 1]$. There exists a steady state $\theta^{ss} > 0$ if and only if

$$\alpha\langle\delta\rangle < \int_{p\in[p_L,p_H]} p \int_{\delta\in P(p)} \delta^2 \; dF(\delta) \; dp. \tag{21}$$

If such a steady state exists, it is unique.

Lemma 1. Let us define the function

$$g(p,\theta) \equiv \frac{\delta^2 p\theta}{\alpha + \delta p\theta}.$$

For fixed sets $\{P(p)\}_{p\in[p_L,p_H]}$, let us define the function

$$G(\theta) \equiv \int_{p\in[p_L,p_H]} \int_{\delta\in P(p)} g(p,\theta) \; dF(\delta) \; dp.$$

Condition (19) can be written as

$$\langle\delta\rangle\theta^{ss} = G(\theta^{ss}). \tag{22}$$

If $\alpha = 0$, then $G(\theta^{ss}) = \langle\delta\rangle$, so that (22) implies $\theta^{ss} = 1$, and all claims of the Lemma hold.

Suppose then that $\alpha \in (0, 1]$. The proof is completed with the following steps.

Step 1. If $\alpha > 0$, then $G(\theta)$ is increasing and concave. To see this, simply note that, if $\alpha > 0$, then $g(p,\theta)$ is increasing and concave in θ. Therefore, $G(\theta)$ is a sum of increasing and concave function, from where it follows that $G(\theta)$ is increasing and concave. It follows from this observation that, if a steady state $\theta^{ss} > 0$ exists, then it is unique.

Step 2. Combining Step 1 with condition (22), if a steady state $\theta^{ss} > 0$ exists, then it must be that $\langle\delta\rangle < G'(0)$. Note that

$$G'(0) = \int_{p\in[p_L,p_H]} \int_{\delta\in P(p)} \frac{p}{\alpha}\delta^2 \; dF(\delta) \; dp.$$

This shows that (21) is necessary for the existence of a steady state $\theta^{ss} > 0$.

Step 3. We finally show sufficiency of condition (21). Note that

$$\lim_{\theta\to\infty} g(p,\theta) = \delta,$$

so that $\lim_{\theta\to\infty} G(\theta) = \langle\delta\rangle$. Since $G(\theta)$ is increasing, it follows that $G(1) < \langle\delta\rangle$. Since $G(\theta)$ is continuous, (21) is sufficient as well as necessary for the existence of a steady state

$\theta^{ss} > 0$. □

According to Lemma 1, for any behaviour at the individual level where there is a positive fraction of the population which does not have perfect protection, a unique steady state with positive infection will exist if α is sufficiently small. Lemma 1 also naturally encompasses the case of a regular society. If, for example, all individuals choose full protection, then the right-hand side of condition (21) simplifies to $p_L\delta^2$, and we obtain that a positive-infection steady exists if and only if $\alpha < p_L\delta$. This is equivalent to $\theta_l^{ss} = 1 - \frac{\alpha}{\delta p_L}$ being strictly positive.

Note that, under all three types of protection, the set of degrees for which protection is profitable is a convex set. For given $\underline{\delta} \in [\varepsilon, 1]$ and $\bar{\delta} \in [\underline{\delta}, 1]$, let us define the function

$$H_F(\theta, \underline{\delta}, \bar{\delta}) := \int_{\varepsilon}^{\underline{\delta}} \frac{f(\delta)\delta}{\langle\delta\rangle} \frac{\delta p_H \theta}{\alpha + \delta p_H \theta} d\delta + \int_{\underline{\delta}}^{\bar{\delta}} \frac{f(\delta)\delta}{\langle\delta\rangle} \frac{\delta p_L \theta}{\alpha + \delta p_L \theta} d\delta + \int_{\bar{\delta}}^{1} \frac{f(\delta)\delta}{\langle\delta\rangle} \frac{\delta p_H \theta}{\alpha + \delta p_H \theta} d\delta. \quad (23)$$

Suppose a degree-δ individual fully protects if $\delta \in (\underline{\delta}, \bar{\delta})$ and does not protect otherwise, and that (21) is satisfied. Then $\theta^{ss}(\underline{\delta}, \bar{\delta}) > 0$ is the unique positive solution to

$$\theta^{ss}(\underline{\delta}, \bar{\delta}) = H_F(\theta^{ss}(\underline{\delta}, \bar{\delta}), \underline{\delta}, \bar{\delta}). \quad (24)$$

The following two results show how $\theta^{ss}(\underline{\delta}, \bar{\delta})$ changes with changes in $\underline{\delta}$, $\bar{\delta}$, and the degree distribution F.

Lemma 2. *$\theta^{ss}(\underline{\delta}, \bar{\delta}) > 0$ is weakly decreasing in $\underline{\delta}$ and weakly increasing in $\bar{\delta}$.*

Lemma 2. I will show that $\theta^{ss}(\underline{\delta}, \bar{\delta}) > 0$ is weakly decreasing in $\underline{\delta}$. The proof of the other statement is analogous and omitted.

For a contradiction, suppose there exist $\underline{\delta}$ and $\underline{\delta}' > \underline{\delta}$ such that $\theta^{ss}(\underline{\delta}', \bar{\delta}) > \theta^{ss}(\underline{\delta}, \bar{\delta}) > 0$. That is,

$$H_F(\theta^{ss}(\underline{\delta}', \bar{\delta}), \underline{\delta}', \bar{\delta}) = \theta^{ss}(\underline{\delta}', \bar{\delta}) > \theta^{ss}(\underline{\delta}, \bar{\delta}) = H_F(\theta^{ss}(\underline{\delta}, \bar{\delta}), \underline{\delta}, \bar{\delta}) > 0. \quad (25)$$

It is easy to see that $H_F(\theta, \underline{\delta}, \bar{\delta})$ is strictly concave in θ. That is, for any $\theta \neq \theta'$ and any $w \in (0, 1)$,

$$wH_F(\theta, \underline{\delta}, \bar{\delta}) + (1 - w)H_F(\theta', \underline{\delta}, \bar{\delta}) < H_F(w\theta + (1 - w)\theta', \underline{\delta}, \bar{\delta}). \quad (26)$$

Note that $H_F(\theta, \underline{\delta}, \bar{\delta})$ is weakly decreasing in $\underline{\delta}$. Therefore, $\underline{\delta}' > \underline{\delta}$ implies that $H_F(\theta, \underline{\delta}', \bar{\delta}) \leq H_F(\theta, \underline{\delta}, \bar{\delta})$ for any θ. In particular, for $\theta = \theta^{ss}(\underline{\delta}, \bar{\delta})$,

$$H_F(\theta^{ss}(\underline{\delta}, \bar{\delta}), \underline{\delta}', \bar{\delta}) \leq H_F(\theta^{ss}(\underline{\delta}, \bar{\delta}), \underline{\delta}, \bar{\delta}). \quad (27)$$

By (25), let us define

$$w := 1 - \frac{\theta^{ss}(\underline{\delta}, \bar{\delta})}{\theta^{ss}(\underline{\delta}', \bar{\delta})} \in (0, 1).$$

Combining the fact that $H_F(0, \underline{\delta}', \bar{\delta}) = 0$ with (27), it follows that

$$\begin{aligned} wH_F(0, \underline{\delta}', \bar{\delta}) + (1-w)H_F(\theta^{ss}(\underline{\delta}', \bar{\delta}), \underline{\delta}', \bar{\delta}) &= \theta^{ss}(\underline{\delta}, \bar{\delta}) \\ &= H_F(\theta^{ss}(\underline{\delta}, \bar{\delta}), \underline{\delta}, \bar{\delta}) \\ &\geq H_F(\theta^{ss}(\underline{\delta}, \bar{\delta}), \underline{\delta}', \bar{\delta}) \\ &= H_F(w0 + (1-w)\theta^{ss}(\underline{\delta}', \bar{\delta}), \underline{\delta}', \bar{\delta}), \end{aligned}$$

which violates (26). □

Lemma 3. *Suppose that, for a degree distribution F with neighbour degree distribution $\tilde{F}$, $\theta^{ss}(\varepsilon, \bar{\delta}) > 0$. For any degree distribution F' such that its associated neighbour degree distribution $\tilde{F}'$ FOSD $\tilde{F}$, $\theta^{ss'}(\varepsilon, \bar{\delta}) > \theta^{ss}(\varepsilon, \bar{\delta})$.*

Lemma 3. Define the function $h : [\varepsilon, 1] \to \mathbb{R}$ where

$$h(\delta) = \begin{cases} \frac{\delta p_L \theta}{\alpha + \delta p_L \theta} & \text{if } \delta \leq \bar{\delta} \\ \frac{\delta p_H \theta}{\alpha + \delta p_H \theta} & \text{if } \delta > \bar{\delta} \end{cases}.$$

Then note that, for a degree distribution $F(\delta)$ with neighbour degree distribution $\tilde{F}(\delta)$,

$$H_F(\theta, \varepsilon, \bar{\delta}) = \mathbb{E}_{\tilde{F}}\big[h(\delta)\big].$$

If $p_L > 0$, then $h(\delta)$ is strictly increasing and hence $\tilde{F}'(\delta)$ FOSD $\tilde{F}(\delta)$ implies that $H_{F'}(\theta, \varepsilon, \bar{\delta}) > H_F(\theta, \varepsilon, \bar{\delta})$. If $p_L = 0$, note that it must be that $\int_{\bar{\delta}}^1 d\tilde{F}(\delta) > 0$, or otherwise $\theta^{ss}(\varepsilon, \bar{\delta}) = 0$. That is, there is positive probability mass on $[\bar{\delta}, 1]$ under $\tilde{F}(\delta)$. Since $h(\delta)$ is strictly increasing over $[\bar{\delta}, 1]$, $\tilde{F}'(\delta)$ FOSD $\tilde{F}(\delta)$ implies that $H_{F'}(\theta, \varepsilon, \bar{\delta}) > H_F(\theta, \varepsilon, \bar{\delta})$. Combining $H_{F'}(\theta, \varepsilon, \bar{\delta}) > H_F(\theta, \varepsilon, \bar{\delta})$ with the fact that, for any F, $H_F(\theta, \varepsilon, \bar{\delta})$ is strictly concave in θ, it follows (by the same steps as in the proof of Lemma 2) that $\theta^{ss'}(\varepsilon, \bar{\delta}) > \theta^{ss}(\varepsilon, \bar{\delta})$. □

Proposition 4. Consider first the direction $\Rightarrow$. Suppose that $\langle\delta\rangle \left(\frac{\alpha}{p_H} - \langle\delta\rangle\right) \geq \sigma^2$, or

$\alpha\langle\delta\rangle \geq p_H\left(\sigma^2 + \langle\delta\rangle^2\right)$. Then note that

$$\begin{aligned}\alpha\langle\delta\rangle &\geq p_H\left(\sigma^2 + \langle\delta\rangle^2\right) \\ &= \int_{\varepsilon}^{1} p_H\delta^2 dF(\delta) \\ &\geq \int_{p\in[p_L,p_H]} p \int_{\delta\in P(p)} \delta^2 \; dF(\delta) \; dp\end{aligned}$$

for any partition $\{P(p)\}_{p\in[p_L,p_H]}$ of $[\varepsilon,1]$. By Lemma 1, a positive-infection steady state cannot exist.

Consider next the direction $\Leftarrow$. Note that in a steady state, the neighbour infection rate is $\theta^{ss}(\varepsilon,\bar{\delta})$, where individuals with degree $\delta < \bar{\delta}$ strictly prefer to protect and those with degree $\delta > \bar{\delta}$ strictly prefer not to protect. Condition (20) implies that $\theta^{ss}(\varepsilon,\varepsilon) > 0$. Three cases are possible.

Case 1: $\theta^{ss}(\varepsilon,\varepsilon) < {\delta_X^*}^{-1}(\varepsilon)$. Since ${\delta_X^*}^{-1}(\delta)$ is increasing in δ, ${\delta_X^*}^{-1}(\varepsilon) \leq {\delta_X^*}^{-1}(\delta)$ for any $\delta \in [\varepsilon,1]$. Since, by Lemma 2, $\theta^{ss}(\varepsilon,\bar{\delta})$ is non-increasing in $\bar{\delta}$, $\theta^{ss}(\varepsilon,\bar{\delta}) \leq \theta^{ss}(\varepsilon,\varepsilon)$ for any $\bar{\delta} \in [\varepsilon,1]$. Combining both observations,

$$\theta^{ss}(\varepsilon,\bar{\delta}) < {\delta_X^*}^{-1}(\delta)$$

for any $\delta \in [\varepsilon,1]$ and any $\bar{\delta} \in [\varepsilon,1]$. Thus, for any possible long-run neighbour infection rate, any individual strictly prefers not to protect. Hence in the unique steady state no individual protects and $\theta^{ss}(\varepsilon,\varepsilon) > 0$ is the neighbour infection rate.

Case 2: $\theta^{ss}(\varepsilon,1) > {\delta_X^*}^{-1}(1)$ and $\frac{(p_H-p_L)(\pi_S-\pi_I)}{p_H c} > 1$. Note that $\frac{(p_H-p_L)(\pi_S-\pi_I)}{p_H c} > 1$ implies ${\delta_X^*}^{-1}(1) > 0$. Hence, in this case $\theta^{ss}(\varepsilon,1) > {\delta_X^*}^{-1}(1) > 0$. Following the same arguments as in Case 1, in this case we have that

$$\theta^{ss}(\varepsilon,\bar{\delta}) > {\delta_X^*}^{-1}(\delta)$$

for any $\delta \in [\varepsilon,1]$ and any $\bar{\delta} \in [\varepsilon,1]$. Thus, for any possible long-run neighbour infection rate, any individual strictly prefers to protect. Hence in the unique steady state all individuals protect and $\theta^{ss}(\varepsilon,1) > 0$ is the neighbour infection rate.

Case 3: $\theta^{ss}(\varepsilon,\varepsilon) \geq {\delta_X^*}^{-1}(\varepsilon)$, and $\theta^{ss}(\varepsilon,1) \leq {\delta_X^*}^{-1}(1)$ or $\frac{(p_H-p_L)(\pi_S-\pi_I)}{p_H c} \leq 1$. Suppose $\theta^{ss}(\varepsilon,1) \leq {\delta_X^*}^{-1}(1)$. Since $\theta^{ss}(\varepsilon,1)$ is non-negative, it must be that $\frac{(p_H-p_L)(\pi_S-\pi_I)}{p_H c} \geq 1$. Otherwise ${\delta_X^*}^{-1}(1) < 0$ and we would have a contradiction. $\frac{(p_H-p_L)(\pi_S-\pi_I)}{p_H c} \geq 1$ implies that $\lim_{\delta\to_{-}1} {\delta_X^*}^{-1}(\delta) > 0$. Define the function $g : [\varepsilon,1] \to \mathbb{R}$, where

$$g(x) := {\delta_X^*}^{-1}(x) - \theta^{ss}(\varepsilon,x).$$

Since ${\delta^*_X}^{-1}(x)$ is strictly increasing and continuous and (by Lemma 2) $\theta^{ss}(\varepsilon, x)$ is non-increasing and continuous in x, $g(x)$ is strictly increasing and continuous. Note that $g(\varepsilon) \leq 0$ and $\lim_{x \to -1} g(x) \geq 0$. Thus, there is a unique x^* such that $g(x^*) = 0$. The unique steady-state neighbour infection rate is given by $\theta^{ss}(\varepsilon, x^*)$; agents with degree smaller than x^* fully protect and agents with degree greater than x^* do not protect; agents with degree equal to x^* choose any level of protection. The case with $\frac{(p_H - p_L)(\pi_S - \pi_I)}{p_H c} \leq 1$ is analogous and omitted.

Now suppose that $\theta^{ss} > 0$ under distribution $F(\delta)$, and that for $F'(\delta)$ we have that $\tilde{F}'(\delta)$ FOSD $\tilde{F}(\delta)$. For a contradiction, suppose that $\theta^{ss'} \leq \theta^{ss}$. Let δ^* ($\delta^{*'}$) denote the threshold such that all degrees up to δ^* ($\delta^{*'}$) protect in the steady state under $F(\delta)$ ($F'(\delta)$). By Lemma 3, $\tilde{F}'(\delta)$ FOSD $\tilde{F}(\delta)$ implies that for any threshold $\bar{\delta}$ such that $\theta^{ss}(\varepsilon, \bar{\delta}) > 0$, $\theta^{ss'}(\varepsilon, \bar{\delta}) > \theta^{ss}(\varepsilon, \bar{\delta})$. Since, by Lemma 2, $\theta^{ss'}(\varepsilon, \bar{\delta})$ is non-increasing in $\bar{\delta}$, for $\theta^{ss'} \leq \theta^{ss}$ it must be that $\delta^{*'} > \delta^*$. This contradicts ${\delta^*_X}^{-1}(\delta)$ being strictly increasing. □

Proposition 5. The direction $\Rightarrow$ is identical to the proof of Proposition 4. For the other direction, the steps are the same as in that proof, but replacing ${\delta^*_X}^{-1}(\delta)$ with ${\delta^{**}_X}^{-1}(\delta)$ and $\theta^{ss}(\varepsilon, \bar{\delta})$ with $\theta^{ss}(\underline{\delta}, 1)$, and noting that ${\delta^{**}_X}^{-1}(\delta)$ is *decreasing* and (by Lemma 2) $\theta^{ss}(\underline{\delta}, 1)$ is *non-decreasing* in $\underline{\delta}$. □

Proposition 6. The necessary condition for existence of a positive steady-state neighbour infection rate follows from Lemma 1.

Suppose $\hat{\theta} \leq \hat{H}_F(\hat{\theta})$. Note that

$$\begin{aligned} \hat{\theta} &= \delta_N^{-1}(1), \\ \hat{H}_F(\theta) &= H_F(\theta, \varepsilon, 1), \end{aligned}$$

where $H_F(\theta, \varepsilon, 1)$ is defined in (23). If $\hat{\theta} = \hat{H}_F(\hat{\theta}) = H_F(\hat{\theta}, \varepsilon, 1)$, then clearly $\hat{\theta} > 0$ is a steady-state neighbour infection rate; in this steady state all individuals protect. Suppose now that $\hat{\theta} < \hat{H}_F(\hat{\theta}) = H_F(\hat{\theta}, \varepsilon, 1)$. Since $H_F(\theta, \varepsilon, 1)$ is increasing and concave in θ, it follows (by the same arguments as in the proof of Lemma 2) that $\theta^{ss}(\varepsilon, 1) > \hat{\theta} = \delta_N^{-1}(1)$. Consider then the following two cases.

Case 1: $\theta^{ss}(\varepsilon, \varepsilon) \geq \delta_N^{-1}(\varepsilon)$. Then $\theta^{ss}(\varepsilon, \varepsilon) \geq \theta^{ss}(\varepsilon, 1) > \hat{\theta} > 0$ is a positive steady-state neighbour infection rate.

Case 2: $\theta^{ss}(\varepsilon, \varepsilon) < \delta_N^{-1}(\varepsilon)$. Since $\theta^{ss}(\varepsilon, \delta)$ and $\delta_N^{-1}(\delta)$ are continuous in δ, there exists $\delta^* \in (\varepsilon, 1)$ such that $\theta^{ss}(\varepsilon, \delta^*) < \delta_N^{-1}(\delta^*)$. That is, there exists a steady state where

individuals of degree less than or equal to δ^* fully protect and individuals of degree greater than δ^* do not protect. The associated neighbour infection rate is positive, since $\theta^{ss}(\varepsilon, \delta^*) \geq \theta^{ss}(\varepsilon, 1) > \hat{\theta} > 0$.

Suppose next that $\hat{\theta} > \hat{H}_F(\hat{\theta})$, and let $\theta^{ss}_{\min}$ denote the smallest steady state where not all agents protect. For a contradiction, suppose this steady state is stable. Then, there must exist $\epsilon > 0$ such that for $\theta = \theta^{ss}_{\min} - \epsilon > \hat{\theta}$,

$$\theta < \theta^*([\varepsilon, \delta_N(\theta)]) = \theta^{ss}(\varepsilon, \delta_N(\theta)),$$

where $\delta_N(\theta) < \delta_N(\hat{\theta}) = 1$ (since $\theta > \hat{\theta}$ and $\delta_N(\cdot)$ is decreasing under the conditions of the proposition). Now note:

$$\hat{\theta} > \hat{H}_F(\hat{\theta}) = \theta^{ss}(\varepsilon, 1) = \theta^{ss}(\varepsilon, \delta_N(\hat{\theta})).$$

Since $\delta_N(\cdot)$ and $\theta^{ss}(\cdot, \cdot)$ are continuous functions, both inequalities combined imply that there exists $\theta' \in (\hat{\theta}, \theta^{ss}_{\min})$ such that

$$\theta' = \theta^{ss}(\varepsilon, \delta_N(\theta')),$$

where $\delta_N(\theta') < \delta_N(\hat{\theta}) = 1$ (since $\theta' > \hat{\theta}$ and $\delta_N(\cdot)$ is decreasing). This contradicts $\theta^{ss}_{\min}$ being the smallest steady state such that not all agents protect. □

Proposition 7. This result follows from Lemma 3 using analogous arguments as the ones used for the proofs of Propositions 4 and 7. □