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The threat of asymptomatic carriers and the benefits of testing

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ABSTRACT

I present a model of infectious disease transmission with asymptomatic carriers, social distancing, and diagnostic testing. First, I study the impact of asymptomatic carriers on the spread of an infectious disease in the absence of testing, to determine when their presence increases the overall prevalence of symptomatic infection and hence unhealthy agents. Then, I consider mass testing and isolation policies to identify and isolate asymptomatic carriers, and incorporate them into my model. I establish that diagnostic testing successfully reduces steady state disease prevalence. I then explore the implications of testing accuracy, explicitly studying the impact of false positive and false negative test results. I find that reducing the rate of false negatives is unambiguously beneficial, since it improves the identification and isolation of asymptomatic carriers. In contrast, reducing the rate of false positives can be detrimental: by limiting the unintended isolation of susceptible individuals, lower rates of false positives reduce the overall level of social distancing in the population and increase disease spread. Hence, I demonstrate how, under certain conditions, false positive results can improve social welfare.

1. Introduction

As the first cases of Covid-19 were recorded in China, experts worried the outbreak could not be contained. Of concern were alleged asymptomatic carriers: people displaying no symptoms, but nonetheless contagious. In their presence, standard symptom-based detection measures would hardly be effective in limiting transmission, as these individuals show no sign of infection. As the virus spread around the world, studies confirmed the existence of asymptomatic infections. Thus, health authorities repeatedly warned countries of the threat posed by asymptomatic carriers and recommended mass testing policies to identify and isolate such cases.

This paper studies the impact of asymptomatic carriers on the spread of an infectious disease and the mitigating role of diagnostic testing. To this end, I extend the model proposed by Chen et al. (2011). In an otherwise standard framework, the authors endogenize the contact rate between individuals, the main driver of epidemics. In particular, they assume that health authorities detect all infected agents in each period and place them in quarantine. Susceptible individuals, by contrast, choose their level of social activity to balance the benefits of interacting with others against the risk of infection.

In my model, I let a share of new infections to be asymptomatic. This type of infections do not affect an individual's health and are therefore unobservable, without testing, both to health authorities and to asymptomatic agents themselves. Symptomatic infections, on the other hand, make an individual unhealthy. Following Chen et al. (2011), I

assume that health authorities detect and quarantine all symptomatic agents in each period. Healthy agents, comprising both susceptible and asymptomatic individuals, instead choose their level of social activity.

Without testing, health authorities cannot identify and isolate asymptomatic carriers. First, I show that asymptomatic carriers can thus play a crucial role in sustaining disease transmission and causing an otherwise containable outbreak to become endemic. By raising the basic reproduction number of an infectious disease, asymptomatic infections can be key for a disease to take root in the population and become endemic, even when traditional symptom-based detection strategies are in place. This dynamic plausibly contributed to the early spread of Covid-19 in China, where asymptomatic carriers likely undermined the extensive containment efforts.

I then examine the effect of asymptomatic carriers on the long-run evolution of an epidemic, focusing on their implications for public health, defined as the long-run prevalence of unhealthy agents in the population, and the degree of social distancing. To this end, I characterize the steady state of the model. I show that both variables can be jointly determined as a solution to a system of two equations. These equations define curves with a simple and intuitive graphical representation, especially convenient for deriving comparative statics.

Thus, I highlight a clear trade-off stemming from asymptomatic transmission. All else equal, more asymptomatic infections mean fewer symptomatic ones, which improves public health. Still, asymptomatic

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agents boost disease transmission, raising the total number of infections. These opposing effects imply that the long-run prevalence of unhealthy individuals can be non-monotonic in the share of asymptomatic cases, and I provide conditions under which this occurs.

Given the limitations of symptom-based detection, the second part of the paper explicitly incorporates diagnostic testing. I assume that health authorities test every healthy individual in each period and isolate those who test positive. Importantly, I allow tests to be imperfect, producing both false positive and false negative results, and examine their implications, which is the main contribution of my paper.

Testing and isolation policies reduce steady state disease prevalence. Tests producing fewer false negative results are better at identifying and isolating infectious individuals and are unambiguously beneficial, as they reduce disease transmission and lower the steady state prevalence of unhealthy agents. In contrast, tests producing fewer false positive results lead to the isolation of fewer susceptible individuals. I show that reducing the rate of false positives can raise steady state disease prevalence and even reduce overall welfare in the population.

False positive results lead to the unintended isolation of some susceptible agents. By reducing the exposure of these individuals at risk of infection, false positive results slow transmission and can reduce overall disease prevalence. Yet, since these individuals are not infectious, one may question the efficiency of restricting their activity. I find that false positives can improve social welfare when the disease is highly transmissible and severe, which are conditions under which the negative externality of individual social activity is plausibly stronger.

1.1. Literature review

The mathematical analysis of the evolution of an epidemic rests upon compartmental models, first introduced by Kermack and McKendrick (1927). Among the most widely used frameworks are the Susceptible–Infected–Susceptible (SIS) and Susceptible–Infected–Recovered (SIR) models. The SIS model applies to diseases that do not confer immunity after recovery, allowing recovered individuals to become susceptible again. The SIR model assumes instead that individuals gain immunity upon recovery, effectively removing them from the susceptible population.

Both models share the same underlying logic: at the onset of an epidemic, a small share of the population is infected, while the remainder is susceptible. Over time, interactions between these groups produce new infections, potentially causing the epidemic to expand. The main insight from the most standard models is that the long-run outcome of an epidemic is fully determined by the basic reproduction number R_0 of the disease, defined as the expected number of secondary infections generated by a single infected individual in a fully susceptible population. When $R_0 < 1$, the epidemic tends to die out rapidly. Conversely, when $R_0 > 1$, a substantial fraction of the population eventually becomes infected, and such proportion increases with R_0 .

Building on this foundation, and following the interest brought about by the HIV epidemic first and more recently by the COVID-19 pandemic, significant research has focused on incorporating more realistic features into these basic models. By introducing asymptomatic carriers, social distancing and diagnostic testing, my work contributes to this literature on economic epidemiology, extensively reviewed in McAdams (2021).

Social Distancing. The main extension relative to the standard models concerns individuals' behavior during epidemics. Many works use expected utility theory to incorporate individuals' incentives to spontaneously reduce their contact rate with others during epidemics.

Kremer (1996) is an early paper incorporating people's incentives to change the amount of sexual partners during an HIV/AIDS epidemic, to show that imperfect vaccines may increase the prevalence of the disease. Then, Chen et al. (2011) and Chen (2012) let susceptible agents engage in social distancing in an SIS model, to show that

it decreases the size of an epidemic and changes some predictions. More recently, Toxvaerd (2020) and Toxvaerd and Makris (2020) incorporate social distancing in an SIR model. The former work points out how agents spontaneously behave as to “flatten the curve”. The latter considers the anticipated arrival of vaccines and treatments, highlighting their different implications for social distancing. Other papers incorporating social distancing in an SIR model are Leung et al. (2018), Eichenbaum et al. (2021), Farboodi et al. (2021), Gans (2022), Maloney and Taskin (2020) and Acemoglu et al. (2023). This latter work is particularly closest to my paper, as I discuss more in a few paragraphs.

Asymptomatic Infections. The standard models neglect asymptomatic carriers. Aguilar et al. (2020) and Aguilar and Gutierrez (2020) introduce them into an SIR model and derive an expression for the basic reproduction number explicitly accounting for the impact of asymptomatic infections. The authors then also consider social distancing, but model it as mechanical reduction in individuals' contact rate, and not as endogenous behavior, like in my paper.

Diagnostic Testing. Several recent works introduce diagnostic testing into epidemic models, including Alvarez et al. (2021), Piguillem and Shi (2022), Brotherhood et al. (2020), Deb et al. (2022), and Drakopoulos and Randhawa (2021). While these papers consider the impacts of testing, none explicitly model how testing influences individuals' social distancing behavior. Philipson and Posner (1993) is an early work considering testing during an epidemic. However, the focus is quite different from mine, as the authors consider voluntary testing and disclosure decisions in the HIV epidemic.

The paper most closely related to mine is Acemoglu et al. (2023), which studies the interplay between testing and social distancing in a network-structured epidemic. The key distinctions are that Acemoglu et al. (2023) model transmission on a network rather than within a standard compartmental model, and most notably, they do not explore the effects of false positive and false negative test results. My paper's main contribution is explicitly accounting for test imperfections (i.e., false negative and false positive test results) and studying their implications for the steady state prevalence of unhealthy agents and social welfare in the population. False positives and false negatives play a role in Ely et al. (2021). However, their work is very different from mine, as it considers a test allocation problem in a static setting.

2. Model without testing

I adapt the SIS model in Chen et al. (2011) to incorporate asymptomatic infections. The SIS model studies infectious diseases that do not provide immunity upon recovery. If recovered individuals become instead immune to reinfection, then the SIR model is more suitable. In this work, I consider the former as (i) Covid-19 infections do not provide permanent immunity and (ii) working with this model is convenient, as it can be solved analytically.

2.1. An SIS model with social distancing

Let time be discrete and consider a continuum of agents. At each point in time, every agent is either susceptible or infected, where the measure of the two compartments is S_t and I_t , respectively. Infected agents recover with probability $\rho \in (0, 1)$ at the end of each period, to become fully susceptible again. Susceptible agents get the disease only if they meet an infected individual. In this case, contagion occurs with probability $\beta \in (0, 1]$, which is the *transmission rate* of the disease.

Chen et al. (2011) assume that all infected agents are put into *quarantine*, which restricts their contact rate to some $\gamma \in (0, 1)$. Instead, susceptible agents choose a *level of social activity* $q_t \in [0, 1]$, balancing the benefits of going out with the cost of potentially contracting the disease. For each unit of activity, the likelihood of meeting an infected

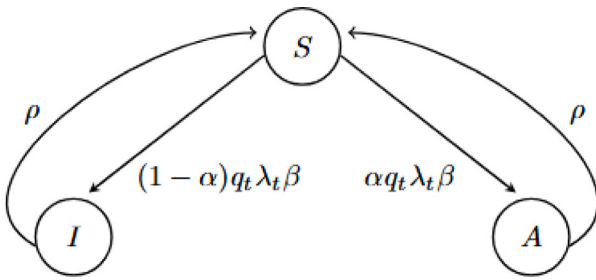


Fig. 1. Transition probability tree. Transition probabilities in arbitrary period t from the susceptible (S) state to the symptomatic (I) and asymptomatic (A) ones, and vice-versa.

agent is equal to their activity-weighted proportion in the population, as shown in the expression for next-period infections

$$I_{t+1} = S_t q_t \lambda_t \beta + (1 - \rho) I_t, \quad \text{where} \quad \lambda_t = \frac{\gamma I_t}{\gamma I_t + q_t S_t}. \quad (1)$$

Variable $\lambda_t \in [0, 1]$ is the *risk in the environment*, increasing in the share of infected agents and their activity level under quarantine, but decreasing in the share of susceptible individuals and their social activity level, as greater exposure by this latter group lowers the likelihood of meeting an agent that is infected, conditional on making a contact.

Susceptible agents are *myopic*: in each period, they choose a level of activity q_t to maximize their expected utility within that period. More activity is preferred to less, but it also raises the risk of infection, which is associated to a health cost $c > 0$. In particular, the authors assume that susceptible agents maximize in every period the utility function $U_t(q_t) = u(q_t) - q_t \lambda_t^e \beta c$, where $u(q)$ is increasing and concave and λ_t^e is their expectation of the risk in the environment.

To estimate the latter, susceptible agents must know the share of infected agents (I_t) and anticipate the activity level (q_t) of her susceptible peers. However, the authors let susceptible agents have *adaptive expectations*, which eliminates any strategic interaction, since susceptible agents then simply best-respond to the activity level observed in the previous period.

2.2. An SIS model with social distancing and asymptomatic carriers

In my model, I also incorporate asymptomatic carriers. Conditional on getting infected, I let an agent be asymptomatic (A) with probability $\alpha \in [0, 1]$ and symptomatic (I) otherwise. I let the recovery rate ρ be the same for both types of infection and assume no transition between them. Hence, the dynamics are as shown in Fig. 1.

Symptomatic cases are observable, as these agents become *unhealthy*. Thus, health authorities detect all symptomatic agents in every period and place them into *quarantine*, which restricts their contact rate to $\gamma \in (0, 1)$. Asymptomatic cases are instead unobservable. Asymptomatic agents remain *healthy* throughout and are thus only *carriers* of the disease, making them indistinguishable from susceptible ones. In each period, healthy agents, which are both asymptomatic carriers and susceptible individuals, choose a social activity level $q_t \in [0, 1]$.

Asymptomatic infections affect healthy agents' decision problem in two *direct* ways. First, conditional on getting infected, a healthy agent incurs the health cost only if the infection is symptomatic, and thus with probability $(1 - \alpha)$. Second, a healthy agent is not necessarily at risk of infection. Indeed, with probability $\mu_t = A_t / (S_t + A_t)$, the healthy agent is currently asymptomatic, and the only possible transition for the agent is into the susceptible compartment. Let μ_t^e denote an healthy agent's belief over the likelihood of being asymptomatic in period t . Assuming

that $u(q) = -(1 - q)^2 / 2$, the optimal level of activity for a healthy agent is determined in each period as

$$q_t = \arg \max_q -\frac{(1 - q)^2}{2} - q(1 - \mu_t^e) \lambda_t^e \beta (1 - \alpha) c. \quad (2)$$

I assume this specific functional form for the benefit component $u(q)$ of a healthy agent's utility function as it is convenient to work with: it ensures existence of a unique optimum and its simple derivative yields an intuitive closed-form solution for the optimal activity level, of the form $q^* = 1 - MC$, where MC refers to the marginal cost of exposure to the population.

Asymptomatic infections affect healthy agents' choice of a social activity level also *indirectly*, through their effect on the risk in the environment. This is now given by the following ratio

$$\lambda_t = \frac{\gamma I_t + q_t A_t}{\gamma I_t + q_t (S_t + A_t)}. \quad (3)$$

When $\alpha = 0$, only unhealthy agents are infected, and these are detected and put into quarantine. When $\alpha > 0$, some healthy individuals are instead asymptomatic carriers. As it can be shown by taking the derivative of expression (3), an increase in healthy agents' activity level q_t still decreases the environment risk, but to a lesser extent. Indeed, it also raises the likelihood of meeting an asymptomatic carrier.

As in Chen et al. (2011), I assume that healthy agents have adaptive expectations. Therefore, their belief over the likelihood of being asymptomatic is $\mu_t^e = \mu_{t-1}$ and their expectation of the risk in the environment is $\lambda_t^e = \lambda_{t-1}$. I discuss the significance of this assumption in Section 2.4. Finally, I let the population have a measure of one, whereby $S_t = 1 - I_t - A_t$.

2.3. Dynamic system without testing

Putting everything together, the model reduces to a dynamic system. This requires two initial conditions: I_0 and q_0 . I assume that $I_0 \approx 0$ and $q_0 = 1$, to capture the onset of an outbreak. The initial conditions then determine

$$A_0 = \frac{\alpha}{1 - \alpha} I_0, \quad S_0 = 1 - I_0 - A_0 \quad \text{and} \quad \lambda_0 = \frac{(\gamma I_0 + q_0 A_0)}{\gamma I_0 + q_0 (1 - I_0)}.$$

Then, in every period $t \geq 0$, we have

$$I_{t+1} = (1 - \rho) I_t + (1 - \alpha) S_t q_t \lambda_t \beta, \quad (4)$$

$$A_{t+1} = (1 - \rho) A_t + \alpha S_t q_t \lambda_t \beta, \quad (5)$$

$$S_{t+1} = 1 - I_{t+1} - A_{t+1}, \quad (6)$$

$$1 - \mu_{t+1}^e = 1 - \frac{A_t}{1 - I_t}, \quad (7)$$

$$q_{t+1} = \max\{0, 1 - (1 - \mu_{t+1}^e) \lambda_t \beta (1 - \alpha) c\}, \quad (8)$$

$$\lambda_{t+1} = \frac{\gamma I_{t+1} + q_{t+1} A_{t+1}}{\gamma I_{t+1} + q_{t+1} (1 - I_{t+1})}. \quad (9)$$

Eq. (8) is healthy agents' optimal decision rule and follows from their decision problem in (2). Note that my model reduces to the standard SIS one when every infection is symptomatic ($\alpha = 0$), there is no quarantine ($\gamma = q_t$) and all agents engage in full activity ($q_t = 1$).

2.4. Discussion

To incorporate asymptomatic carriers, I assumed that some infections present clear and recognizable symptoms while others leave an individual completely unaffected. In practice, infections can be associated with a wide spectrum of symptoms. Still, I believe modeling such heterogeneity is not necessary to study the role of asymptomatic carriers. Also, I let every symptomatic individual incur a health cost c .

This captures the *severity* of the disease, which depends on the extent to which its symptoms are debilitating, as well as on its fatality rate. The latter could be explicitly included in the model without substantial changes to its workings.

This binary characterization implies that symptom-based detection strategies allow health authorities to detect only symptomatic infections, without testing. The most they can do, then, to limit transmission is systematically force symptomatic agents to isolate. As these individuals face no risk of infection, successfully isolating them requires strong enforcement powers, as China demonstrated during Covid-19. Otherwise, by the assumption of a continuum of agents, parameter γ can be interpreted as the average contact rate under quarantine, when enforcement is imperfect.

Without testing, asymptomatic carriers cannot be detected. Thus, they belong to the same pool as susceptible individuals: that of healthy individuals. Full activity is optimal in the absence of disease, and an activity level can thus be interpreted as a degree of *social distancing* ($1 - q_t$). This might correspond to a percentage reduction in the frequency/duration of social activities, like meetings with friends and relatives, or visits to shops and entertainment venues.

A healthy agent selects their social activity level to maximize their own utility. However, their social distancing also affects the utility to other individuals in the population, through its impact on future disease prevalence. This positive externality implies that healthy agents engage in too little socially distancing, relative to what a social planner would prescribe.

I assume healthy individuals are boundedly rational when choosing a social activity level: they are myopic and rely on adaptive expectations. Myopia means that they select an activity level in period t to maximize their utility within that period, thus neglecting the impact of their decisions on their future utility and hence the full intertemporal dimension of their problem. As McAdams (2021) points out, this is a reasonable middle ground between entirely naive behavior (i.e., ignoring social distancing altogether) and fully forward-looking rationality, which may be unrealistic given its high cognitive burden.

Adaptive expectations imply that individuals use data from period $t - 1$ as estimates of the variables influencing their decision in period t , which are the share of infected agents and the social activity level of their healthy peers. Instead of forming accurate expectations, they adopt simple heuristics serving as cognitive shortcuts to reduce complexity. This behavior can be justified by the cognitive load involved in accurately estimating these variables and a lack of experience with epidemics, which are complex and infrequent events. Alternatively, individuals might misinterpret recent data as current, not recognizing that reports reflect past infection rates due to incubation periods or reporting delays.

As shown in the next section, this bounded rationality means individuals fail to fully maximize their utility in the short-term, when the epidemic is not in a steady state, whereas utility is instead maximized in the long run, when the epidemic reaches its steady state.

3. Analysis of model without testing

Before introducing testing, I study the case in which asymptomatic carriers cannot be detected. I consider the implications for the Steady State of the dynamic model in 2.3.

3.1. Disease-free and endemic steady state

Some time following the outbreak of an epidemic, the share of infected agents stops changing over time and the system reaches a Steady State (SS). Formally, a SS of the dynamical system in (4)–(9) is a vector $(I^*, A^*, S^*, \mu^*, q^*, \lambda^*)$ such that, if $(I_t, A_t, S_t, \mu_t, q_t, \lambda_t) = (I^*, A^*, S^*, \mu^*, q^*, \lambda^*)$ for some $t > 0$, then $(I_s, A_s, S_s, \mu_s, q_s, \lambda_s) = (I^*, A^*, S^*, \mu^*, q^*, \lambda^*)$ for every $s > t$.

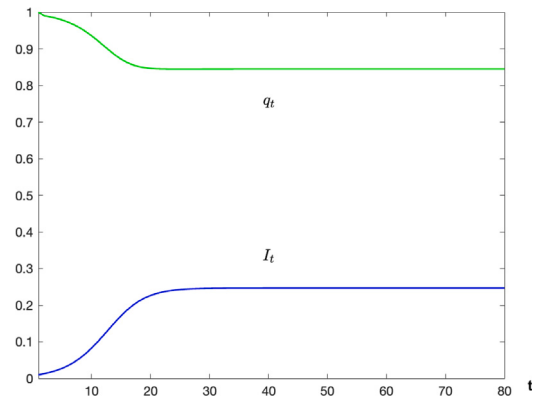


Fig. 2. Epidemic evolution simulation. Evolution of the prevalence of unhealthy agents (I_t) and of the level of social distancing (q_t) of healthy agents for every $t = 1, \dots, 80$, given parameter values $(\rho, \beta, \gamma, \alpha, c) = (.2, .7, .3, .6, 2)$.

Of interest is the prevalence of unhealthy agents I^* and the degree of social distancing q^* in the population. Importantly, the prevalence of unhealthy agents corresponds to the fraction of the population with a symptomatic infection at each point in time. This measure does not include asymptomatic agents, since these individuals are healthy throughout. Asymptomatic agents are important only as vehicles for the disease to spread further, potentially affecting the overall number of symptomatic and hence unhealthy individuals in the population.

An epidemic either converges to the *Disease-Free SS*, where the disease is eradicated ($I^* = 0$) and people stop distancing ($q^* = 1$), or to an *Endemic SS*, where a share $I^* > 0$ of the population is unhealthy at each point in time and people perpetually transition between the susceptible and infected state, maintaining some degree of social distancing.

Fig. 2 plots the evolution of the prevalence of unhealthy agents I_t and the social activity level q_t of healthy individuals given $(\rho, \beta, \gamma, \alpha, c) = (.2, .7, .3, .6, 2)$. Under this plausible set of parameters, inspired by the simulations in Chen et al. (2011) and used throughout this article to provide intuitions for my results, the system converges to an Endemic SS: about 25% of the population is unhealthy in each period and the level of social activity is reduced by about 15%.

In Fig. 2, the epidemic converges to an Endemic SS. Notice that healthy agents are not fully maximizing their utility during convergence. This is because adaptive expectations lead to inaccurate estimates of infection levels and of the activity level of the other healthy individuals. At the Endemic SS, variables are constant over time and expectations become instead accurate. Thus, individuals are maximizing their actual expected utility in the Endemic SS. Moreover, healthy agents' choices of a social activity level constitute a Nash equilibrium, as each agent is indeed best responding to others. The same considerations apply also to the Disease-Free SS.

The Disease-Free SS always exists. Proposition 1 characterizes the Endemic SS.

Proposition 1. (i) An Endemic SS exists if and only if

$$R_0 = \frac{\alpha\beta + (1-\alpha)\beta\gamma}{\rho} \geq 1. \quad (10)$$

(ii) If an Endemic SS exists, then (I^*, q^*) can be determined as a solution to

$$\begin{cases} I = \frac{(1-\alpha)q(\alpha\beta q + (1-\alpha)\beta\gamma - \rho)}{q(\alpha\beta q + (1-\alpha)\beta\gamma) - (1-\alpha)\rho(q-\gamma)}; \\ I = \frac{q(1-q)}{q(1-q) + c\rho}. \end{cases} \quad (11)$$

Then, $A^* = [\alpha/(1-\alpha)] I^*$, and S^* , μ^* and λ^* can be found through Eqs. (6), (7) and (9).

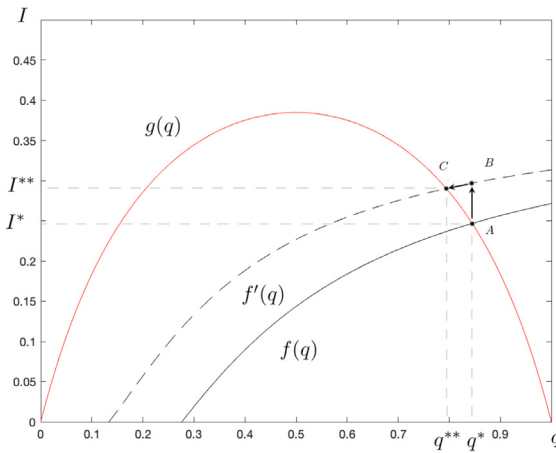


Fig. 3. Characterization of endemic SS and graphical analysis. The graph shows functions $f(q)$ and $g(q)$, as defined by the first and second lines of the RHS of Eq. (11). The first function describes an increasing curve. The second a parabola, which is always positive and crosses the origin and point (1,0). When $R_0 > 1$, function $f(q)$ is positive at $q = 1$, and the two curves intersect. In this case, an Endemic SS exists, with prevalence of unhealthy agents and activity level of healthy individuals corresponding to the coordinates of the intersection point. Intersection point A pins down the Endemic SS in Fig. 2. Increasing the transmissibility β of the disease shifts curve $f(q)$ upwards and moves the Endemic SS to point C.

When $R_0 < 1$, there exists only the Disease-Free SS: more people recover in each period than get infected, and the disease eventually dies out. When $R_0 \geq 1$, there exists also an Endemic SS, characterized by a pair of values (I^*, q^*) solving system (11). In this case, the disease may spread further and take root in the population, therefore becoming endemic.

Without quarantine and asymptomatic carriers, a disease has its natural basic reproduction number $R_0 = \beta/\rho$. Symptom-based detection and isolation measures lower this to $R_0 = (\beta\gamma)/\rho$. As asymptomatic carriers cannot be detected and put into quarantine, their presence raises the value of R_0 back towards its natural value.

Corollary 1. *For some set of parameters' values, the dynamic system in (4) to (9) converges to the Disease-Free SS when $\alpha = 0$, but can converge to an Endemic SS when $\alpha > 0$.*

Hence, the outbreak of an infectious disease that could be neutralized through symptoms-based detection and isolation measures may spread out of control when some infections are instead asymptomatic, as it presumably happened with Covid-19 in China.¹

3.2. Endemic steady state and asymptomatic carriers

In an Endemic SS, the prevalence of unhealthy agents and the activity level of the healthy ones solve system (11). Define function $f(q)$ and $g(q)$ by the top and bottom lines of the RHS of (11), respectively. As shown in Fig. 3, the first function describes an increasing curve. The second describes a parabola, which is always positive and crosses the origin and point (1,0). If $R_0 \geq 1$, then $f(q)$ is positive at $q = 1$. The two curves intersect, and the intersection is the Endemic SS.

Intersection point A in Fig. 3 pins down the Endemic SS in Fig. 2. Higher transmissibility β shifts curve $f(q)$ upwards. All else equal, the prevalence of unhealthy agents rises to B. However, healthy individuals react by reducing activity, dampening the effect (point C).

Simulating the model suggests that the case in Fig. 3 with a unique intersection point at some $q > .5$ is the common one to arise. Still, the existence of multiple Endemic SSs cannot be ruled out theoretically. The two curves might intersect more than once, both to the left of the parabola's vertex and to its right. Alternatively, the two curves might intersect only once at some $q < .5$. This is problematic, since the location of the intersection point relative to the parabola's vertex can affect the direction of comparative statics.

For example, in Fig. 3, a higher transmissibility rate β would have resulted in a decrease in the prevalence of unhealthy agents, rather than an increase, if the original intersection point had occurred at some $q < .5$. In this case, individuals' social distancing response would have more than offset the increased transmission potential of the disease, leading to a lower prevalence of infections in the SS. This is an interesting feature of the model: a higher R_0 can, under certain conditions, result in a lower prevalence of infection once behavioral responses are taken into account, which is a phenomenon already noted in previous work (e.g., Chen et al. 2012).

My simulations suggest that intersection points to the left of the parabola's vertex represent special cases, in my model. To sharpen the main intuition of the paper, I therefore rule out this possibility and focus only on Endemic SSs where $q^* > .5$. If such an Endemic SS exists, then it is the unique one with this characteristic, as the two curves have opposite slope when $q > .5$. Also, I restrict attention to Endemic SSs such that $q^* > \gamma$, i.e., where the activity level of healthy individuals is higher than that under quarantine, which seems plausible. Overall, I therefore restrict attention to Endemic SS such that $q^* > \max\{\gamma, .5\}$. Although violations of this restriction appear rare in my simulations, it nonetheless limits the generality of the results.

Asymptomatic Carriers. Consider an infectious disease where $(\beta\gamma)/\rho \geq 1$. In this case, an Endemic SS exists, even when all infections are symptomatic.² I am interested in determining the impact of increasing the fraction of asymptomatic infections on the prevalence of unhealthy agents in the Endemic SS.

All else equal, a higher share of asymptomatic infections necessarily translates into a lower share of symptomatic ones, and hence of unhealthy agents. Still, asymptomatic carriers boost the spread of a disease, therefore increasing the number of total infections. The overall impact on the prevalence of unhealthy agents then depends on which of these two effects dominates.

For the same set of parameters as in Fig. 2, Fig. 4 plots the SS prevalence of unhealthy agents for all $\alpha \in [0, 1]$. Relative to the case in which all infections are symptomatic, this more than doubles when 1 out of 4 infections is instead asymptomatic. The prevalence of unhealthy agents is obviously zero when $\alpha = 1$. This highlights an interesting non-monotonic relationship between the fraction of new asymptomatic infections and the prevalence of unhealthy agents.

Suppose we replaced a symptomatic agent with an asymptomatic carrier. For the prevalence of unhealthy agents to rise, adding the asymptomatic carrier must boost disease transmission at least to compensate for the lost symptomatic infection. The higher the share α of new infections that are asymptomatic, the harder it is to produce one that is instead symptomatic.

Next, I establish when this non-monotonicity result arises.

Proposition 2. *The SS prevalence of unhealthy agents is non-monotonic in the fraction of new asymptomatic infections if and only if*

$$\frac{\beta\gamma}{\rho} < 1 + \frac{q_0^* - \gamma}{q_0^*}, \quad (12)$$

where q_0^* is the activity level of healthy agents in the Endemic SS with $\alpha = 0$.

² Otherwise, the epidemic converges to the Disease-Free SS when all infections are symptomatic. In this case, if inequality (10) does not hold at $\alpha = 1$, then the prevalence of unhealthy agents is equal to zero for any fraction of asymptomatic infections. Otherwise, this is either zero or it jumps at some value of $\alpha < 1$.

¹ see Figs. 9 and 10 in Appendix A.

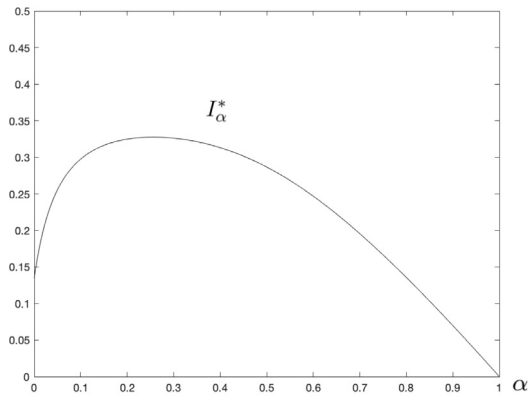


Fig. 4. Non-monotonic relationship between SS prevalence of unhealthy agents and share of asymptomatic infections. For the same set of parameters as in Fig. 2, this graph plots the SS prevalence of unhealthy agents I_α^* as a function of the fraction α of new infections that are asymptomatic. The relationship is non-monotonic. Relative to the case where all infections are symptomatic, the SS prevalence of unhealthy agents rises substantially as the fraction of new asymptomatic infections increases to about 1/4. Then, the SS prevalence of unhealthy agents decreases in α and reaches zero when $\alpha = 1$.

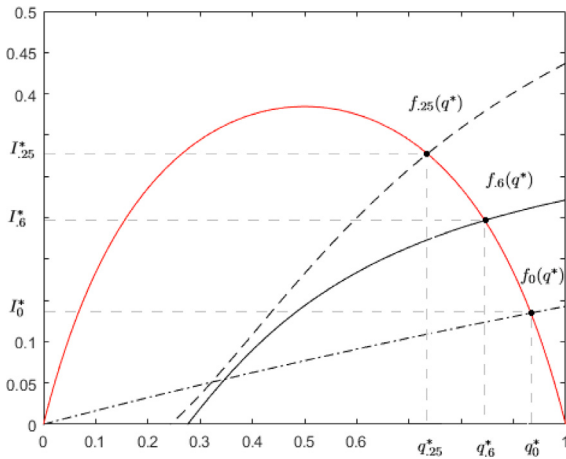


Fig. 5. Non-monotonicity in the endemic SS diagram. Relative to the case $\alpha = 0$, curve $f(q)$ shifts upwards when $\alpha = .25$. The new intersection point lies north of the old one and the prevalence of unhealthy agent increases. Still, a further increase to $\alpha = .6$ yields the opposite: curve $f(q)$ shifts downwards and the new intersection point lies south of the old one.

As Fig. 5 shows, changing α does not affect parabola $g(q)$, but shifts curve $f(q)$. The curve shifts upwards when α increases from 0 to 25% and the prevalence of unhealthy agents rises. A further increase to 60% produces instead the opposite effect.

For the result to hold, curve $f(q)$ must shift upwards for at least some $\alpha \in [0, 1]$. I find that this must hold at $\alpha = 0$. This suggests that adding the first asymptomatic carrier is associated to the highest marginal damage, as when all infections are symptomatic it is easier to generate another unhealthy individual. This analysis leads to condition (12).

To interpret condition (12), suppose infections were only symptomatic. When $(\beta\gamma)/\rho$ is high, an epidemic generates a large prevalence of unhealthy agents and induces healthy individuals to lower their activity level substantially, due to the high environment risk. In this case, replacing a symptomatic agent with an asymptomatic carrier does not boost transmission that much, since the difference in activity level between the two types of infectious agents is relatively small. The opposite holds, instead, when $(\beta\gamma)/\rho$ is small.

This non-monotonicity result is analogous the main result in [Acmoglu et al. \(2023\)](#), which shows that increased testing capacity can initially be successful at reducing infections, but may later increase them, as a result of inducing riskier behavior in susceptible individuals. My result, on the other hand, pertains to the fraction of new infections that are asymptomatic.

4. Mass testing and isolation policies

As seen in the previous section, the presence of asymptomatic infections during an epidemic can be detrimental to public health. Asymptomatic carriers can boost disease transmission and raise the overall incidence of infections that are instead symptomatic, and hence the number of people who are eventually unhealthy. This type of infections amplify the spread of an infectious disease in my model mostly because asymptomatic carriers cannot be identified and isolated.

In this section, I consider *mass testing and isolation* policies: in every period, I assume that health authorities test every healthy agent, to isolate those resulting positive.

Testing. A test is an experiment $\sigma : \{A, S\} \rightarrow \Delta(\{p, n\})$, which maps the status of each healthy agent into a probability distribution over a positive (p) and negative (n) result. I assume tests are *informative*: asymptomatic agents are more likely to test positive than susceptible individuals, which requires the rate of true positives to be higher than that of false positives ($\sigma_{p|A} > \sigma_{p|S}$).³ Notice, however, that this does not imply any clear restriction on the rate of false positives and false negatives, but testing can be arbitrarily accurate.⁴

Covid-19 diagnostic tests are usually described in terms of their *sensitivity* and *specificity* parameters, which are the probabilities of a correct outcome. Although several tests are used in practice at the same time, I assume that every healthy agent is diagnosed using the same test.

Model. At the start of each period $t \geq 1$, I let health authorities test every healthy individual and force positive ones into quarantine, which restricts their contact rate to $\gamma \in (0, 1)$. Negative agents do instead choose their level of social activity.

Before choosing a level of social activity, I let negative agents incorporate their negative test result into their assessment of the likelihood of being asymptomatic, and hence not at risk of infection for that given period. Given their negative test result, I assume that negative agents apply Bayes' rule and update their belief μ_t^e to

$$\mu_t^n = \frac{\mu_t^e \sigma_{n|A}}{\mu_t^e \sigma_{n|A} + (1 - \mu_t^e) \sigma_{n|S}}. \quad (13)$$

Since testing is informative, we have that $\mu_t^n < \mu_t^e$. That is, testing negative can only decrease the assessment of the likelihood of being asymptomatic. When tests produce no false negative, we have that $\mu_t^n = 0$, and an agent testing negative is certain of being susceptible.⁵

³ This implies that susceptible individuals are more likely to test negative than asymptomatic ones, since $\sigma_{n|A} = 1 - \sigma_{p|A}$ and $\sigma_{n|S} = 1 - \sigma_{p|S}$.

⁴ To see this, consider two tests: one assigning a positive result to all asymptomatic agents, but also to 9 out of 10 susceptible ones, and one assigning a positive result to 1 out of 10 asymptomatic agents, but never to susceptible ones. Both tests are informative, but the former produces many false negatives and no false positives, and the latter is the opposite.

⁵ Belief updating plays a limited role in my model and is not a primary driver of the results. Positive agents are placed into quarantine and the way in which they update their beliefs is thus inconsequential. Belief updating matters only for negative agents and I introduce it in my model with testing primarily to be rigorous and ensure consistency with the model without testing. There, agents assess their likelihood of being asymptomatic, before choosing their level of social activity. Upon receiving a negative result, agents must revise this belief somehow. While agents have adaptive expectations and are myopic, I assume they process their test results rationally, through Bayes' theorem.

Given their updated belief, negative agents choose the level of social activity given by

$$q_t = \arg \max_q -\frac{(1-q)^2}{2} - q(1-\mu_t^n) \lambda_t^e (1-\alpha) c. \quad (14)$$

By the assumption of a continuum of agents, $A_t \sigma_{s|A}$ is the share of asymptomatic carriers with test result $s \in \{p, n\}$, and similarly for susceptible individuals. Whether contagious or not, any healthy agent testing positive is placed into quarantine and thus assigned activity level γ . Any healthy agent testing negative engages in some level of social activity q_t . Hence, the risk in the environment is now given by

$$\lambda_t = \frac{\gamma I_t + A_t (\gamma \sigma_{p|A} + q_t \sigma_{n|A})}{\gamma (I_t + S_t \sigma_{p|S} + A_t \sigma_{p|A}) + q_t (S_t \sigma_{n|S} + A_t \sigma_{n|A})}, \quad (15)$$

and negative agents' estimate therefore is $\lambda_t^e = \lambda_{t-1}$, due to adaptive expectations.

4.1. Dynamic system with testing

The model reduces to an updated dynamical system. As initial conditions, I set $q_0 = 1$ and $I_0 \approx 0$. These determine A_0 , S_0 and λ_0 as in the dynamical model in Section 2.3. In period $t = 0$, no testing occurs, to reflect lack of awareness about asymptomatic infections. Hence, we have I_{t+1} , A_{t+1} , S_{t+1} and μ_{t+1}^e as in Eqs. (4) to (7). At the start of each period $t \geq 1$, health authorities test every healthy agent to detect asymptomatic carriers. Test results are revealed and those testing positive are isolated alongside symptomatic individuals. Those testing negative update their belief over the likelihood of being asymptomatic and choose an activity level for that period. Finally, new infections are generated and a fraction of infected individuals recovers.

Accordingly, in every period $t \geq 0$, we have

$$\mu_{t+1}^e = \frac{\mu_{t+1}^e \sigma_{n|A}}{\mu_{t+1}^e \sigma_{n|A} + (1 - \mu_{t+1}^e) \sigma_{n|S}} \quad (16)$$

$$q_{t+1} = \max\{0, 1 - (1 - \mu_{t+1}^e) \lambda_t \beta (1 - \alpha) c\}, \quad (17)$$

$$\lambda_{t+1} = \left(\frac{\gamma I_{t+1} + A_{t+1} (\gamma \sigma_{p|A} + q_{t+1} \sigma_{n|A})}{\gamma (I_{t+1} + S_{t+1} \sigma_{p|S} + A_{t+1} \sigma_{p|A}) + q_{t+1} (S_{t+1} \sigma_{n|S} + A_{t+1} \sigma_{n|A})} \right). \quad (18)$$

Furthermore, in every period $t \geq 1$, we have

$$I_{t+1} = (1 - \rho) I_t + (1 - \alpha) S_t (\sigma_{p|S} \gamma + \sigma_{n|S} q_t) \lambda_t \beta, \quad (19)$$

$$A_{t+1} = (1 - \rho) A_t + \alpha S_t (\sigma_{p|S} \gamma + \sigma_{n|S} q_t) \lambda_t \beta, \quad (20)$$

and S_{t+1} and μ_{t+1}^e as in expression (6) and (7), respectively. Note that the model reduces to that in Chen et al. (2011) when there are no asymptomatic carriers ($\alpha = 0$) and testing produces no false positives ($\sigma_{p|S} = 0$).

5. Analysis of the model with mass testing and isolation policies

Through mass testing, health authorities aim to screen asymptomatic carriers and isolate them. Still, tests might not be perfectly accurate, but produce false negative and false positive results.

First, I determine that mass testing and isolation policies generically improve public health, as they always lead to a reduction in the SS prevalence of unhealthy agents, given the restrictions discussed in Section 3.2. Then, I consider the implications of false negative and false positive results: some asymptomatic carrier may be left exposed to the

population while some susceptible agent may be mistakenly isolated. I find that false negatives are detrimental as they always raise the SS prevalence of unhealthy agents. False positives, instead, can lower disease prevalence and even improve welfare in the population.

Endemic SS. Given some arbitrary test σ , an Endemic SS exists if and only if

$$R_0 = \frac{\alpha \beta (\sigma_{n|A} + \sigma_{p|A} \gamma) + (1 - \alpha) \beta \gamma}{\rho} > 1. \quad (21)$$

In this case, there exists an Endemic SS where the prevalence of unhealthy agents (I_σ^*) and the activity level of negative individuals (q_σ^*) solve the following system

$$\begin{cases} I = \frac{(1 - \alpha) [\beta (\gamma + \alpha \sigma_{n|A} (q - \gamma)) - \rho] (\gamma + \sigma_{n|S} (q - \gamma))}{\beta (\gamma + \sigma_{n|S} (q - \gamma)) (\gamma + \alpha \sigma_{n|A} (q - \gamma)) - \rho (q - \gamma) (\sigma_{n|S} - \alpha \sigma_{n|A})}; \\ I = \frac{(1 - \alpha) (1 - q) [\sigma_{n|S} (\sigma_{n|S} q + \sigma_{p|S} \gamma)]}{(1 - q) [(\sigma_{n|S} - \alpha \sigma_{n|A}) (q \sigma_{n|S} + \gamma \sigma_{p|S})] + (1 - \alpha) \sigma_{n|S} c \rho}. \end{cases} \quad (22)$$

The LHS of condition (21) is lower than the LHS of condition (10), since $\sigma_{n|A} + \sigma_{p|A} \gamma < 1$. Thus, mass testing and isolation policies reduce the basic reproduction number of a disease, the more so the more accurate are tests at screening asymptomatic carriers. When tests produce no false negatives (i.e., when $\sigma_{p|A} = 1$), we have that $R_0 = (\beta \gamma) / \rho$ and the presence of asymptomatic carriers does not prevent the early containment of an outbreak.

If $R_0 > 1$, then there exists an Endemic SS. I apply the same restrictions as in the model without testing. Thus, I consider only sets of parameters such that, in the model without testing, an Endemic SS where $q^* \geq \max\{\gamma, .5\}$ exists. As previously observed, if such an Endemic SS exists, then it is unique, which allows to obtain sharper results. Proposition 3 summarizes the impact of mass testing and isolation policies.

Proposition 3. *Mass testing and isolation policies reduce the SS prevalence of unhealthy agents.*

Define function $f_\sigma(q)$ and $g_\sigma(q)$ by the first and second lines of the RHS of Eq. (22). For every $q > \gamma$, these curves lie below $f(q)$ and $g(q)$, and therefore the new intersection point lies to the south of the old one, which implies a reduction in the prevalence of unhealthy agents.

Fig. 6 illustrates the results for *partially informative* tests with $\sigma_{p|A} = .8$ and $\sigma_{p|S} = .2$. In any period, 80% of asymptomatic carriers and 20% of susceptible agents are isolated. This reduces the risk in the environment and the spread of the disease (A to B). Still, lower risk in the environment induces negative agents to increase their activity level. Note that the system reaches point C, rather than C'. All else equal, testing decreases the level of social activity of those individuals who are not in isolation. Indeed, as already pointed out below Eq. (13), testing negative implies a higher chance of being susceptible than simply being healthy, and therefore an higher likelihood of being at risk of infection.

Testing and Increment in Infections. By curbing the spread of a disease, mass testing and isolation measures improve public health. Still, they may raise the risk in the environment. To see this, suppose that $\sigma_{p|S} > 0$ and $\alpha \approx 0$. Then, the numerator of expression (15) is the same as that of (3), but the denominator is much smaller. Since there are no asymptomatic carriers, testing results only in the isolation of some susceptible agents, which increases the probability of meeting one who is instead infected and therefore the risk in the environment.

However, as shown in Eq. (19), the increment in infections is determined by the product of the environment risk and the average activity level of susceptible individuals. False positives decrease susceptible individuals' average activity level to $\sigma_{n|S} q_t + \sigma_{p|S} \gamma$, and the overall effect on the increment in infections is negative. Without asymptomatic carriers, mass testing and isolation policies are in fact equivalent to a *lockdown*, reducing infections not by restraining infected agents (who

Allowing for simpler heuristics (e.g., fully trusting the test result) would not significantly alter the results. A potentially interesting extension would be to incorporate belief updating biases, such as base-rate neglect (Tversky and Kahneman, 1974), which is particularly relevant in medical testing.

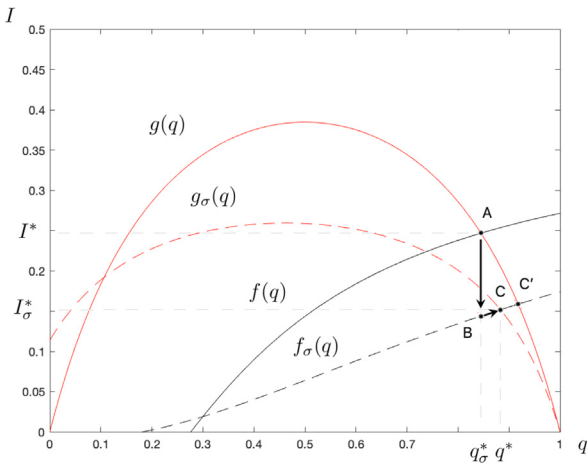


Fig. 6. Impact of mass testing and isolation policies on endemic SS. For the same set of parameters of Fig. 4, and given partially informative tests with $\sigma_{p|A} = .8$ and $\sigma_{p|S} = .2$, this diagram compares the Endemic SS without and with mass testing and isolation policies. The latter is characterized by the intersection point between curve $f_\sigma(q)$ and $g_\sigma(q)$, as defined by the first and second lines of the RHS of (22). The two curves lies below $f(q)$ and $g(q)$ for every $q > \gamma$ and so the Endemic SS with mass testing and isolation policies (point C) always features a lower prevalence of unhealthy agents than the Endemic SS without (point A).

are indeed already into quarantine), but by limiting the exposure of those who are instead at risk of infection.

False Negative and False Positive Results. The environment risk is always minimized with *fully informative* tests, which correctly diagnose the status of every healthy individual (i.e., tests with $\sigma_{p|A} = \sigma_{n|S} = 1$). These tests achieve *perfect sorting*: every infectious agent is placed into quarantine, whereas all susceptible individuals are free to choose their own activity level.

Tests producing less false negatives are more accurate at detecting asymptomatic carriers and isolating them. This decreases the risk in the environment and the increment in infections. Thus, lower rates of false negatives always reduce the prevalence of unhealthy agents.⁶

Proposition 4. *Lower rates of false negatives reduce the SS prevalence of unhealthy agents.*

This is not necessarily true for tests producing less false positives. By inducing the isolation of a lower share of susceptible individuals, they increase their average activity level, which can lead to an higher increment in infections and to an increase in the prevalence of unhealthy agents.

Proposition 5. *Lower rates of false positives can increase the SS prevalence of unhealthy agents.*

Fig. 7 shows how the intersection point in Fig. 6 moves north when tests do not produce any false positive, leading to a higher prevalence of unhealthy agents (A to B).⁷ The lockdown effect thus operates also in the presence of asymptomatic carriers. By mechanically restraining the activity level of those individuals at risk of infection, false positive

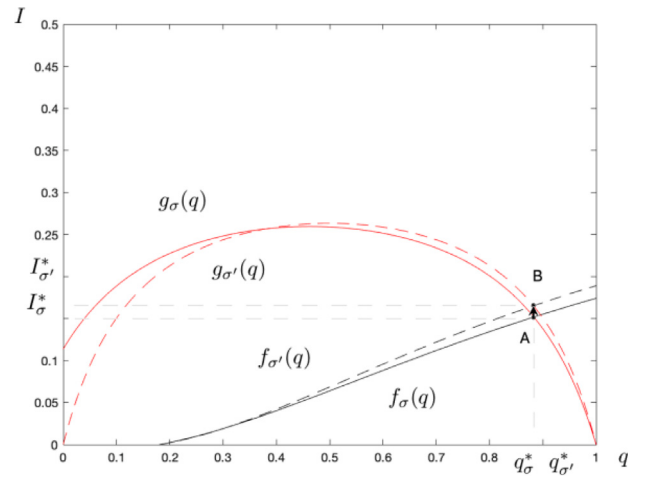


Fig. 7. Impact of lower rate of false positives on endemic SS. The diagram shows that the Endemic SS that arises when testing does not produce any false positives (point B) is associated to a higher prevalence of unhealthy agents than the Endemic SS that arises when testing has a rate of false positives equal to 20%.

results reduce disease transmission and lead to a lower SS prevalence of unhealthy agents.

False Positives and Welfare. Designing tests that produce false positives can thus improve public health. Inducing the isolation of some susceptible agents reduces their risk of infection and can ultimately lower the prevalence of unhealthy agents. Yet, these individuals would prefer to engage in their optimal level of activity, rather than being put into quarantine. Hence, I ask whether this extreme measure to limit contagion can improve welfare. This might be the case if the benefits in terms of public health outweigh the costs imposed on false positive agents.

Consider some arbitrary test σ . The population's welfare in the Endemic SS is given by

$$W_\sigma^* = - \left[A_\sigma^* \left(\sigma_{n|A} \frac{(1 - q_\sigma^*)^2}{2} + \sigma_{p|A} \frac{(1 - \gamma)^2}{2} \right) + S_\sigma^* \left(\sigma_{n|S} \frac{(1 - q_\sigma^*)^2}{2} + \sigma_{p|S} \frac{(1 - \gamma)^2}{2} \right) + c I_\sigma^* \right]. \quad (23)$$

Let testing be fully informative. Raising the rate of false positives induces the isolation of an ever larger share of susceptible agents, thus reducing the expected utility within this group. However, this policy can also lower disease prevalence, and hence increase the size of this compartment.

The transmission rate (β) and severity (c) of a disease are of particular importance for the result. The higher the former, the greater the impact on public health of isolating an additional susceptible individual. The higher the latter, the greater the impact on welfare. Fig. 12 in Appendix A shows that welfare decreases monotonically as the rate of false positives increases when $(\beta, c) = (0.7, 2)$, while the opposite holds when $(\beta, c) = (0.8, 3)$. Fig. 8 suggests that there exist threshold values of (β, c) beyond which the effect of false positives on welfare changes sign, shifting from strictly negative to strictly positive.

The threshold displayed in Fig. 8 is a curve. For $(\beta, c) = (0.805, 2)$ and $(0.746, 3)$, welfare decreases as the rate of false positives rises. Still, the opposite holds when $(\beta, c) = (0.7755, 2.5)$, which lies on the segment connecting these two points. Consequently, the region in the $(\beta - c)$ space where higher false positive rates lead to decreased welfare is convex, which highlights the role of the interaction between (β) and (c) in shaping the observed outcomes.

⁶ Fig. 11 in Appendix A shows that the intersection point in Fig. 6 moves to the south-east when tests do not generate any false negative.

⁷ Two competing effects imply that the activity level is almost unchanged, in this example. Lower risk in the environment induces negative agents to raise their activity level. However, lower rates of false positives increase the likelihood of being at risk of infection, conditional on a negative result, which has the opposite effect.

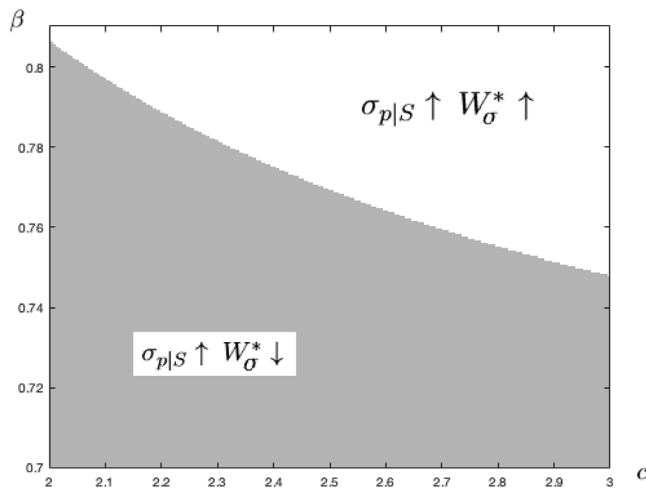


Fig. 8. Welfare effect of increasing the rate of false positives. For the same set of parameters of Fig. 4, and given $\sigma_{p|A} = 1$, the diagram shows the effect of raising the rate of false positives on welfare, for every $(\beta, c) \in [0.7, .8] \times [2, 3]$. The graph indicates the existence of threshold values for (β, c) at which the monotonic relationship between the rate of false positives and welfare reverses direction.

It is well known that social distancing during an epidemic generates positive externalities. By reducing their level of social activity, individuals not only lower their own risk of infection but also reduce the likelihood of transmitting the disease to others. This externality implies that, in decentralized settings, there is typically underinvestment in social distancing relative to what a benevolent social planner would choose. This is a key reason why false positives can improve welfare: by mechanically reducing the activity level of susceptible individuals, false positive test results narrow the gap between the actual level of social distancing and the socially optimal one.

The strength of this positive externality, and so the potential welfare gains from false positive test results, plausibly increases with both the transmission rate and the severity of the disease. All else equal, the higher the former, the more an increase in social activity levels raises infection rates. The higher the latter, the more “costly” a symptomatic infection becomes. By lowering social activity in situations where the negative externality from interaction is greatest, false positive results contribute more substantially to mitigating the spread and impact of the disease.

6. Conclusion

In this paper, I built on the epidemic model in Chen et al. (2011) to study the role of asymptomatic carriers in infectious disease transmission. Then, I incorporated diagnostic testing into the model to evaluate the effectiveness of mass testing and isolation policies.

First, I considered the case in which health authorities lack testing to identify asymptomatic carriers, and can only rely on symptom-based strategies to limit transmission. I showed how, in this case, asymptomatic carriers can be crucial for an epidemic to take root in the population and an infectious disease to become endemic. Then, I considered their impact on the long-run evolution of an epidemic. I characterized the Steady State of my model, which admits a simple graphical representation, and showed how a small fraction of asymptomatic infections can significantly raise the long-run prevalence of unhealthy agents.

Given the shortcomings of symptom-based strategies in the presence of asymptomatic carriers, I introduced mass testing and isolation policies. I assumed health authorities test all healthy agents in every period,

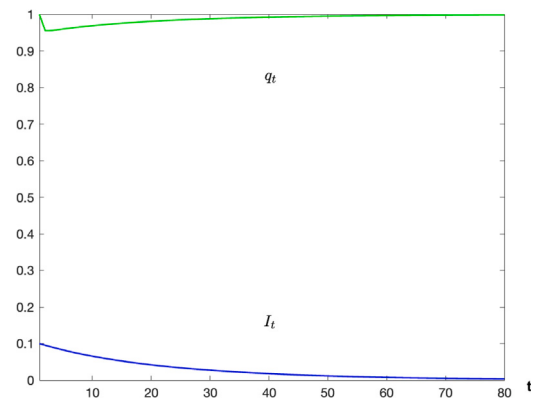


Fig. 9. Convergence to disease-free SS. When $\alpha = 0$, we have $R_0 = .9$. As shown in the above simulation, the system converges to the Disease-Free SS.

and isolate those that test positive. I let testing produce false negative and false positive results to check their implications.

First, I found that mass testing and isolation policies improve public health. Then, I considered the impact of false negative and false positive results. I showed that false negative results are always detrimental during an epidemic, as they raise the SS prevalence of unhealthy agents, but false positive results can actually improve public health. By forcing some susceptible agents to isolate, false positive results decrease the average activity level of those individuals who are at risk of infection, thus limiting transmission and the long-run prevalence of unhealthy agents. I illustrate how false positive results can improve the population’s welfare when the infectious disease has high severity and transmissibility.

A limitation of my results is that they hold for parameter values such that $q^* > \max\{\gamma, 5\}$. Thus, for epidemics such that healthy individuals’ SS level of social activity does not fall below 50% of normal levels, or below the activity level of symptomatic agents in quarantine if higher. As discussed in Section 3.2, this restriction is plausible and rarely violated in practice. Nevertheless, it limits the generality of my findings.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Figures

The following graphs assume that $(\rho, \beta, \gamma, \alpha, c) = (.2, .7, .3, .6, 2)$ and $I_0 = .01$, unless specified otherwise.

Figs. 9 and 10 provide an example for . Suppose that $\rho = .25$. If $\alpha = 0$, then $R_0 = .84$ and the system converges to the Disease-Free SS. This is shown in Fig. 9, where I let $I_0 = .1$ to better illustrate the result. As the share of recovered individuals in each period exceeds that of new infections, the prevalence of infected agents I_t decreases over time until it reaches zero. Healthy individuals’ activity levels q_t initially decrease slightly, to then converge and return to $q = 1$. Symptoms-based detection and isolation strategies effectively contain the outbreak, preventing epidemic escalation and allowing individuals to resume full activity.

If $\alpha = .6$, then $R_0 \approx 2$ and the system converges to an Endemic SS (Fig. 10). When there are asymptomatic carriers, symptoms-based detection and isolation strategies are not effective at containing an outbreak. The disease becomes endemic and people engage in social distancing.

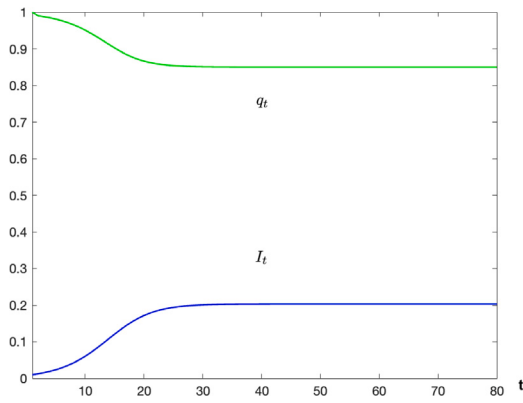


Fig. 10. Convergence to endemic SS. When $\alpha = .6$, we have $R_0 = 2.16$, instead. As shown in the above simulation, the system converges to an Endemic SS..

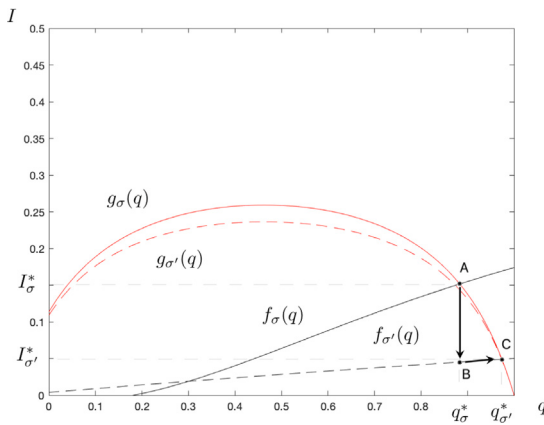


Fig. 11. Impact of lower rate of false negatives on endemic SS. The diagram shows that the Endemic SS to which the epidemic converges to when testing does not produce any false negatives (point B) is associated with a lower prevalence of unhealthy agents than the Endemic SS to which the epidemic converges when testing has a 20% rate of false negatives.

Fig. 11 show the effect on the Endemic SS of a reduction in the rate of false negatives, from $\sigma_{p|A} = .8$ to $\sigma'_{p|A} = 1$. By inducing the isolation of a larger share of asymptomatic carriers, less false negative results lower the environment risk and, in turn, the prevalence of unhealthy agents (B). However, individuals respond by raising their activity level, which dampens the effect.

Finally, given $\sigma_{p|A} = 1$, Fig. 12 plots the SS prevalence of unhealthy agents and total welfare in the population for every $\sigma_{p|S} \in [0, 1]$. As shown in the left panel, increasing the rate of false positives reduces welfare when $\beta = .7$. However, as the right panel shows, the opposite holds when $\beta = .8$.

Appendix B. Proofs

Model without testing

Proposition 1. (i) Assume that an Endemic SS exists. Then, there exists a vector of values $(I, A, S, \mu, q, \lambda)$ such that $I > 0$ and $(I_t, A_t, S_t, \mu_t, q_t, \lambda_t) = (I, A, S, \mu, q, \lambda)$ in some period t implies that $(I_s, A_s, S_s, \mu_s, q_s, \lambda_s) = (I, A, S, \mu, q, \lambda)$ in all periods $s > t$. This must satisfy the following equations simultaneously

$$\rho I = (1 - \alpha) S q \lambda \beta, \quad (24)$$

$$\rho A = \alpha S q \lambda \beta, \quad (25)$$

$$S = 1 - I - A, \quad (26)$$

$$1 - \mu = 1 - \frac{A}{1 - I}, \quad (27)$$

$$q = 1 - c(1 - \alpha)(1 - \mu)\lambda\beta, \quad (28)$$

$$\lambda = \frac{\gamma I + q A}{\gamma I + q(1 - I)}. \quad (29)$$

First, notice that:

- (a) If $\alpha = 1$, then $I = 0$ and $q = 1$, which follows directly from (24) and (28).
- (b) We must have $I \neq 1$. Otherwise, $S = 0$ and Eq. (24) never holds.
- (c) We must have $q \neq 0$. Otherwise, $I = A = 0$ by Eqs. (24) and (25), which implies that $\lambda = 0$ and therefore, by Eq. (28), that we must have $q = 1$.

By point (a), I consider only $\alpha \in [0, 1)$. Next, I derive expression $g(q)$ and $f(q)$ in system (11). To obtain the former, I apply the following steps:

$$1 - \mu = \frac{1 - I - A}{1 - I} = \frac{S}{1 - I} \rightarrow S = (1 - \mu)(1 - I). \quad (30)$$

Substituting in (24) and isolating $(1 - \mu)\lambda\beta$ gives

$$(1 - \mu)\lambda\beta = \frac{\rho I}{(1 - \alpha)(1 - I)} \frac{1}{q}. \quad (31)$$

Notice that the denominator on the RHS is never equal to zero, by points (a) to (c). Substituting in expression (28) yields

$$q = 1 - c(1 - \alpha) \frac{\rho I}{(1 - \alpha)(1 - I)} \frac{1}{q}. \quad (32)$$

Multiplying both sides by q and rearranging terms, I can write

$$q(1 - q) = \frac{c \rho I}{(1 - I)}. \quad (33)$$

Finally, by inverting the previous equation, I obtain the expression for $g(q)$.

Now, I derive $f(q)$. Dividing Eq. (25) by (24), I can write $A = \frac{\alpha}{1 - \alpha} I$. Then, substituting in (29) gives

$$\lambda = \frac{(1 - \alpha) I \gamma + \alpha I q}{(1 - \alpha)(\gamma I + q(1 - I))} \quad (34)$$

and $S = \frac{(1 - \alpha) - I}{1 - \alpha}$. Substituting these two expressions into (24) I can write

$$(1 - \alpha)(\gamma I + q(1 - I))\rho I = ((1 - \alpha) - I)q\beta((1 - \alpha) I \gamma + \alpha I q), \quad (35)$$

which can be written as

$$I^2 \left[q(\alpha\beta q + (1 - \alpha)\beta\gamma - (1 - \alpha)\rho) + (1 - \alpha)\rho\gamma \right] = I \left[(1 - \alpha)q(\alpha\beta q + (1 - \alpha)\beta\gamma - \rho) \right]. \quad (36)$$

Dividing both sides by $I > 0$ and rearranging terms yields the desired expression.

(ii) To see that condition (10) is necessary, substitute Eq. (35) into (24). Then, multiplying both sides by the denominator gives

$$(I\gamma + (1 - I)q)\rho I = Sq\beta((1 - \alpha)\gamma + \alpha q)I \quad (37)$$

Subtracting $Sq\rho I$ on both sides, I can write

$$\rho I(I\gamma + (1 - I)q - Sq) = Sq\beta((1 - \alpha)\gamma + \alpha q - \rho)I. \quad (38)$$

The LHS is always positive, since $1 - I \geq S$. Hence, the RHS must also be positive, which implies condition (10). In this case, curve $f(q)$ is positive at $q = 1$ and the two curves intersect at some $I^* > 0$. $\square$

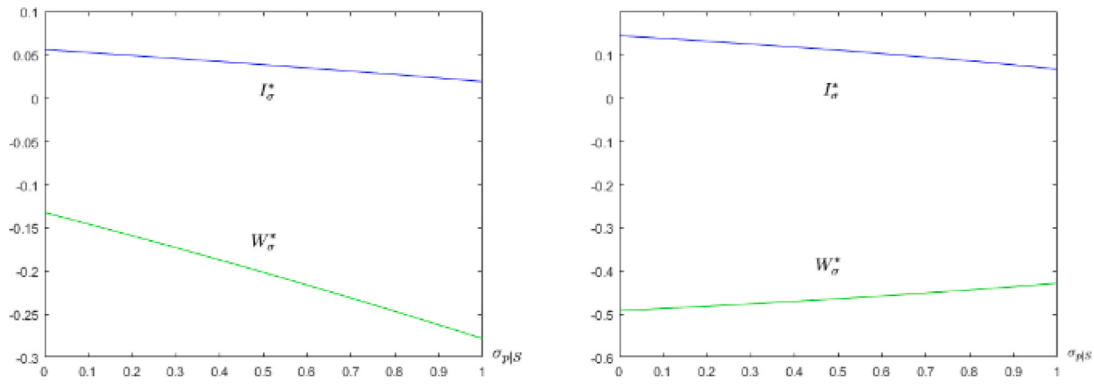


Fig. 12. Impact of higher rate of false positives on welfare. Given $\sigma_{p|A} = 1$, the graphs plot the SS prevalence of unhealthy agents and total welfare in the population for every $\sigma_{p|S} \in [0, 1]$. As shown in the left panel, increasing the rate of false positives reduces welfare when $\beta = .7$. However, as shown in the right panel, the opposite holds when $\beta = .8$.

Corollary 1. This immediately follows, since R_0 increases in parameter α . $\square$

Proposition 2. To prove the result I must show that, relative to the case in which $\alpha = 0$, there must exist some $\alpha \in [0, 1]$ at which the prevalence is higher. Changing α does not affect parabola $g(q)$, but moves curve $f(q)$. This must shift upwards at some $\alpha \in [0, 1]$, which requires the numerator of its derivative to be positive. Thus,

$$\begin{aligned} & (\beta q^2 - 2\alpha\beta q^2 - 2\beta q\gamma + 2\alpha\beta q\gamma + q\rho) \times \\ & \times (\alpha\beta q^2 + \beta q\gamma - \alpha\beta q\gamma - q\rho + \alpha q\rho + \rho\gamma - \alpha\rho\gamma) - \\ & (\beta q^2 - \beta q\gamma + q\rho - \rho\gamma) \times \\ & \times (\alpha\beta q^2 - \alpha^2\beta q^2 + \beta q\gamma - 2\alpha\beta q\gamma + \alpha^2\beta q\gamma - q\rho + \alpha q\rho) > 0. \end{aligned} \quad (39)$$

This can be simplified and written as

$$\begin{aligned} & (1 - \alpha)\beta q\rho\gamma - 2\alpha(1 - \alpha)\beta q\rho\gamma + (1 - \alpha)\beta q\rho\gamma + 2\alpha(1 - \alpha)\beta q^2\rho - \\ & - 2\alpha(1 - \alpha)\beta^2 q^2\gamma - (1 - \alpha)^2\beta^2 q\gamma^2 - (1 - 2\alpha)\beta\rho\gamma^2 - \alpha^2\beta^2 q^3 > 0. \end{aligned} \quad (40)$$

I notice that the derivative with respect to α of the LHS of this latter inequality is negative. Indeed, its numerator can be written as

$$-2(q - \gamma) [2\alpha\beta q\rho + \beta\rho\gamma + \beta q(\beta\gamma - \rho) + \alpha\beta^2 q(q - \gamma)] < 0. \quad (41)$$

Hence, if the inequality in (40) holds for some $\alpha > 0$, then it also holds at $\alpha = 0$. Therefore, it is necessary that (40) holds for $\alpha = 0$. Substituting, I obtain

$$\begin{aligned} & \beta q_0^* \rho\gamma + \beta q_0^* \rho\gamma - \beta^2 q_0^{*2} \gamma^2 - \beta\rho\gamma^2 > 0 \\ & q_0^* \rho + q_0^* \rho - \beta q_0^* \gamma - \rho\gamma > 0 \end{aligned} \quad (42)$$

$$\begin{aligned} & \rho(q_0^* - \gamma) - q_0^* (\beta\gamma - \rho) > 0 \\ & \beta\gamma - \rho < \frac{\rho(q_0^* - \gamma)}{q_0^*} \rightarrow \frac{\beta\gamma}{\rho} < 1 + \frac{q_0^* - \gamma}{q_0^*}. \end{aligned} \quad (43)$$

If condition (43) holds, then $f(q)$ shifts upward when α increases from $\alpha = 0$ to some $\alpha > 0$. Thus, curve I_α^* is non-monotonic in parameter α . $\square$

B.1. Model with testing

The derivation of system (22) and inequality (21), which describe the Endemic SS in the model with testing and the condition for its existence, is very similar to that for the model without testing. In an Endemic SS, it must be that

$$\rho I = (1 - \alpha) S(\sigma_{p|S} \gamma + \sigma_{n|S} q) \lambda \beta, \quad (44)$$

$$\rho A = \alpha S(\sigma_{p|S} \gamma + \sigma_{n|S} q) \lambda \beta, \quad (45)$$

$$S = 1 - I - A, \quad (46)$$

$$\mu_e = \frac{A}{1 - I}, \quad (47)$$

$$\mu_n = \frac{\mu_e \sigma_{n|A}}{\mu_e \sigma_{n|A} + (1 - \mu_e) \sigma_{n|S}} \quad (48)$$

$$q = 1 - c(1 - \mu_n)(1 - \alpha) \lambda \beta, \quad (49)$$

$$\lambda = \left(\frac{\beta(\gamma I + A(\gamma \sigma_{p|A} + q \sigma_{n|A}))}{\gamma(I + S \sigma_{p|S} + A \sigma_{p|A}) + q(S \sigma_{n|S} + A \sigma_{n|A})} \right). \quad (50)$$

We have

$$1 - \mu_e = \frac{1 - I - A}{1 - I} \rightarrow 1 - \mu_n = \frac{\frac{1 - I - A}{1 - I} \sigma_{n|S}}{\frac{1 - I - A}{1 - I} \sigma_{n|S} + \frac{A}{1 - I} \sigma_{n|A}}.$$

Since $A = \frac{\alpha}{1 - \alpha} I$, we have $1 - I - A = \frac{(1 - \alpha) - I}{1 - \alpha}$. Substituting in (48) gives

$$1 - \mu_n = \frac{((1 - \alpha) - I) \sigma_{n|S}}{((1 - \alpha) - I) \sigma_{n|S} + \alpha \sigma_{n|A}}. \quad (51)$$

Since $S = 1 - I - A = ((1 - \alpha) - I)/(1 - \alpha)$, substituting in expression (44) gives

$$\rho I = ((1 - \alpha) - I) [\sigma_{p|S} \gamma + \sigma_{n|S} q] \lambda \beta, \quad (52)$$

by which

$$\lambda \beta = \frac{\rho I}{((1 - \alpha) - I)(\sigma_{p|S} \gamma + \sigma_{n|S} q)}. \quad (53)$$

Substituting (51) and (53) into expression (49) yields

$$q = 1 - \frac{((1 - \alpha) - I) \sigma_{n|S} (1 - \alpha) c \rho I}{((1 - \alpha) - I)(\sigma_{p|S} \gamma + \sigma_{n|S} q)[((1 - \alpha) - I) \sigma_{n|S} + \alpha I \sigma_{n|A}]}. \quad (54)$$

Isolating I on the LHS and collecting terms gives the expression for $g_\sigma(q)$.

Next, I show how to derive $f_\sigma(q)$. Substituting $S = ((1 - \alpha) - I)/(1 - \alpha)$ and $A = (\alpha/(1 - \alpha))I$, the risk in the environment λ can be written as

$$\lambda(I) = \frac{((1 - \alpha)I\gamma + \alpha I(\sigma_{p|A}\gamma + \sigma_{n|A}q))}{\gamma((1 - \alpha)I + \alpha I\sigma_{p|A}) + ((1 - \alpha) - I)\sigma_{p|S} + q(\alpha I\sigma_{n|A} + ((1 - \alpha) - I)\sigma_{n|S})}.$$

Substituting $\lambda(I)$ into expression (52), multiplying both sides by the denominator of the RHS and collecting I on both sides gives

$$\begin{aligned} I^2 [\beta(\gamma + \sigma_{n|S}(q - \gamma))(\gamma + \alpha\sigma_{n|A}(q - \gamma)) - \rho(q - \gamma)(\sigma_{n|S} - \alpha\sigma_{n|A})] = \\ = I [(1 - \alpha) [\beta(\gamma + \alpha\sigma_{n|A}(q - \gamma)) - \rho] (\gamma + \sigma_{n|S}(q - \gamma))]. \end{aligned} \quad (55)$$

Dividing both sides by $I > 0$ and rearranging terms yields $f_\sigma(q)$.

To see that condition (21) is necessary for the Endemic SS to exist, substitute $\lambda(I)$ into expression (44) and multiply both sides by the denominator of the RHS. Rearranging terms on the LHS, we can write

$$\rho I \left[((1-\alpha) - I)(\sigma_{p|S}\gamma + \sigma_{n|S}q) + \alpha I(\sigma_{p|A}\gamma + \sigma_{n|A}q) + (1-\alpha)I\gamma \right] = (1-\alpha)S(\sigma_{p|S}\gamma + \sigma_{n|S}q) \left[(1-\alpha)\beta\gamma + \alpha\beta(\sigma_{p|S}\gamma + \sigma_{n|S}q) \right] I \quad (56)$$

Then, subtracting $(1-\alpha)S(\sigma_{p|S}\gamma + \sigma_{n|S}q)I\rho$ on both sides, we can write

$$\rho I \left[((1-\alpha) - I)(\sigma_{p|S}\gamma + \sigma_{n|S}q) + \alpha I(\sigma_{p|A}\gamma + \sigma_{n|A}q) + (1-\alpha)I\gamma \right] - (1-\alpha)S(\sigma_{p|S}\gamma + \sigma_{n|S}q)I\rho = (1-\alpha)S(\sigma_{p|S}\gamma + \sigma_{n|S}q) \left[(1-\alpha)\beta\gamma + \alpha\beta(\sigma_{p|S}\gamma + \sigma_{n|S}q) - \rho \right] I \quad (57)$$

Substituting $S = ((1-\alpha) - I)/(1-\alpha)$ on the LHS, the equality can be written as

$$\rho I \left[\alpha I(\sigma_{p|A}\gamma + \sigma_{n|A}q) + (1-\alpha)I\gamma \right] = (1-\alpha)S(\sigma_{p|S}\gamma + \sigma_{n|S}q) \left[(1-\alpha)\beta\gamma + \alpha\beta(\sigma_{p|S}\gamma + \sigma_{n|S}q) - \rho \right] I. \quad (58)$$

The LHS is always positive. The RHS is positive if and only if the term inside the square brackets is positive for some $q \in [0, 1]$. Since this increases in parameter q , we can let $q = 1$ and we obtain the condition in (21). $\square$

Proposition 3. If $g_\sigma(q) \leq g(q)$ and $f_\sigma(q) \leq f(q)$ for every $q \geq \gamma$ and the intersection point between $f(q)$ and $g(q)$ lies weakly to the right of $q = .5$, then the intersection point between the two curves with testing and isolation policies always lies to the south of the intersection point between the two curves with no testing.

First, I show that: (i) if $q \geq \gamma$, then we have $g_\sigma(q) \leq g(q)$. This holds iff

$$(1-\alpha)(1-q) \left[\sigma_{n|S}(q\sigma_{n|S} + \gamma\sigma_{p|S}) \right] (q(1-q) + c\rho) \leq q(1-q) \left[(1-q) \left[(\sigma_{n|S} - \alpha\sigma_{n|A})(q\sigma_{n|S} + \gamma\sigma_{p|S}) \right] + (1-\alpha)\sigma_{n|S}c\rho \right]. \quad (59)$$

where I multiplied both sides by the product of the denominators. Simplifying term $(1-q)$, taking everything to the RHS and collecting terms gives

$$q(1-q) \left[q\sigma_{n|S} + \gamma\sigma_{p|S} \right] \left[\sigma_{n|S} - \alpha\sigma_{n|A} - (1-\alpha)\sigma_{n|S} \right] + (1-\alpha)\sigma_{n|S}\sigma_{p|S}(q-\gamma)c\rho \geq 0. \quad (60)$$

The first line is always positive, since $\sigma_{n|S} > \sigma_{n|A}$ by the assumption of informative tests. The second is non-negative whenever $q \geq \gamma$, which proves the result.

Next, I show that (ii) $f_\sigma(q) \leq f(q)$ for all $q \geq \gamma$. Hence, that the following inequality always holds

$$\frac{(1-\alpha)q(\alpha\beta q + (1-\alpha)\beta\gamma - \rho)}{q(\alpha\beta q + (1-\alpha)\beta\gamma - (1-\alpha)\rho(q-\gamma))} - \frac{(1-\alpha) \left[\beta(\gamma + \alpha\sigma_{n|A}(q-\gamma)) - \rho \right] (\gamma + \sigma_{n|S}(q-\gamma))}{\beta(\gamma + \sigma_{n|S}(q-\gamma)) (\gamma + \alpha\sigma_{n|A}(q-\gamma)) - \rho(q-\gamma)(\sigma_{n|S} - \alpha\sigma_{n|A})} \geq 0. \quad (61)$$

Bringing the two terms to a common denominator, the inequality can be expressed as $\frac{N}{D} \geq 0$, where the denominator D is given by

$$D = \beta(\gamma + \sigma_{n|S}(q-\gamma)) (\gamma + \alpha\sigma_{n|A}(q-\gamma)) [q(\alpha\beta q + (1-\alpha)\beta\gamma - \rho(1-\alpha)(q-\gamma)) - \rho(q-\gamma)(\sigma_{n|S} - \alpha\sigma_{n|A})] [q(\alpha\beta q + (1-\alpha)\beta\gamma - \rho(1-\alpha)(q-\gamma))];$$

and the numerator N by

$$N = q(\alpha\beta q + (1-\alpha)\beta\gamma - \rho) \left[\beta(\gamma + \sigma_{n|S}(q-\gamma))(\gamma + \alpha\sigma_{n|A}(q-\gamma)) - \rho(q-\gamma)(\sigma_{n|S} - \alpha\sigma_{n|A}) \right] - \left[\beta(\gamma + \alpha\sigma_{n|A}(q-\gamma)) - \rho \right] (\gamma + \sigma_{n|S}(q-\gamma)) [q(\alpha\beta q + (1-\alpha)\beta\gamma - (1-\alpha)\rho(q-\gamma))].$$

Next, I show that both D and N are always positive when $q > \gamma$ and $\frac{\beta\gamma}{\rho} > 1$, which implies that the inequality in (61) always holds. I start from the denominator D . Since $\sigma_{n|S} > \sigma_{n|A}$ by the assumption of informative experiments, to show that this is positive, one must show that the following two inequalities hold.

$$\beta(\gamma + \sigma_{n|S}(q-\gamma)) (\gamma + \alpha\sigma_{n|A}(q-\gamma)) - \rho(q-\gamma)(\sigma_{n|S} - \alpha\sigma_{n|A}) > 0$$

$$q(\alpha\beta q + (1-\alpha)\beta\gamma - \rho(1-\alpha)(q-\gamma)) > 0.$$

The former inequality can be rewritten as

$$\beta\gamma^2 + \alpha\beta\sigma_{n|S}\sigma_{n|A}(q-\gamma)^2 + (\beta\gamma - \rho)\sigma_{n|S}(q-\gamma) + \alpha(\beta\gamma + \rho)\sigma_{n|A}(q-\gamma) > 0.$$

This shows that the former inequality always holds given the restrictions on the parameters, as $q \geq \gamma$ and $\beta\gamma > \rho$. The second inequality can be written as

$$q(\alpha\beta q + (1-\alpha)(\beta\gamma - \rho)) + (1-\alpha)\rho\gamma,$$

which shows that also the latter inequality always holds, since $\beta\gamma > \rho$. Hence, we have $D > 0$.

Next, I consider the numerator N . After lengthy algebra, this can be rewritten as

$$N = (q-\gamma)\rho[\alpha\beta\gamma\sigma_{n|A}(q-\gamma)(1-\sigma_{n|S}) + \alpha q(\sigma_{n|S} - \sigma_{n|A})\rho + \alpha^2\beta\sigma_{n|A}(1-\sigma_{n|S})(q-\gamma)^2 + (1-\alpha)(1-\sigma_{n|S})\gamma(\beta\gamma - \rho) + \alpha\beta q\gamma(1-\sigma_{n|S})].$$

This demonstrates that also the numerator is always (weakly) positive under the restrictions on the parameters, since $q \geq \gamma$, $\beta\gamma > \rho$, $\sigma_{n|S} \leq 1$ and $\sigma_{n|S} \geq \sigma_{n|A}$. Hence, since the denominator is always positive and the numerator always non-negative, we have $\frac{N}{D} \geq 0$, which implies that the inequality in (61) always hold and thus that statement (ii) is true. Together with statement (i), this proves the result in . $\square$

Proposition 4. The lower the rate of false negatives, the lower the SS prevalence of unhealthy agents and the higher the level of public activity. Indeed, a decrease in the rate of false positives shifts $f_\sigma(q)$ and $g_\sigma(q)$ downward, which implies that the new intersection point lies to the south-west of the old one.

The numerator of the derivative of $g_\sigma(q)$ with respect to $\sigma_{p|A}$ is

$$-\alpha(1-\alpha)(1-q)^2\sigma_{n|S} \left(\sigma_{n|S}q + \sigma_{p|S}\gamma \right)^2, \quad (62)$$

which is always negative. The numerator of the derivative of $f_\sigma(q)$ with respect to $\sigma_{p|A}$ is given by

$$\begin{aligned} & \left(\beta(\gamma + \sigma_{n|S}(q-\gamma))\alpha(q-\gamma) - \alpha\rho(q-\gamma) \right) \times \\ & \left((1-\alpha) \left[\beta \left(\gamma + \alpha\sigma_{n|A}(q-\gamma) \right) - \rho \right] \left(\gamma + \sigma_{n|S}(q-\gamma) \right) \right) - \\ & - \alpha(1-\alpha)\beta \left(\gamma + \sigma_{n|S}(q-\gamma) \right) \times \\ & \left[\beta \left(\gamma + \sigma_{n|S}(q-\gamma) \right) \left(\gamma + \alpha\sigma_{n|A}(q-\gamma) \right) - \rho(q-\gamma)(\sigma_{n|S} - \alpha\sigma_{n|A}) \right]. \end{aligned} \quad (63)$$

It turns out that most terms can be simplified and that we remain with $-\rho^2 < 0$. Hence, given two tests σ and σ' such that $\sigma_{p|S} = \sigma'_{p|S}$ and $\sigma_{p|A} > \sigma'_{p|A}$, we must have that $I_\sigma^* < I_{\sigma'}^*$ and $q_\sigma^* > q_{\sigma'}^*$. $\square$

Proposition 5. The result follows from the case highlighted in Fig. 6, where the unhealthy prevalence increases following a reduction in the false positives rate.

Data availability

No data was used for the research described in the article.

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