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Economic development with deadly communicable diseases and public prevention

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Abstract

Infectious diseases have been a major determinant of human mortality in history and the key regulator of population size, including the first epoch of the Industrial Revolution (until the 1950s) in Western countries and still now in developing countries, especially in Sub-Saharan Africa. In recent times, a new vein of economic research dealing with the interplay between communicable diseases and economic development has grown. However, pioneering previous research has analysed this issue in a framework where prevention decisions were the outcome of private individual rational choices. This assumption neither seems to hold for least-developed countries, primarily due to a lack of resources, nor for developed countries, where prevention policies are mostly planned by the public authority through its (public) health system, as also well documented by the current COVID-19 crisis. Our aim in this article is twofold. First, we pinpoint the properties of Chakraborty et al.'s (2010, 2016) basic epidemiological equation to fully enlighten its usability in economic-epidemiology modelling. Second, we apply this framework to analyse prevention activities against a range of infectious diseases by endogenous public (rather than

private) health expenditures. Our results identify the relationships governing the interplay between—on the one hand—typical epidemiological phenomena, namely invasion (i.e., the tendency of infection to establish in a population) versus endemicity (i.e., the tendency of infection to persist in the long term) and—on the other hand—economic variables, such as capital accumulation, GDP, and taxation. This is done by identifying threshold quantities, depending on both epidemiological and economic parameters, and by bifurcation analysis showing the effects that public intervention can have on previously uncontrolled infectious diseases. Both direct and indirect, that is, partial and general equilibrium, effects of control interventions are identified.

1 | INTRODUCTION

Infectious diseases have been a major determinant of human mortality in history and the key regulator of population size. In Western countries, communicable infections claimed an important death toll during a large part of the industrial revolution (Livi-Bacci, 2017), continuing until the introduction of mass vaccination in the 1950s. Still now, communicable diseases—ranging from major killers, such as malaria, tuberculosis (TB) and AIDS, to lower-trait respiratory infections and diarrheal diseases—represent the major component of mortality in least-developed countries, especially Sub-Saharan Africa (SSA) (e.g., Bloom & Canning, 2004; Lorentzen et al., 2008; Murray et al., 2017).

The ongoing COVID-19 pandemic is dramatically changing the perspectives on the economic effects of infectious diseases in the industrialised world and is opening up a growing literature (see, e.g., Acemoglu et al., 2020; Auray & Eyquem, 2020; Alvarez et al., 2021; Eichenbaum et al., 2020; Goenka et al., 2021; Gollier, 2020; Gori, Manfredi, Marsiglio, & Sodini, 2021), which was previously confined in a niche largely focused on specific topics, such as the effects of deadly infections as HIV/AIDS and malaria in SSA, and their effects on economic development, or the effects of vaccine refusal in relation to vaccine preventable infections.

In relation to the key topic of the impact of communicable infections on development, a seminal effort has been done by Chakraborty et al. (2010, 2016), who were first in setting an explicit, parsimonious representation of the dynamics of infection prevalence (i.e., the proportion of infective individuals at any time in the population) within a finite lived overlapping generations (OLG) growth model. They built on a standard Diamond-like OLG set-up, where rational (two-period lived) individuals choose their private health prevention investments. Though their representation is a stylised one, using a simplistic time frame of infection dynamics, which is taken identical to the OLG time (and therefore appropriate only for infections spreading over long scales of time, as is the case of the HIV/AIDS epidemic or has been the case of TB spread in history), it nonetheless represents a very useful tool for qualitative interpretations of the interplay between economic development and infectious diseases.

The present work departs from Chakraborty et al. (2010, 2016), but replaces their main assumption of individual rational behaviour applied to (private) infection prevention with the alternative hypothesis that infection prevention is primarily conducted by the public authority. Indeed, in modern industrialised countries, interventions against infectious diseases were mostly set within public prevention programmes through national public health systems. This has a rationale, namely the inefficiency of markets for private prevention (Stiglitz, 1988). The most important instance is that of vaccine-preventable infectious diseases and related public vaccination programmes targeted to childhood (WHO, 2019). However, the span of public intervention against infectious diseases goes beyond vaccine-preventable infections: for example, interventions against chronic costly sequelae of infectious diseases such as cancer induced by hepatitis (B or C) or by human papillomas (HPV) or even the highly active antiretroviral therapies against HIV could never be afforded by the single individual and therefore require an underlying public health effort. More than this, the hypothesis of rational individual preventive behaviour is hardly tenable for least-developed countries, especially SSA, where interventions against infections are heavily supported by international donors within ad hoc public policies (Katz et al., 2014). Not to say about the current COVID-19 epidemic, which has shown the extent of the lack of ability of the private sector to internalise the disease-related negative externalities.

From these viewpoints, we believe that the framework developed by Chakraborty and coauthors may adequately explain prevention for (i) settings where agents are endowed of both economic and education assets, and (ii) special infections, for example, those for which public programmes are not in place (e.g., mild or nonlife-threatening sexually transmitted diseases). A fortiori, their approach can hardly be used to describe infectious disease dynamics in low-resource settings, such as SSA, where prevailing conditions do not allow individuals to undertake health prevention actions and education investments, which represent a precondition for developing individual awareness about infection-related risks. Therefore, in this article we consider a simple OLG macroeconomic dynamic framework for developing economies, in which infectious diseases are controlled by endogenous public health expenditures.

Our aim is achieving a theoretical understanding of the interplay between epidemiological and economic variables in an economic growth framework explicitly incorporating the transmission dynamics of a "representative" (socially or sexually transmitted) infection, controlled by a public health system financed by general taxation. The taxation schedule includes an exogenous component and a prevalence-dependent component. In particular, we consider a widely circulating, deadly infection generating a substantial burden for the community both in terms of morbidity and economic costs. The infection-related mortality impacts on accumulation through the propensity to save.

Compared to previous works, we deepen the relationships governing the interplay between, on the one hand, typical epidemiological phenomena, namely invasion (i.e., the tendency of infection to establish in a population, initially in an epidemic form), versus endemicity (i.e., the tendency of an infection that initially invaded a population in an epidemic form to persist in the long term), and, on the other hand, main economic variables, such as capital accumulation, gross domestic product (GDP) and taxation.

First, on the issue of invasion, we identify precise threshold quantities, depending on both epidemiological and economic parameters. Second, we investigate the issue of the long-term conflict among infection, population and economic policies designed to control the disease, by systematically using bifurcation diagrams showing the interplay between epidemiological and policy parameters on the long-term equilibria of a developing economy with an endemic infection. Notably, these results extend to a more articulated modelling setting, namely a

macroeconomic dynamic general equilibrium model including an explicit dynamic for transmissible infections, the typical features of classical epidemiological models, namely the duality between an infection-free equilibrium and the presence of an endemic state mediated by suitable threshold parameters. To allow readers to fully grasp these aspects, we also provide a detailed discussion of the properties of the "epidemiological equation" proposed by Chakraborty et al.'s (2010, 2016).

The rest of the article is organised as follows. Section 2 reviews the economic epidemiology literature by setting it within the broader framework of public health interventions against communicable infections. Section 3 presents the model. Section 4 discusses some key properties of Chakraborty et al.'s epidemiological equation as a useful building block for general economic-epidemiology models. Sections 5 and 6 report the main static and dynamic results of our general model. Concluding remarks and suggestions for future studies follow in Section 7. The technical arguments are relegated in the appendix.

2 | ECONOMIC EPIDEMIOLOGY, ECONOMICS OF INFECTIOUS DISEASES AND PUBLIC HEALTH: A REVIEW OF THE PRE-COVID-19 ERA

Widespread individual health is a universal value and a necessary condition for the prosperity of a community and its development perspectives from a broader standpoint (Bloom et al., 2018). High mortality and generally the widespread lack of health in Africa, especially among SSA populations, have been often documented as key determinants of underdevelopment and poverty in that region (Bloom & Canning, 2004; Bloom et al., 2018; Lorentzen et al., 2008).

As a heritage of the Enlightenment («health as a civil right»), the idea of the public protection of health in contemporary Western countries has developed and spread around modern public health systems, whose creation went along (but also contributed to) the birth of National States as autonomous political entities. This was allowed by the simultaneous onset of the new scientific disciplines of epidemiology, demography and, clearly, statistics as basic knowledge for a scientific approach to disease prevention (Porter, 2005). As well documented, this process has not been linear but a complex one with an endless list of facets. Public health systems are continuously contributing to shaping modern States, due to the evolving concept of «health citizenship» in modern democracies (Porter, 2005), but also to the critical role of health expenditure on the governments' budget—and their dynamic evolution—to respond to the changing socio-demographic needs, for example, about the current wave of massive population ageing in Western countries (UN, 2021).

Much of the concern of the burgeoning modern National public health systems (PHSs) in their early lifecycles had been with the control of communicable diseases. The early development of the industrial revolution and the contemporary flowing of the demographic transition, with the ensuing massive population growth and urbanisation, initially generated overcrowded, over poor and unhealthy urban environments that played a critical role in strengthening the endemic character of many infectious diseases in an epoch when these still represented the major source of the overall mortality burden of humans (Livi-Bacci, 2017; Porter, 2005). About this, infections as smallpox and cholera represented the first major battlefield for the new-born PHSs by their two major prevention tools namely, vaccination for the case of smallpox, thanks to the development of the first vaccine in mankind history, versus sanitation, through clean potable water for the case of cholera (Livi-Bacci, 2017). This original footprint has amazingly

evolved due to the dramatic progress of western societies concerning the control of infectious diseases. Widespread prevention through vaccination of major deadly socially transmitted infections as diphtheria, poliomyelitis, pertussis, and measles has allowed exceptional degrees of control of diseases that were still highly prevalent at the beginning of the second half of the 20th century. Quantitative public health and epidemiological studies have confirmed the beneficial impact of previous mass childhood immunizations—as the key area of public health about communicable diseases in Western countries—on infant and child mortality in the industrialised world (see e.g., van Wijhe et al., 2016, for the Netherlands). At a later stage, further momentum was given by the introduction of new vaccines against a range of further infections among which viral hepatitis B, a multiroute infection, and human papilloma, a sexually transmitted infection, both responsible for the onset of specific cancer diseases. The dramatic global benefit of vaccination in terms of mortality avoidance, especially in low-resource countries, where in many cases fully developed PHS are still rare, was proved in a recent multicountry study (Li et al., 2021, see also the commentaries in Bärnighausen et al., 2014 and Bloom et al., 2018). There is now widespread agreement that mass vaccinations administered through a public health system have some beneficial "secondary indirect effects" at the broader societal level, that go well beyond the classical direct (protection of the vaccinated individual) and indirect (protection of unvaccinated individuals through the reduced virus circulation) effects typically described in basic epidemiological and economic epidemiology textbooks (e.g., Bärnighausen et al., 2014; Gessner et al., 2017, and references therein).

However, the current landscape of priorities about infectious disease control at a global level is still widely articulated. On one extreme, in modern industrialised countries most infectious diseases are well controlled by public health prevention systems, to the extent that vaccine opposition is spreading due to low perceived risks from infections (Manfredi & d'Onofrio, 2013), and major efforts are pointing towards preparedness against major future challenges such as pandemic risks or the potential development of large-scale antibiotic resistance (WHO, 2021). On the other extreme, in low resource settings, first of all, SSA, infectious diseases, ranging from major killers, such as malaria, tuberculosis (TB) and AIDS, to lower-trait respiratory infections and diarrheal diseases, still represent a major component of overall mortality and morbidity (Murray et al., 2017), thereby emerging as a major obstacle to economic development (Bloom & Canning, 2004; Bloom et al., 2018). Focusing on these issues has made the applied mathematical modelling of infectious diseases—as a rational approach to infection control—a dramatically growing area of public health sciences.

Despite the central importance of the topic, and the basic principles of infectious diseases transmission and control are widely established since several decades (Anderson & May, 1991; Bailey, 1975; Capasso, 1990), the interest of economic theory for such issues has remained somewhat absent, despite already as early as 1963 a master as Kenneth Arrow already pinpointed the special nature of welfare economics of health markets, particularly in relation with communicable diseases "The concept of marketability is somewhat broader than the traditional divergence between private and social costs and benefits... In the medical field, the obvious example is the spread of communicable diseases. An individual who fails to be immunized not only risks his own health, a disutility which presumably he has weighed against the utility of avoiding the procedure, but also that of others" (Arrow, 1963, p. 945). Indeed, to the best of our knowledge, the first contribution in modern economic epidemiology possibly stem from Stiglitz's remarks on vaccination and the free-rider problem, suggesting that immunization systems must be public as vaccination markets are typically not efficient to ensure the positive externality allowed by vaccination to reach adequate levels of infection control (Stiglitz, 1988).

After Stiglitz (1988), the emerging field of *economic epidemiology* was built upon the unifying concept of prevalence-dependent responses by agents. This concept, which was proposed first in basic extensions of the classical SIR model (Capasso, 1990, and references therein), states that individuals will demand more prevention or less risk (e.g., by vaccinating more or by undertaking a more prudent social/sexual behaviour, i.e., by increasing social/sexual distancing) the more prevalent the infection is. This concept shows per se the difficulty to bring many infections under high levels of control (up to elimination) because the better the infection is controlled, that is, the lower the prevalence, the lower will also be the incentive to undertake preventive behaviours. Two major directions were initially developed by economics scholars. The first one focused on the need to understand to effects of individual behavioural choices on the spread of HIV/AIDS and related interventions (e.g., Kremer, 1996, and references therein). The second one (Brito et al., 1991; Francis, 1997; Philipson, 2000, and references therein; Anderson et al., 2013; Gersovitz, 2013; Gersovitz & Hammer, 2003, and references therein; Manski, 2017) focused on issues as the dynamic effects of vaccination, the free rider problem, centralised versus decentralised decisions, elimination versus eradication of infections as well as the welfare effects of infection control with a special attention to common vaccine-preventable diseases, like measles. This discussion highlighted substantive policy implications including the need for public policies (e.g., by mandatory immunization) to overcome the difficulties in maintaining high degrees of control over time and eventually in achieving elimination (Manfredi & d'Onofrio, 2013). Beyond theory, Philipson's contribution reports a rich survey on econometric articles aimed at documenting the empirical substance on the concept of prevalence dependency covering the epoch before 2000. Fenichel (2013) has been a first economic attempt to investigate—long before the COVID-19 era—the effects of social distancing in contaning a deadly epidemic.

Most previous contributions on economic epidemiology tended to have a simple structure, namely they had as a building block a prevalence-dependent epidemiological set-up augmented with simple economic objectives. Later, a broader emerging discipline on the *economics of infectious diseases* attempted to set infectious diseases dynamics and their impacts into more general economic frameworks and narratives. In some among these contributions, the dynamics of infections was represented only implicitly, for example, through exogenous mortality shocks (see, among others, Azomahou et al., 2016; Bell & Gersbach, 2009, 2013; Boucekkine & Laffargue, 2010; Boucekkine et al., 2009; Corrigan et al., 2005; Greenwood et al., 2019; Momota et al., 2005; Young, 2005). Innovative contributions have—well before the COVID-19 era—attempted to endogenously integrate infection transmission and macroeconomic dynamics into unifying frameworks. As was pointed out in the introduction, a seminal effort in this direction has been done by Chakraborty et al. (2010, 2016). By using their approach, Gori et al. (2020) have investigated, within a Unified Growth Theory (UGT) model with endogenous mortality and fertility, one of the main conundra of current development, that is, the possibility that HIV/AIDS might alter the fertility transition in SSA, whereas Gori, Manfredi, and Sodini (2021) have analysed the effects of different ways of financing interventions against HIV/AIDS, namely international donations versus managing endogenously the public budget by the afflicted country. Finally, Gori et al. (2019) have considered HIV transmission within a general UGT setting with physical and human capital. Further, Goenka and Liu (2012, 2020) and Goenka et al. (2014) have analysed the interplay between infectious diseases and economic growth by setting a standard susceptible-infectious-susceptible (SIS) epidemiological transmission model within either discrete-time (Goenka & Liu, 2012) or continuous-time (Goenka & Liu, 2020; Goenka et al., 2014) infinite-lived Ramsey-like macroeconomic models. A similar effort was also performed by Bosi and Desmarchelier (2018).

Beyond the previous review, the explosion of contribution in the COVID-19 era is showing that finally economic theory and infectious diseases have definitely met. However, to the best of our knowledge, empirical studies aimed to document the key role of public health systems (whose presence and critical role are implicit in most of the theoretical works previously cited), especially prevention by vaccination, seem to rather rare in applied health economics. This is a matter sometimes acknowledged in public health studies (Gessner et al., 2017; Neumann et al., 2008). Filling this gap will surely be important in future research.

3 | THE MODEL

3.1 | Individuals

Consider a general equilibrium OLG (macro)economy à la Diamond (1965) closed to international trade and inhabited by a stationary population (normalised to one) composed of two-period lived rational and identical individuals. The life of the representative agent of generation $t \in \mathbb{N}$ (where $t = 0, 1, 2, 3...$ is the time index) is divided into youth—intended as the young adulthood phase (young, henceforth, for the sake of simplicity)—and old age. When young, individuals are economically active and they are endowed with one unit of labour, which is inelastically supplied to firms (producing in competitive markets) in exchange for wage income at the rate w_t. Moreover, they work, consume and save and they will retire at the onset of old age. Individuals are also socially and sexually active and therefore at risk of infections either socially or sexually transmitted. They are assumed to be *uninfected* (U) at entry in the young phase, but can acquire the infection becoming *infected* (I). Unlike Gori et al. (2020), where childhood was explicitly modelled to explain a demographic transition including the transition of infant mortality, here we do not consider a childhood generation, which exists but is dealt with as a fictitious cohort not playing any demo-economic role, as is usual in the basic Diamond-like OLG context. From this standpoint, the adopted infection model would in principle be better suited for infections circulating only among young adult individuals, therefore primarily sexually transmitted infections such as, for example, HIV/AIDS, viral hepatitis B (HBV), human papillomavirus infection (HPV), and so on. However, we believe that the proposed model and analysis have a wider purpose and in principle could be extended also to young people by just adding the related cohort. For the sake of simplicity, we consider a unique "representative" infection. Nonetheless, nothing prevents us from thinking in terms of the infection considered as the cumulative burden from different types of infections, as also postulated in Chakraborty et al. (2010). Infected individuals will die at the onset of old age with a probability of $1 - \phi$, where $\phi \in [0, 1]$ is the corresponding survival probability. We disregard morbidity effects on infected individuals, meaning, for example, that their productivity is unaffected. This choice is restrictive as it implies that the infection only has a mortality effect, which is however postponed at the entry into old age. Nonetheless, this can be considered an acceptable approximation for, for example, HIV/AIDS in SSA in the first decades of the epidemic when no effective therapies were available. Indeed, HIV has a long incubation period (defined as the period between the moment the individual acquires the infection and the moment he subsequently develops AIDS disease), in the range of 10–15 years during which the individual is typically in good health. This implies that even an early infected individual, who, for example, acquired the infection by age 20, would probably experience good health (in the absence of comorbidity) up to age 35, thereby being fully economically active up to that age.

Following Chakraborty et al. (2016), let $0 < p_t \leq 1$ denote the probability that a susceptible young individual acquires the infection. This is defined as follows:

$$p_t = 1 - (1 - i_t \pi_t)^{\mu}, \tag{1}$$

where $0 \leq i_t \leq 1$ is the *infection prevalence* at time t, representing the fraction of young adult individuals that are in the infected state at t, $0 < \pi_t \leq 1$ is the probability of acquiring the infection per single (sexual or social) contact/partnership with an infected individual and $\mu > 0$ represents the average number of (sexual or social) contacts of a young individual during his entire adulthood. The transmission probability π_t can be reduced either by interventions on (sexual or social) individuals' behaviour and lifestyle. In the case of a sexually transmitted infection, these interventions might include a range of strategies: (i) direct prevention activities (e.g., distribution and use of condoms or quarantine measures), (ii) pharmaceutical treatments of infected individuals aimed, for example, at reducing infectivity (e.g., in the case of HIV antiretroviral treatments reducing the viral load allow to decrease the probability of transmission), (iii) the diffusion of safe health practices such as male circumcision, which reduce the probability of infection per single sexual episode, (iv) the communication of risks to the population aimed to increase awareness in at-risk individuals. In the public health practice, the heterogeneity of these interventions makes their implementation quite articulated. However, from a modelling perspective, we model this range of different interventions in a simplified manner by using a direct relationship between the probability of transmission per single at-risk contact and the per worker public health expenditure against the disease. Relying on Chakraborty et al.'s formulation, we include the public health expenditure endogenously managed by the government in the transmission probability as follows:

$$\pi_t = \pi(h_t) = \frac{\pi_0}{1 + \pi_1 h_t} \in (0, \pi_0], \tag{2}$$

where $h_t \geq 0$ represents the amount of public expenditures against the infection on a per worker basis, $0 < \pi_0 \leq 1$ is the transmission probability in the absence of any public interventions and $\pi_1 > 0$ is an exogenous parameter tuning the effectiveness of public expenditures in reducing π_t. From (2), we have that $\pi'(h_t) < 0, \pi(0) = \pi_0$ and $\pi(\infty) = 0$. Equation (2) modifies Chakraborty et al.'s analogous equation, which included private prevention activities, by considering the public component h_t. As the relationship $p_t = i_{t+1}$ holds (Chakraborty et al., 2010, 2016), the dynamic equation of prevalence can be written as follows:

$$i_{t+1} = 1 - (1 - i_t \pi(h_t))^{\mu}. \tag{3}$$

Preferences follow Chakraborty et al. (2010, 2016). Infected (I) and uninfected (U) individuals of generation t have expected lifetime utility functions (V_t^I and V_t^U, respectively) defined over material consumption when young (c_t^j) and when old (d_{t+1}^j), $j = \{I, U\}$. The utility function of the infected is

$$V_t^I = \ln\left(c_t^I\right) + \phi\beta \ln\left(d_{t+1}^I\right), \tag{4}$$

where $0 < \phi < 1$ is the constant probability of surviving from the first period to the second one and $0 < \beta < 1$ is the subjective discount factor, which is assumed to be the same for agents of

type I and type U, whereas the utility function of the uninfected (who do not suffer infection-related mortality) is

$$V_t^U = \ln\left(c_t^U\right) + \beta \ln\left(d_{t+1}^U\right). \tag{5}$$

Equations (4) and (5) imply that the infected save less because they face a shorter quantity of life ($0 < \phi < 1$) and get (ceteris paribus) a lower utility compared to the uninfected, who do not face any risk of dying before reaching old age. The present approach is also consistent with the case where infected individuals (though not suffering specific mortality) may discount the future at a higher rate than the uninfected (Chakraborty, 2004). Unlike Chakraborty et al. (2010, 2016), we do not speculate about the possible different quality of life of infected and uninfected individuals as the lifetime utility only depends on material consumption. Consequently, the utility functions of the two groups give the same utility flow from young-age and old-age consumption bundles (ceteris paribus). In any case, this assumption would not be relevant for the individual optimum. The lifetime budget constraints of infected and uninfected individuals are respectively given by the following equations:

$$c_t^I + \frac{\phi d_{t+1}^I}{R_{t+1}^e} = w_t(1 - \tau_t) \tag{6}$$

and

$$c_t^U + \frac{d_{t+1}^U}{R_{t+1}^e} = w_t(1 - \tau_t), \tag{7}$$

where R_{t+1}^e is the interest factor that an individual of generation t expects to prevail from time t to time $t + 1$ (it will become the realised interest factor at the beginning of the period $t + 1$) and $0 \leq \tau_t < 1$ is the tax rate levied by the government on labour income of people of both groups.

The difference in the left-hand side of the expressions in (6) and (7) stems from the assumption of a perfect market for annuities where savings are intermediated through mutual funds (Chakraborty, 2004; Chakraborty et al., 2010, 2016). Unlike Chakraborty et al. (2010, 2016), we did not include a parameter capturing a productivity loss due to the infection-related morbidity (as discussed above).

The maximisation of lifetime utility (4) (resp. 5) is carried out subject to the lifetime budget constraint (6) (resp. 7), by taking as given factor prices, the public health expenditure and the health tax rate. This allows us to get the saving functions of both infected (z_t^I) and uninfected (z_t^U), which are given by:

$$z_t^I = \frac{\phi\beta}{1 + \phi\beta} w_t(1 - \tau_t) \tag{8}$$

and

$$z_t^U = \frac{\beta}{1 + \beta} w_t(1 - \tau_t), \tag{9}$$

where $z_t^I < z_t^U$.

3.2 | Government

The government uses the revenues collected by labour income taxes ($\tau_t w_t L_t$, where $L_t = 1$ is the labour force employed in the market, i.e., full employment) to finance public health prevention investments against the infection (H_t) at a balanced budget $H_t = \tau_t w_t L_t$. On a per worker basis, we have that

$$h_t = \tau_t w_t = [\tau_0 + \tau(i_t)]w_t, \tag{10}$$

where $h_t := H_t/L_t$, $\tau_t := \tau_0 + \tau(i_t) < 1$, and $\tau(i_t) = \tau_1 i_t$. The rule in (10) includes two components. The first one represents exogenous taxation (independent of the infection prevalence) to finance prevention expenditures in the absence of the infection. This resource allocation aims to prevent an emerging epidemic, that is, used in a situation where the infection is temporarily absent (i.e., prevalence equal to zero) but it might emerge in the future due to the importation of infected cases. Referencing to the COVID-19 case, whose risk will persist in the population in the long term, this component might include, for example, (i) long-term vaccination expenditures, (ii) communication aimed to maintain high awareness against the risk of infection, (iii) containment activities aimed to prevent the return of the virus. The second ("prevalence-dependent") component is endogenous and implies that public prevention expenditures proportionally increase with the infection prevalence. This is a realistic situation that might occur, for example, during a seasonal influenza epidemic, where the needs of public budget depend on the epidemic severity (e.g., number of hospitalisations, treatments, etc.), which cannot be predicted in advance. The choice of a linear specification of $\tau(i_t)$ is a first approximation: for example, Goenka et al. (2014) and Goenka and Liu (2020) showed that the optimal health expenditure can be nonlinear with prevalence.

3.3 | Firms

Firms are identical and act competitively in the market. At time t, the representative firm produces a homogeneous good (Q_t) by combining capital (K_t) and labour (L_t) through the following production function:

$$Q_t = AK_t^{\alpha} L_t^{1-\alpha} + bL_t, \tag{11}$$

where $0 < \alpha < 1$ is the output elasticity of capital, $A > 0$ is a scaling constant and $b \geq 0$ is a parameter capturing natural endowments (Chakraborty et al., 2010). We note that case $b = 0$ represents the standard Cobb–Douglas production function. The representative firm maximises profits $AK_t^{\alpha} L_t^{1-\alpha} + bL_t - w_t L_t - R_r K_t$ by taking factor prices as given. Therefore, the wage and the interest factor are equal to the marginal product of labour and capital, respectively, that is:

$$w_t = w(k_t) := (1-\alpha)Ak_t^{\alpha} + b, \tag{12}$$

$$R_t = R(k_t) := \alpha A k_t^{\alpha-1}, \tag{13}$$

where $k_t := K_t/L_t$ is the stock of capital per worker at time t ($L_t = 1$ for any t).

3.4 | General equilibrium

Equilibrium in the capital market is determined by the equation $K_{t+1} = Z_t$, where $Z_t := p_t z_t^I + (1 - p_t) z_t^U$ is aggregate saving, which is obtained as a weighted sum of the savings of infected and uninfected individuals. As $k_{t+1} := K_{t+1}/L_{t+1}$ and using (8) and (9), we get:

$$k_{t+1} = \left[p_t \frac{\phi\beta}{1+\phi\beta} + (1 - p_t) \frac{\beta}{1+\beta} \right] w_t (1 - \tau_t). \tag{14}$$

Therefore, the equilibrium dynamics of the economy is described by the following two-dimensional system of difference equations:[1]

$$k_{t+1} = \left\{ p(k_t, i_t) \frac{\phi\beta}{1+\phi\beta} + [1 - p(k_t, i_t)] \frac{\beta}{1+\beta} \right\} w(k_t)(1 - \tau_0 - \tau_1 i_t), \tag{15}$$

$$i_{t+1} = 1 - [1 - i_t \pi(k_t, i_t)]^{\mu}, \tag{16}$$

where $w(k_t)$ is given by the expression in (12), while

$$p(k_t, i_t) = 1 - [1 - i_t \pi(k_t, i_t)]^{\mu}, \tag{17}$$

directly follows from (1) and (16), and finally

$$\pi(k_t, i_t) = \frac{\pi_0}{1 + \pi_1(\tau_0 + \tau_1 i_t) w(k_t)}, \tag{18}$$

follows from (2), (10), and (12).

3.5 | The final dynamic system

For analytical convenience, let us now rewrite Equations (15) and (16) as follows:

$$T : \begin{cases} x' := f(x, y) = [1 - (\tau_0 + \tau_1 y)][(1 - \alpha) A x^{\alpha} + b] \frac{\phi\beta(1+\beta) + \beta(1-\phi)\gamma(x,y)}{(1+\phi\beta)(1+\beta)}, \\ y' := g(x, y) = 1 - \gamma(x, y), \end{cases} \tag{19}$$

where we adopted the notation $x' := k_{t+1}, x := k_t, y' := i_{t+1}, y := i_t$ and

$$\gamma(x, y) := \left(1 - \frac{\pi_0 y}{1 + (\tau_0 + \tau_1 y)\pi_1 [(1 - \alpha) A x^{\alpha} + b]} \right)^{\mu}. \tag{20}$$

[1]Including a parameter capturing a productivity loss due to morbidity, as in Chakraborty et al. (2010, 2016), would have prevented obtaining a closed-form expression for the dynamics of capital. This is because in their work $L_{t+1} = 1 - \theta p_{t+1}$ and $p_{t+1} = p(k_{t+1}, i_{i+1})$.

The following remark summarises the main differences between the present formulation and the main reference works in the literature, namely those proposed by Chakraborty et al. (2010, 2016).

- The key link function between the epidemiological and the economic system is represented by the endogenous transmission probability π.
- Individuals in Chakraborty et al. (2010, 2016) attempt to determine π choosing private prevention investments against communicable diseases by maximising their utility functions subject to the related budget constraints.
- Unlike Chakraborty et al. (2010, 2016), in the present model the government attempts to determine π by public health expenditures financed by labour income taxation. Taxation includes both a prevalence-dependent component and a prevalence-independent component mirroring prevention in the absence of infections. Other contributions still using the Chakraborty et al.'s equation are Gori et al. (2019, 2020) and Gori, Manfredi, and Sodini (2021).

4 | RESULTS: CHAKRABORTY ET AL.'S EQUATION OF INFECTION DIFFUSION

The dynamic system given by Equations (19) and (20) links an equation of infection diffusion à la Chakraborty et al. (2010, 2016) with an OLG model describing the dynamics of the (macro) economy. The feedback between the two equations is given by public prevention expenditures and the probability of infection transmission. We pinpoint that Chakraborty et al.'s equation aims to represent the evolution of infection prevalence over a time frame which is the same as the underlying OLG cohorts. As such, their equation is primarily valid for infection processes showing a significant evolution over the long time scales characterising human long-term demographic and economic decisions. These interpretations hold if an individual is infective for the entire adult period, which in turn requires that infective individuals are essentially generated at the beginning of the young adult phase. This can appear somewhat restrictive but it is a consequence of the simplicity of the OLG framework adopted. Nonetheless, this might represent an appropriate description at least for some infections with a long infective period, as HIV/AIDS that is devastating SSA since four decades. Clearly, it might fail to describe short epidemic episodes, as was, for example, the deadly outbreak of SARS in 2003 and hopefully will be the case with COVID-19.

Summing up, Chakraborty et al.'s equation represents a general useful tool for qualitative predictions and understanding of the interplay between communicable diseases and economic processes. In technical terms, their equation can be interpreted, at a first glance, as a pure infection process, that is a susceptible-infectious (SI) process in epidemiological jargon. Obviously, given its peculiar definition of the time frame, the predictions of Chakraborty et al.'s equation are hardly comparable with, for example, the discrete- or continuous-time SI and SIS equations adopted in other economic studies explicitly modelling infection dynamics within macroeconomic frameworks (Bosi & Desmarchelier, 2018; Goenka & Liu, 2012, 2020; Goenka et al., 2014).

However, the formal properties of Chakraborty et al.'s equation as an infection model have not been so far characterised in detail. Therefore, we discuss here the main dynamic features of Chakraborty et al.'s equation in the simplest setting, that is, in the absence of any link with the

economy, namely under the simplifying assumption that the infection parameters μ and π_t are both constant (i.e., in the absence of public or private prevention), with $\pi_t = \pi_0$. In this case,

$$i_{t+1} = G(i_t) = 1 - (1 - \pi_0 i_t)^{\mu}. \tag{21}$$

where G is a one-dimensional smooth map invariant in $[0, 1]$, that is, $G([0, 1]) \subseteq [0, 1]$. The following lemma gives the global dynamics of (21).

Lemma 1 (Existence)**.** *Map G in (21) always admits the infection-free equilibrium (IFE) $i_0 = 0$. It also admits a unique interior equilibrium $i^* \in (0, 1)$ for $\pi_0\mu > 1$. [Stability] (i) If $\pi_0\mu < 1$ then the IFE is globally asymptotically stable; (ii) if $\pi_0\mu > 1$ then the IFE is unstable and all trajectories starting from positive initial conditions monotonically converge to i^*; (iii) at $\pi_0\mu = 1$ a tangent bifurcation occurs.*

Proof. The proof is trivial and directly follows from the geometrical properties of map f as summarized in the following.

- $G(0) = 0 \ \ \forall\, \pi_0$ and $\forall\, \mu$ values;
- G is strictly increasing and concave in $[0, 1]$ and $G(1) < 1$, hence at most one positive fixed point exists in $(0, 1)$;
- as $\lim_{i_t \to 0^+} G'(i_t) = \pi_0\mu$ then the following cases may occurr. Map G is below the main diagonal for all $i_t \in (0, 1]$ as long as $\pi_0\mu \leq 1$; when $\pi_0\mu$ increases and crosses 1 from below, then a fold bifurcation occurs; finally, $\forall\, \pi_0\mu > 1$ an interior fixed point $i^* \in (0, 1)$ exists and it attracts all trajectories exiting from initial conditions different from zero.

□

With obvious caveats due to the peculiar time frame, Lemma 1 can be given a deep epidemiological interpretation. As μ represents the (average) number of at-risk contacts during the entire infective period (that formally coincides with the length of the young period) and π_0 is the transmission probability per single contact in the absence of interventions, the product $\pi_0\mu$ represents the total number of cases of infections that an infective individual would generate during his infective lifetime in the case all her encounters were with uninfected individuals. Given the previous interpretation, $\pi_0\mu$ can be interpreted as the basic *reproduction number* of the infection, typically denoted by R_0 in epidemiological jargon. Consequently, Lemma 1, which holds in the absence of any interventions, implies that (1) if $R_0 := \pi_0\mu < 1$ only the infection-free equilibrium exists and is globally stable. In this case, any initial infective seed will never cause epidemics and will "rapidly" disappear. The condition $R_0 < 1$ means that an infective case generates less than one new infective case during the individual infective lifetime in a fully susceptible population, and therefore the infection cannot spread; (2) a globally stable endemic equilibrium with positive infection prevalence exists if and only if $R_0 > 1$. The condition $R_0 > 1$ means that an infective case generates more than one new infective case, therefore the infection has the potential to spread, eventually reaching, by a monotonic pattern, a positive equilibrium i^*. The magnitude of the equilibrium prevalence i^* is increasing in R_0.

The previous features indicate that Chakraborty et al.'s equation actually represents a flexible formulation exhibiting a threshold character, with the separation between an infection-free equilibrium and an endemic equilibrium characterised by an infection prevalence lower

than 100%. From this viewpoint, the model shows mixed features, shared by both the epidemic susceptible-infectious-recovered (SIR) model and by the SIS endemic model. In relation to the SIR epidemic model, the Chakraborty et al.'s equation in the supercritical case ($R_0 > 1$) can be taken to represent a *cumulative incidence curve* of an epidemic process. However, the fact that (still for $R_0 > 1$) Equation (21) eventually reaches a meaningful equilibrium implies that the model can be used to represent situations where the infection becomes endemic at the *endemic equilibrium* i^*, still by a monotonic pattern of prevalence. In this perspective, Equation (21) can be taken to represent a meaningful description of the cumulative prevalence of an endemic communicable disease of the SIS type (Capasso, 1990). This property was used in both Chakraborty et al. (2010) and Gori et al. (2020) to represent the long-term interplay between development processes and major infective killers as malaria, TB and HIV/AIDS. The prevalence function i_t generally shows a monotonic behaviour with respect to both its characteristic parameters μ and π_0. This was expected as it implies to consider infections characterised by a larger reproductive number. Previous considerations make Equation (21) a parsimonious and flexible tool for meaningful economic-epidemiology modelling.

A noteworthy extension of Chakraborty et al.'s equation can be given by endogeneising its two parameters, as follows:

$$i_{t+1} = 1 - (1 - \pi_0(i_t)i_t)^{\mu(i_t)}. \tag{22}$$

Under appropriate hypotheses on functions $\mu(i_t)$ and $\pi_0(i_t)$, Equation (22) can be used to represent the effects of behavioural responses in an implicit manner. More interesting extensions are obtained by making behavioural responses fully explicit as the outcome of the interplay between epidemiologic and economic processes, as represented by our general model given by (19) and (20), where endogeneisation of μ and π_0 results from public prevention activities of the government. In presence of endogeneisation, the monotonic pattern of i_t can be lost. In this case, the quantity i_t can simply be taken to represent the actual infection prevalence at a certain moment in time.

5 | RESULTS: ENDOGENOUS PUBLIC HEALTH EXPENDITURES

Let us now turn to our general analysis of the interplay between the infection and economic variables resulting from the full macroeconomic model (19) and (20). This analysis will systematically rely on extensions of the properties of Chakraborty et al.'s epidemiological equation.

5.1 | Preliminary mathematical properties

As capital per worker x varies over the nonnegative half plane and infection prevalence y is bounded in $[0, 1]$, then the state space is defined by $S := \mathbb{R}_+ \times [0, 1]$. Therefore, the dynamic system defined by map T is feasible if and only if S is positively invariant for any given initial condition, that is $(x(0), y(0)) \in S$ implies $T^t(x(0), y(0)) \in S, t = 1, 2, 3, \ldots$ In practical terms, this means that if the system starts from a meaningful initial condition of the economy and the infection, it will be meaningful forever. This implies that the model is well posed. Moreover, it can be shown that the long-term dynamics of system T are always bounded and that system T

admits an attractor $\Lambda \subset [0, \bar{T}\,] \times [0, 1-(1-\pi_0)^{\mu}]$, $\bar{T} < \infty$. The formal statement of the well-posedness of (T, S), of the boundedness of T, of the existence of the attractor Λ and of the corresponding proofs are relegated in the appendix.

5.2 | Infection spread and economic development: The infection-free equilibrium of the economy and its stability

To describe the structure of the attractor Λ, we observe that $T(x, 0) = (x', 0)$ for all parameter values. This corresponds to the situation of full absence of the infection, that is an infection-free economy. Let $K_{IFE} = \{(x, y) \in S : y = 0\}$ define the set containing the infection-free trajectories of the economy. Then, it is fully immediate to show that set K_{IFE} is invariant under map T. The implication of this result is that if $y(0) = 0$ that is, the economy is infection-free at time $t = 0$, then it will remain infection-free forever.

The dynamics of T on the invariant set K_{IFE} are governed by the Diamond-like one-dimensional map $F_{K_{IFE}}(x) = f(x, 0)$, given by

$$x' = F_{K_{IFE}}(x) = (1-\tau_0)\frac{\beta}{1+\beta}[(1-\alpha)Ax^{\alpha} + b]. \tag{23}$$

To characterise the long-term dynamics of this infection-free economy, we observe that for all parameter values map $F_{K_{IFE}}(x)$ admits as the unique equilibrium the positive steady state $x^*_{K_{IFE}} > 0$. In the particular case $b = 0$, the expression in (23) implies that (i) the origin $x^0 = 0$ is a further fixed point of $F_{K_{IFE}}(x)$, and (ii) there exists a closed-form expression of $x^*_{K_{IFE}}$, that is:

$$x^*_{K_{IFE}} = \left[(1-\tau_0)\frac{\beta}{1+\beta}(1-\alpha)A\right]^{\frac{1}{1-\alpha}}. \tag{24}$$

Based on previous arguments, map T admits the fixed point $E^*_{K_{IFE}} = (x^*_{K_{IFE}}, 0)$ for all parameter values.

As the economy always admits an infection-free equilibrium, the part of Lemma 1 dealing with the stability of IFE in Chakraborty et al.'s equation can be extended to the case where the infection interacts nontrivially with the (macro)economy. We now introduce the following definition to distinguish the case of Lemma 1, where a pure epidemiological dynamics was considered, from the current case, where also the (macro)economy is studied.

Definition 1. Let $E^*_{K_{IFE}} = (x^*_{K_{IFE}}, 0)$ be the IFE of the (macro)economy defined by system T.

As set K_{IFE} is invariant, then a trajectory starting from K_{IFE} will remain there for all t. As $F_{K_{IFE}}(x)$ is continuous, strictly increasing and concave, then $x^*_{K_{IFE}}$ is globally asymptotically stable for all $x(0) \geq 0$ if $b \geq 0$. Therefore, in the absence of infection the model (qualitatively) reproduces a Diamond-like growth path (Diamond, 1965), implying long-term convergence towards the IFE for any $x(0) > 0$.

The aim of the previous discussion was purely that of clarifying the behaviour of an economy in the trivial situation of the full absence of any infections. The truly interesting question deals with the risk that the economy is invaded by an onsetting infection, that is, what happens when a few infectious individuals enter a previously infection-free economy. This leads to the analysis of the local stability of $E^*_{K_{IFE}}$ when some infection seeds are introduced at time $t = 0$, that is, $y(0) > 0$.

Then, let us now consider the *transverse* eigenvalue associated to set K_{IFE}:

$$\lambda_2 = \left.\frac{\partial g}{\partial y}\right|_{y=0} = \frac{R_0}{1 + \tau_0 \pi_1 w(x)} \coloneqq R_c(x). \tag{25}$$

The previous expression tells us that K_{IFE} is locally stable if the previously defined quantity fulfilfs $R_c(x) < 1$. This has a nice interpretation. Note first that $R_c(x) \leq R_0$, with equality holding in a subsistence nonproductive economy, that is, when $x = 0$, or in a productive economy ($x > 0$) without dedicated resources allocated for *containing an incoming infection at onset*, that is, the exogenous tax rate $\tau_0 = 0$. The latter case might be considered as a case where the economy is missing a "preparedness plan." In these circumstances, the economy will suffer the threat of the incoming infection (this has been the case with the novel SARS-CoV-2 virus at its onset in winter 2020) at its maximal level R_0.

Instead, if the economy is productive and a preparadness plan is available ($\tau_0 > 0$), then $R_c(x) < R_0$. This allows to interpret $R_c(x)$ as the *current reproductive number* of the infection at onset when the stock of capital is at its generic level x and $w(x) \coloneqq (1 - \alpha)Ax^\alpha + b$ represents the corresponding wage rate.

In relation to (25), we remark that if the infection is not productive in the absence of any control measure ($R_0 < 1$), then $\lambda_2 = R_c(x)$ will be a fortiori lower than 1 and the infection cannot spread in the economy. The infection-free equilibrium $E^*_{K_{IFE}}$ remains globally stable.

Passing to the case of main interest, that is when the economy is at its infection-free equilibrium and the incoming infection is productive in the absence of any control measure ($R_0 > 1$), the condition $R_c(x^*_{K_{IFE}}) < 1$, where $R_c(x^*_{K_{IFE}})$ denotes the current reproductive number evaluated at the equilibrium, ensures that the population will not be invaded by the pathogen. We note among other things that the threshold parameter $R_c(x^*_{K_{IFE}})$ depends in an articulated manner on the exogenous tax rate τ_0, which also appears in the equilibrium wage rate, $w^*(\tau_0)$ (at the IFE $x^*_{K_{IFE}}$). We denote this by the following notation:

$$R^*_c(\tau_0) = \frac{R_0}{1 + \tau_0 \pi_1 w^*(\tau_0)}, \tag{26}$$

to suggest that the current reproduction number can differ from the basic reproduction number at onset of an incoming infection only in the presence of a public preparadness plan (funded by the exogenous taxation component τ_0).

As the steady-state wage rate $w^*(\tau_0)$ depends negatively on τ_0, the role played by the exogenous taxation on (26) is not univocal. This is because $R^*_c(\tau_0)$ is affected by two counterbalancing effects. The first one is the direct reduction induced by prevention taxation on the transmission probability. The second one is the indirect general equilibrium effect arising through the effect that taxation has on capital accumulation by reducing available income

eventually leaving less resources for prevention. In particular, $E^*_{K_{IFE}}$ remains locally asymptotically stable provided that $R_c^*(\tau_0) < 1$. Note that the local dynamics of the infection prevalence occurring close to K_{IFE} are monotonic as $\lambda_2 > 0$. These results can be summarised in the following proposition.

Proposition 1. *If* $1 < R_0 < 1 + \tau_0\pi_1 w^*(\tau_0)$ *then the IFE of* T *is locally asymptotically stable even if the infection is productive* ($R_0 > 1$).

The shape of the dependency of the threshold parameter $R_c^*(\tau_0)$ on τ_0 can be studied analytically for $b = 0$. In this case, by taking constant the remaining parameters, $R_c^*(\tau_0)$ can be written as follows:

$$R_c^*(\tau_0) = \frac{R_0}{1 + D\tau_0(1-\tau_0)^{\frac{\alpha}{1-\alpha}}}, \tag{27}$$

where D is a positive constant defined as $D := \pi_1\left(\frac{\beta}{1+\beta}\right)^{\frac{\alpha}{1-\alpha}}[(1-\alpha)A]^{\frac{1}{1-\alpha}}$. From (27), it is easy to show that the denominator is a one-hump function of τ_0 having its maximum at $\tau_0^{\max} := 1 - \alpha < 1$. Consequently $R_c^*(\tau_0)$ is decreasing up to $\tau_0^{\max}$ and increasing thereafter. Therefore, exogenous taxation is effective in reducing the reproduction of infection (for purposes of containment at onset) up to a threshold level. Beyond this level, taxation becomes counterproductive due to the erosion of resources at the general equilibrium level.

The previous discussion leads to a number of remarks. Under our working assumption that the economy has already achieved its long-term stationary state and by recalling that public prevention expenditure is the sum of an exogenous component ($\tau_0 w(x)$), undertaken even when infection prevalence is zero, and a prevalence-dependent one ($\tau(y)w(x) = \tau_1 yw(x)$), which is activated when the infection penetrates and persists in the population, then the parameter $R_c(x)$ from (25) represents the correct reproduction parameter of the infection that is relevant under initial epidemic conditions, that is, the conditions resulting when, for example, infective immigrants enter a previously infection-free economy. Therefore, $R_c^*(\tau_0) < 1$ represents the correct rule of epidemic *containment at onset* in an infection-free economy. This rule states the exact condition for containment at onset through a public policy based on (exogenous) taxation. The "containment tax rate" (only relying on the exogenous component of taxation) is obtained by solving the equation $R_c^*(\tau_0) = 1$ with respect to τ_0. In view of the shape of the function $R_c^*(\tau_0)$, this equation has two solutions on which the lower one, $\tau_0^- < \tau_0^{\max} := 1 - \alpha$, is always meaningful, while the other, $\tau_0^+ > \tau_0^{\max}$, does not need to be. In any case, only τ_0^- would be desirable from an economic point of view.

A further remark holding in the general situation where the economy is not necessarily at its steady state and $b > 0$ is the following. As $\frac{\partial\lambda_2}{\partial x} < 0$, it follows that $\max_{\{x\}}\lambda_2 = \lambda_2^{\max} := \lambda_2|_{x=0} = \frac{R_0}{1+\tau_0\pi_1 b}$. This parameter assigns an epidemiological *worst case*: indeed, it corresponds to the situation of a least-developed economy where GDP per young person is essentially close to zero and therefore no resources are made available to the public sector for prevention of emerging epidemics. This possibly well describes the situation of many current SSA countries (Gori, Manfredi, & Sodini, 2021), which are potentially under the threat of any future emerging infection because generalised poverty conditions prevent any endogenous mobilization of resources for containment at onset. The condition $\lambda_2^{\max} < 1$ is a strong one ensuring that K_{IFE} is

an attracting set and $E^*_{K_{IFE}}$ is an asymptotically stable fixed point (also guaranteeing containment at onset in the worst case), can be summarised by the following proposition.

Proposition 2. *If $R_0 < 1 + \tau_0\pi_1 b$ then the IFE of T is locally asymptotically stable even if the infection is productive.*

The corresponding taxation prevention threshold in this case is fully explicit and given by $\tau_0 > \frac{R_0 - 1}{\pi_1 b} := \bar{\tau}_0$, which obviously will be economically meaningful only if $\bar{\tau}_0 < 1$.

5.2.1 | The stability boundary in the (τ_0, μ) plane

We complete the discussion on the stability boundary of the IFE by focusing on the interplay between the contact parameter μ, which is the key parameter in Chakraborty et al.'s equation and the exogenous taxation parameter τ_0 introduced in this study by distinguishing between $b = 0$ and $b > 0$. This will be useful for the subsequent investigation of endemic equilibria of the economy, where the pair of parameters (τ_0, μ) will allow to sharply simplify the analysis.

For $b = 0$ the necessary and sufficient condition specified above for the local stability of the IFE in terms of all the model parameters is the following:

$$R_0 = \pi_0\mu < 1 + \tau_0\pi_1\left[\frac{(1-\tau_0)\beta}{1+\beta}\right]^{\frac{1}{1-\alpha}}[A(1-\alpha)]^{\frac{\alpha}{1-\alpha}}. \tag{28}$$

Figure 1a,b depicts the stability boundary $\mu = \omega(\tau_0)$ defined by (28) in the plane (τ_0, μ)—whose interplay is critical for the containment of the epidemic at onset—for different choices of A (which eventually tunes the level of the equilibrium GDP and consequently the amount of resources available to the public health system) given the other model parameters. Therefore, points below (resp. above) $\omega(\tau_0)$ identify parameter combinations ensuring the local stability (resp. instability) of IFE. When $\omega(\tau_0)$ is crossed from below, the transverse eigenvalue crosses +1 and the IFE becomes a saddle. The curves depicted in Figure 1 have a minimum in $\tau_0 = 0$ and a maximum in $\tau_0 = \tau_M$.

Define $\mu_0 = \omega(0)$ and $\mu_M = \omega(\tau_M)$. If $\mu \in (\mu_0, \mu_M)$ then there exists a set of values of τ_0 given by $\Omega = (\tau_a, \tau_b)$ such that the IFE is locally asymptotically stable for all $\tau_0 \in \Omega$. In contrast, if $\mu > \mu_M$ no tax rate level can be fixed to avoid infection in the long term. The public health and economic interpretations of the latter result is that if the rate of social/sexual partners per year is very large then the effective reproductive number $R_c(x)$ of the infection is too large, given the prevailing GDP and the resulting dimension of the public health budget, to allow containment of the epidemic at onset.

For $b > 0$, $x^*_{K_{IFE}}$ cannot be solved in closed form and the local stability of $E^*_{K_{IFE}}$ must be studied numerically. The straight line $C = \{(\tau_0, \mu) \in [0, 1 - \tau_1] \times [2, +\infty) : \tau_0 = \frac{R_0 - 1}{\pi_1 b}\}$ separates the plane $[0, 1 - \tau_1] \times [2, +\infty)$ in two regions: points below C correspond to parameter combinations where the IFE is locally stable; points above curve C imply that the IFE can be locally stable or unstable and, in this last case, the economy will converge to a different attractor. This is because the sufficient condition stated in Proposition 2 does not hold.

To obtain sufficient conditions for the IFE to be locally stable we can proceed numerically as follows. We fix all the parameter values but μ and τ_0 and notice that $x^*_{K_{IFE}} = x^*_{K_{IFE}}(\tau_0)$ while

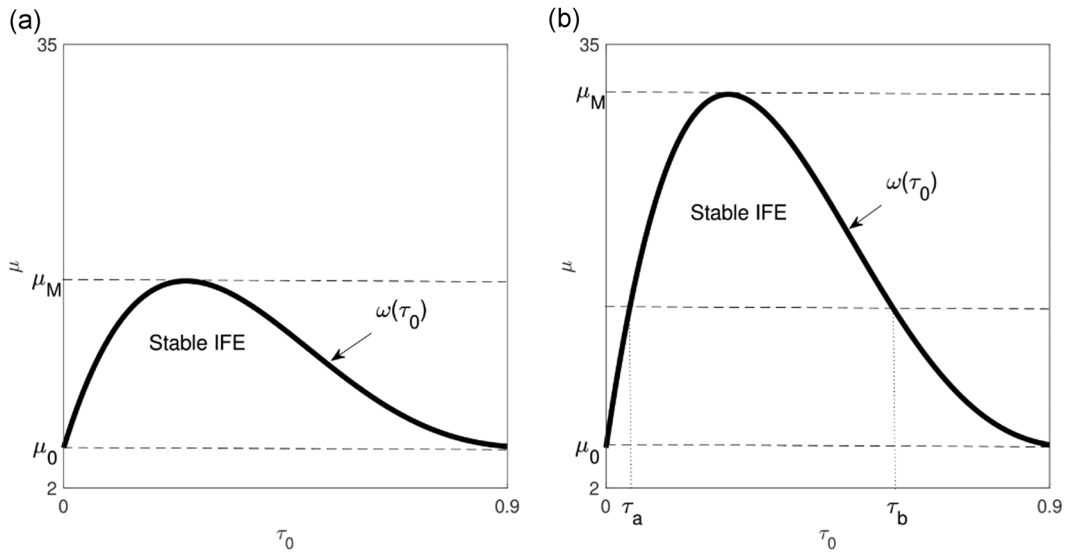

FIGURE 1 Stability boundary of the IFE in the (τ_0, μ) plane and related stability/instability regions. The points below the stability boundary in both panels represents parameter combinations in which the IFE is locally stable. When A increases (moving from (a) to (b)) the stability region widens. Parameter values: $\alpha = 0.67$, $\pi_0 = 0.2$, $\pi_1 = 35$, $\beta = 0.28$, $\phi = 0.62$, $b = 0$ and $\tau_1 = 0.1$. (a) $A = 24.18$. (b) $A = 35$. IFE, infection-free equilibrium

$\lambda_2|_{x=x^*_{K_{IFE}}} = \lambda_2(\tau_0, \mu) > 0$. If $\lambda_2(\tau_0, \mu) < 1$ holds then the IFE is locally stable. Define $C_1 = \{(\tau_0, \mu) \in [0, 1 - \tau_1] \times [2, +\infty) : \lambda_2(\tau_0, \mu) = 1\}$ as the curve that separates the parameter plane (τ_0, μ) in two regions: a parameter space corresponding to the local stability of the IFE (dark grey) and a parameter space corresponding to which the local dynamics will converge to another attractor characterised by positive infection prevalence (light grey). This is shown in Figure 2a (Figure 2b represents an enlargement in which line C is depicted). Points below C verify the sufficient condition for the local stability of the IFE. However, also parameter combinations above C and below C_1 are such that the IFE is locally stable. Even if we could not prove the global stability of the IFE, numerical simulations suggested that if $b > 0$ and the IFE is locally stable then it is also globally stable (the same result holds for $b = 0$).

It is important to underline that since $\lim_{\mu \to \infty} \lambda_2(\mu)|_{x^*_{K_{IFE}}} = +\infty$ then $\lambda_2(\mu)|_{x^*_{K_{IFE}}} > 1$ for all $\mu > \mu_\infty$ (where if $b = 0$ then $\mu_\infty = \mu_M$ as depicted in Figure 1), other things being equal. This confirms that for all $b \geq 0$ there exists a threshold value μ_∞ such that for $\mu > \mu_\infty$ the IFE is a saddle point. In such a case, no tax rate τ_0 exists to promote intervention allowing containment of the epidemic at onset.

5.3 | Infection spread and economic development: The growth equilibria with endemic infection (GEEI)

As map T always admits an attractor at finite distance and the IFE can be locally unstable, as shown in the previous section, we now investigate the existence of other attractors characterised by a strictly positive infection prevalence. This is also motivated by the fact that Chakraborty et al.'s equation can have an *endemic equilibrium* (at most one), as shown in

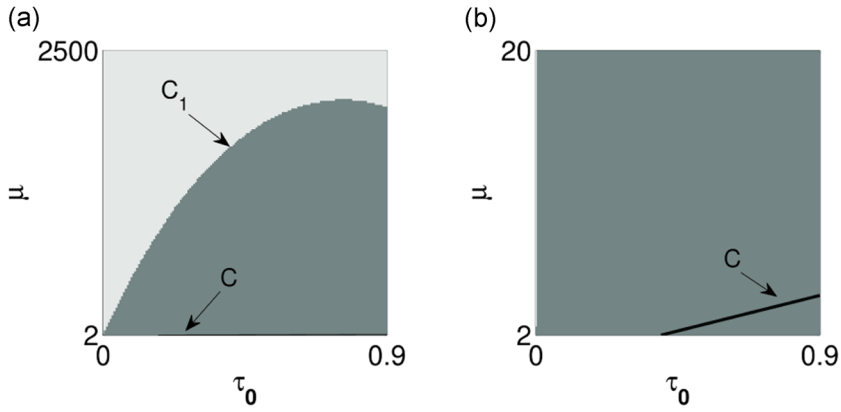

FIGURE 2 Stability boundary of the IFE in the (τ_0, μ) plane. (a) the long term dynamics for a given initial conditions are depicted in different colors for different combinations of couples (τ_0, μ). The dark grey region captures long-term dynamics converging to the IFE. The light grey region captures long-term dynamics converging to a different attractor. (b) enlarged view of a portion of the dark-grey region in which line C is depicted. Parameter values: $\alpha = 0.67, A = 24.18, \pi_0 = 0.2, \pi_1 = 35, \beta = 0.28, \phi = 0.62, b = 20$ and $\tau_1 = 0.1$. IFE, infection-free equilibrium

Section 4. Such endemic equilibria would represent situations where the infection persists in the economy in the long term.

5.3.1 | Fixed point on the boundaries of the state space

A preliminary question is related to the existence of other fixed points on the boundaries of set S. In relation to this, it can be easily observed that if $b = 0$ then $T(0, y) = (0, y')$. This situation describes the infection evolution in a "poorest-poor" economy with GDP equal to zero. As this deals with the case of a freely circulating infection due to the impossibility to enact any control interventions (given the lack of resources), Lemma 1 applies. Consequently, the following proposition holds for this case.

Proposition 3. *Let system T given by (19). If $b = 0$ then set $N = \{(x, y) \in S : x = 0\}$ is positively invariant.*

If $b = 0$ the dynamics of T on N are described by the map:

$$F_N(y) = g(0, y) = 1 - (1 - \pi_0 y)^{\mu}, y \in [0, 1],$$

whose dynamics are exhaustively described by the properties of Chakraborty et al.'s equation in Lemma 1. Moreover, the transverse eigenvalue associated to N, $\lambda_1 = \frac{\partial f(x,y)}{\partial x}|_{x=0}$, tends to $+\infty$ as $x \to 0^+, \forall\, y > 0$. As a consequence N is a repelling set so that in the long term an initial positive capital per worker cannot produce dynamics converging to a situation with zero capital per worker regardless of the level of infection prevalence, as expected from the basic nature of the model. Therefore, once the economy lies on a trajectory with positive capital accumulation,

it will become capable to supply resources for health expenditure and to provide some control of infection. To sum up, unlike Gori et al. (2019) this model is not able to generate a poverty trap induced by the infection.

Let us finally consider set $J = \{(x, y) \in S : y = 1\}$, which describes the dynamics of the economy in the presence of the upper bound of prevalence ($y = 100\%$). As $g(x, 1) < 1 \; \forall \; x \geq 0$, all initial conditions characterised by 100% prevalence ("total infection"), that is, $(x(0), 1)$, leave J at the first iteration thus proving that in the long term no economically meaningful situations with 100% prevalence can occur.

5.3.2 | Proper endemic equilibria

Let us now investigate the existence of proper equilibria with strictly positive (and strictly lower than 100%) infection prevalence. This would represent (in the event they are stable) situations where the infection is *endemic* and persistently frightens the population in an economy which is by itself at a stationary equilibrium with a strictly positive capital per worker. We term these equilibria as *growth equilibria with endemic infection* (GEEI) and generally define them by $E_P^* = (x_P^*, y_P^*) \in S$, where $x_P^* > 0$ and $y_P^* \in (0, 1)$. Given the nonlinearity of map T, one has to tackle the following two main issues, namely the number of GEEIs and their local stability properties. As their investigation is analytically cumbersome, in what follows we combine analytical remarks with numerical simulations focusing on the role played by the two key parameters of the model, namely the number of at risk contacts μ and the (exogenous) health tax rate τ_0 along the lines exposed in Section 5.2.

Regarding the first issue, that is, number of GEEIs, extensive numerical simulations suggested that if map T admits a GEEI then it is unique. Though this could not be proved analytically, much insight can be obtained by the analysis of two limit maps. The first one is associated to $\mu \to 1^+$ implying that $R_c(x) \to \frac{\pi_0}{1 + \tau_0 \pi_1 w(x)} < 1$, meaning that the infection has no reproduction potential irrespective of the economic conditions.[2] In such a case, system T tends to system T_1, whose second equation is given by

$$y' = g_1(x, y) = \frac{y}{1 + (\tau_0 + \tau_1 y)\pi_1 [(1 - \alpha)Ax^\alpha + b]}.$$

The previous equation has a unique solution for all $x \geq 0$ given by $y = 0$. Therefore, no fixed points characterised by strictly positive infection prevalence exist for T_1. As system T is continuous with respect to both the state variables and the system parameters, then $\exists \; \mu_m$ such that no GEEI exists for all μ such that $1 < \mu < \mu_m$, where μ_m can be obtained numerically from Equation (25). In such a case, based on previous results, only the IFE exists and attracts all economically meaningful initial conditions, that is, $(x(0), y(0)), x(0) > 0$. *The substantive intuition of this result is that endemic equilibria can only establish provided that each agent has more than one adequate (social or sexual) contact during his infective lifetime.*

[2]Theoretically speaking, μ can range $[0, +\infty)$, where the lower bound corresponds to a situation of a perfectly segregated community. Note however that if $\mu = 1$, then $R_0 = \pi_0 < 1$, meaning that the infection is not productive even in the absence of any control intervention. An infection with such a characteristic would never be observed. Therefore, $\mu = 1$ represents the relevant lower bound for our analysis.

Next, let us consider the case $\mu \to \infty$. Then $\gamma(x, y) \to 0$ and system T tends to the following limiting form:

$$T_\infty : \begin{cases} x' = f_\infty(x, y) = [1 - (\tau_0 + \tau_1)][(1 - \alpha)Ax^\alpha + b]\left[\frac{\beta\phi}{1 + \phi\beta}\right], \\ y' = g_\infty(x, y) = 1, \end{cases} \tag{29}$$

where the population is entirely infected and set $J = \{(x, y) \in S : y = 1\}$ is invariant, as shown above. *In this case, the substantive intuition is that the infection always becomes endemic around a stable equilibrium where every adult individual is infected*. In relation to this case, consider now an initial condition $(x(0), y(0)) \in S$, then $T_\infty(x(0), y(0)) = (x(1), 1)$, that is, all initial conditions are mapped into J at the first iterate. The dynamics of capital on J are described by map f_∞, where f is the first component of two-dimensional map (19). About the fixed points of f_∞ and its dynamics, considerations analogous to those related to set K_{IFE} imply that if $b > 0$ there exists $x_J^* > 0$ that is globally attracting on set J, while if $b = 0$ the point $x_J^* > 0$ still exists and attracts all trajectories starting from J with $x(0) > 0$. This proves the following proposition about the long-term dynamics of system T_∞.

Proposition 4. *Let T_∞ be given by (29). Then set $J = \{(x, y) \in S : y = 1\}$ is invariant and globally attracting. If $b > 0$ then all initial conditions produce trajectories converging towards the unique steady-state equilibrium $E_J^* = (x_J^*, 1), x_J^* > 0$. If $b = 0$ then E_J^* will attract all trajectories starting from $S - \{(0, y(0))\}$.*

As it has been previously pinpointed, no steady states of system T can belong to set J. However, the limiting map T_∞ always admits a fixed point with total infection. Let us now introduce the following definition.

Definition 2. The steady state $E_J^* = (x_J^*, 1)$ appearing as the limiting case described by system T_∞ is the total infection equilibrium of the economy (TIE).

The previous results allow us to conclude that if $\mu \to +\infty$ then GEEI $\to$ TIE. By recalling that for finite values of μ the TIE cannot be a steady state of map T. However, in view of the continuity of T, if μ is large enough, that is, $\mu > \mu^M$, then a unique stable GEEI exists. This equilibrium tends to the TIE when μ increases without bounds.

The results just discussed about the two limiting maps T_1 and T_∞ prove the following Proposition.

Proposition 5. *Let T be given by (19). Then $\exists$ (μ_m, μ_M) both belonging to $(1, +\infty)$, with $\mu_m < \mu_M$, such that:*

(i) *if $\mu \in (1, \mu_m)$ then there exists no GEEI and the IFE attracts all trajectories starting from $x(0) > 0$;*
(ii) *if $\mu > \mu_M$ then, besides the IFE, there exist a unique GEEI. The GEEI is locally (and globally) stable and the IFE is unstable.*

The panels of Figure 3 illustrate the previous findings by considering the equilibrium isoclines of map $T = (x', y')$, that is the sets of points where $x' = x$ and $y' = y$, for three pairs of values of the key parameters μ and τ_0. Note that the isocline $y' = y$ (bolded black) is given by two branches: the x axis and a downward-sloping curve. Points between the two branches are characterised by increasing prevalence, while points above the second branch correspond to decreasing prevalence. The isocline $x' = x$ (bolded dashed black) is also a decreasing curve. Figure 3a reports the benchmark case $\mu = 6000$ (as used by Chakraborty et al., 2016) and

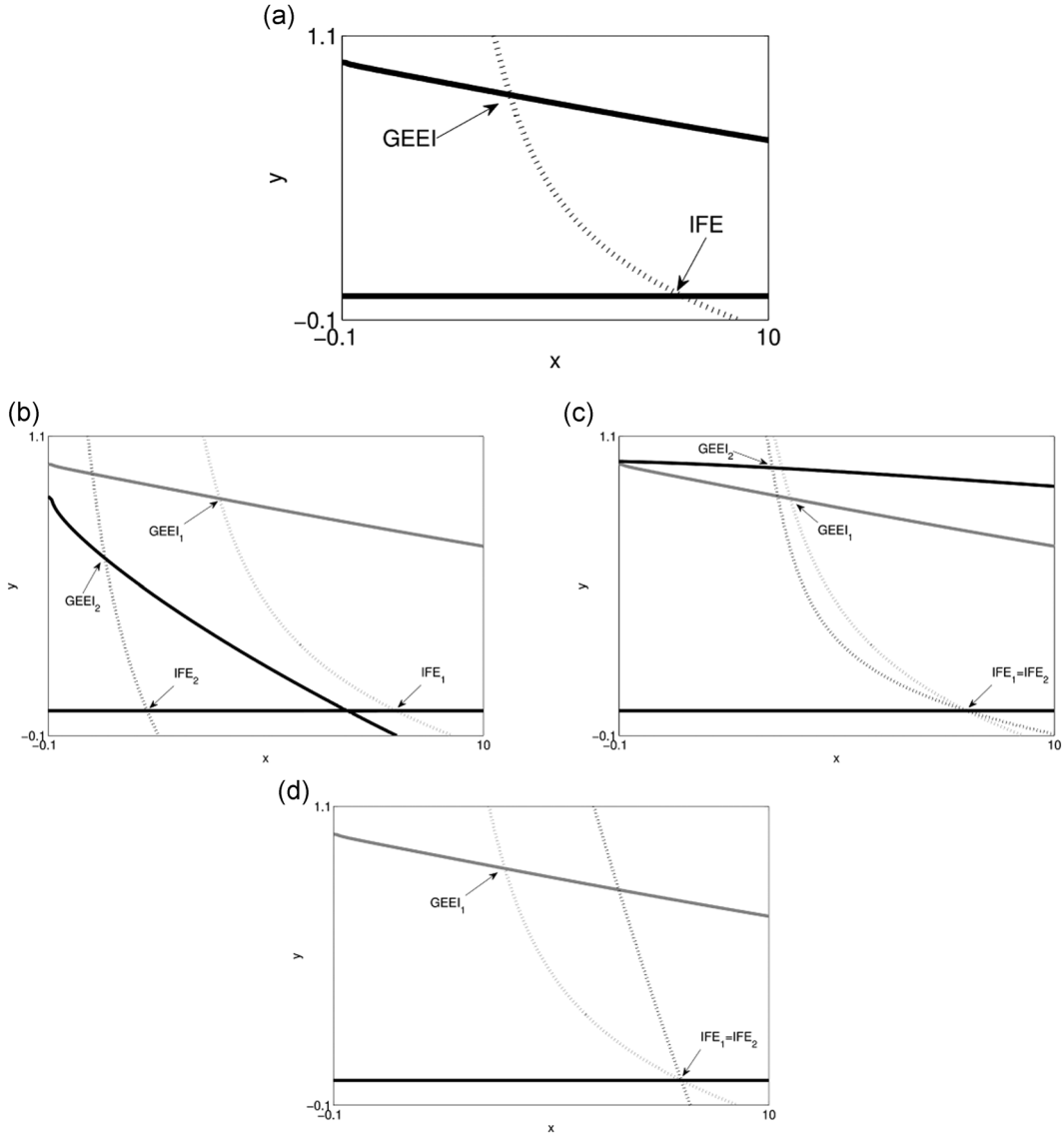

FIGURE 3 Sensitivity of IFE and GEEI (as intersections between the relevant isoclines) for different values of the critical parameters μ and τ_0. (a) The baseline case $\mu = 6000$ (as used by Chakraborty et al., 2016) and $\tau_0 = 0.3$. (b) Increases τ_0 to 0.7 (μ kept constant). (c) Increases μ to 10000 (τ_0 kept constant). (d) Decreases μ to 60 (τ_0 kept constant). Other parameter values: $\alpha = 0.67$, $A = 24.18$, $\pi_0 = 0.2$, $\pi_1 = 35$, $\beta = 0.28$, $\phi = 0.62$, $b = 20$ and $\tau_1 = 0.1$. GEEI, growth equilibria with endemic infection; IFE, infection-free equilibrium

$\tau_0 = 0.3$, showing the existence of a unique GEEI, as the inner intersection between the two isoclines, and the IFE being the intersection point between the two isoclines along the x-axis. If τ_0 is increased (Figure 3b), no qualitative change is observed. This new case is depicted in black leaving the previous case in gray for comparison purposes (the x-axis is an isocline for both cases). In quantitative terms, both equilibrium capital and infection prevalence decline as large public prevention investments reduce the long-term infection prevalence trading off however with lower capital and GDP. Figure 3c shows the effects of a higher value of μ implying a higher equilibrium infection prevalence and a lower capital per worker at the unique GEEI. Finally, Figure 3d presents the situation corresponding to a sufficiently small level of μ so that the IFE is the unique equilibrium point, as stated in part (i) of Proposition 5.

6 | SYSTEM BEHAVIOUR

In this section, we illustrate the system behavior (Figures 4 and 5) by resorting to one-dimensional bifurcation diagrams reporting the steady-state levels $x^*(.), y^*(.)$ of the two state variables (x, y). In these diagrams, we represent the values of $x^*(.), y^*(.)$ as one-dimensional functions of the three key parameters used throughout the work namely, (i) the number of lifetime contacts μ, (ii) the exogenous tax rate τ_0, (iii) the endogenous taxation coefficient τ_1. Additionally, we look at how the resulting diagrams are perturbed when the other two parameters are (separately) varied. The diagrams are generated by simulating the joint economic-epidemiology dynamics represented by map T for different parameter values by departing from a positive initial condition on both x and y (we recall that if the infection prevalence is equal to zero at $t = 0$, it will remain zero for all $t > 0$ and the economy will approach the IFE). Given the theoretical spirit of this study, the adopted parameter values are not intended to represent any real-world situations but rather aim to be illustrative of the spectrum of possible scenarios generated by the interplay of the infection and the economy.

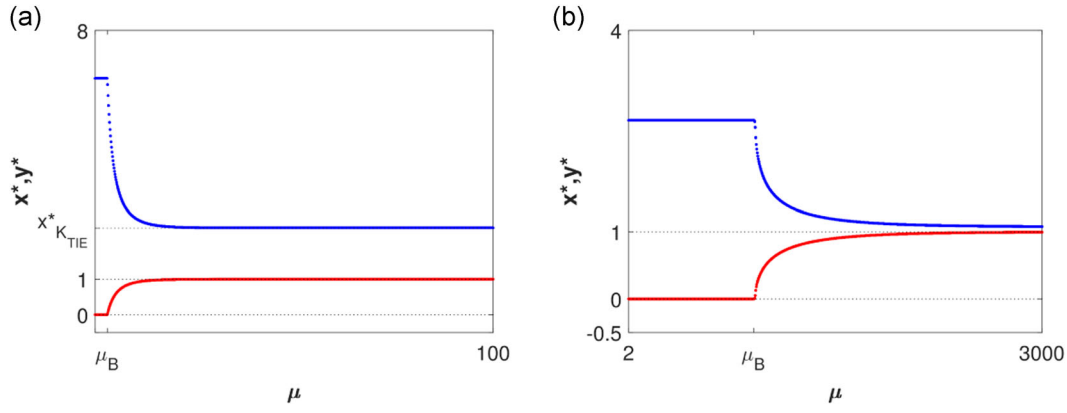

FIGURE 4 One-dimensional bifurcation diagrams of the system equilibrium levels of capital and infection prevalence as functions of μ. Red curve: equilibrium infection prevalence; blue curve: equilibrium capital per worker. (a) The baseline case of no intervention ($\tau_0 = \tau_1 = 0$), showing a bifucation level of $\mu_B \simeq 5$. (b) The case of intervention based on exogenous taxation only ("containment at onset") for $\tau_0 = 0.3$. In this case $\mu_B \simeq 910$. Other parameter values: $\alpha = 0.67, A = 24.18, b = 2, \pi_0 = 0.2, \pi_1 = 35, \beta = 0.28$, and $\phi = 0.62$

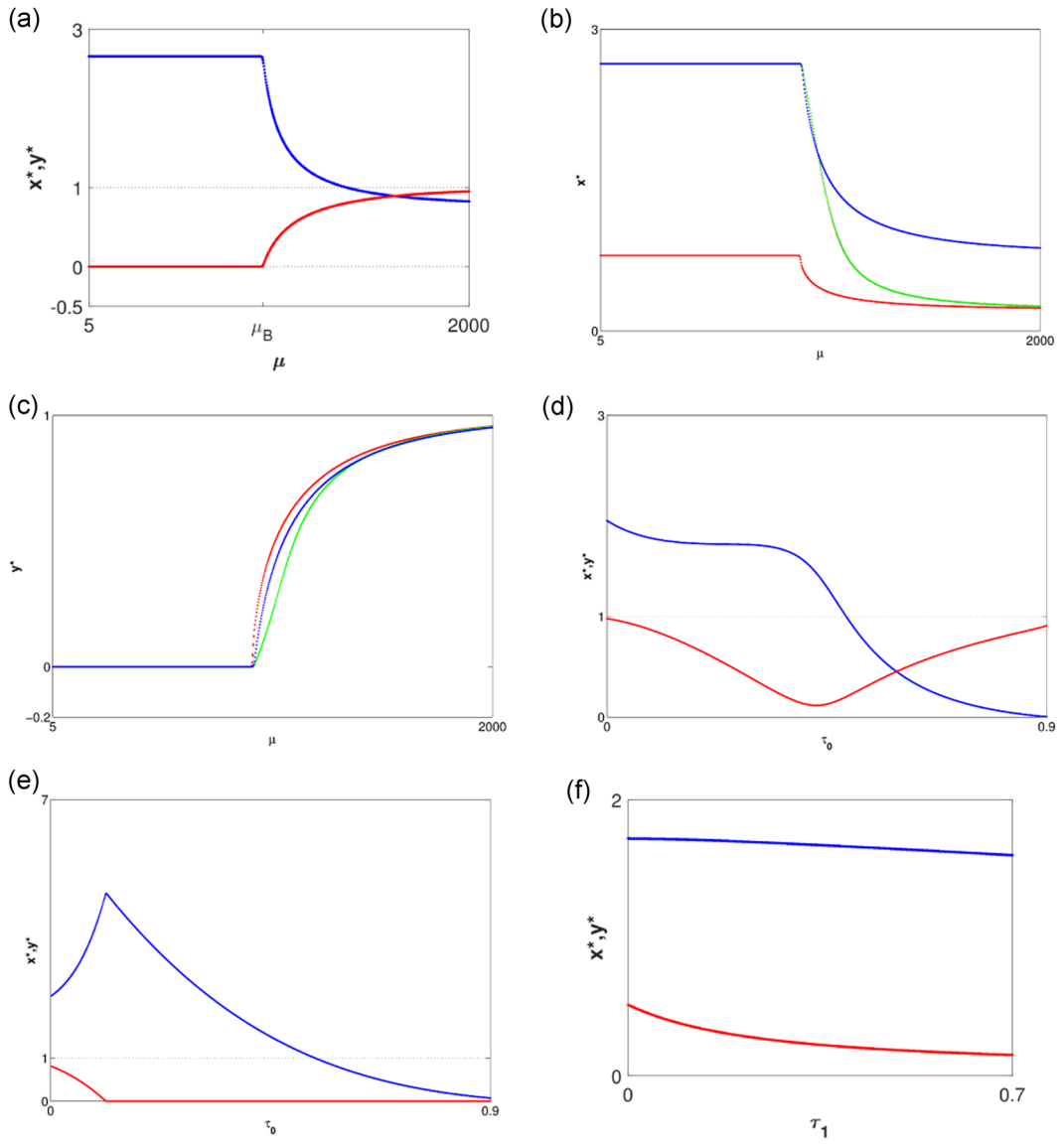

FIGURE 5 One-dimensional bifurcation diagrams of the system equilibrium levels of capital and infection prevalence as functions of the main infection or control parameters. (a) One-dimensional bifurcation diagram as a function of μ ($\tau_0 = 0.3$, $\tau_1 = 0.1$). In particular $\mu_B \simeq 917.7$. Red curve: equilibrium infection prevalence; blue curve: equilibrium capital per worker. (b) One-dimensional bifurcation diagram of equilibrium capital as a function of μ by considering the parameter values as in (a) (blue), $\tau_0 = 0.6$ (red) or $\tau_1 = 0.4$ (green). (c) One-dimensional bifurcation diagram of equilibrium prevalence as a function of μ by considering the parameter values as in (a) (blue), $\tau_0 = 0.6$ (red) or $\tau_1 = 0.4$ (green). (d) and (e) One-dimensional bifurcation diagrams of the system equilibrium levels of capital and infection prevalence as functions of τ_0 when $\mu = 1000$ (d) and $\mu = 500$ (e) $\tau_1 = 0.1$. (f) One-dimensional bifurcation diagrams of the system equilibrium levels of capital and infection prevalence as functions of τ_1 ($\tau_0 = 0.3$ and $\mu = 1000$). Other parameters values: $\alpha = 0.67$, $A = 24.18$, $b = 2$, $\pi_0 = 0.2$, $\pi_1 = 35$, $\beta = 0.28$ and $\phi = 0.62$

Figure 4a reports the bifurcation diagram $x^*(\mu)$ and $y^*(\mu)$ (capital per worker: blue line, infection prevalence: red line) drawn for the baseline case of a free infection in the absence of any interventions ($\tau_0 = \tau_1 = 0$). The figure shows the existence of a threshold value μ_B in the lifetime number of at risk contacts (and consequently in the basic reproduction number of the infection) such that the IFE—which was stable for $\mu < \mu_B$—loses its stability (through a saddle-node bifurcation at $\mu = \mu_B$) and a GEEI (the positive increasing branch of the prevalence curve) emerges that is locally stable for any $\mu > \mu_B$. For $\mu > \mu_B$ the curve $y^*(\mu)$ is increasing and concave (in the same way as in classical SIS and SIR epidemiological models with a stable endemic state) showing that a larger μ implies, other parameters being equal, a larger equilibrium prevalence. Correspondingly, the curve $x^*(\mu)$ is constant at its infection-free equilibrium for any $\mu < \mu_B$, and it is monotonically declining for $\mu > \mu_B$. In words, as far as μ lies below the threshold μ_B, the IFE is stable, that is, the infection cannot spread and is eventually eliminated as its basic reproductive rate R_0 will lie below unity, and both capital accumulation and GDP are unaffected by the disease. However, as soon as the number of lifetime contacts overtakes the threshold μ_B, the infection becomes endemic with an equilibrium prevalence which is strictly increasing in μ (and, other things being equal, in R_0). This leads to a decreasing equilibrium capital stock due to the larger burden of disease mortality (tuned by the infection extra mortality ϕ) resulting from the larger prevalence caused by the higher value of μ.

Figure 4b illustrates the case of a baseline, nonprevalence dependent intervention, that is, one purely aimed to containment at onset by exogenous taxation. Things go much in the same way as Figure 4a (note however the expansion of the scale of the right axis). The larger the infection prevalence at equilibrium, the larger the amount of resources allocated for public prevention and the lower the resulting accumulation of capital.

More articulated prevention policies can be obtained by varying either or both taxation parameters. Their effects are reported in Figure 5a–f. Such policies can aim to (i) further strengthening the ability of the economy to contain the epidemic risk at onset, or (ii) further reducing the equilibrium prevalence of infection (if prevalence is positive) and correspondingly increasing capital accumulation at equilibrium. As was explained in the theoretical discussion of the previous section, target (i) can only be achieved by further increasing the exogenous tax rate τ_0 and is totally insensitive to the level of τ_1, while target (ii) should mainly be achieved by tuning τ_1. Figure 5a amends Figure 4b by adding some prevalence-dependent taxation ($\tau_1 = 0.1$) showing no effects at all on the bifurcating value of μ.

Next, we investigate the effects of increasing τ_0 ($\tau_0 = 0.6$, τ_1 being fixed) and τ_1 ($\tau_1 = 0.4$, τ_0 being fixed). This is shown in Figure 5b, which reports the equilibrium levels of capital per worker, $x^*(\mu)$, and Figure 5c, which reports the equilibrium prevalence, $y^*(\mu)$. The blue curves represent the benchmark scenario of Figure 5a. The red curves refer to an increase of τ_0 only. The green curves refer to an increase of τ_1 only. While the effects on capital per worker are expected, the effects on equilibrium prevalence $y^*(\mu)$ are not necessarily intuitive. On the one hand, the endogenous prevention action has some moderate success only at low and intermediate prevalences (i.e., for low-moderate values of μ), and is totally ineffective thereafter. This is expected as very large values of μ imply, in presence of a prevalence-dependent control action (funded by the τ_1 component), larger and larger control efforts that in turn cause a harmful effect on capital accumulation and therefore on resources available for prevention. On the other hand, increasing the exogenous prevention effort always increases endemic prevalence (i.e., for all values of μ in the increasing branch of the $y^*(\mu)$ curve), again suggesting a dominance of the general equilibrium effect on capital accumulation and related available prevention resources.

Figure 5d,e illustrate the nice effects of τ_0 as a bifurcation parameter for widely different levels of μ (Figure 5d, $\mu = 1000$; Figure 5e, $\mu = 500$). Consider first $\mu = 1000$. In this case, the infection is highly reproductive and no value of τ_0 exists capable to achieve elimination. Nonetheless, the pattern of infection prevalence (as a function of τ_0) is complicated: departing from the case of no intervention, increasing levels of exogenous taxation allow to decrease prevalence up to a threshold level beyond which further intervention becomes counterproductive due to its negative general equilibrium effects on capital accumulation (and consequently on resources available for prevention). The pattern of capital per worker is even more interesting as it is convex for low levels of τ_0, then it increases mildly at intermediate levels of taxation (when the general equilibrium effect is not important yet), and finally declines fast (to zero) when both the direct and indirect effects of taxation add up, eventually fully eroding capital accumulation and prevention resources. Instead, when $\mu = 500$, the infection is less reproductive and there exists a value of τ_0 capable to achieve elimination, as predicted previously (Section 5). Beyond this threshold level, any preventive intervention will be counterproductive and therefore only harmful for capital accumulation and prevention due to the full dominance of the general equilibrium effect.

Additionally, an intervention only expanding endogenous taxation expenditures τ_1 has straightforward effects on endemic prevalence and equilibrium accumulation of capital (Figure 5f). Overall, Figure 5f confirms the low effectiveness of endogenous prevention.

7 | CONCLUDING REMARKS

This article was written before the explosion of economic epidemiology works in the COVID era. At that time, the economics of infectious diseases was a niche with quite a few contributions dealing with a few topics, such as the effects of vaccination refusal and the free-rider problem or the interplay between health and economic development with special focus on SSA. This study has the aims of (1) providing a general approach to the interplay between the dynamics of communicable diseases and economic growth and development when intervention activities are carried out within a public health system financed by general taxation, (2) trying to shed light on the complicated effects of infectious diseases pinpointing the most appropriate (public) interventions to be used to fight current and future epidemics.

Surprisingly, the critical topic dealt with in this article has not yet received in-depth attention in the macroeconomic literature. We departed from the seminal contribution of Chakraborty et al. (2010), who made a nice methodological proposal allowing to combine in a single unified OLG framework the dynamics of a developing economy with the one of a range of infectious diseases. Nonetheless, we identified two main shortcomings in their work. The first one is the emphasis on what they call in their introduction the "rational approach to disease prevention." Their agents rationally choose their prevention expenditure against communicable diseases at the beginning of their active life. This suffers a twofold criticism: it can hold only for a few modern industrialised countries, and it ignores that interventions against infectious diseases worldwide have always been carried out with public resources through ad hoc public measures. The second one relates to the fact that though they developed a nice framework, they virtually did not provide any theoretical results by mainly resorting to simulations.

This study has attempted to deal with these shortcomings. To this aim, we developed an alternative framework to Chakraborty et al.'s contributions where we considered a deadly

communicable infection whose prevention is entirely carried out within the framework of a public health system designing public intervention programmes and financing them by taxation. This intervention aims at reducing the probability of infection transmission per single social or sexual contact. The financing of the policy is enacted by two different fiscal tools. The first one is based on general taxation on labour income. The second one is based on a disease-specific tool, which we defined prevalence-dependent labour income taxation.

Our results appear to have a general-purpose. First, we enlightened the relationships governing the interplay between typical epidemiological phenomena, namely epidemic invasion versus endemicity, and public interventions, their financing through taxation, and their general equilibrium repercussions on capital accumulation. Second, on the issue of invasion, we identified the threshold quantities allowing containment of a fatal epidemic at onset and the dependencies of these thresholds on taxation parameters. Notably, these results extended to a more articulated modelling setting combining macroeconomic and epidemic dynamics the classical findings of mathematical epidemiology (Capasso, 1990), namely the duality between an IFE and the presence of an endemic state mediated by a suitable threshold parameter. As for endemicity, the merit of a general approach including the dynamics of the economy is that of enlightening the complicated relationships between the disease and the economy that emerge at the general equilibrium level. The most important result here is that while in a purely *descriptive* epidemiological setting ignoring the macroeconomic structure, a larger degree of control would always be beneficial because only the direct effect of intervention would be considered, instead in a general equilibrium setting this does not need to be the case. Indeed, as shown here, costly interventions might erode accumulation and eventually negatively feedback on resources available for prevention. More precisely, we showed that intervention policies are always effective up to some intervention thresholds above which the policy returns become negative because taxation reduces resources available for development, thereby causing a strong negative effect on individual available income and eventually on prevention resources. Though these general equilibrium effects might hardly be prominent in industrialised countries, they are well evident in poor-resource settings especially after the HIV-AIDS crisis in SSA. Indeed, economic studies of the epidemiology and control of HIV documented that the order of magnitude of the financial resources needed for bringing the epidemic under full control would be in the range of the entire GDP of those countries (Remme et al., 2016; Resch et al., 2015).

Our analysis showed that general versus prevalence-dependent fiscal tools is by no means equivalent, rather they must be considered as highly specific tools to be used for adequately targeted interventions. A policy issue emerging from previous remarks is that situations where development is severely compromised by the burden of multiple coexisting infections as SSA, possibly cannot afford infection control by using domestic resources, which make generalised international donation a fundamental tool for fighting communicable diseases without compromising chances to develop (Gori, Manfredi, & Sodini, 2021).

The present model can be considered as a template to be improved in future works. For example, we could not find articulated dynamics, including poverty traps, multiple endemic states and multistability, which might appear by adding more realistic representations of the demo-economics. The model did not include demographic dynamics and therefore did not allow to account for the possible effects of the infection on demographic variables such as mortality and fertility. The analysis of this issue will be the object of future works. Preliminary efforts in this direction—devoted to the study of the impact of HIV/AIDS on the fertility transition in SSA—are Gori et al. (2020) and Gori, Manfredi, and Sodini (2021).

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CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

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REFERENCES

Acemoglu, D., Chernozhukov, V., Werning, I., & Whinston, M. D. (2021). Optimal targeted lockdowns in a multi-group SIR model. *American Economic Review: Insights*, *3*, 487–502.

Alvarez, F. E., Argente, D., & Lippi, F. (2021). A simple planning problem for COVID-19 lockdown. *American Economic Review: Insights*, *3*, 367–382.

Anderson, R., & May, R. (1991). *Infectious diseases of humans: Dynamics and control*. Oxford University Press.

Anderson, S. T., Laxminarayan, R., & Salant, S. W. (2013). Diversify or focus? Spending to combat infectious diseases when budgets are tight. *Journal of Health Economics*, *31*, 658–675.

Arrow, K. J. (1963). Uncertainty and the welfare economics of medical care. *American Economic Review*, *53*, 941–973.

Auray, S., & Eyquem, A. (2020). The macroeconomic effects of lockdown policies. *Journal of Public Economics*, *190*, 104260.

Azomahou, T. T., Bouccekkine, R., & Diene, B. (2016). HIV/AIDS and development: A reappraisal of the productivity and factor accumulation effects. *American Economic Review*, *106*, 472–477.

Bailey, N. (1975). *The mathematical theory of infectious diseases and its applications* (2nd ed.). Hafner Press.

Bärnighausen, T., Bloom, D. E., Cafiero-Fonseca, E. T., & O'Brien, J. C. (2014). Valuing vaccination. *Proceedings of the National Academy of Sciences of the United Sates of America*, *111*, 12313–12319.

Bell, C., & Gersbach, H. (2009). The macroeconomics of targeting: The case of an enduring epidemic. *Journal of Health Economics*, *28*, 54–72.

Bell, C., & Gersbach, H. (2013). Growth and enduring epidemic diseases. *Journal of Economic Dynamic and Control*, *37*, 2083–2103.

Bloom, D. E., & Canning, D. (2004). The health and wealth of Africa. *World Economics*, *5*, 57–81.

Bloom, D. E., Fan, V. Y., & Sevilla, J. P. (2018). The broad socioeconomic benefits of vaccination. *Science Translational Medicine*, *10*, eaaj2345.

Bosi, S., & Desmarchelier, D. (2018). Pollution and infectious diseases. *International Journal of Economic Theory*, *14*, 351–372.

Boucekkine, R., Desbordes, R., & Latzer, H. (2009). How do epidemics induce behavioral changes? *Journal of Economic Growth*, *14*, 233–264.

Boucekkine, R., & Laffargue, J. P. (2010). On the distributional consequences of epidemics. *Journal of Economic Dynamic and Control*, *34*, 231–245.

Brito, D. L., Sheshinski, E., & Intriligator, M. D. (1991). Externalities and compulsary vaccinations. *Journal of Public Economics*, *45*, 69–90.

Capasso, E. (1990). *The mathematical structure of epidemic systems*. Springer.

Chakraborty, S. (2004). Endogenous lifetime and economic growth. *Journal of Economic Theory*, *116*, 119–137.

Chakraborty, S., Papageorgiou, C., & Pérez Sebastián, F. (2010). Diseases, infection dynamics, and development. *Journal of Monetary Economics*, *57*, 859–872.

Chakraborty, S., Papageorgiou, C., & Pérez Sebastián, F. (2016). Health cycles and health transitions. *Macroeconomic Dynamics*, *20*, 189–213.

Corrigan, P., Glomm, G., & Mendez, F. (2005). AIDS crisis and growth. *Journal of Development Economics*, *77*, 107–124.

Diamond, P. A. (1965). National debt in a neoclassical growth model. *American Economic Review*, *55*, 1126–1150.

Eichenbaum, M. S., Rebelo, S., & Trabandt, M. (2020). *The macroeconomics of epidemics* (NBER Working Paper No. 26882).

Fenichel, E. P. (2013). Economic considerations for social distancing and behavioral based policies during an epidemic. *Journal of Health Economics*, *32*, 440–451.

Francis, P. J. (1997). Dynamic epidemiology and the market for vaccinations. *Journal of Public Economics*, *63*, 383–406.

Gersovitz, M. (2013). Mathematical epidemiology and welfare economics. In P. Mafredi, & A. D'Onofrio (Eds.), *The impact of human behavior on the transmission and control of infectious diseases* (pp. 185–202). Springer.

Gersovitz, M., & Hammer, J. S. (2003). Infectious diseases, public policy, and the marriage of economics and epidemiology. *World Bank Research Observer*, *18*, 129–157.

Gessner, B. D., Kaslow, D., Louis, J., Neuzil, K., O'Brien, K. L., Picot, V., Pang, T., Parashar, U. D., Saadatian-Elahi, M., & Nelson, C. B. (2017). Estimating the full public health value of vaccination. *Vaccine*, *35*, 6255–6263.

Goenka, A., & Liu, L. (2012). Infectious diseases and endogenous fluctuations. *Economic Theory*, *50*, 125–149.

Goenka, A., & Liu, L. (2020). Infectious diseases, human capital and economic growth. *Economic Theory*, *70*, 1–47.

Goenka, A., Liu, L., & Nguyen, M.-H. (2014). Infectious diseases and economic growth. *Journal of Mathematical Economics*, *50*, 34–53.

Goenka, A., Liu, L., & Nguyen, M.-H. (2021). SIR economic epidemiological models with disease induced mortality. *Journal of Mathematical Economics*, *93*, 102476.

Gollier, C. (2020). Cost-benefit analysis of age-specific deconfinement strategies. *Journal of Public Economic Theory*, *22*, 1746–1771.

Gori, L., Lupi, E., Manfredi, P., & Sodini, M. (2020). A contribution to the theory of economic development and the demographic transition: Fertility reversal under the HIV epidemic. *Journal of Demographic Economics*, *86*, 125–155.

Gori, L., Manfredi, P., Marsiglio, S., & Sodini, M. (2021). COVID-19 epidemic and mitigation policies: Positive and normative analyses in a neoclassical growth model. *Journal of Public Economic Theory*. (forthcoming). https://doi.org/10.1111/jpet.12549

Gori, L., Manfredi, P., & Sodini, M. (2019). HIV/AIDS, demography and development: Individual choices versus public policies in SSA. In A. Bucci, A. Fürnkranz-Prskawetz, & K. Prettner (Eds.), *Human capital and economic growth: The impact of health, education and demographic change* (Chapter 10, pp. 319–352). Palgrave MacMillan.

Gori, L., Manfredi, P., & Sodini, M. (2021). A parsimonious model of longevity, fertility, HIV transmission and development. *Macroeconomic Dynamics*, *25*, 1155–1174.

Greenwood, J., Kircher, P., Santos, C., & Tertilt, M. (2019). An equilibrium model of the African HIV/AIDS epidemic. *Econometrica*, *87*, 1081–1113.

Katz, I., Routh, S., Bitran, R., Hulme, A., & Avila, C. (2014). Where will the money come from? Alternative mechanisms to HIV donor funding. *BMC Public Health*, *14*, e956.

Kremer, M. (1996). Integrating behavioral choice into epidemiological models of AIDS. *Quarterly Journal of Economics*, *111*, 549–573.

Li, X., Mukandavire, C., Cucunubá, Z. M., Londono, S. E., Abbas, K., Clapham, H. E., et al., & Vaccine Impact Modelling Consortium. (2021). Estimating the health impact of vaccination against ten pathogens in 98 low-income and middle-income countries from 2000 to 2030: A modelling study. *Lancet*, *397*, 398–408.

Livi-Bacci, M. (2017). *A concise history of world population* (6th ed.). Wiley.

Lorentzen, P., McMillan, J., & Wacziarg, R. (2008). Death and development. *Journal of Economic Growth*, *13*, 81–124.

Manfredi, P., & D'Onofrio, A. (Eds.). (2013). *Modeling the interplay between human behavior and the spread of infectious diseases*. Springer-Verlag.

Manski, C. F. (2017). Mandating vaccination with unknown indirect effects. *Journal of Public Economic Theory*, *19*, 603–619.

Momota, A., Tabata, K., & Futagami, K. (2005). Infectious disease and preventive behavior in an overlapping generations model. *Journal of Economic Dynamics and Control*, *29*, 1673–1700.

Murray, et al. (2017). Global, regional, and national under-5 mortality, adult mortality, age-specific mortality, and life expectancy, 1970-2016: A systematic analysis for the Global Burden of Disease Study 2016. *Lancet*, *390*, 1084–1150.

Neumann, P. J., Jacobson, P. D., & Palmer, J. A. (2008). Measuring the value of public health systems: The disconnect between health economists and public health practitioners. *American Journal of Public Health*, *98*, 2173–2180.

Philipson, T. (2000). Economic epidemiology and infectious disease. In A. J. Cuyler, & J. P. Newhouse (Eds.), *Handbook of health economics* (Vol. 1B, pp. 1761–1799). North Holland.

Porter, D. (2005). *Health, civilization and the state: A history of public health from ancient to modern times*. Routledge.

Remme, M., Siapka, M., Sterck, O., Ncube, M., Watts, C., & Vassall, A. (2016). Financing the HIV response in sub-Saharan Africa from domestic sources: Moving beyond a normative approach. *Social Science & Medicine*, *169*, 66–76.

Resch, S., Ryckman, T., & Hecht, R. (2015). Funding AIDS programmes in the era of shared responsibility: An analysis of domestic spending in 12 low-income and middle-income countries. *Lancet Global Health*, *3*, 52–61.

Stiglitz, J. E. (1988). *Economics of the public sector*. Norton.

UN. (2021). World population prospects.

van Wijhe, M., McDonald, S. A., de Melker, H. E., Postma, M. J., & Wallinga, J. (2016). Effect of vaccination programmes on mortality burden among children and young adults in the Netherlands during the 20th century: A historical analysis. *Lancet Infectious Diseases*, *16*, 592–598.

WHO. (2019). Communicable diseases. Fact sheets. www.who.org.

WHO. (2021). Weekly Epidemiological Update—September 2021.

Young, A. (2005). The gift of the dying: The tragedy of AIDS and the welfare of future African generations. *Quarterly Journal of Economics*, *120*, 423–466.

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APPENDIX A

This appendix reports some basic mathematical results about the economic-epidemiological model presented in the main text.

A.1 | Well-posedness, boundedness, and existence of an attractor for map T

We report here the main technical results on the well-posedness, boundedness and existence of an attractor for the model described by Equations (19) and (20). Regarding the feasibility of (T, S), the following proposition holds.

Proposition 6. *Let T be given by (19). Then set S is positively invariant.*

Proof. Let $x \geq 0, y \in [0,1]$. Then, taking into account equation $g(x,y)$ in (19), $y' \in [0, 1-(1-\pi_0)^{\mu}] \subset [0,1]$. Furthermore, as $f(x,y) \geq 0, \forall\, (x,y) \in S$, then $x' \geq 0$. Hence $S = \mathbb{R}_+ \times [0,1]$ is positively invariant. □

According to Proposition 6 we have the following remarks.

- As feasibility conditions hold for all parameter values and for any $t \in \mathbb{N}$, the model is well posed.
- If map T admits an attractor at finite distance Λ then $\Lambda \subseteq S$. As $T(S) \subseteq \mathbb{R}_+ \times [0, 1-(1-\pi_0)^{\mu}] := S_1$ then $\Lambda \subseteq S_1$.

To investigate the existence of an attractor Λ for map T and describe the long-term evolution of the economy for a given initial condition, we provide the following proposition showing that unbounded growth is ruled out.

Proposition 7. *The long-term dynamics of T are bounded.*

Proof. Let $(x,y) \in S$ and notice that $\gamma(x,y) \in [0,1]$ so that $g(x,y) \in [0,1]$. Hence $y(t)$ is bounded $\forall\, t \geq 0$. To prove that also variable x cannot diverge we observe that

$$\begin{aligned} x' = f(x,y) &\leq (1-\tau_0-\tau_1)[(1-\alpha)Ax^{\alpha}+b]\tfrac{\beta}{1+\beta} \\ &= \tfrac{\beta}{1+\beta}(1-\tau_0-\tau_1)(1-\alpha)Ax^{\alpha} + \tfrac{b(1-\tau_0-\tau_1)\beta}{1+\beta} = j(x). \end{aligned}$$

Notice that $j(x) = Mx^{\alpha} + N, M > 0$ and $N \geq 0$. Since $j(x)$ is continuous, strictly increasing and concave then the sequence $x_{t+1} = j(x_t)$ is convergent $\forall\, x_0 \geq 0$ hence $j(x)$ is bounded that is $0 \leq j(x) < L, \forall\, x \geq 0$.

Being $f(x,y) \leq j(x)\ \forall\, y \in [0,1]$ then x cannot diverge. □

Proposition 7 ensures that map T admits an attractor $\Lambda \subset [0, \bar{T}\,] \times [0, 1-(1-\pi_0)^{\mu}]$, $\bar{T} < \infty$.