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Stochastic Disease Spreading and Containment Policies under State-Dependent Probabilities

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Abstract

We analyze the role of disease containment policy in the form of treatment in a stochastic economic-epidemiological framework in which the probability of the occurrence of random shocks is state dependent, namely it is related to the level of disease prevalence. Random shocks are associated with the diffusion of a new strain of the disease which affects both the number of infectives and the growth rate of infection, and the probability of such shocks realization may be either increasing or decreasing in the number of infectives. We determine the optimal policy and the steady state of such a stochastic framework, which is characterized by an invariant measure supported on strictly positive prevalence levels, suggesting that complete eradication is never a possible long run outcome where instead endemicity will prevail. Our results show that: (i) independently of the features of the state-dependent probabilities, treatment allows to shift leftward the support of the invariant measure; and (ii) the features of the state-dependent probabilities affect the shape and spread of the distribution of disease prevalence over its support, allowing for a steady state outcome characterized by a distribution alternatively highly concentrated over low prevalence levels or more spread out over a larger range of prevalence (possibly higher) levels.

Keywords: Economic Epidemiology, Invariant Distribution, Optimal Policy, State-Dependent Probability
JEL Classification: C60. H50, I10

1 Introduction

Infectious diseases have historically played a major role in shaping the prospects of economic development both in industrialized and developing countries through a variety of microeconomic

and macroeconomic channels (Acemoglu and Johnson, 2007; Lopez et al., 2006; Boucekkine et al., 2009; Adda, 2016; Bloom et al., 2022). The ongoing COVID-19 pandemics has shown more clearly than ever that understanding how to contain the spread of communicable diseases is essential not only to protect human lives but also to preserve economic prosperity (World Bank, 2020; McKee and Stuckler, 2020). The economic epidemiology literature has extensively discussed the role of disease containment policies, mainly in the form of pharmaceutical interventions (generally classified as either preventive or treatment measures), in both limiting the spread of epidemic diseases (Philipson, 2000; Goldman and Lightwood, 2002; Gersovitz and Hammer, 2004; Anderson et al, 2010) and supporting economic activity (Goenka and Liu, 2012, 2019; Goenka et al., 2014; La Torre et al., 2020). The issue has been become even more popular following the COVID-19 outbreak, when a huge and growing number of works has analyzed from a normative perspective the optimal policy response to balance the economic and health trade-off involved in non-pharmaceutical interventions, such as social distancing, lockdowns and travel bans (Acemoglu et al., 2021; Alvarez et al., 2021; La Torre et al., 2021; Eichenbaum et al., 2021). Despite the high level of uncertainty associated with epidemic dynamics most of the studies have assumed that disease spreading is entirely deterministic, and very limited are those exploring the implications of stochasticity on the determination of the optimal containment policy (Federico and Ferrari, 2021; Hong et al., 2021; Shevchenko et al., 2021). Federico and Ferrari (2021) analyze how randomness in the disease transmission rate as well as in the time horizon impact policymakers' optimal response. Hong et al. (2021) shows that accounting for stochasticity in disease transmission yields richer optimal mitigation strategies than those derived in deterministic contexts. Shevchenko et al. (2021) discuss how stochastic epidemic shocks affect economic and environmental conditions analyzing their impact on optimal climate change policies. In all these works the probability of shocks affecting disease spreading is constant and thus completely independent of the level of disease prevalence. This is a strong simplification of reality where prevalence determines the likelihood of epidemic-related shocks by influencing disease incidence and individuals' behavioral responses. Our paper tries contributing to this scant literature by exploring how the optimal containment policy is related to stochastic shocks under state-dependent probabilities, that is the probability of shock realization depends on disease prevalence. State-dependent probabilities are a straightforward generalization of constant probabilities which allow to account for the mutual relation between epidemic shocks and epidemic dynamics.

Specifically, we develop a stylized economic-epidemiological framework in which the social planner needs to choose the optimal mitigation policy to limit the spread of an infectious disease by determining the intensity of treatment measures, accounting for the effects of stochastic shocks. Random shocks are associated with the diffusion of a new strain of the disease, which affects disease prevalence both additively (by increasing the number of infectives) and multiplicatively (by modifying the growth rate of infection), and the probability of shocks realization is state-dependent. In particular, we allow the shocks probability to be either increasing or decreasing in the number of infectives to account for the eventual presence or absence of individuals' behavioral changes in an attempt to reduce their disease exposure, respectively. Such two alternative setups may be well suited to describe individuals' response to different types of infections (common diseases vs. potentially deadly diseases), and thus allow us to characterize from a normative perspective how the optimal policy may change according to the specific features of the epidemic threat. In this context we explicitly derive the optimal policy by solving in closed-form the Bellman equation associated with our stochastic framework with state-dependent probabilities. This allows us to analyze its stochastic steady state which is represented by an invariant distribution of disease prevalence, with support on strictly positive

values meaning that complete eradication is never a possible outcome. We also characterize how the properties of the invariant distributions are related to the characteristics (in terms of monotonicity and steepness) of the probability function. We derive two interesting sets of conclusions. First, the optimal policy is independent of the features of the state-dependent probabilities, and independently of them treatment allows to shift leftward the support of the invariant measure. This suggests that the disease containment efforts are effective in reducing the possible endemic prevalence levels associated with the steady state outcome. Second, the features of the state-dependent probabilities do matter as they affect the distribution of disease prevalence (in particular its shape and spread) over its support. In particular, their monotonicity property determines the shape of the invariant distribution: whenever the probability function is decreasing the steady state outcome is characterized by a skewed distribution highly concentrated over extremely low or high prevalence levels, while whenever it is increasing the disease outcome is associated with epidemic waves giving rise to a distribution more evenly spread out over a large range of prevalence (possibly higher) levels. The steepness property of the state-dependent probabilities instead determines where most of the mass is concentrated, that is whether the probability of low prevalence levels is higher or lower; however, the likelihood of low prevalence depends in a nontrivial way on the interactions between the monotonic and steepness characteristics of the state-dependent probability function. Moreover, we present a new result, more general than those discussed in extant literature (Mitra et al., 2003; Shmerkin, 2014), determining sufficient conditions for the invariant measure to be either singular or absolutely continuous with respect to the Lebesgue measure, showing that this ultimately depends on the relative magnitude of the net infectivity rate and the weight attached to potential infections in the objective function.

By introducing state-dependent probabilities in the determination of the optimal disease containment policy, our paper makes some interesting contributions in two different branches of the literature. With respect to the economic epidemiology literature (Goldman and Lightwood, 2002; Gersovitz and Hammer, 2004; Goenka et al., 2014; La Torre et al., 2020) which discusses that the economy may converge to a situation of eradication or endemicity according to the effectiveness of disease containment policies, we show that complete eradication is not possible and the steady state outcome is represented by an endemic state in which the distribution of disease prevalence may be more or less concentrated around lower or higher levels according to the characteristics of the shock probabilities. Methodologically, instead, we rely on the theory of iterated function systems with state-dependent probabilities to characterize the long run properties of the dynamic system associated with our economic-epidemiological framework. Iterated function systems (IFS) with constant probabilities have been extensively employed in economic applications to characterize the fractal properties of the steady state in stochastic optimal growth models (Montrucchio and Privileggi, 1999; Mitra et al., 2003; Mitra and Privileggi, 2009; La Torre et al., 2015), while iterated function systems with state-dependent probabilities (IFSSDP) have been frequently employed only in the mathematics literature (Barnsley et al., 1985; Stenflo, 2002) and only seldom in economics (La Torre et al., 2019). Different from La Torre et al. (2019) who analyze how state-dependent probabilities affect the long run outcome in a purely dynamic context, we determine their implications on the optimal policy in a normative framework where the social planner specifically accounts for the role of state-dependent probabilities in its policy decisions. To the best of our knowledge, ours is the first attempt to address a stochastic dynamic optimization problem under state-dependent probabilities in economics.

The paper proceeds as follows. Section 2 reviews some concepts on the IFS theory and it focuses in particular on the theory of IFSSDP. Section 3 introduces our stochastic epidemiolog-

ical framework where random shocks associated with the diffusion of a new disease strain occur with state-dependent probabilities. Section 4 introduces our economic framework in which the social planner determines the optimal treatment policy accounting for the state-dependency of such probabilities. Section 5 explicitly derives the optimal solution discussing the role of the optimal policy in determining the steady state outcome and the role of state-dependent probabilities. Section 6 discusses the characteristics of the invariant measure in terms of singularity vs absolute continuity. Section 7 as usual presents concluding remarks and highlights directions for future research. All the proofs of our main results are presented in appendix A.

2 Iterated Function Systems

We now review some basic concepts and the main results in the theory of Iterated Function Systems (IFSs) with constant and state-dependent probabilities. The notion of IFS was firstly introduced by Barnsley et al. (1990) and Hutchinson (1981) and then extended in different contexts (see Kunze et al., 2012, and the references therein).

Given a compact metric space (X, d), an N-map *Iterated Function System* (IFS) on X, $\mathbf{w} = \{w_1, \ldots, w_N\}$, is a set of N contraction mappings on X, *i.e.*, $w_i : X \to X$, $i = 1, \ldots, N$, with contraction factors $c_i \in [0, 1)$. It can be proved that under these assumptions the following set-valued mapping $\hat{\mathbf{w}}$ defined on the space $\mathcal{H}(X)$ of nonempty compact subsets of X:

$$\hat{\mathbf{w}}(S) := \bigcup_{i=1}^{N} w_i(S), \qquad S \in \mathcal{H}(X).$$

is a contraction on the complete metric space $\mathcal{H}(X)$ endowed with the classical Hausdorff distance h defined as:

$$h(A, B) = \max\left\{\sup_{x\in A}\inf_{y\in B} d(x, y), \sup_{x\in B}\inf_{y\in A} d(x, y)\right\}.$$

This result implies the existence and uniqueness of a fixed point A such that $\hat{\mathbf{w}}(A) = A$. Moreover, A is self-similar, that is, it is the union of distorted copies of itself and it is also attracting, that is, for any $B \in \mathcal{H}(X)$, $h(A, \hat{\mathbf{w}}^t B) \to 0$ as $t \to \infty$.

An N-map *iterated function system with (constant) probabilities* $(\mathbf{w}, \mathbf{p})$ is an N-map IFS $\mathbf{w}$ with associated probabilities $\mathbf{p} = \{p_1, \ldots, p_N\}$, $\sum_{i=1}^{N} p_i = 1$. It can be proved that the Markov operator defined by $\nu(S) = (M\mu)(S)$:

$$\nu(S) = (M\mu)(S) = \sum_{i=1}^{N} p_i \mu\left(w_i^{-1}(S)\right).$$

is a contraction mapping on the space $\mathcal{M}(X)$ composed by all probability measures on (Borel subsets of) X with respect to the *Monge-Kantorovich* distance defined as follows: For any pair of probability measures $\mu, \nu \in \mathcal{M}(X)$, we have

$$d_{MK}(\mu, \nu) = \sup_{f\in Lip_1(X)}\left[\int f d\mu - \int f d\nu\right],$$

where $Lip_1(X) = \{f : X \to \mathbb{R} : |f(x) - f(y)| \leq d(x, y)\}$. These assumptions imply the existence of a unique attracting measure $\bar{\mu} \in \mathcal{M}(X)$.

The family of IFS with state-dependent probabilities extends the above definitions. Within this framework, the probabilities p_i are no longer constant but they are are state-dependent, *i.e.*, $p_i : X \to [0,1]$ such that:

$$\sum_{i=1}^{N} p_i(x) = 1, \qquad \text{for all } x \in X. \tag{1}$$

The result is an N-map *IFS with state-dependent probabilities* (IFSSDP). The Markov operator $M : \mathcal{M}(X) \to \mathcal{M}(X)$ associated with an N-map IFSSDP, $(\mathbf{w}, \mathbf{p})$, is defined as:

$$\nu(S) = M\mu(S) = \sum_i \int_{w_i^{-1}(S)} p_i(x)\, d\mu(x), \tag{2}$$

where $\mu \in \mathcal{M}(X)$ and $S \subset X$ is a Borel set.

Theorem 1 (La Torre et al., 2018a) *Given M as defined in equation (2), then M maps $\mathcal{M}(X)$ to itself. In other words, if $\mu \in \mathcal{M}(X)$, then $\nu = M\mu \in \mathcal{M}(X)$.*

Under appropriate conditions, the above Markov operator can be contractive with respect to the Monge-Kantorovich metric.

Theorem 2 (La Torre et al., 2018a) *Let (X, d) be a compact metric space and $(\mathbf{w}, \mathbf{p})$ an N-map IFSSDP with IFS maps $w_i : X \to X$ with contraction factors $c_i \in [0,1)$. Furthermore, assume that the probabilities $p_i : X \to \mathbb{R}$ are Lipschitz functions, with Lipschitz constants $K_i \geq 0$. Let $M : \mathcal{M}(X) \to \mathcal{M}(X)$ be the Markov operator associated with this IFSSDP, as defined in (2). Then for any $\mu, \nu \in \mathcal{M}(X)$,*

$$d_{MK}(M\mu, M\nu) \leq (c + KDN)\, d_{MK}(\mu, \nu),$$

where $c = \max_i c_i$, $K = \max_i K_i$ and $D = \operatorname{diam}(X) < \infty$.

Theorem 3 (La Torre et al., 2018a) *Under the same assumptions as in the above Theorem, if $c + KDN < 1$ then the Markov operator M has a unique fixed point μ in $\mathcal{M}(X)$. Furthermore, for any $\nu \in \mathcal{M}(X)$, the orbit $M^n\nu$ converges to μ in d_{MK} when $n \to +\infty$.*

We now describe the so-called *Chaos Game* for an IFS with probabilities. Start with $x_0 \in X$, and define the sequence $x_t \in X$ by:

$$x_{t+1} = w_{\sigma_t}(x_t),$$

where $\sigma_t \in \{1, 2, \ldots, N\}$ is chosen according to the probabilities $p_i(x_t)$ (that is, $P[\sigma_t = i] = p_i(x_t)$). We note that the sequence (x_t) is a Markov chain with values in X. The following theorem (from results in Elton, 1987; and Barnsley et al., 1988) gives conditions as to when an IFSSDP has a unique stationary distribution μ and the Chaos Game "converges" to μ in a distributional sense.

Theorem 4 (Elton, 1987; Barnsley et al., 1988) *Suppose that there is a $\delta > 0$ so that $p_i(x) > \delta$ for all $x \in X$ and $i = 1, 2, \ldots, N$ and suppose further that the moduli of continuity of the p_is satisfy Dini's condition (see Elton, 1987; and Barnsley et al., 1988). Then there is a unique stationary distribution $\bar{\mu}$ for the Markov operator. Furthermore, for each continuous function $f : X \to \mathbb{R}$,*

$$\frac{1}{t+1} \sum_{i=0}^{t} f(x_i) \to \int_X f(x)\ d\bar{\mu}(x). \tag{3}$$

Theorem 4 can be used to show the following result.

Corollary 1 *Suppose that the IFSSDP* $\{\mathbf{w}, p_i\}$ *satisfies the hypothesis of Theorem 4. Then the support of the invariant measure* $\bar{\mu}$ *of the* N*-map IFSSDP* $(\mathbf{w}, \mathbf{p})$ *is the attractor* A *of the IFS* $\mathbf{w}$*, i.e.,*

$$\operatorname{supp} \bar{\mu} = A.$$

Therefore the invariant measure μ satisfies the following equation

$$\mu(S) = \sum_i \int_{w_i^{-1}(S)} p_i(x)\, d\mu(x), \tag{4}$$

for any subset S of X. This equation shows how the invariant measure can be obtained by combining different distorted copies of itself. This justifies why the invariant measure is a self-similar object. These basic concepts related to the theory of IFSSDP will be useful to derive the steady state equilibrium and understand its characteristics in our economic-epidemiological model.

3 The Epidemiological Model

We start discussing the epidemiological context abstracting completely from containment policies in order to clarify our setup and the role of state-dependent probabilities. We develop a very simple framework to characterize the spread of a communicable disease, which may be either a common disease (i.e., the seasonal flu, the common cold) or a potentially deadly infection (i.e., SARS, COVID-19). Different from traditional epidemiological setups in which the interactions between different population groups (i.e., susceptibles and infectives) drive the epidemic dynamics, we focus only on the determinants of disease prevalence. In such a simplistic context we account for the uncertainty associated with infection diffusion by considering the role played by the arise of a new disease strain and by endogeneizing the likelihood with which random shocks occur assuming that the their probability is state dependent.

The population size, which is constant and normalized to unity without loss of generality, $N \equiv 1$, is composed by healthy individuals who are susceptible to the disease, S_t, and the infectives who have already contracted the disease and can transmit it by getting in contact with susceptibles, I_t, thus at any moment in time we have that $1 = S_t + I_t$. We assume that the disease dynamics is described by the following equation:

$$I_{t+1} = \Omega z_t I_t + z_t, \tag{5}$$

where $\Omega > 0$ measures the net infectivity rate (i.e., the infectivity rate net of the recovery rate) and z_t denotes random shocks that can take one of the two values, r_1 or r_2, such that $0 < r_1 < r_2$. The equation above states that the dynamics of disease prevalence depends on the biological features of the disease (Ω) and the realization of random shocks (z_t). Biological factors combined with the disease prevalence determine the disease incidence ΩI_t which characterizes the pace of disease diffusion in the presence of only one strain of the disease. The random shock term captures the twofold impact of a new disease strain on the evolution of the disease. (i) A new strain is discovered when an individual is found to be infected with a genetic variant of the microorganism (i.e., a virus or bacterium) causing the infectious disease. Thus, the diffusion of a new strain gives rise to some new infections not related to the single-strain disease incidence, captured by the additive term $+z_t$. (ii) A new strain is characterized by different infectivity

and recovery rates with respect to the original strain of the disease, such that the biological disease parameters change from one strain to the next. Thus, with the origin of a new strain the average biological parameters of the disease between strains change, such that the net infectivity rate may become higher or lower following the discovery of a new strain, captured by the multiplicative term $\Omega z_t I_t$.

The probability of the realization of such random shocks is not constant but state dependent, that is it depends on the level of disease prevalence $p(I_t)$. Specifically, $\{z_t\}_{t=0}^{\infty}$ is a Bernoulli process such that at each date t:

$$z_t = \begin{cases} r_1 & \text{with probability } p\left(I_t\right) \\ r_2 & \text{with probability } 1 - p\left(I_t\right) \end{cases}, \tag{6}$$

where either $p' < 0$, that is the probability that the smaller shock value, r_1, (larger shock value r_2) is decreasing (increasing) in the number of infectives, or $p' > 0$, that is the probability that the smaller shock value, r_1, (larger shock value r_2) is increasing (decreasing) in the number of infectives. The former case represents a situation in which individuals do not automatically implement behavioral changes in response to increases in disease prevalence, and this in turn expands the spread of the disease and thus also the eventual diffusion of a new strain. The latter case instead describes a situation in which individuals do automatically implement behavioral changes in response to increases in disease prevalence by reducing their possible exposure to the disease and this in turn limits the spread of the disease and thus also the likelihood of diffusion of a new strain. We believe that both scenarios are realistic conceptualizations of how a disease may spread following individuals' behavioral response, because such a response may largely depend on the biological features of specific diseases. For example, when dealing with common diseases (such as the seasonal flu) individuals rarely implement behavioral changes to limit their exposure thus the $p' < 0$ case may apply, while when dealing with potentially deadly diseases (such as COVID-19) behavioral changes may become predominant thus the $p' > 0$ case may apply instead. In the following we shall consider both scenarios and analyze how the features of the probability function $p(\cdot)$ may affect our conclusions.

Equation (5) describes the evolution of disease prevalence based on the idea that there exists some universality in the features of epidemic dynamics independently of the specific epidemiological model underlying disease spreading (i.e., SI, SIS, SIR, SIRS...). A similar setup is frequently used in empirical applications to perform estimation and forecasting of the evolution of the number of infectives without specifying a particular epidemiological model (Zakharov et al., 2020; Remuzzi and Remuzzi, 2020), and also in epidemiological studies to describe infection dynamics in early stages of an epidemic when the number of infectives is relatively small compared to the susceptible population (Chowell et al., 2016; Ma, 2020). Basically, the net infectivity rate determines the growth factor of infection and if this is large enough disease prevalence will tend to increase over time, while if it is small prevalence will tend to decrease. By affecting the magnitude of such a growth factor, the state dependency of shocks realization may give rise to periods of positive and negative prevalence growth, resulting eventually in the occurrence of different disease waves. Specifically, in the $p' > 0$ case when prevalence is low the probability of the larger shock value is high and this tends to increase prevalence giving rise to an expansionary period of infection, but as prevalence increases also the probability of the smaller shock value rises and this tends to lower prevalence giving rise to a contractionary period of infection. Overall, periods of growing and shrinking prevalence may alternate one another over time characterizing multiple epidemic waves. In the $p' < 0$ case instead when prevalence is low the probability of the larger shock value is low and this tends to decrease prevalence giving rise to a contractionary period of infection, deterring the possibility of fast

infection growth. Overall, periods of shrinking (or growing) prevalence may tend to persist over time characterizing monotonic epidemic dynamics. Note that if the probability of shocks were constant (i.e., $p' = 0$) such alternative outcomes would not be possible because the evolution of infection would resemble a random walk.

Despite the simplicity of (5) in describing epidemic dynamics, we believe that its ability to characterize endogenously the occurrence of periods characterized by monotonic epidemic dynamics or by multiple epidemic waves makes it a good benchmark to understand the working mechanisms of disease containment policies. Indeed, traditional mathematical epidemiological models cannot account endogenously for such alternative outcomes, as they are generally characterized by monotonic epidemic dynamics in which there is no room for multiple waves, and in order to explain alternate periods of growing and shrinking infections they usually rely on ad-hoc assumptions, such as the exogenous introduction of a periodic term to capture some cyclicality in disease transmission (Grassly and Fraser, 2006; Jodar et al., 2008). Thus, the ability of our setup to describe within the same stylized framework monotonic epidemic dynamics and multiple epidemic waves, whose alternative occurrence depends on the specific features of the probability function, represents a novel approach to conceptualize disease spreading which may help us to better understand the role of containment policies in limiting the spread of infectious diseases. In particular we wish to clarify how the characteristics of the state dependent probability function impacts epidemic dynamics and policymakers' optimal policy response.

A central role in our analysis needs thus to be placed on the features of the state dependent probabilities. In this context, note that as the number of infectives I_t in each period t must lie in the interval $[0, 1]$, also the state-dependent probabilities have the same domain, that is, $p : [0, 1] \to [0, 1]$. In order to analyze explicitly the role of state dependent probabilities, we introduce the following hyperbolic forms for $p(\cdot)$, defined for $I \in [0, 1]$:

$$p(I) = \frac{a}{BI^2 + 1} \quad \text{and} \quad 1 - p(I) = 1 - \frac{a}{BI^2 + 1}, \quad \text{or} \tag{7}$$

$$p(I) = 1 - \frac{a}{BI^2 + 1} \quad \text{and} \quad 1 - p(I) = \frac{a}{BI^2 + 1}, \tag{8}$$

where $0 < a \leq 1$ and $B > 0$ are parameters. Note that, because $0 < a \leq 1$, $p(I)$ and $1 - p(I)$ according to (7) actually have values in $\left[\frac{a}{B+1}, a\right] \subset (0, 1]$ and $\left[1 - a, 1 - \frac{a}{B+1}\right] \subset [0, 1)$ respectively, while $p(I)$ and $1 - p(I)$ according to (8) have values in $\left[1 - a, 1 - \frac{a}{B+1}\right] \subset [0, 1)$ and $\left[\frac{a}{B+1}, a\right] \subset (0, 1]$ respectively. Moreover, $p(I)$ in (7) is such that $p'(I) < 0$, so that the probability of the smaller shock value, r_1 (larger shock value, r_2) is decreasing (increasing) in the number of infectives; conversely, $p(I)$ in (8) is such that $p'(I) > 0$, so that the probability of the smaller shock value, r_1 (larger shock value, r_2), is increasing (decreasing) in the number of infectives. Therefore, (7) defines two (Lipschitz) continuous state-dependent probability functions satisfying $0 < p(I) \leq 1$ and $0 \leq 1 - p(I) < 1$ for all $0 \leq I \leq 1$, and (8) defines two (Lipschitz) continuous state-dependent probability functions satisfying $0 \leq p(I) < 1$ and $0 < 1 - p(I) \leq 1$ for all $0 \leq I \leq 1$. We shall see in the following that the dynamics of I_t will always remain trapped in a proper sub-interval of $[0, 1]$, so that the values of both $p(I)$ and $1 - p(I)$ will always be bounded away from 0 and 1, as required by Theorem 4 and Corollary 1 to establish the existence and uniqueness of the invariant measure.

The dynamics in (5) can be rewritten in terms of the following IFSSDP:

$$I_{t+1} = \begin{cases} r_1 \Omega I_t + r_1 & \text{with probability } p(I_t) \\ r_2 \Omega I_t + r_2 & \text{with probability } 1 - p(I_t), \end{cases} \tag{9}$$

which can be analyzed through the tools described in the previous Section 2, which ensure the existence of a unique stationary distribution μ for such an IFSSDP supported on the interval

$[I_1^{st}, I_2^{st}] \subset [0,1]$, where the endpoints are the steady states of the two affine maps in (9) respectively:

$$I_1^{st} = \frac{r_1}{1 - r_1\Omega} \qquad \text{and} \qquad I_2^{st} = \frac{r_2}{1 - r_2\Omega}. \tag{10}$$

In order to keep the dynamics of I_t defined by IFSSDP (9) inside the interval $[0,1]$ and rule out the trivial case $r_1 = 0$, the steady states of the above maps must satisfy the following parameter condition:

$$0 < r_1 < r_2 \leq \frac{1}{1+\Omega}. \tag{11}$$

Note that (11) imposes an upper limit to the larger shock value, which turns out to be strictly lower than unity because $\Omega > 0$. Indeed, under both the larger and smaller shock values, the diffusion of a new disease strain decreases the growth rate of infection, which in turn ensures that the dynamics in (9) remains trapped in a subset of $[0,1]$. Note that, since the smaller shock value needs to be strictly larger than zero, the steady states of the maps in (10), which determines the left endpoint of the support of the invariant measure, do not include $I = 0$, which suggests that full eradication will never be possible. Since the diffusion of new disease strains affects additively epidemic dynamics, some new infectives will always be adding to the existing stock of infectives precluding the possibility for full eradication.

It is also interesting to note that the characteristics of the probability function do not affect the support of the invariant measure, but they may affect the distribution of disease prevalence over its support. Unfortunately, characterizing this explicitly is not possible thus in the following we shall present some numerical example to illustrate the implications of different shapes of the probability function on the steady state distribution of disease prevalence. Specifically, we shall numerically approximate the time evolution of a given probability density according to the affine IFSSDP (9). To this purpose, we apply a Maple algorithm[1] that approximates successive iterations of the Markov operator (2) associated with the IFSSDP which is based on Algorithm 1 in La Torre et al. (2019), in order to have a qualitative idea on what the limiting invariant measure may look like. In the following numerical examples, we assume that the initial density is uniform and given by $\mu_0(I) \equiv \frac{1}{I_2^{st} - I_1^{st}}$, and we set the parameter values arbitrarily as follows:

$$\Omega = 2, \qquad r_2 = \frac{1}{\Omega + 1} = \frac{1}{3}, \qquad r_1 = \frac{r_2}{2} = \frac{1}{6}, \tag{12}$$

while we consider the two alternative values $B = \{3.571, 14.286\}$ in order to perform some comparative dynamics and to allow comparability with what we will present later when we determine the optimal disease containment policy. Indeed, as it will become clearer in section 5, our goal is to obtain a closed-form solution for a planning problem and for this to be possible some parameter restrictions are required, and in particular the constant B needs to take on one of the specific values we have considered in our parametrization. Figure 1 plots probabilities $p(I)$ (left panels) and $1 - p(I)$ (right panels) defined as in (7) for $B = 3.571$ (top panels) and $B = 14.286$ (bottom panels): clearly, $p(I)$ is decreasing in Figures 1(a) and 1(c) while $1 - p(I)$ is increasing in Figures 1(b) and 1(d); the difference between the top and bottom figures is related to the more pronounced steepness in the bottom panels.

Under the parametrization in (12), our IFSSDP (9) reads as follows:

$$I_{t+1} = \begin{cases} \Omega r_1 I_t + r_1 = \dfrac{1}{3} I_t + \dfrac{1}{6} & \text{with probability } p(I_t) \\ \Omega r_2 I_t + r_2 = \dfrac{2}{3} I_t + \dfrac{1}{3} & \text{with probability } 1 - p(I_t), \end{cases} \tag{13}$$

[1]The detailed code is available upon request.

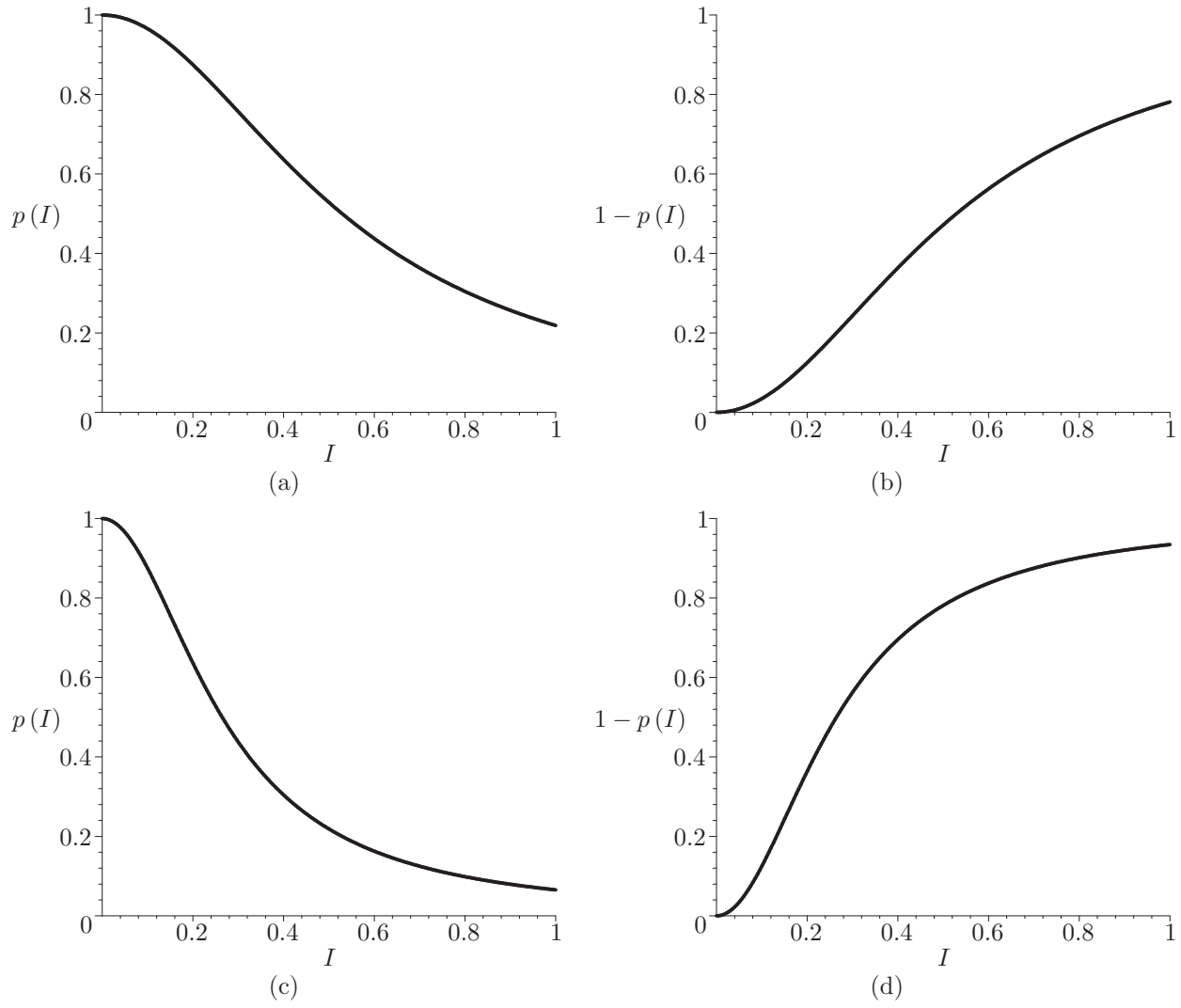

FIGURE 1: state-dependent probabilities, $p(I) = \frac{1}{BI^2+1}$ (left) and $1 - p(I) = 1 - \frac{1}{BI^2+1}$ (right) as defined in (7), associated with schocks $r_1 = \frac{1}{6}$ and $r_2 = \frac{1}{3}$ respectively, for $B = 3.571$ (top) and $B = 14.286$ (bottom).

and it has $[I_1^{st}, I_2^{st}] = [0.25, 1]$ as trapping interval (the fixed point of the upper map is 1 due to our choice on the larger shock to be exactly its admissible upper bound: $r_2 = \frac{1}{\Omega+1}$). As the fixed point of the lower map, $I_1^{st} = 0.25$, is bounded away from 0, both $p(I_t)$ and $1 - p(I_t)$ are such that $0 < p(I_t) < 1$ and $0 < 1 - p(I_t) < 1$ for all $I \in [0.25, 1]$. Hence, the dynamics of I_t will always remain trapped in the proper sub-interval $[0.25, 1] \subset [0, 1]$.

We consider first the scenario in which $p(I)$ is decreasing and specifically $p(I_t)$ takes the form in (7). In this case, because the left endpoint of the trapping interval $[0.25, 1]$—recall that, according to Corollary 1, $[0.25, 1]$ is the support of the invariant measure—is bounded away from 0, the values of both $p(I)$ and $1 - p(I)$ will always be bounded away from 0 and 1, as required by Theorem 4 to establish existence and uniqueness of the invariant measure. Figure 2 shows the initial uniform density $\mu_0(I) \equiv \frac{1}{I_2^{st} - I_1^{st}}$ (left panels), the 1^{st} (mid panels) and 6^{th} (right panels) iterations of our Maple algorithm for the IFSSDP (13) whenever $B = 3.571$ (top panels) or $B = 14.286$ (bottom panels). As convergence toward the unique invariant measure is geometric, *i.e.* very fast, Figures 2(c) and 2(f) can be considered as good approximations

of the invariant measure itself. As $\frac{1}{3}+\frac{1}{6}=\frac{1}{2}=\frac{2}{3}\frac{1}{4}+\frac{1}{3}$, the images of the two affine maps in (13) *almost* do not overlap, having in common the only point $\frac{1}{2}$ so that the invariant measure has the full interval $[0.25,1]$ as support. In the case of a small B, Figure 2(b) shows that the IFSSDP concentrates a large probability mass of the uniform density in Figure 2(a) close to the lower fixed point $I_1^{st}=0.25$, and this process is being reinforced after each iteration so to obtain, after 6 iterations, Figures 2(c), in which the mass concentrated in the vicinity of I_1^{st} has become predominant. In the case of a large B, Figure 2(e) shows that the IFSSDP concentrates a large probability mass of the uniform density close to the higher fixed point $I_2^{st}=1$, such that after 6 iterations in Figures 2(f) larger mass is concentrated in the vicinity of I_2^{st}. Therefore, whenever $p(I_t)$ is decreasing, in the medium-long run the epidemic dynamics are characterized by a monotonic variation in infections. If B is small (large) such a monotonic dynamic is associated with a reduction (increase) in infections which may increase only (also) because of the additive shock induced by the diffusion of a new disease strain, such that the level of disease prevalence tends to be concentrated to a large extent near the lower (upper) extreme of the support of the invariant measure and the steady state outcome is represented by an endemic state with low (high) prevalence.

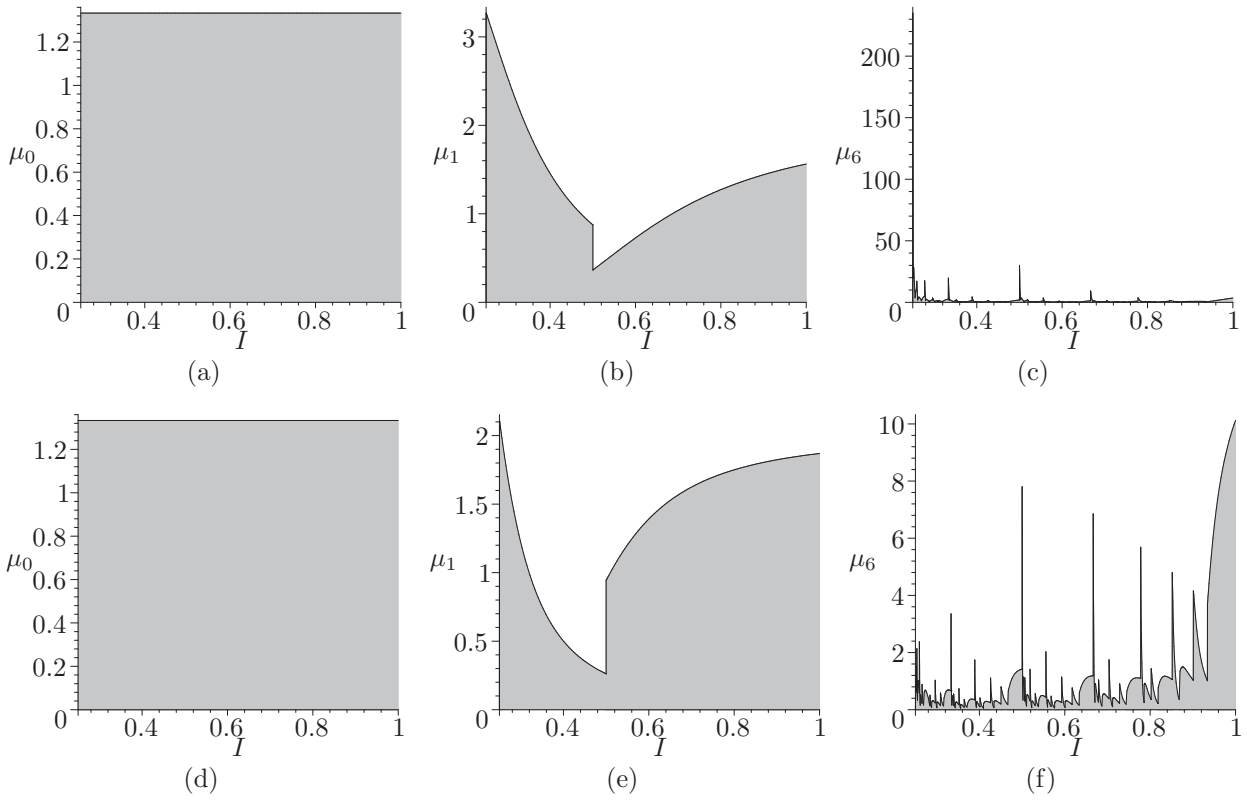

FIGURE 2: Initial uniform density over $[0.25,1]$ (left), 1^{st} (mid) and 6^{th} (right) iterations of our Algorithm to approximate the Markov operator (2) associated to the IFSSDP (13) whenever $p(I)=\frac{1}{BI^2+1}$ with $B=3.571$ (top) or $B=14.286$ (bottom).

We consider now the scenario in which $p(I)$ is increasing and specifically $p(I_t)$ takes the form in (8). Because the trapping interval is still $[0.25,1]$, again the values of both $p(I)$ and $1-p(I)$ are bounded away from 0 and 1 and Theorem 4 applies. Figure 3 shows the initial uniform density $\mu_0(I)\equiv\frac{1}{I_2^{st}-I_1^{st}}$ (left panels) and the 1^{st} (mid panels) and 6^{th} (right panels) iterations of our Maple algorithm for the IFSSDP (13) whenever $B=3.571$ (top panels) or $B=14.286$

(bottom panels). In the case of a small B, Figure 3(b) shows that the IFSSDP concentrates a large probability mass of the uniform density in Figure 3(a) around the interval $[0.4, 0.5]$, Such a pattern, although scattered across all pre-fractals of the interval $[0.25, 1]$ emerging after each iteration, is clearly preserved in the medium-long term approximation of the probability measure plotted in Figure 3(c). In the case of a large B, Figure 3(e) shows that the IFSSDP concentrates a large probability mass of the uniform density in Figure 3(d) to the left of 0.5, such that after 6 iterations in Figure 3(f) a larger mass is concentrated close to the lower fixed point $I_1^{st} = 0.25$.Therefore, whenever $p(I_t)$ is increasing, in the medium-long run the epidemic dynamics are characterized by fluctuations in the level of infections, giving rise to multiple epidemic waves: the additive shocks combined with the higher incidence due to the diffusion of a new disease strain lead the number of infectives to continually rise and fall. If B is small (large) such fluctuations are associated on average with a larger (lower) number of infections, such that the level of disease prevalence tends to be dispersed but more densely concentrated toward to upper (lower) extreme of the support of the invariant measure and the steady state outcome is represented by an endemic state with diffuse prevalence.

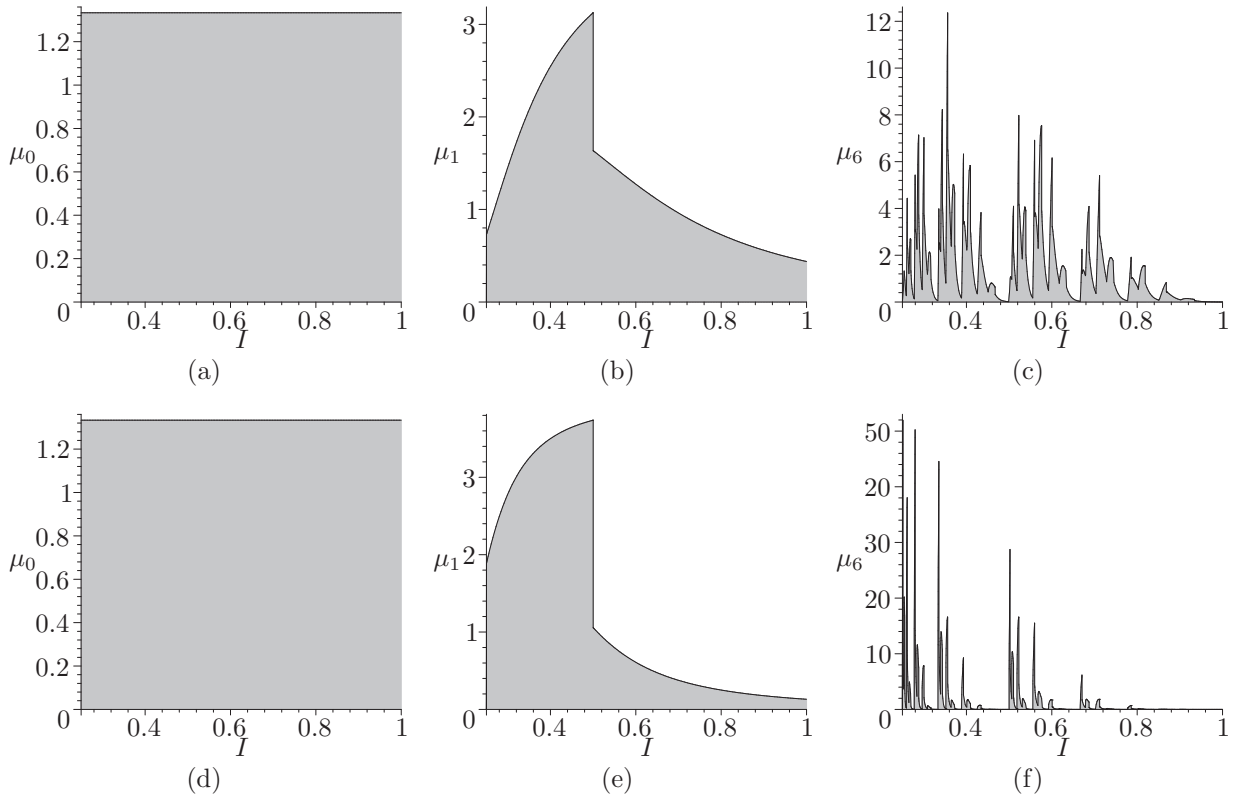

FIGURE 3: Initial uniform density over $[0.25, 1]$ (left), 1^{st} (mid) and 6^{th} (right) iterations of our Algorithm to approximate the Markov operator (2) associated to the IFSSDP (13) whenever $p(I) = 1 - \frac{1}{BI^2+1}$ with $B = 3.571$ (top) or $B = 14.286$ (bottom).

By comparing Figures 2 and 3, we can observe that, despite the fact that the support of the invariant measure does not change with the characteristics of the probability function, the properties of the state-dependent probability (both in terms of the sign and size of its first derivative) critically determine the distribution of disease prevalence over its support. The sign of the derivative of the probability function determines the shape of the invariant distribution, and in particular whether this tends to be skewed or more symmetric over its

support. Whenever $p' < 0$ the distribution tends to be more skewed toward one of the two extremes of the support (Figure 2), while whenever $p' > 0$ it tends to be more symmetric and evenly distributed (Figure 3). The size of the derivative of the probability function (i.e., the magnitude of B) instead determines where most of the mass is concentrated, and in particular whether the probability of low prevalence levels is higher or lower. However, the size of the derivative and its sign jointly contribute to determine such a feature of the invariant probability. When $p' < 0$ a high B leads the distribution to be more concentrated toward the upper extreme of the support (Figures 2), while when $p' > 0$ a high B leads it to be more concentrated toward the lower extreme (Figure 3).

We can conclude that the monotonicy (increasingness vs decreasingness) and the steepness (low vs high steepness) properties of the state-dependent probability function jointly contribute to determine a wide variety of possible outcomes. There may be situations in which it is fair to expect that the steady state outcome will be associated with positive but low levels of disease prevalence, and as the spread of the invariant distribution is particularly small in the stochastic steady state it is almost possible to deterministically determine the arising prevalence level ($p' < 0$ and B small – see Figure 2, top panels). Alternatively, it may happen that the spread of the invariant distribution is as large as its support such that in the stochastic steady state it is almost impossible to forecast the arising prevalence level ($p' < 0$ and B large – see Figure 2, bottom panels). But is may also happen that, despite the more limited spread, disease prevalence is evenly spread across the support such that we cannot really understand whether prevalence will tend to be characterized by low or high values ($p' > 0$, both with small and large B – see Figure 3).

Our results surprisingly suggest that behavioral changes aiming at reducing individuals' exposure to the disease ($p' > 0$) may not always be that desirable in improving the long run health outcome, since people's behavioral response to changes in disease prevalence combined with the random diffusion of new disease strains may result in perpetual epidemic waves characterized by eventually high prevalence. But also the absence of behavioral changes to minimize disease exposure ($p' < 0$) may not be more desirable as in this case this may generate monotonic epidemic dynamics giving rise to high prevalence. Therefore, whenever the probability of epidemic shocks is state dependent it may be more important than ever to rely on public intervention to improve the long run health outcomes. We thus now investigate the role of public disease containment policies in shaping the invariant distribution of prevalence under state-dependent probabilities.

4 The Economic Model

We now introduce our economic framework by analyzing how a social planner decides the intensity of the policy measure to reduce the spread of a communicable disease, whose evolution is characterized as in the previous section, in order to minimize the social cost associated with the epidemic management program. As in epidemic periods the control of the spread of the communicable disease becomes the main priority for policymakers, we assume that the resources available to contain the epidemic are unconstrained, that is policymakers may always rely on international borrowing to finance their mitigation expenditure needs. For the sake of simplicity we do not model international borrowing, but we simply assume that the resources available for public health policy are exogenously given and large enough to meet policymakers' expenditure needs. The disease dynamics is characterized as in the previous section by the following equation: $I_{t+1} = \Omega z_t I_t + z_t - X_t$, where the last term, $X_t \geq 0$, captures the effects of treatment measures which, by favoring recovery, reduces the number of infectives. The social

cost is the discounted sum ($0 < \beta < 1$ is the discount factor) of the one-period losses associated with the epidemic management program. The one-period loss function depends on the level of disease incidence, $z_t I_t$, the potential infections associated with the diffusion of a new strain of the disease, $z_t S_t$ and the intensity of policy intervention, and is assumed to take the following additively separable quadratic form: $\ell(I_t, S_t, z_t) = \gamma_1 z_t^2 I_t^2 + \gamma_2 z_t^2 S_t^2 + X_t^2$, where $\gamma_1 > 0$ and $\gamma_2 > 0$ measure the relative weight of incidence and potential infection with respect to economic policy, respectively. Note that the potential infections due to a new disease strain depend on the share of susceptibles, since only susceptible individuals may be subject to infection (infectives are already exposed to the disease, thus the diffusion of a new strain may affect the economy only up to the extent that its population is susceptible). The random shock term z_t directly affects the instantaneous losses since the diffusion of a new strain determines disease incidence and potential infections. By recalling that $S_t = 1 - I_t$ and denoting with $\mathbb{E}_0$ the expectation operator at time $t = 0$, the social planner's problem can be summarized by the following stochastic dynamic programming model:

$$V(I_0, z_0) = \min_{\{X_t\}} \mathbb{E}_0 \sum_{t=0}^{\infty} \beta^t \left[\gamma_1 z_t^2 I_t^2 + \gamma_2 z_t^2 (1 - I_t)^2 + X_t^2\right] \tag{14}$$
$$\text{s.t.} \begin{cases} I_{t+1} = \Omega z_t I_t + z_t - X_t, \\ 0 \leq I_t \leq 1,\ X_t \geq 0\ \forall t \geq 0, \\ 0 \leq I_0 \leq 1 \text{ and } z_0 \in \{r_1, r_2\} \text{ are given,} \end{cases}$$

where $\{z_t\}_{t=0}^{\infty}$ is the Bernoulli process (6) taking positive values r_1, r_2 such that $r_1 < r_2$ with *state-dependent probabilities* $p(I_t)$ and $1 - p(I_t)$ discussed in the previous section, and where the probability function is alternatively specified as in (7) or (8).

As by assumption $X_t \geq 0$ must hold for all $t \geq 0$, $I_{t+1} = \Omega z_t I_t + z_t - X_t \leq \Omega z_t I_t + z_t \leq 1$ holds for all $t \geq 0$, where the last inequality is a consequence of condition (11), $0 < r_1 < r_2 \leq \frac{1}{\Omega+1}$, discussed in Section 3. Moreover, the value $I_{t+1} = \Omega z_t I_t + z_t - X_t = 0$ is always feasible for any I_t value and shock realization z_t because X_t can be taken large enough. Hence, we can substitute $X_t = \Omega z_t I_t + z_t - I_{t+1}$ from the dynamic constraint into the one-period objective function so that the reduced problem associated with (14) can be stated as follows:

$$V(I_0, z_0) = \min_{\{I_t\}} \mathbb{E}_0 \sum_{t=0}^{\infty} \beta^t \left[\gamma_1 z_t^2 I_t^2 + \gamma_2 z_t^2 (1 - I_t)^2 + (\Omega z_t I_t + z_t - I_{t+1})^2\right] \tag{15}$$
$$\text{s.t.} \begin{cases} 0 \leq I_{t+1} \leq \Omega z_t I_t + z_t,\ \forall t \geq 0, \\ 0 \leq I_0 \leq 1 \text{ and } z_0 \in \{r_1, r_2\} \text{ are given,} \end{cases}$$

where the constraint $0 \leq I_t \leq 1$ for all $t \geq 0$ is guaranteed by condition (11). Note that the probability $p(I_t)$ determines the occurrence of the random shock z_t at the same time t in which the actual number of infectives is I_t; hence, such a probability, through the realization of one of the two shocks $z_t \in \{r_1, r_2\}$, affects both the instantaneous losses in the objective function at time t and the number of infectives in the next period $t+1$ through the dynamic constraint.

Because the z_t-sections of the graph $G = \{(I_t, I_{t+1}, z_t) : I_{t+1} \in \Gamma(I_t, z_t)\}$ of the optimal correspondence $\Gamma(k_t, z_t) = \{I_{t+1} : 0 \leq I_{t+1} \leq \Omega z_t I_t + z_t\}$ are convex sets and the one-period objective function is quadratic, (15) is clearly a *convex problem* defined over the state space $[0, 1]$.

5 The Optimal Policy and Dynamics

In order to explicitly determine the optimal policy in our economic-epidemiological model, we need to solve in closed-form the Bellman equation associated with (15), which reads as follows:

$$V(I,z) = \min_{0\leq y\leq \Omega zI+z} \left[\gamma_1 z^2 I^2 + \gamma_2 z^2 (1-I)^2 + (\Omega zI + z - y)^2 + \beta \mathbb{E}_y V(y, z')\right],$$

where $\mathbb{E}_y$ denotes the expectation operator that depends on the probabilities of both realizations of the random variable z' occurring in the next period, themselves depending on the choice y, which corresponds to the number of infectives in the next period; that is, $\Pr(z' = r_1) = p(y) = \frac{a}{By^2+1}$ and $\Pr(z' = r_2) = 1 - p(y) = 1 - \frac{a}{By^2+1}$ if probabilities are taken according to (7), or $\Pr(z' = r_1) = p(y) = 1 - \frac{a}{By^2+1}$ and $\Pr(z' = r_2) = 1 - p(y) = \frac{a}{By^2+1}$ if probabilities are taken according to (8) (recall that, for given y, the random variable z' is independent of past realizations). In order to solve the above Bellman equation we consider separately the two cases in which the state-dependent probabilities have either the form in (7) or in (8).

5.1 The $p' < 0$ Case

By assuming that the probabilities are given by (7) the expectation $\mathbb{E}_y$ can be directly evaluated and the Bellman equation can be rewritten in the following form:

$$\begin{aligned}
V(I,z) &= \min_{0\leq y\leq \Omega zI+z} \left\{\gamma_1 z^2 I^2 + \gamma_2 z^2 (1-I)^2 + (\Omega zI + z - y)^2 \right. \\
&\quad \left. + \beta p(y) V(y, r_1) + \beta [1 - p(y)] V(y, r_2)\right\} \\
&= \min_{0\leq y\leq \Omega zI+z} \left[\gamma_1 z^2 I^2 + \gamma_2 z^2 (1-I)^2 + (\Omega zI + z - y)^2 \right. \\
&\quad \left. + \frac{\beta a}{By^2+1} V(y, r_1) + \beta \left(1 - \frac{a}{By^2+1}\right) V(y, r_2)\right] \\
&= \min_{0\leq y\leq \Omega zI+z} \left\{\gamma_1 z^2 I^2 + \gamma_2 z^2 (1-I)^2 + (\Omega zI + z - y)^2 \right. \\
&\quad \left. + \frac{\beta a}{By^2+1} [V(y, r_1) - V(y, r_2)] + \beta V(y, r_2)\right\}.
\end{aligned}$$

In order to search for a closed-form solution of our optimization problem, we guess the following form for the value function in the Bellman equation:

$$V(I,z) = Az^2 \left(BI^2 + 1\right) + C,$$

where A, B and C are constants to be determined; specifically, B is the same constant in the denominator of the state-dependent probability $p(I) = \frac{a}{BI^2+1}$. For such a quadratic guess the Bellman equation becomes:

$$\begin{aligned}
V(I,z) = Az^2 \left(BI^2 + 1\right) + C &= \min_{0\leq y\leq \Omega zI+z} \left\{\gamma_1 z^2 I^2 + \gamma_2 z^2 (1-I)^2 + (\Omega zI + z - y)^2 \right. \\
&\quad \left. + \frac{\beta a}{By^2+1} \left[Ar_1^2 \left(By^2+1\right) - Ar_2^2 \left(By^2+1\right)\right] + \beta A r_2^2 \left(By^2+1\right) + \beta C\right\} \\
&= \min_{0\leq y\leq \Omega zI+z} \left[\gamma_1 z^2 I^2 + \gamma_2 z^2 (1-I)^2 + (\Omega zI + z - y)^2 + \beta A B r_2^2 y^2 \right. \\
&\quad \left. + \beta a A \left(r_1^2 - r_2^2\right) + \beta A r_2^2 + \beta C\right].
\end{aligned} \tag{16}$$

The following result characterizes the closed-form solution for the Bellman equation (16) under some conditions on the model's parameters.

Proposition 1 *Assume that* $0 < \beta < 1$, $0 < a \leq 1$, $\gamma_2 > 0$, $\Omega > 0$ *and the shocks' values satisfy the feasibility condition (11),* $0 < r_1 < r_2 \leq \frac{1}{\Omega+1}$. *If, moreover,* $\gamma_2 < \Omega$ *and parameter* γ_1 *is given by*

$$\gamma_1 = \left[\frac{1}{\beta\left(\Omega - \gamma_2\right) r_2^2} - (\Omega + 1)\right] \gamma_2, \tag{17}$$

then, the solution of the Bellman equation (16) is the function:

$$V(I, z) = Az^2\left(BI^2 + 1\right) + C$$

where:

$$A = \frac{\Omega + 1}{\Omega}\gamma_2, \tag{18}$$

$$B = \frac{\gamma_1 + (\Omega + 1)\gamma_2}{(\Omega + 1)\gamma_2}\Omega, \tag{19}$$

$$C = \frac{\beta(\Omega + 1)\left[ar_1^2 + (1 - a) r_2^2\right]\gamma_2}{(1 - \beta)\Omega}. \tag{20}$$

The optimal policy for the number of infectives is affine in I_t^* *and has the following form:*

$$I_{t+1}^* = h\left(I_t^*, z_t\right) = \left(\Omega - \gamma_2\right) z_t I_t^* + \frac{\Omega - \gamma_2}{\Omega} z_t, \tag{21}$$

while the corresponding optimal policy parameter is given by:

$$X_t^* = \gamma_2 z_t I_t^* + \frac{\gamma_2}{\Omega I_t^*} z_t = \gamma_2 \left(I_t^* + \frac{1}{\Omega}\right) z_t. \tag{22}$$

The proof is in Appendix A, where it is shown that the expression of γ_1 in (28) is strictly positive. Clearly, as the constants A and B in (18) and (19) are strictly positive, the RHS of the Bellman equation (16) is strictly convex in y, so that the solution characterized in Proposition 1, including the optimal policy (21), is unique. As $\gamma_2 < \Omega$, clearly the optimal policy in (21) satisfies $0 < I_{t+1}^* < \Omega z_t I_t^* + z_t \leq 1$ for all $t \geq 0$; similarly, the optimal policy parameter in (22) satisfies $0 < X_{t+1}^* < \Omega z_t I_t^* + z_t \leq 1$ for all $t \geq 0$.

The affine optimal policy in (21) can be rewritten in terms of the following IFSSDP:

$$I_{t+1} = \begin{cases} r_1\left(\Omega - \gamma_2\right) I_t + \dfrac{\Omega - \gamma_2}{\Omega} r_1 & \text{with probability } p\left(I_t\right) = \dfrac{a}{BI_t^2 + 1} \\ r_2\left(\Omega - \gamma_2\right) I_t + \dfrac{\Omega - \gamma_2}{\Omega} r_2 & \text{with probability } 1 - p\left(I_t\right) = 1 - \dfrac{a}{BI_t^2 + 1}, \end{cases} \tag{23}$$

where the constant B corresponds to the value in (19). Similar to what discussed in Section 3, it is possible to prove the existence of a unique stationary distribution μ for such an IFSSDP supported on the interval $[I_1^{st}, I_2^{st}] \subset [0, 1]$, where the endpoints are the steady states of the two affine maps in (23) respectively:

$$I_1^{st} = \frac{\left(\Omega - \gamma_2\right) r_1}{\Omega\left[1 - r_1\left(\Omega - \gamma_2\right)\right]} \qquad \text{and} \qquad I_2^{st} = \frac{\left(\Omega - \gamma_2\right) r_2}{\Omega\left[1 - r_2\left(\Omega - \gamma_2\right)\right]}. \tag{24}$$

It is straightforward to show that the steady states of the maps above are strictly lower than those in the absence of policy intervention—see (10). This suggests that containment policy is effective as it moves leftward the support of the invariant distribution, meaning that the steady

state disease prevalence will tend to be characterized by lower values than what we would observe without any containment effort. Moreover, as both expressions in (24) are increasing in Ω and decreasing in γ_2, the smaller Ω and/or the larger γ_2, the larger the leftward shift of the support.

We now present a numerical example to clarify how optimal behavior by a social planner may affect the characteristics of the invariant distribution with respect to that approximated in Figure 2(c) of Section 3 for the same epidemiological parameter values as in (12), and setting the remaining economic parameters as follows:

$$\beta = 0.96, \qquad \gamma_2 = 0.25, \tag{25}$$

which from expressions (17), (18), (19) and (20) in Proposition 1 imply that

$$\gamma_1 = 0.589, \qquad A = 0.375, \qquad B = 3.571, \qquad C = 0.25.$$

Note that the value of the parameter B is exactly the same we have used in Section 3 for the first case characterized by $p' < 0$, i.e., when $p(I_t)$ and $1 - p(I_t)$ are defined according to (7); this allows us to compare the steady state outcome arising in the same setting with and without mitigation policy.

According to (21) the optimal policy is represented by the following IFSSDP:

$$I_{t+1} = \begin{cases} 0.292 I_t + 0.146 & \text{with probability } p(I_t) = \dfrac{1}{3.571 I_t^2 + 1} \\ 0.583 I_t + 0.292 & \text{with probability } 1 - p(I_t) = 1 - \dfrac{1}{3.571 I_t^2 + 1}, \end{cases} \tag{26}$$

which has $[I_1^{st}, I_2^{st}] = [0.206, 0.7]$ as trapping interval. As the fixed point of the lower map, $I_1^{st} = 0.206$, is bounded away from 0, clearly both $p(I_t)$ and $1 - p(I_t)$ are such that $0 < p(I_t) < 1$ and $0 < 1 - p(I_t) < 1$ for all $I \in [0.206, 0.7]$. Figure 4 shows the initial uniform density $\mu_0(I) \equiv \frac{1}{I_2^{st} - I_1^{st}}$ and the 1^{st} and 6^{th} iterations of our Maple algorithm for the IFSSDP (26), where the last plot can be considered as a good approximation of the invariant measure in this case. Note that, as $0.292 I_2^{st} + 0.146 = 0.35 < 0.412 = 0.583 I_1^{st} + 0.292$, the images of the two affine maps in (26) do not overlap, so that the invariant measure is singular as it is supported on a Cantor-like set.

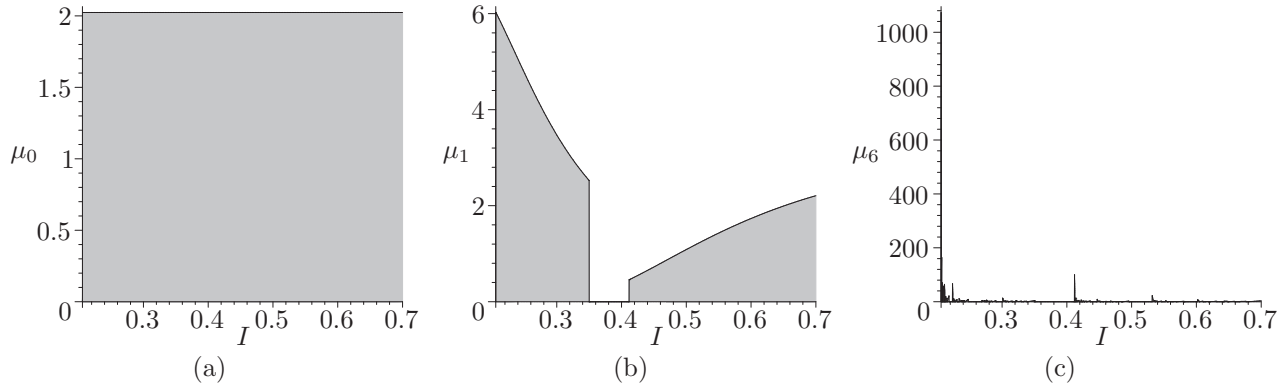

FIGURE 4: a) initial uniform density over $[0.206, 0.7]$, b) 1^{st} and c) 6^{th} iterations of our Algorithm to approximate the Markov operator (2) associated to the IFSSDP (26).

By comparing Figure 2 (top panels) with Figure 4, exactly as we have discussed before, we can observe that the support of the invariant probability measure in the latter is characterized

by lower extremes than those in the absence of containment policy of the former. Moreover, we can see that the effects of the optimal mitigation policy consist of concentrating the value of disease prevalence more closely toward to the lower extreme of the support, $I_1^{st} = 0.206$, as it becomes apparent by comparing directly Figures 2(c) and 4(c); in fact, the latter plot exhibits a much higher spike close to I_1^{st} than that in the former figure. Thus, containment policy not only reduces on average the possible steady state values of disease prevalence, but it also increases the likelihood that prevalence will be associated with its possible lowest values.

Note that the Bellman equation (16) is specifically defined for the $p' < 0$ case, therefore Proposition 1 cannot be applied for the $p' > 0$ case when $B = 3.571$. That is, we have no comparison between the optimal dynamics determined by containment policies and the dynamics described in Figure 3 (top panels). To compare the dynamics with and without containment policies when $p' > 0$ we must resort to the result in the next Proposition 2 specifically designed for the increasing $p(I)$ and $B = 14.286$ case.

5.2 The $p' > 0$ Case

By assuming that the probabilities are given by (8) the Bellman equation becomes:

$$V(I,z) = \min_{0\leq y\leq \Omega zI+z} \left\{ \gamma_1 z^2 I^2 + \gamma_2 z^2 (1-I)^2 + (\Omega zI + z - y)^2 \right.$$
$$\left. + \beta V(y, r_1) + \frac{\beta a}{By^2+1} \left[V(y, r_2) - V(y, r_1) \right] \right\}.$$

We guess the same form for the value function in the Bellman equation as before: $V(I,z) = Az^2(BI^2+1) + C$, where A, B and C are constants to be determined; specifically, B is the same constant in the denominator of the state-dependent probability $p(I) = 1 - \frac{a}{BI^2+1}$. For such a quadratic guess the Bellman equation becomes:

$$V(I,z) = Az^2\left(BI^2+1\right) + C = \min_{0\leq y\leq \Omega zI+z} \left[\gamma_1 z^2 I^2 + \gamma_2 z^2 (1-I)^2 + (\Omega zI + z - y)^2 \right. \quad (27)$$
$$\left. + \beta AB r_1^2 y^2 + \beta A r_1^2 + \beta C + \beta a A \left(r_2^2 - r_1^2\right) \right].$$

The following result is very similar to Proposition 1 and characterizes the closed-form solution for the Bellman equation (27) under the same conditions on the parameters of the model, except for γ_1. It yields the same results as in Proposition 1 except for the values of the constants B and C.

Proposition 2 *Assume that $0 < \beta < 1$, $0 < a < 1$, $\gamma_2 > 0$, $\Omega > 0$ and the shocks' values satisfy the feasibility condition (11), $0 < r_1 < r_2 \leq \frac{1}{\Omega+1}$. If, moreover, $\gamma_2 < \Omega$ and parameter γ_1 is given by*

$$\gamma_1 = \left[\frac{1}{\beta(\Omega - \gamma_2) r_1^2} - (\Omega+1) \right] \gamma_2, \quad (28)$$

then, the solution of the Bellman equation (27) is the function:

$$V(I,z) = Az^2\left(BI^2+1\right) + C$$

where:

$$A = \frac{\Omega+1}{\Omega}\gamma_2, \quad (29)$$

$$B = \frac{\gamma_1 + (\Omega+1)\gamma_2}{(\Omega+1)\gamma_2}\Omega, \quad (30)$$

$$C = \frac{\beta(\Omega+1)\left[(1-a)r_1^2 + a r_2^2\right]\gamma_2}{(1-\beta)\Omega}. \quad (31)$$

The optimal policy for the number of infectives is affine in I_t^* and has the following form:

$$I_{t+1}^* = h\left(I_t^*, z_t\right) = \left(\Omega - \gamma_2\right) z_t I_t^* + \frac{\Omega - \gamma_2}{\Omega} z_t, \tag{32}$$

while the corresponding optimal policy parameter is given by:

$$X_t^* = \gamma_2 z_t I_t^* + \frac{\gamma_2}{\Omega I_t^*} z_t = \gamma_2 \left(I_t^* + \frac{1}{\Omega}\right) z_t. \tag{33}$$

The proof is in Appendix A and the same comments after Proposition 1 apply also to Proposition 2. Thus, it turns out then that the affine optimal policy in (32) is the same as that of Proposition 1 in the previous subsection, when state-dependent probabilities are given by (8) instead of (7):

$$I_{t+1} = \begin{cases} r_1\left(\Omega - \gamma_2\right) I_t + \dfrac{\Omega - \gamma_2}{\Omega} r_1 & \text{with probability } p\left(I_t\right) = 1 - \dfrac{a}{BI_t^2 + 1} \\ r_2\left(\Omega - \gamma_2\right) I_t + \dfrac{\Omega - \gamma_2}{\Omega} r_2 & \text{with probability } 1 - p\left(I_t\right) = \dfrac{a}{BI_t^2 + 1}, \end{cases} \tag{34}$$

where the constant B corresponds to the new value in (30). Also in this case it is possible to prove the existence of a unique stationary distribution μ for such an IFSSDP supported on the interval $[I_1^{st}, I_2^{st}] \subset [0, 1]$, where the endpoints are the steady states of the two affine maps in (34) respectively:

$$I_1^{st} = \frac{\left(\Omega - \gamma_2\right) r_1}{\Omega\left[1 - r_1\left(\Omega - \gamma_2\right)\right]} \qquad \text{and} \qquad I_2^{st} = \frac{\left(\Omega - \gamma_2\right) r_2}{\Omega\left[1 - r_2\left(\Omega - \gamma_2\right)\right]}. \tag{35}$$

Since the steady state of the maps above perfectly coincide with those earlier found in the $p' < 0$ case, exactly the same comments apply: containment policy is effective as it results in a leftward shift of the support of the invariant distribution, meaning that steady state disease prevalence will be characterized on average by lower values than in the absence of policy intervention. Moreover, the smaller Ω and/or the larger γ_2, the larger the leftward shift of the support.

We continue to illustrate numerically how optimal behavior by a social planner may affect the characteristics of the invariant distribution with respect to that approximated in Figure 3(f) of Section 3 for the same epidemiological parameter' values as in (12) and (25). Expressions (28), (29), (30) and (31) in Proposition 2 imply that:

$$\gamma_1 = 4.607, \qquad A = 0.375, \qquad B = 14.286, \qquad C = 1.$$

Note that also in this case the value of the parameter B is exactly the same of that used in Section 3 for the second case characterized by $p' > 0$; this allows us to compare the steady state outcome arising in the same setting with and without mitigation policy. Note that now the constant B has a much larger value than in the $p' < 0$ case, itself triggered by a much larger value of γ_1 provided by (28); this feature translates into much steeper state-dependent probabilities. According to (32) the optimal policy is represented by the following IFSSDP:

$$I_{t+1} = \begin{cases} 0.292 I_t + 0.146 & \text{with probability } p\left(I_t\right) = 1 - \dfrac{1}{14.286 I_t^2 + 1} \\ 0.583 I_t + 0.292 & \text{with probability } 1 - p\left(I_t\right) = \dfrac{1}{14.286 I_t^2 + 1}, \end{cases} \tag{36}$$

which has the same interval, $[I_1^{st}, I_2^{st}] = [0.206, 0.7]$, as the IFSSDP (26) as trapping interval. Figure 5, as usual, shows the initial uniform density $\mu_0(I) \equiv \frac{1}{I_2^{st} - I_1^{st}}$ and the 1^{st} and 6^{th} iterations of our Maple algorithm for the IFSSDP (36). As the affine maps are the same as in (26), again their images do not overlap and the invariant measure is singular as it is supported on a Cantor-like set.

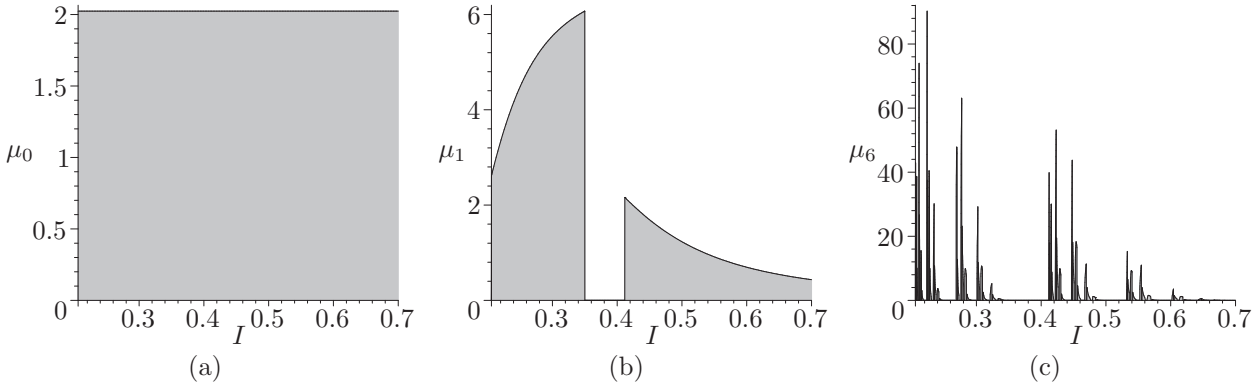

FIGURE 5: a) initial uniform density over $[0.206, 0.7]$, b) 1^{st} and c) 6^{th} iterations of our Algorithm to approximate the Markov operator (2) associated to the IFSSDP (36).

By comparing Figure 3 (bottom panels) with Figure 5, exactly as in the $p' < 0$ case, we can observe that the support of the distribution is characterized by lower extremes than in the absence of containment policy, and that the optimal mitigation policy results in concentrating the value of disease prevalence more closely toward to the lower extreme of the support, as it becomes apparent by comparing directly Figures 3(f) and 5(c); in fact, the latter plot exhibits higher spikes close to I_1^{st} than those in the former figure, although in a less pronounced fashion than in our earlier comparison between Figures 2(c) and 4(c). Thus, once more, containment policy not only reduces on average the possible steady state values of disease prevalence, but it also increases the likelihood that prevalence will be associated with its possible lowest values.

Also in this case a comparison between the dynamics under optimal containment policies for the $p' < 0$ case when $B = 14.286$ and the dynamics without policies described by Figure 3 is not available, as the Bellman equation (27) is specifically defined for the $p' > 0$ case, and thus also Proposition 2 can be applied only for this case.

6 Singularity versus Absolute Continuity

One important question, especially from a policy perspective, regarding the nature of the invariant distribution ν is related to its properties in terms of absolutely continuity or singularity. Specifically, if it is absolutely continuous then it will be represented by a density and so it could be estimated in terms of a few parameters, while if it is singular then there will be no convenient way to represent it and we will have to list the value of the function for every point in its domain. Clearly, absolutely continuous measures are easier to work with and more well-behaved than singular measures as they allow for a more precise forecasting of future dynamics, and so it is valuable to know conditions under which the invariant measure may be absolutely continuous. There is also a long history of works in this area and it is still a very active area of theoretical research. The strongest results are when the contraction factors are all the same (so-called *equi-contractive IFS*). Our situation with unequal scaling factors is more delicate and so we are

only able to give an incomplete characterization. In the constant probability case, recent work in Saglietti et. al (2018) shows that for each fixed choice of probability the invariant measure is absolutely continuous for almost every (α, β) in the so-called "super-critical region". Our situation with state-dependent probabilities is quite a bit more intricate so our result is less comprehensive. The description of the region Θ is complicated and can be found in Ngai and Wang (2005).

Theorem 5 *Take the two-map IFS on $\mathcal{R}$ given by $\{\alpha x + \tau_1, \beta x + \tau_2\}$, with $\alpha, \beta \in [0, 1)$ along with the two probability functions $p_1(x) = p(x)$ and $p_2(x) = 1 - p(x)$. Assume that $\delta < p(x) < 1 - \delta$ for all x and some $\delta > 0$ and also that p is Hölder continuous. Let $\mu_{\alpha,\beta}$ be the invariant measure of this state-dependent IFS.*

1. *If $0 \leq \alpha + \beta < 1$ then $\mu_{\alpha,\beta}$ is singular with respect to Lebesgue measure.*
2. *If $\alpha + \beta = 1$ then $\mu_{\alpha,\beta}$ is either singular with respect to Lebesgue measure or is equal to the (normalized) Lebesgue measure on the closed interval with endpoints $\frac{\tau_1}{1-\alpha}$ and $\frac{\tau_2}{1-\beta}$ and $p(x) = \alpha$.*
3. *For each $\alpha + \beta > 1$, let $h_{\alpha,\beta}$ be defined by*

$$h_{\alpha,\beta} = -\int \{p(x)\ln[p(x)] + [1 - p(x)]\ln[1 - p(x)]\} d\mu_{\alpha,\beta}(x)$$

and

$$\chi_{\alpha,\beta} = -\log(\beta) + [\log(\beta) - \log(\alpha)] \int p(x)\ d\mu_{\alpha,\beta}(x).$$

Then $\mu_{\alpha,\beta}$ is singular for every α, β with $h_{\alpha,\beta} < \chi_{\alpha,\beta}$.

Furthermore, there is an open subset $\Theta \subset \{(\alpha, \beta) \in (0, 1)^2 : \alpha + \beta > 1\}$ so that $\mu_{\alpha,\beta}$ is absolutely continuous with respect to Lebesgue measure for Lebesgue almost every $(\alpha, \beta) \in \Theta$ such that $h_{\alpha,\beta} > \chi_{\alpha,\beta}$.

Theorem 5 states that the singularity vs absolute continuity properties of the invariant measure depend ultimately on the contraction factors, which in our IFSSDPs is given by the $\Omega - \gamma_2$, and thus it depends on the relative magnitude of the net infectivity rate and the weight attached to potential infections in the objective function. While it is straightforward to check whether one of the first two cases of the theorem applies, the third case is quite a bit more delicate and deserves some further clarification. In fact, the condition $h_{\alpha,\beta} > \chi_{\alpha,\beta}$ is generally difficult to check since it involves integrals with respect to μ. Moreover, unfortunately for any specific choice of parameters it is not a simple task to determine if μ is absolutely continuous even if this condition holds. All we would know is that μ is absolutely continuous for almost all choices of the parameters in some open subset. Even in the case of equal contraction factors it would be difficult to know if a specific choice of parameters results in an absolutely continuous invariant measure. We would do know, however, that the invariant distribution is a (weakly) continuous function of the parameters.

Returning to our epidemiological framework, all the IFSSDPs that have analyzed, both in the case of presence and absence of optimal disease containment policies, fit into the scheme of Theorem 5 since the IFS maps are all one-dimensional and affine and the probabilities are smooth functions. The IFSSDPs with optimal policy given in (26) and (36) are both in the first case of the theorem (where $\alpha + \beta < 1$) and thus their invariant measures are singular, and this is true no matter the form of the probability function $p(x)$. The IFSSDP without mitigation

policy given in (13) is in the second case where $\alpha + \beta = 1$. However, since the probability functions are not constant (and equal in value to the corresponding contraction ratios), the invariant measure is singular also in this case. While none of our specific parametrizations have resulted in a IFSSDP fitting the third case where $1 < \alpha + \beta < 2$, this could be true in principle for any of our IFSSDPs, both without optimal policy – given in (9) – and with optimal policy – given in (23) and (34) for the scenario $p' < 0$ and $p' > 0$, respectively.

7 Conclusion

The ongoing COVID-19 pandemic has brought to light the need to understand the working mechanisms of disease containment policies in order to effectively save human lives and preserve economic conditions. A huge number of works in literature has analyzed from different points of view how the optimal policy should be determined in deterministic settings, but very few have been the attempt to relate containment policies and stochastic epidemiological dynamics. In this context all the works have assumed that the probability with which shocks affect epidemic dynamics are constant and thus unrelated to disease prevalence. In this paper we contribute to this literature by analyzing the implications of state-dependent probabilities, that is probabilities depending on disease prevalence, for optimal policymaking. We have developed a stylized economic-epidemiological stochastic framework in which random shocks determine the diffusion of a new strain of the disease and the social planner needs to choose the intensity of treatment in order to minimize the social cost of the epidemic management program, accounting for the state-dependency of probabilities. Our results show that in the stochastic steady state complete eradication is never a possible long run outcome where instead disease will always be endemic. Moreover, independently of the features of the state-dependent probabilities, treatment allows to shift leftward the support of the invariant measure, reducing the possible endemic prevalence levels associated with the steady state outcome. However, the features of the state-dependent probabilities are not irrelevant as they affect the shape and spread of disease prevalence over its support, allowing for a steady state outcome characterized by a distribution either highly concentrated over low prevalence levels or more spread out over a larger range of prevalence (possibly higher) levels. Moreover, we characterize the properties of the invariant self-similar measure in terms of singularity and absolutely continuity with respect to the Lebesgue measure, showing that this is ultimately related to the magnitude of the relative magnitude of the net infectivity rate and the weight attached to potential infections in the objective function.

To the best of our knowledge, ours is the first attempt to introduce state-dependent probabilities in the analysis of the optimal policy in economic-epidemiological frameworks. Therefore, in order to allow for the analytical tractability needed to clarify the main arguments underlying our analysis we have relied on simplifying assumptions limiting the nature of our conclusions. In particular, the abstraction of the epidemic dynamics from the social interactions between infectives and susceptibles has brought us to depart substantially from standard epidemiological models making the comparison of our results with those traditionally discussed in literature particularly complicated. Moreover, we have focused on containment policies taking the form of treatment without exploring how results may change under different types of policies, such as preventive or social distancing measures. Extending our analysis along these directions is currently a priority in our research agenda.

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A Proofs of Propositions 1 and 2

As the RHS in (16) is strictly convex in y whenever the values of constraints A and B are positive, to guarantee interiority of the minimum value of y we check the sign of the the derivative with respect to y is negative on the left endpoint of the constraint $0 \leq y \leq \Omega z I + z$ and is positive on its right endpoint; in fact, this is the case provided that $AB > 0$:

$$\frac{\partial}{\partial y}\left(RHS\right) = -2\left(\Omega z I + z - y\right) + 2\beta AB r_2^2 y = \begin{cases} -2\left(\Omega z I + z\right) < 0 & \text{if } y = 0 \\ 2\beta AB r_2^2\left(\Omega z I + z\right) > 0 & \text{if } y = \Omega z I + z. \end{cases}$$

The FOC with respect to y yields the unique solution

$$y^* = \phi z\left(\Omega I + 1\right), \qquad \text{with } \phi = \frac{1}{1 + \beta AB r_2^2}. \tag{37}$$

Substituting y^* as in (37) into the RHS of (16) after some algebra yields

$$\begin{aligned} V\left(I, z\right) &= Az^2\left(BI^2 + 1\right) + C = ABz^2I^2 + Az^2 + C \\ &= \gamma_1 z^2 I^2 + \gamma_2 z^2\left(1 - I\right)^2 + \left[\Omega z I + z - \phi z\left(\Omega I + 1\right)\right]^2 + \beta AB r_2^2 \phi^2 z^2\left(\Omega I + 1\right)^2 \\ &\quad + \beta A\left[a r_1^2 + \left(1 - a\right) r_2^2\right] + \beta C \\ &= \left(\gamma_1 + \gamma_2 + \Psi\Omega^2\right) z^2 I^2 + 2\left(\Psi\Omega - \gamma_2\right) z^2 I + \left(\gamma_2 + \Psi\right) z^2 + \beta A\left[a r_1^2 + \left(1 - a\right) r_2^2\right] + \beta C, \end{aligned}$$

where in the fourth equality we have set $\Psi = \left(1 - \phi\right)^2 + \beta AB r_2^2 \phi^2$.

By equating all similar terms in both sides and setting the coefficient of $z^2 I$ equal to 0 we find that a solution of the Bellman equation (16) is given by the constants A, B and C that satisfy

$$\begin{cases} AB = \gamma_1 + \gamma_2 + \Psi\Omega^2 \\ \Psi\Omega - \gamma_2 = 0 \\ A = \gamma_2 + \Psi \\ C = \beta A\left[a r_1^2 + \left(1 - a\right) r_2^2\right] + \beta C. \end{cases}$$

From the second equation we get $\Psi = \frac{\gamma_2}{\Omega}$, so that, after substituting this in the third equation, we easily find the value of A as in (18), while, after substituting both values of Ψ and A into the first equation, one has

$$AB = \frac{\Omega + 1}{\Omega}\gamma_2 B = \gamma_1 + \gamma_2 + \gamma_2\Omega = \gamma_1 + \left(\Omega + 1\right)\gamma_2, \tag{38}$$

which implies that

$$B = \frac{\Omega\gamma_1}{\left(\Omega + 1\right)\gamma_2} + \Omega,$$

which is equivalent to the expression in (19). Finally, after substituting the value of A as in (18) into the fourth equation one immediately gets the value of C as in (20).

Recalling that $\Psi = (1-\phi)^2 + \beta ABr_2^2\phi^2$ and, from (37), $\phi = \frac{1}{1+\beta ABr_2^2}$, the second equation implies that γ_2 is related to all other parameters according to

$$\gamma_2 = \Psi\Omega = \left[(1-\phi)^2 + \beta ABr_2^2\phi^2\right]\Omega = \left[\left(1 - \frac{1}{1+\beta ABr_2^2}\right)^2 + \frac{\beta ABr_2^2}{\left(1+\beta ABr_2^2\right)^2}\right]\Omega$$
$$= \frac{\beta\left[\gamma_1 + (\Omega+1)\gamma_2\right]r_2^2}{1+\beta\left[\gamma_1 + (\Omega+1)\gamma_2\right]r_2^2}\Omega, \tag{39}$$

where in the last equality we used (38). Note that the last expression requires that $0 < \gamma_2 < \Omega$ must hold.

By further algebraic manipulation, from (39) we easily get the value of γ_1 as in (17):

$$\gamma_2 = \frac{\beta\left[\gamma_1 + (\Omega+1)\gamma_2\right]r_2^2}{1+\beta\left[\gamma_1 + (\Omega+1)\gamma_2\right]r_2^2}\Omega \quad \Longleftrightarrow \quad \gamma_1 = \left[\frac{1}{\beta(\Omega-\gamma_2)r_2^2} - (\Omega+1)\right]\gamma_2.$$

Note that $\gamma_1 > 0$ because the term in square brackets of the last expression is always strictly positive; in fact, condition (11) implies that $\frac{1}{r_2^2} \geq (\Omega+1)^2$, so that

$$\frac{1}{\beta(\Omega-\gamma_2)r_1^2} - (\Omega+1) \geq \frac{(\Omega+1)^2}{\beta(\Omega-\gamma_2)} - (\Omega+1) = \left[\frac{\Omega+1}{\beta(\Omega-\gamma_2)} - 1\right](\Omega+1),$$

and the last expression is strictly positive because, recalling that $(\Omega-\gamma_2) > 0$,

$$\frac{\Omega+1}{\beta(\Omega-\gamma_2)} > 1 \quad \Longleftrightarrow \quad (1-\beta)\Omega + 1 + \beta\gamma_2 > 0.$$

By rewriting (39) as

$$\gamma_2 = \frac{\beta\left[\gamma_1 + (\Omega+1)\gamma_2\right]r_2^2}{1+\beta\left[\gamma_1 + (\Omega+1)\gamma_2\right]r_2^2}\Omega \quad \Longleftrightarrow \quad r_2^2 = \frac{\gamma_2}{\beta\left[\gamma_1 + (\Omega+1)\gamma_2\right](\Omega-\gamma_2)},$$

and replacing the value r_2^2 just obtained into (37) together with the value of the product AB as in (38) easily yields the optimal policy as in (21):

$$y^* = h(I,z) = \phi z(\Omega I+1) = \frac{z(\Omega I+1)}{1+\beta ABr_2^2} = \frac{z(\Omega I+1)}{1 + \dfrac{\beta\left[\gamma_1 + (\Omega+1)\gamma_2\right]\gamma_2}{\beta\left[\gamma_1 + (\Omega+1)\gamma_2\right](\Omega-\gamma_2)}}$$
$$= \frac{\Omega-\gamma_2}{\Omega}z(\Omega I+1).$$

Finally, as for each $z \in \{r_1, r_2\}$, the value function $V(\cdot, z)$ is defined over the compact interval $[0,1]$ and continuous, $V(I,z) = ABz^2I^2 + Az^2 + C$ is bounded over $[0,1]$; this is enough to apply the standard verification principle and establishes that, in fact, $V(I,z)$ is the value function.

The proof of Proposition 2 is very similar to that of Proposition 1 just described; the key differences lie in the terms $\beta ABr_2^2 y$ and $\beta A\left[ar_1^2 + (1-a)r_2^2\right]$ appearing in the Bellman equation (16) that become $\beta ABr_1^2 y$ and $\beta A\left[(1-a)r_1^2 + ar_2^2\right]$ respectively in the Bellman equation (27); we omit the details.

B Proof of Theorem 5

First we reduce to the case where the IFS is $\{\alpha x, \beta x + 1 - \beta\}$ acting on $[0,1]$. This is possible because for any values of $\alpha, \beta, \tau_1, \tau_2$, the closed interval with endpoints $\frac{\tau_1}{1-\alpha}$ and $\frac{\tau_2}{1-\beta}$ is invariant under the IFS and thus contains the support of $\mu_{\alpha,\beta}$. A simple affine change of variables then gives the IFS $\{\alpha x, \beta x + 1 - \beta\}$ on $[0,1]$.

1) The first conclusion is clear since whenever $\alpha + \beta < 1$ the measure $\mu_{\alpha,\beta}$ is supported on a Cantor set with zero Lebesgue measure.

3) By the results in Ngai and Wang (2005) the IFS $\{\alpha x, \beta x + 1 - \beta\}$ satisfies the transversality condition for all $(\alpha, \beta) \in \Omega$ and then the conclusion follows by Theorem 1.1 in Bárány (2015).

2) Since we have α, β fixed, we use μ rather than $\mu_{\alpha,\beta}$ to avoid extraneous clutter on our notation. With no loss of generality we assume that $0 < \alpha \leq \beta = 1 - \alpha < 1$.

Recall that the "Markov operator" is given by

$$M\nu(S) = \sum_i \int_{w_i^{-1}(S)} p_i(x)\, d\nu(x) = \sum_i \int_S p_i\left(w_i^{-1}(x)\right) d\nu\left(w_i^{-1}(x)\right)$$

and that μ satisfies $\mu(S) = (M\mu)(S)$. Suppose that μ is absolutely continuous with density function $f(x)$. Then we obtain the equation

$$\begin{aligned}\int_S f(x)\, dx &= \sum_i \int_{w^{-1}(S)} p_i(y)\, f(y)\, dy \\ &= \int_S \frac{1}{\alpha} p_1(x/\alpha) f(x/\alpha) \chi_{[0,\alpha]}(x) + \frac{1}{\beta} p_2(\frac{x-1+\beta}{\beta}) f(\frac{x-1+\beta}{\beta}) \chi_{[1-\beta,1]}(x)\ dx,\end{aligned}$$

where $\chi_A(x)$ is the characteristic function of the set A. For this to be true for all Borel sets S we must have that, for almost every x, the two equations

$$f(x) = \frac{1}{\alpha} p_1(x/\alpha) f(x/\alpha), \quad 0 \leq x \leq \alpha;$$

and

$$f(x) = \frac{1}{\beta} p_2(\frac{x-1+\beta}{\beta}) f(\frac{x-1+\beta}{\beta}), \quad \alpha = 1 - \beta \leq x \leq 1.$$

Doing a simple change of variable these become for $0 \leq y \leq 1$

$$f(\alpha y) = \frac{1}{\alpha} p_1(y) f(y) \quad \Longrightarrow \quad p_1(y) = \frac{\alpha f(\alpha y)}{f(y)} \tag{40}$$

and

$$f(\beta y + 1 - \beta) = \frac{1}{\beta} p_2(y) f(y) \quad \Longrightarrow \quad p_2(y) = \frac{\beta f(\beta y - 1 + \beta)}{f(y)}. \tag{41}$$

Then the condition that $p_1(y) + p_2(y) = 1$ implies that

$$f(y) = \alpha f(\alpha y) + \beta f(\beta + 1 - \beta) \tag{42}$$

for Lebesgue almost every $y \in [0,1]$. Thus $f(x)$ is the fixed point of the operator

$$T(g)(x) = \alpha g(\alpha y) + \beta g(\beta y + 1 - y).$$

We show that the only fixed point of T which is a density function is the constant function $g(x) = 1$. It is easy to see that Tg is a density if g is a density. Suppose that $g \in C^1[0,1]$. Then $(Tg)'(x) = \alpha^2 g'(\alpha y) + \beta^2 g'(\beta y + 1 - \beta)$ and so (since $t^2 + (1-t)^2 \leq \max(t, 1-t)$ for $0 \leq t \leq 1$) we have $\|(Tg)'\|_\infty \leq \beta \|g'\|_\infty$. By induction this means that

$$\left\|(T^n g)'\right\|_\infty \leq \beta^n \left\|g'\right\|_\infty . \tag{43}$$

Next, suppose that we have a density function $g \in C^1[0,1]$ with $|g'(x)| \leq m$ for all $x \in [0,1]$. Then for all $x \in [0,1]$ we have

$$g(0) - mx \leq g(x) \leq g(0) + mx$$

and thus, integrating over $[0,1]$, we have

$$g(0) - m/2 \leq 1 \leq g(0) + m/2 \Rightarrow |g(0) - 1| \leq m/2$$

and so

$$|g(x) - 1| \leq mx + m/2 \leq \frac{3}{2}m \Rightarrow \|g - 1\|_\infty \leq \frac{3}{2}m. \tag{44}$$

Next for two functions $f, g \in L^1[0,1]$, integrating the inequality

$$|T(f)(x) - T(g)(x)| \leq \alpha |f(\alpha x) - g(\alpha x)| + \beta |f(\beta x + 1 - \beta) - g(\beta x + 1 - \beta)|$$

over $[0,1]$ we get

$$\begin{aligned}
&\int_0^1 |T(f)(x) - T(g)(x)|\, dx \\
&\qquad \leq \alpha \int_0^1 |f(\alpha x) - g(\alpha x)|\, dx + \beta \int_0^1 |f(\beta x + 1 - \beta) - g(\beta x + 1 - \beta)|\, dx \\
&\qquad\qquad = \int_0^\alpha |f(u) - g(u)|\, du + \int_\alpha^1 |f(u) - g(u)|\, du = \int_0^1 |f(u) - g(u)|\, du,
\end{aligned}$$

and thus $\|T(f) - T(g)\|_1 \leq \|f - g\|_1$. Let f be a density function and let $\epsilon > 0$ be given. Then there is some density function $g \in C^1[0,1]$ so that $\|f - g\|_1 \leq \epsilon/2$. Then we have

$$\begin{aligned}
\|T^n(f) - 1\|_1 &\leq \|T^n(f) - T^n(g)\|_1 + \|T^n(g) - 1\|_1 \\
&\leq \|T^n(f) - T^n(g)\|_1 + \|T^n(g) - 1\|_\infty \\
&\leq \|f - g\|_1 + \frac{3}{2} \left\|(T^n g)'\right\|_\infty \\
&\leq \|f - g\|_1 + \frac{3\beta^n}{2} \|g'\|_\infty \leq \epsilon
\end{aligned}$$

for sufficiently large n. Thus $T^n f \to 1$ in $L^1[0,1]$ for any density f and so the only density function which satisfies (42) is $f(x) = 1$.

From (40) we get $p_1(x) = \alpha$ as claimed.

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