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RESEARCH

Dynamical analysis of an OLG model with interacting epidemiological and environmental domains

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Abstract
We study a model encompassing economic, epidemiological, and environmental domains, which feature reciprocal interactions. The economy is described by an overlapping generations model in which productivity and agents' preferences are affected by the epidemiological situation. The evolution of an epidemic is represented by a susceptible–infected–susceptible model, in which disease spread depends on the pollution level and can be reduced through government expenditure. The pollution level increases during the production process and can be reduced by allocating resources to its abatement. Resources are collected through the capital taxation and the regulator must decide how to share them between healthcare and environmental protection. For the resulting model, we show the possible existence of a unique steady state, either characterized by the presence of epidemics or disease-free. We study the comparative statics with respect to the policy parameter that governs the share of resources devoted to improving the epidemiological situation relative to environmental protection. We investigate the emergence of dynamics that do not converge to equilibrium, including complex and quasi-periodic trajectories.

Keywords OLG model · Epidemiological and environmental domains · Dynamical analysis · Bifurcations

JEL Classification C61 · O11 · Q56

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1 Introduction

Over recent decades, it has become clear that studying economic growth in isolation cannot provide a reliable basis for effective policy design. It is crucial to adopt an integrated approach that allows for assessing the reciprocal influences of different domains. For example, the economic impact of the recent pandemic highlighted the need to carefully plan healthcare policies to prevent epidemics. Similarly, environmental degradation negatively affects both economic growth and health outcomes.

Research began to tackle these challenges by developing modeling approaches that investigate the effects of interactions among different domains. The effect of consumption on environmental quality was studied in the seminal work by John and Pecchenino (1994). Coupling an overlapping generations model (OLG) with an equation describing the evolution of environmental quality, they showed how environmental quality deteriorates due to the consumption of the produced good. They also showed that this effect can be counteracted if the young agents invest in environmental maintenance. The improvements of environmental quality can also be addressed from the public investment perspective, through taxation (Horan et al. 1998) and/or the promotion of environmental awareness (Constant and Davin 2019; Grassetti et al. 2024). In particular, Horan et al. (1998) analyzes the effectiveness of ambient taxes in the context of non-point source pollution, showing that such policies can achieve cost-effective pollution control by inducing firms to adopt efficient abatement methods even when individual emissions are unobservable. Conversely, Constant and Davin (2019) develops a growth model with endogenously evolving environmental awareness, demonstrating that environmental taxation, funding abatement, or education can sustain growth by strengthening environmental policies. Finally, Grassetti et al. (2024) shows that when capital and environmental quality coevolve under government intervention, multiple equilibria may emerge: higher awareness can support both environmental quality and capital accumulation, whereas resource-use restrictions may be detrimental.

The literature investigates the interrelation between the economic and environmental domains from macroeconomic, microeconomic, and evolutionary perspectives. At the macroeconomic level, we can mention the contribution by Wei and Aadland (2021), which proposes an overlapping generations model incorporating physical capital, human capital, and pollution's health effects. The primary finding shows the existence of coexisting balanced-growth paths. One is beneficial, featuring high growth and lower pollution, whereas the other features high pollution and low growth. Moreover, in this latter scenario, pollution can create intergenerational welfare trade-offs, in which current generations benefit from increased production while future generations bear health costs, highlighting the importance of incorporating health impacts into environmental policy design. Menuet et al. (2023) reexamines from a theoretical perspective how environmental quality evolves during economic growth processes. The main result provides conditions under which environmental quality can improve along the growth path, challenging the traditional environmental Kuznets curve and showing that institutional factors and policy design are crucial for achieving simultaneous economic growth and environmental improvement. Finally, Cavalli et al. (2024a) considered an integrated economy–environment model (with Solow-type economic

dynamics coupled to an environmental block) and studied how an environmental tax affects the joint system in discrete time. The main result is a characterization of parameter/tax conditions under which taxation can simultaneously improve environmental outcomes while still allowing economic growth, and it highlights that taxation can also generate non-trivial dynamical out-of-equilibrium regimes.

From a microeconomic perspective, Matsumoto et al. (2020) investigates a two-stage Bertrand duopoly with non-point source pollution, showing that environmental taxation is effective when applied at the pricing stage, while its impact is ambiguous when introduced at the technology choice stage. Matsumoto and Szidarovszky (2022) analyzes a Cournot duopoly with emission charges, highlighting that policy effectiveness depends on model parameters and may differ between static equilibria and dynamic adjustment processes once stability and bifurcations are considered. Finally, Naimzada and Pireddu (2023) examines a dynamic Cournot duopoly with differentiated goods, showing that the effectiveness of output-based emission charges crucially depends on whether goods are substitutes or complements and on the degree of their interdependence.

An evolutionary perspective is first adopted by Zeppini (2015), who studies technological transitions between dirty and clean technologies in a discrete-choice framework, showing that transitions are path-dependent and that policy instruments such as pollution taxes can induce a shift toward clean technologies. Building on this, Cavalli et al. (2024b) incorporates explicit pollution dynamics, demonstrating that the success of green transitions depends on policy and tax settings that affect relative profitability and technological selection, potentially leading to clean technology adoption alongside persistently high pollution levels.

For further contributions, we refer to the surveys by Brock and Taylor (2005) and Levin and Xepapadeas (2021). An epidemic outbreak can severely affect productivity and, by shaping expectations about the future, also influence agent behavior. A first direct effect is that infected agents may be unable to work, with negative repercussions for production and, hence, economic growth. This was described by Goenka and Liu (2012), embedding epidemiological (infectious-disease) dynamics into a one-sector growth framework to study how disease prevalence interacts with macroeconomic decisions over time. The nonlinear feedback between the economy and infections can generate endogenous macro fluctuations (cycles/instability) even without exogenous shocks, so disease dynamics can be a source of business-cycle-like movements. Goenka and Liu (2019) reconsidered the problem with respect to the risk of being locked in poverty traps. Conversely, Bell and Gersbach (2013) analyzed economic growth when an epidemic disease persists over time and causes premature adult mortality, focusing on how this affects human capital formation and intergenerational transmission. Enduring epidemics can depress long-run growth by weakening human-capital accumulation mechanisms, helping explain persistent development gaps across otherwise similar economies.

Other research strands have focused on effective policies to counter the spread of disease. Anderson et al. (2012) study the optimal dynamic allocation of health budgets in a nonconvex susceptible–infected–susceptible framework and show that, under certain conditions, concentrating resources on a single group is optimal. Momota et al. (2005) consider disease prevalence and preventive behavior in a small open OLG

model, showing that prevalence-responsive investments can generate cyclical disease dynamics and that, despite externalities, one-shot foreign medical aid does not necessarily yield Pareto improvements. Gori et al. (2021a) embeds epidemiology into an OLG development model with publicly financed prevention, identifying threshold conditions under which public intervention shifts the system from endemicity to disease control, with implications for capital accumulation. Finally, Gori et al. (2021b) integrates a susceptible–infected–susceptible model into a Solow-type growth framework to evaluate the dynamic and welfare effects of non-pharmaceutical interventions, showing that long-run social distancing is not viable and potentially harmful, while investment in testing–tracing–isolation yields high welfare returns.

The present contribution expands upon that in Cavalli et al. (2024c), which considered the interaction between the economic and epidemiological sides, and draws inspiration from the work by Davin et al. (2022), which considers the economic, epidemiological, and environmental spheres simultaneously. In Davin et al. (2022), the economic side is described by an OLG model. It is assumed that only healthy agents are able to work, and their saving preferences for retired age are negatively affected by the spread of epidemics. The epidemiological domain consists of a susceptible–infected–susceptible (SIS) model, in which the contact rate can be mitigated by a suitable use of resources collected from taxation. Moreover, the contact rate is positively affected by pollution, as quantified by the output level, a proxy for pollution. The government can also issue debt to provide subsidies to agents who are unable to work due to illness. The goal of Davin et al. (2022) is to study the effectiveness of redistributive measures on the overall welfare of the population. A drawback of their model is the highly stylized representation of the environmental sector. Inherently, taxes can be used only for healthcare, and no interventions to improve environmental quality are possible. Finally, the model's dynamical analysis is quite limited.

The present research aims to overcome the previous issues. First of all, the model is enriched by a more detailed description of the dynamics of pollution, which is emitted by firms during the production process and can be partially reduced through natural absorption or by means of suitable investments in abatement. The regulator chooses how to allocate the collected resources between healthcare and environmental protection. Prioritizing healthcare directly helps control disease spread and benefits the economy, but it also reduces pollution abatement, which can worsen epidemiological outcomes. The converse occurs for the opposite choice. The proposed setting allows addressing the problem of developing a balanced policy that takes into account all these reciprocal effects, without disregarding their dynamical consequences.

Firstly, we study a baseline model in which the government does not issue a debt and does not provide subsidies for non-working agents. After showing that, as in Davin et al. (2022), a unique steady state characterized by epidemic exists together with a disease-free steady state, we focus on the effects on stability of the parameter regulating the distribution of resources between healthcare and the environment. In particular, we show that a flip bifurcation can occur if the investments in healthcare are too low, due to instabilities arising from the epidemiological side. Conversely, a Neimark–Sacker bubbling phenomenon can take place as resources for the pollution abatement are decreased, as a consequence of instabilities arising in the environmental

dynamics. We then show that the baseline model provides reliable guidance also when debt and subsidies are introduced.

The remainder of the manuscript is organized as follows. In Section 2 we present the general model. We start studying the limit case without subsidies in Section 3, both from the static and dynamical point of view. We then consider a particular case study for it in Section 4, together with numerical investigations. The complete model with debt and subsidies is investigated in Section 5. Conclusions and future research perspectives are reported in Section 6. Proofs of propositions are collected in the Appendix.

2 The model

The baseline model is built along the lines of what is proposed by Davin et al. (2022) and consists of three interacting domains: epidemiological, economic, and environmental. Before delving into the mathematical description of each domain, we briefly outline the main constitutive elements. The evolution occurs in discrete time periods, $t \in \mathbb{N}$. The focus is on a population composed of overlapping generations of adult and elderly agents, with each group having a constant size over time. In what follows, we use capital letters to denote variables referring to the non-normalized population, whereas lowercase letters will denote fractions and per capita amounts, normalized to 1.

The fraction $0 \leq s_t \leq 1$ of healthy adult agents works and supplies one unit of labor. Hence, the fraction i_t of infected individuals amounts to $1 - s_t$. A healthy (either adult or retired) agent can become infected depending on the characteristics of the epidemic, the environmental quality, and the government's healthcare policy.

At time t, healthy adult agents produce a homogeneous per capita output y_t, which can be used for consumption or saved as capital per capita k_t. The epidemiological situation influences the preference for saving or consumption: the better the situation, the greater the preference for saving, whereas the opposite occurs when the healthcare scenario deteriorates.

The government taxes production, issues a debt, and allocates a fraction $\omega \in [0, 1]$ of the collected resources to healthcare, while the remaining fraction $1 - \omega$ is devoted to environmental policies. We emphasize that ω is an exogenous parameter representing the regulator policy choice, on which we will focus the analytical study and the numerical investigations of the model. Finally, the environmental situation is described by the evolution of the pollution stock p_t, which increases with production, decays naturally, and can be reduced through publicly funded abatement.

In what follows, we provide details on how each domain is modeled, and at the end, we provide a summary table for the reader.

The environmental sphere

The pollutant stock p_t present in the environment at each time period evolves due to three factors:

1. the production process emits pollutants at a constant rate α, which is proportional to output per capita y_t;
2. pollution naturally decays at a constant rate $\delta \in (0, 1)$;
3. public environmental expenditures contribute to pollution abatement[1], with the effectiveness of the abatement process measured by the constant $\lambda > 0$.

Environmental transition equation

This process is described by the equation:

$$p_{t+1} = \max\{(1-\delta)p_t + \alpha y_t - \lambda(1-\omega)g_t, 0\}, \tag{1}$$

where $(1-\omega)g_t$ represents the fraction of resources allocated by the government to environmental improvement.

Unlike Davin et al. (2022), who proxies pollution with output, we model pollution as a stock with its own dynamics.[2] This approach allows us to account for the dynamic evolution of pollution and its influence on other spheres.

Moreover, in Davin et al. (2022), taxation was solely used to improve the epidemiological sector, whereas the introduction of Eq. 1 enables an investigation of the trade-off in allocating resources between healthcare and environmental policies.

Finally, we note that a zero-pollution level corresponds to the so-called "virgin state", in which the environment is completely unpolluted. This occurs when abatement is so effective that it eliminates all pollution. This also explains the use of the max function in Eq. 1.

The epidemiological sphere

As in Davin et al. (2022), we assume that the epidemic follows a classic SIS model. In each period, there are two generations of agents: adults and retired individuals. The population size for each generation remains constant over time and is equal to N.

The number of infected (respectively susceptible) adults and retired agents is the same and is denoted by I_t (respectively S_t). Thus, at each time t, we have $2N = 2I_t + 2S_t$, from which we define the fractions $s_t = S_t/N$ and $i_t = I_t/N$, representing the proportions of susceptible and infected agents, respectively.[3]

Since the entire adult and retired population is either susceptible or infected, it follows that $i_t + s_t = 1$.

The infection rate θ depends on healthcare policies and the environmental situation. Specifically, it is modeled as a decreasing function $\theta : [0, +\infty) \to (0, +\infty)$ that depends on the ratio $\omega g_t / p_t$, where ωg_t represents the portion of resources allocated

[1] For the implementation of resources aimed at improving environmental conditions, we refer, for instance, to the contributions by Beladi et al. (2013); Palivos and Varvarigos (2016); Wei and Aadland (2026).

[2] More precisely, it corresponds to y_{t+1}. Unlike Davin et al. (2022), we assume that emissions are proportional to y_t. The resulting pollution dynamics in Eq. 1 align with the related existing literature (see, e.g., Cavalli et al. (2024a) and the references therein).

[3] As in Davin et al. (2022), we assume that the probability of infection is identical across age groups. This modeling choice reduces age-related heterogeneity while preserving the possibility that individuals may become infected at different stages of their life cycle.

to public healthcare. A higher value of this ratio leads to a lower infection (or contact) rate θ, slowing the spread of the disease. Conversely, the negative dependence on the pollution stock p_t reflects the worsening health conditions as environmental pollution increases. The quotient form is appropriate, gives the direct and inverse relation typical of the problem, and avoids the possibility of negative terms arising from a difference form. It is also in line with the previous literature (see Davin et al. (2022)).

Epidemiological transition equation

The resulting SIS model is given by

$$\begin{cases} s_{t+1} = s_t \left(1 - \theta \left(\frac{\omega g_t}{p_t}\right) i_t\right) + \gamma i_t \\ i_{t+1} = (1-\gamma) i_t + \theta \left(\frac{\omega g_t}{p_t}\right) i_t s_t \\ s_0, i_0 > 0, \quad s_0 + i_0 = 1. \end{cases} \tag{2}$$

where $0 < \gamma \leq 1$ is the recovery rate.

To ensure positive trajectories, we impose condition $\theta \left(\frac{\omega g_t}{p_t}\right) \in \left(0, \left(1+\sqrt{\gamma}\right)^2\right)$ (see Allen (1994)) has to be imposed. Finally, we note that only one of the two equations in Eq. 2 is needed to model the epidemic's evolution. Henceforth, it is sufficient to study s_t, since $i_t = 1 - s_t$.

The economic sphere

The economy is described by an OLG model with production, along the lines of that in Davin et al. (2022). The main difference from the model in Davin et al. (2022) lies in the alternative characterization of public intervention, following the framework in Cavalli et al. (2024c).

Households

Every adult $i \in \{1, \ldots, N\}$ decides how to allocate consumption between adulthood $c_{i,t}$ and retirement $d_{i,t+1}$ by solving the following maximization problem

$$\begin{cases} \max_{c_{i,t}, d_{i,t+1}} u(c_{i,t}, d_{i,t+1}) = \max_{c_{i,t}, d_{i,t+1}} \left(\ln c_{i,t} + \beta(s_t) \ln d_{i,t+1}\right) \\ \text{subject to} \\ \sigma_{i,t} + c_{i,t} = \Omega_{i,t} + \tau_{i,t} \\ d_{i,t+1} = \frac{r_{t+1}}{\beta(s_t)} \sigma_{i,t} \end{cases} \tag{3}$$

where function $u(c_{i,t}, d_{i,t+1})$ captures the preferences of agent i and $\beta : [0,1] \to (0,1]$ the survival probability, which in turn influences the willingness to save for old age. Function β is increasing ($\beta'(x) \geq 0$ for any $x \in [0,1]$) and concave. We stress that in Davin et al. (2022) the utility function depends on $\beta(s_{t+1})$. This implies that agents anticipate the future evolution of the epidemic when making their decisions. In contrast, we assume agents observe only the current epidemic state and base their consumption and saving choices on it. This assumption, which requires a more limited informational endowment about epidemiological dynamics, provides a more plausible and suitable setting for the agents. Moreover, it allows for the analytical tractability

of the model[4] The constraint concerning $d_{i,t+1}$ is modified accordingly. Utility is maximized subject to the budget constraints given by the last two conditions in Eq. 3. Moreover, in the former constraint in Eq. 3, $\Omega_{i,t}$ represents the labor income while $\tau_{i,t}$ is the subsidy paid by the government. If adult agent i is able to work, we have $\Omega_{i,t} = w_t > 0$ and $\tau_{i,t} = 0$. Conversely, if adult agent i is unable to work and hence $\Omega_{i,t} = 0$, we assume that the government supplies an amount $\tau_{i,t} > 0$, which represents a subsidy and can be interpreted as health insurance or paid sick leave. The labor/subsidy income is split between savings $\sigma_{i,t}$ and consumption $c_{i,t}$, while the latter constraint expresses the relationship between savings at period t and the consumption at period $t + 1$. Specifically, future consumption is directly proportional to the marginal productivity of capital r_{t+1} and inversely proportional to the survival probability.

Solving Eq. 3 provides

$$c_{i,t} = \frac{1}{1+\beta(s_t)}(\Omega_{i,t}+\tau_{i,t}),\ \sigma_{i,t} = \frac{\beta(s_t)}{1+\beta(s_t)}(\Omega_{i,t}+\tau_{i,t}),\ d_{i,t+1} = \frac{r_{t+1}}{1+\beta(s_t)}(\Omega_{i,t}+\tau_{i,t}).$$

Firms

We assume for firms the neoclassical production function with constant returns to scale

$$Y_t(L_t, K_t) = A(s_t)L_t^{1-a}K_t^a,$$

where factor inputs L_t and K_t are, respectively, the labor and the capital, $a \in (0, 1)$, and increasing function $A : [0, 1] \to (0, 1]$ represents the normalized productivity factor. The per capita production function is obtained as

$$y_t(l_t, k_t) = A(s_t)l_t^{1-a}k_t^a,$$

where factor inputs l_t and k_t are, respectively, the labor and the capital per capita. In line with Davin et al. (2022), we assume that only healthy adult agents work, so $l_t = s_t$, and that productivity is increasing with respect to the fraction of healthy people[5]. If the whole adult population is healthy, we assume $A(1) = 1$.

Total factor productivity (TFP) changes with respect to the number of workers, as discussed in Davin et al. (2022) and its references. However, the relationship between A and s_t is complex and difficult to estimate accurately, as it involves the impact of

[4] Simulations show that this choice in the model has no significant impact in the results.

[5] We note that in the proposed model the productivity factor depends only on s_t, in line with Davin et al. (2022), whereas in reality it may also depend on the pollution level p_t (see, for instance, Cavalli et al. (2024a) and the related references). Anyway, we stress that the productivity factor indirectly depends on the evolution of pollution through the influence of p_t on s_t in Eq. 2.

changes in human capital on TFP[6]. Firms may face uncertainty about this relationship; thus, they assume they can observe TFP but not its marginal response to s_t. Accordingly, for small changes in s_t, firms treat TFP as locally constant, as Cavalli et al. (2024c). Note that the explicit expressions for the function $A(s_t)$ used in Davin et al. (2022), Cavalli et al. (2024c) and in the present work exhibit constant behavior over certain ranges.

The government charges firms a tax rate $\tau \in [0, 1]$, assumed exogenous and constant over time. The maximization of firm profits, which corresponds to

$$\pi(s_t, k_t) = (1 - \tau) y_t(s_t, k_t) - r_t k_t - w_t s_t,$$

provides the marginal productivity of capital and the wage[7]

$$r_t = (1 - \tau) a A(s_t) \left(\frac{s_t}{k_t}\right)^{1-a}, \quad w_t = (1 - \tau)(1 - a) A(s_t) \left(\frac{s_t}{k_t}\right)^{-a}.$$

Government

Now we detail the modeling of the subsidy. It is reasonable to link its amount to the economic performance. For this reason, we consider a constant, basic level of subsidy, described by an exogenous parameter $\tau^{ex} > 0$, supplemented by an endogenous component financed by tax revenues. This latter contribution is described by $\tau^{end} Y_t$, in which $0 \leq \tau^{end} < \tau$, and the goal is to provide the fraction $\tau^{end} Y_t / N$ to each infected adult, with an overall expenditure that then amounts to $\tau^{end} Y_t I_t / N = \tau^{end} Y_t i_t$. For each infected agent, the government allocates a subsidy equal to $\tau^{ex} + \tau^{end} Y_t / N$.

The government budget constraint can be written as

$$B_{t+1} + \tau Y_t = r_t B_t + G_t + \tau^{ex} I_t + \tau^{end} Y_t i_t,$$

where B_t is the total amount of public debt, $r_t B_t$ corresponds to the interest on it, and G_t is the public expenditures net of subsidies. We assume a time constant debt[8] $B_t \equiv B$, so that normalizing and rearranging the previous equation, we obtain

$$g_t = b(1 - r_t) + \tau y_t - \tau^{ex}(1 - s_t) - \tau^{end} y_t (1 - s_t), \tag{4}$$

[6] Although the effect of human capital on TFP is well-established (see, e.g., Miller and Upadhyay (2000)), it is hardly realistic to assume that firms have precise knowledge of how changes in human capital affect TFP. A variation of s_t determines in turn a change in the human capital of the workforce, and hence it influences TFP through alterations in the stock of knowledge and skills, social interactions, and individual capabilities. For an investigation on the methods used to measure the impact of human capital on TFP, we refer to Männasoo et al. (2018), which also outlines the challenges in this process

[7] According to the assumption of the knowledge of $A(s_t)$ by firms, we have that they act as if $A'(s_t) = 0$.

[8] In line with the setting considered by Davin et al. (2022), new debt cannot be issued. This assumption allows us to better focus on the role of the policy parameter ω and its effects, in particular those related to dynamics. The problems of a time-varying debt and of its sustainability are left for future research.

where g_t represents the residual per capita resources net of subsidies, which the government can allocate to healthcare and the environment. In Eq. 4 we assume $b > \tau^{ex}$.

Finally, the intertemporal equilibrium condition becomes $k_{t+1} + b = \sum_{i=1}^{N} \sigma_{i,t}/N$, from which we obtain

$$k_{t+1} = \frac{\beta(s_t)}{1+\beta(s_t)} \Big[(1-\tau)(1-a)A(s_t)s_t^{1-a}k_t^a + \tau^{ex}(1-s_t) + \tau^{end}A(s_t)s_t^{1-a}k_t^a(1-s_t) \Big] - b. \tag{5}$$

We note that $\beta(s_t)/(1+\beta(s_t))$ represents the saving propensity (see Chakraborty (2004)). Finally, in the remainder of the paper, we assume that functions θ, β, and A are sufficiently regular to be able to compute all the involved derivatives. For this, since up to third-order derivatives are involved, it is sufficient to have $\theta, \beta, A \in C^3$, at least almost everywhere on their domains. Moreover, since the model henceforth depends only on normalized variables, we will no longer use expressions such as "per capita" for the corresponding quantities. We summarize in Table 1 the variables, functions, and parameters involved in the models we are going to study in the remainder of the paper.

3 Analysis of the baseline model without subsidies

We study the static and dynamical properties of the model in which $\tau^{ex} = \tau^{end} = b = 0$. This represents the limit case in which infected agents, who are unable to work, have no income during the second period of their lives. This starting point is motivated by the analytical tractability of the resulting model. Moreover, in Section 5 we reconsider the model by taking into account government debt and subsidies, showing that the outcomes found for the baseline model still hold true for the improved one.

Table 1 List of variables, functions and parameters involved in the model

Variables			
s_t	fraction of susceptible agents	g_t	resources allocated to healthcare and environment
k_t	capital	p_t	pollution
Functions			
$\theta\left(\frac{\omega g_t}{p_t}\right)$	infection rate	$\beta(s_t)$	survival probability
$A(s_t)$	total factor productivity		
Parameters			
γ	recovery rate	b	debt
τ	output taxation rate	a	capital elasticity of output
τ^{ex}	basic level of subsidy	τ^{end}	taxation fraction for subsidies
α	emission rate	δ	natural decay rate of pollution
λ	effectiveness of abatement	ω	resources fraction allocated to healthcare

The resulting model is described by function $M : [0, 1] \times [0, +\infty) \times [0, +\infty) \to [0, 1] \times [0, +\infty) \times [0, +\infty)$, $\boldsymbol{\xi}_t = (s_t, p_t, k_t) \mapsto M(s_t, p_t, k_t)$ defined by means of the first equation in Eq. 2 (in which i_t is replaced by $1 - s_t$), and Eqs. 5 and 1, so that we have

$$\begin{cases} s_{t+1} = M_1(\boldsymbol{\xi}_t) = s_t \left[1 - \theta\left(\frac{\omega g_t}{p_t}\right)(1 - s_t)\right] + \gamma(1 - s_t) \\ k_{t+1} = M_2(\boldsymbol{\xi}_t) = \frac{\beta(s_t)}{1+\beta(s_t)}(1 - \tau)(1 - a)A(s_t)s_t^{1-a}k_t^a \\ p_{t+1} = M_3(\boldsymbol{\xi}_t) = (1 - \delta)p_t + \alpha A(s_t)s_t^{1-a}k_t^a - \lambda(1 - \omega)g_t \end{cases} \tag{6}$$

in which we introduced functions $M_i, i = 1, 2, 3$ to represent each component of $M(s_t, p_t, k_t)$. We note that even if the dynamics of the stock of pollution can become null, we avoid focusing on this case, studying situations characterized by $p_t > 0$. The reason is that the occurrence of the virgin state is quite unrealistic, and would presume quite extreme conditions to realize. The situation corresponding to $p_t = 0$ can be viewed as a limit case, impossible to reach in practice, and the analysis will focus only on the behavior as the pollution level approaches the virgin state, since it represents a boundary situation that is theoretically feasible only in principle. Henceforth, we will analytically account for pollution positivity in the steady-state analysis, while, from the dynamical point of view, we will monitor $p_t > 0$ in the numerical simulations. Finally, note that if $p_t > 0$, the argument of the function θ is well defined.

3.1 Static analysis

In this section, we study the possible steady states $\boldsymbol{\xi}^* = (s^*, p^*, k^*)$ of Eq. 6 and how they change with respect to the most relevant parameters of the model. Consistently with the possible steady states of the SIS model, we speak of an endemic steady state if $s^* > 0$ and of a disease-free steady state if $s^* = 1$. We avoid discussing steady states with zero capital, as they are economically irrelevant.

To formulate the results, in what follows, we make use of

$$\eta = \frac{\omega g}{p} = \frac{\omega\tau A(s)s^{1-a}k^a}{p}, \tag{7}$$

which, as in Davin et al. (2022), represents the relative government expenditure for healthcare with respect to the pollution level. Moreover, we define

$$\eta^* = \frac{\delta\omega\tau}{\alpha - \lambda(1 - \omega)\tau}. \tag{8}$$

Proposition 1 *Model Eq. 6 always has a disease-free steady state* $\boldsymbol{\xi}^*_{df} = (s^*_{df}, p^*_{df}, k^*_{df})$

$$\begin{cases} s^*_{df} = 1, \\ k^*_{df} = \left[(1 - \tau)(1 - a)\frac{\beta(1)}{1+\beta(1)}\right]^{\frac{1}{1-\alpha}}, \\ p^*_{df} = \frac{\alpha-\lambda(1-\omega)\tau}{\delta}(k^*_{df})^a, \end{cases}$$

provided that

$$\alpha - \lambda(1-\omega)\tau > 0. \tag{9}$$

If $\theta(\eta^*) > \gamma$ *and if Eq. 9 holds, there exists a unique endemic steady state* $\boldsymbol{\xi}^* = (s^*, p^*, k^*)$ *with positive components at which*

$$\begin{cases} s^* = \frac{\gamma}{\theta(\eta^*)}, \\ k^* = \left[(1-\tau)(1-a)\frac{\beta(s^*)}{1+\beta(s^*)}A(s^*)\right]^{\frac{1}{1-a}} s^*, \\ p^* = \frac{\alpha-\lambda(1-\omega)\tau}{\delta} A(s^*)(s^*)^{1-a}(k^*)^a. \end{cases} \tag{10}$$

If $\theta(\eta^*) \leq \gamma$*, no endemic steady state exists.*

Note that η^* in Eq. 8 actually corresponds to the value of Eq. 7 when the system is at an endemic steady state.

The disease-free steady state always exists in case Eq. 9 holds, and can coexist with a unique endemic steady state. We note that the result of Proposition 1 is in line with that obtained by Davin et al. (2022), while much more complicated steady state scenarios can arise in the simplified setting studied by Cavalli et al. (2024c), in which the environmental side was not considered. The motivation for this can be ascribed to the influence of pollution level on the contact rate, in particular, as described in both Eq. 6 and Davin et al. (2022). The effect is to eliminate the possibility of having more than one single endemic steady state[9]. The existence of the endemic steady state is guaranteed under two conditions. The former one, $\theta(\eta^*) > \gamma$, is a generalization of the similar requirement in the classic SIS model, while the latter one, $\alpha - \lambda(1-\omega)\tau > 0$, ensures the positivity of p^*.

Since the focus of the present research is on the role of the policy parameter ω, we restrict ourselves to situations in which we can study its behavior on its whole range of values. For this reason, in what follows we always assume that Eq. 9 holds true for any $\omega \in [0, 1]$, which requires $\alpha > \lambda\tau$. We note that this condition is automatically fulfilled if $\alpha > \lambda$, which is an agreeable and realistic setting, since it means that the rate of abatement is lower than the emission rate. So we make the following assumption

Assumption 1 *The rate of abatement is lower than the rate of emission of new pollutant, i.e.,* $\alpha > \lambda$.

From now on, all the results are presented and proved under Assumption 1.

In the next proposition, we study comparative statics of $\boldsymbol{\xi}^*$. To this end, let us define $g^* : [0, 1] \to [0, 1]$ as

$$g^*(s) = \tau(A(s))^{\frac{1}{1-a}} \left((1-\tau)(1-a)\frac{\beta(s)}{1+\beta(s)}\right)^{\frac{a}{1-a}} s, \tag{11}$$

[9] In particular, by simulative investigations, the endemic steady states that were observed in the economic-epidemiological model in Cavalli et al. (2024c) and that are ruled out when the environmental side is considered are those characterized by higher levels of susceptible agents. The effect of pollution on the epidemic spread is then to select the least desirable one.

which represents the government expenditure when the economic domain is at the steady state k^* depending on an exogenous fraction s of susceptible agents[10]. Below, for a given function f depending on a variable x we denote by $E_f(x)$ the elasticity of f at x. Let us introduce function $E_\theta(g)$, defined where θ is differentiable and representing the elasticity of θ at g, i.e.,

$$E_\theta(g) = \frac{g\theta'(g)}{\theta(g)}.$$

In the suitable domain, we can also define

$$E_{\frac{\beta}{1+\beta}}(s) = \frac{s\left(\frac{\beta(s)}{1+\beta(s)}\right)'}{\frac{\beta(s)}{1+\beta(s)}} = \frac{s\beta'(s)}{\beta(s)(\beta(s)+1)}, \quad E_A(s) = \frac{sA'(s)}{A(s)},$$

respectively representing the elasticity of the saving propensity and of the total factor productivity with respect to the fraction of susceptible agents. Similarly, function $E_{g^*}(s)$, defined where g^* is differentiable and representing the elasticity of g^* at s, can be written

$$\begin{aligned} E_{g^*}(s) &= \frac{sg^{*\prime}(s)}{g^*(s)} = \frac{a}{1-a}\frac{\beta'(s)}{s\beta(s)(1+\beta(s))} + \frac{1}{1-a}\frac{sA'(s)}{A(s)} + 1 \\ &= \frac{a}{1-a}E_{\frac{\beta}{1+\beta}}(s) + \frac{1}{1-a}E_A(s) + 1. \end{aligned} \tag{12}$$

We remark that since θ is strictly decreasing, we have $E_\theta(g) < 0$ for $g > 0$, while since both β and A are non-decreasing, we have $E_{g^*}(s) \geq 1$.

Proposition 2 *Let us consider the endemic steady state $\boldsymbol{\xi}^* = (s^*, p^*, k^*)$. Under Assumption 1, on increasing ω, we have that s^*, k^* and p^* increase.*

The main outcome of Proposition 2 is that, in the present setting, moving resources from environmental protection to healthcare necessarily induces a deterioration of the environmental quality for an improvement of the epidemiological situation and of the economic growth. This latter aspect is an effect of the direct negative influence of the disease spread on productivity. As a consequence, balanced welfare concerns should take into account the trade-offs in the distribution of resources.

3.2 Dynamical analysis

We recall (see e.g. Allen (1994)) that in the SIS model with exogenous contact rate the endemic steady state is locally asymptotically stable provided that $\theta - \gamma < 2$. Similarly, if s and k are assumed exogenous in the dynamical equation for the pollution, the unique steady state is always locally asymptotically stable.

For the disease-free steady state, the next result mirrors the standard SIS case.

[10] Expression 11 can be obtained by replacing in Eq. 4 the equilibrium expression of k for a generic s (see the second equation in Eqs. 10 or A2).

Proposition 3 *Let $\gamma > \theta(\eta^*)$ so that the endemic steady state $\boldsymbol{\xi}^* = (s^*, p^*, k^*)$ does not exist. We then have that $\boldsymbol{\xi}^*_{df}$ is locally asymptotically stable.*

We remark that when conversely the endemic steady state exists, $\boldsymbol{\xi}^*_{df}$ is unstable. Now we turn our attention to the stability of the endemic steady state. We note that if the endemic steady state exists for $\omega = 0$ (i.e. if $\gamma < \theta(0)$), since as ω increases, we have that η^* increases and hence $\theta(\eta^*)$ decreases, so condition $\gamma < \theta(\eta^*)$ may be violated for suitably large ω. This leads to the disappearance of $\boldsymbol{\xi}^*$ (which actually becomes unfeasible, with $s^* \geq 1$) and $\boldsymbol{\xi}^*_{df}$ becomes stable. This behavior actually recalls that of a transcritical bifurcation, which will be confirmed by the numerical simulations of the next sections.

Proposition 4 *Let $\gamma < \theta(\eta^*)$ so that the endemic steady state $\boldsymbol{\xi}^* = (s^*, p^*, k^*)$ exists. Under Assumption 1, we have that $\boldsymbol{\xi}^*$ is locally asymptotically stable provided that*

$$\begin{cases} c_{11}(\theta(\eta^*) - \gamma) + c_{10} > 0, \\ c_{22}(\theta(\eta^*) - \gamma)^2 + c_{21}(\theta(\eta^*) - \gamma) + c_{20} > 0, \\ c_{31}(\theta(\eta^*) - \gamma) + c_{30} > 0, \end{cases} \tag{13}$$

where

$$\begin{aligned}
c_{11} &= -(a+1)(2-\delta) - 2E_\theta(\eta^*)\left(E_{g^*}\left(\frac{\gamma}{\theta(\eta^*)}\right)(1-a) - 2aE_{\beta/1+\beta}\left(\frac{\gamma}{\theta(\eta^*)}\right)\right), \\
c_{10} &= 2(a+1)(2-\delta), \\
c_{22} &= a\left(1-\delta-E_\theta(\eta^*)E_{\beta/1+\beta}\left(\frac{\gamma}{\theta(\eta^*)}\right)\right)\left(a\delta+1-a+(1-a)E_\theta(\eta^*)E_{g^*}\left(\frac{\gamma}{\theta(\eta^*)}\right)\right) \\
c_{21} &= (1-a)E_\theta(\eta^*)\left((1-a+a\delta)E_{g^*}\left(\frac{\gamma}{\theta(\eta^*)}\right) - a\delta E_{\beta/1+\beta}\left(\frac{\gamma}{\theta(\eta^*)}\right)\right) \\
&\quad + 4a\delta - \delta - 2a - a\delta^2 - 3a^2\delta + a^2 + 2a^2\delta^2 + 1 \\
c_{20} &= \delta(1-a)(a\delta+1-a), \\
c_{31} &= 1 + a - \delta + (1-a)E_\theta(\eta^*)\left(E_{g^*}\left(\frac{\gamma}{\theta(\eta^*)}\right) - \frac{2a}{1-a}E_{\beta/1+\beta}\left(\frac{\gamma}{\theta(\eta^*)}\right)\right), \\
c_{30} &= 3 - (1+a)(1-\delta) - a.
\end{aligned}$$

A flip bifurcation can occur only when the first condition in Eq. 13 is violated, while a Neimark–Sacker bifurcation can occur only when the second condition in Eq. 13 is violated.

The expressions of stability conditions Eq. 13 are very convoluted, as the involved coefficients c_{ij} can depend on η^* and it is not possible to make explicit the role of each parameter, in particular of ω. Nonetheless, several observations can be made. Firstly, we recall that the last condition in Eq. 13 is not involved in the possible emergence of bifurcations, so we avoid taking it into account (see e.g. Lines et al. (2020), in which an equivalent formulation of this condition is considered).

A key point is that, as ω changes, the main source of a potential bifurcation is the change in the contact rate $\theta(\eta^*)$. This may suggest that, in the present setting, instabilities arise from the epidemiological side and are then transmitted to the other domains.

This can be directly ascribed to the presence of the environmental side.[11] Let us focus on the first condition in Eq. 13. The role of the contact rate $\theta(\eta^*)$ can be amplified or damped by its elasticity and that of the government expenditure and of the saving propensity. Let us assume constant total factor productivity and survival probability. If we consider an exogenous contact rate, we find the same stability condition related to the SIS model. If θ is endogenous, $\theta(\eta^*) - \gamma$ decreases as ω increases. In the case of an isoelastic contact rate[12] we have that c_{11} is constant and increasing ω, we can have that the first condition in Eq. 13 is either always/never fulfilled or it becomes true for sufficiently large ω, which is then stabilizing. Indeed, this behavior may be altered in the case of general contact rate functions or by endogenous A and β.

Similarly, the second condition in Eq. 13 seems to suggest that ω can lead to a double stability change for the endemic steady state[13] even if also in this case the role of coefficients c_{22} and c_{21} may alter the occurrence of this scenario. Finally, we note that for $\theta(\eta^*) - \gamma = 0$, both conditions in Eq. 13 are fulfilled, and this guarantees that the endemic steady state must become stable as it sufficiently approaches the disease-free one. This means that if $\theta(\eta^*) - \gamma = 0$ occurs for some $\omega_{tr} \in (0, 1)$, we have that $\boldsymbol{\xi}^*$ is stable on a left neighborhood of ω_{tr}. In the next sections, we look for simplified settings that provide sufficient conditions on the endogenous elements of the model for the occurrence of at most one stability change (respectively two stability changes) arising from the first (respectively second) condition in Eq. 13. To this end we will deal with a case of study, in view of which we reformulate Proposition 4 for scenarios of increasing complexity.

4 Case study with increasing complexity

In this section, we study stability by introducing the endogenous mechanisms one at a time. The goal is to obtain specialized versions of Proposition 4 that allow investigating the role of each sphere in being the source of instabilities. Moreover, for each framework, we provide sufficient conditions under which the arising scenarios match those discussed after Proposition 4.

4.1 Endogenous θ

When A and β are constant, conditions Eq. 13 simplify as follows. Since the calculations are straightforward, we do not provide a proof.

[11] In studying dynamical properties, Davin et al. (2022) imposed restrictive conditions in order to obtain stable steady states, since their goal is to focus on this situation. For this reason, and for the introduction in the present contribution of a detailed description of the environmental side, it is awkward to compare the dynamical outcomes of the two models.

[12] Due to the upper bound on the contact rate to preserve the positivity of trajectories, function θ can be isoelastic just for $\eta^* > \bar{\eta}$ with $\theta(\bar{\eta}) < (1+\sqrt{\gamma})^2$. However, it is possible to consider a piecewise isoelastic contact rate $\theta(\eta) = \min\{\theta_0, \theta_0/\eta^{\xi}\}$, to which we can apply the next discussion.

[13] For example by considering the same setting sketched for the first stability condition, with isoelastic θ and exogenous A and β.

Corollary 5 *Under Assumption 1, if $A(s) \equiv A$ and $\beta(s) \equiv \beta$ and $\gamma < \theta(\eta^*)$ conditions Eq. 13 become*

$$\begin{cases} E_\theta(\eta^*) < \dfrac{(a+1)(2-\delta)[2-(\theta(\eta^*)-\gamma)]}{2(1-a)(\theta(\eta^*)-\gamma)}, \\ E_\theta(\eta^*) > -\dfrac{(1-a+a\delta)[1-a+a(\theta(\eta^*)-\gamma)][\delta+(1-\delta)(\theta(\eta^*)-\gamma)]}{(\theta(\eta^*)-\gamma)(1-a)[1-a(1-\delta)+a(1-\delta)(\theta(\eta^*)-\gamma)]}, \\ E_\theta(\eta^*) > \dfrac{(1+a)(1-\delta)+a-3-(1+a-\delta)(\theta(\eta^*)-\gamma)}{(1-a)(\theta(\eta^*)-\gamma)}. \end{cases} \tag{14}$$

We remark that, since total factor productivity does not depend on s_t, the marginal total factor productivity is null, as assumed by the firms, and therefore this case of study is not affected by such an element of uncertainty. Note that the right-hand side of the first condition in Eq. 14 is positive when $\theta(\eta^*) - \gamma < 2$, which corresponds to the stability conditions of the endemic steady state in the SIS model, while the left-hand side is negative. This means that when the endemic state is stable in an SIS model characterized by a contact rate $\theta_0 = \theta(0)$, the first condition in Eq. 14 is fulfilled for any ω. However, because the right-hand side of the second inequality in Eq. 14 is negative, the Neimark–Sacker stability condition may fail even when the epidemiological block is not a source of instability. Depending on θ, many different scenarios can arise when Eq. 14 is violated. A simple situation is described in the next proposition. In what follows, we denote bifurcation thresholds for ω of potential transcritical, flip and Neimark–Sacker bifurcations by subscripts tr, f and ns.

Proposition 6 *If $\theta'(\eta) \neq 0$, $E'_\theta(\eta) < 0$ and*

$$E''_\theta(\eta) > E'_\theta(\eta)\left(\frac{\theta''(\eta)}{\theta'(\eta)} - \frac{2\theta'(\eta)}{\theta(\eta)-\gamma}\right), \tag{15}$$

then we have that, depending on the parameters defining the model and function θ, the endemic steady state $\boldsymbol{\xi}^*$ for $\omega \in [0, \omega_{tr}] \cap [0, 1]$ can incur

- *at most one period halving bifurcation at ω_f;*
- *at most a couple of Neimark–Sacker bifurcations at $\omega_{1,ns}, \omega_{2,ns}$.*

Different sequences of bifurcations are possible, and, increasing ω, are characterized by a recover of stability at ω_f and $\omega_{2,ns}$, and a loss of stability at $\omega_{1,ns}$.

From Proposition 6, we have that if at the equilibrium the elasticity of the contact rate with respect to the relative government expenditure is decreasing and "not too concave", we can have up to three stability inversions for $\boldsymbol{\xi}^*$, with a possible final transcritical bifurcation. We note that ω_{tr} is the threshold at which the endemic steady state can disappear and disease free steady state can become stable; ω_f is the threshold value at which a period-halving bifurcation can take place, while we can have up to two Neimark–Sacker bifurcations at $\omega_{1,ns}$ and $\omega_{2,ns}$.

To support discussion of these scenarios, we also rely on numerical simulations. To this end, we introduce

$$\theta(\eta) = \theta_0 e^{-\theta_1 \eta^{\theta_2}}, \tag{16}$$

where $\theta_0, \theta_2 > 0$ and $\theta_1 \geq 0$. We note that setting $\theta_1 = 0$ we have an exogenous contact rate θ_0, which we consider as a limit case. In what follows, we focus on $\theta_1 > 0$, in which case θ_0 represents the maximum possible contact rate, occurring when the government expenditure is null. Parameters θ_1 and θ_2 determine the steepness and concavity of the function θ. Function Eq. 16 fulfills the requirements of Proposition 6, as shown in the next corollary.

Corollary 7 *Let θ be defined by Eq. 16. For $\eta \in \left(0, \min\left\{\frac{\delta\tau}{\alpha}, \theta^{-1}\{\gamma\}\right\}\right)$, we have that $E_\theta(\eta)$ is strictly decreasing and fulfills condition Eq. 15.*

We start investigating the occurrence of all the possible[14] scenarios arising from Proposition 6 by means of numerical investigations using function Eq. 16. In what follows, we set $A = 1, \beta = 1, \theta_1 = 1, a = 0.3, \gamma = 0.999, \alpha = 0.1, \delta = 0.3$ and $\lambda = 0.075$. In Fig. 1 we report in the first row the two-dimensional bifurcation diagrams in (ω, τ) plane. Each point is colored according to the type (and period) of the attractor reached for that parameter pair. White denotes convergence to a steady state, and blue denotes a period-2 cycle. Higher-period cycles are shown with other colors, while cyan indicates either very high-period cycles or quasi-periodic/chaotic dynamics. Crossing the dashed black line (upper-right region), $\boldsymbol{\xi}^*$ disappears and $\boldsymbol{\xi}^*_{df}$ becomes stable. Accordingly, trajectories converge to $\boldsymbol{\xi}^*$ the region to the left/below the line, and to $\boldsymbol{\xi}^*_{df}$ otherwise. Below each two-dimensional bifurcation diagram, we report three examples of one-dimensional bifurcation diagrams related to it, obtained for different values of τ as *omega* increases.

The simulations reported in the first column of Fig. 1 are obtained for a large θ_0, such that the endemic steady state is unstable for an isolated SIS model characterized by such a contact rate. We note that numerical evidence from the simulations indicates that the first condition in Eq. 14 is violated for small values of τ and ω, whereas the second condition is satisfied for all $\omega \in [0, 1]$. The resulting scenarios are consistent with the isolated SIS case and display a stabilizing role of taxation, in line with Cavalli et al. (2024c). Similarly, ω is stabilizing as well. For small taxation rates we have that $\boldsymbol{\xi}^*$ is unstable for any $\omega \in [0, 1]$ (panel (d)), while as τ increases it is possible to stabilize dynamics if the share of resources devoted to healthcare is suitably large (panel (g)), and even recover the disease-free steady state (panel (j)). Note that the bifurcation diagram in Fig. 1 (j) provides numerical evidence of a transcritical bifurcation occurring for $\omega = \omega_{tr}$ the endemic and disease-free steady states merge and exchange stability. This is also confirmed by several other bifurcation diagrams, both in Fig. 1 and in subsequent figures. In the simulations reported in the first column of Fig. 1, the interaction with the environmental side does not introduce new scenarios with respect to those observed in an SIS model or in Cavalli et al. (2024c).

Conversely, in panel (b) of Fig. 1 we consider a setting for which the first condition in Eq. 13 is fulfilled for any $\omega \in [0, 1]$. Note that θ_0 is small, so the endemic steady state is stable for the classic SIS model with contact rate θ_0, which is the rate obtained in the present model with $\tau = 0$. For small taxation rates, we have that $\boldsymbol{\xi}^*$ is stable for

[14] Among the possible combinations, those in which $\omega_{1,ns} = 0$ or $0 < \omega_{1,ns} < \omega_f < \omega_{2,ns}$ or $0 < \omega_{1,ns} < \omega_{2,ns} < \omega_f$ seem not to be possible, but they would provide scenarios characterized by degrees of complexity qualitatively similar to others that occur.

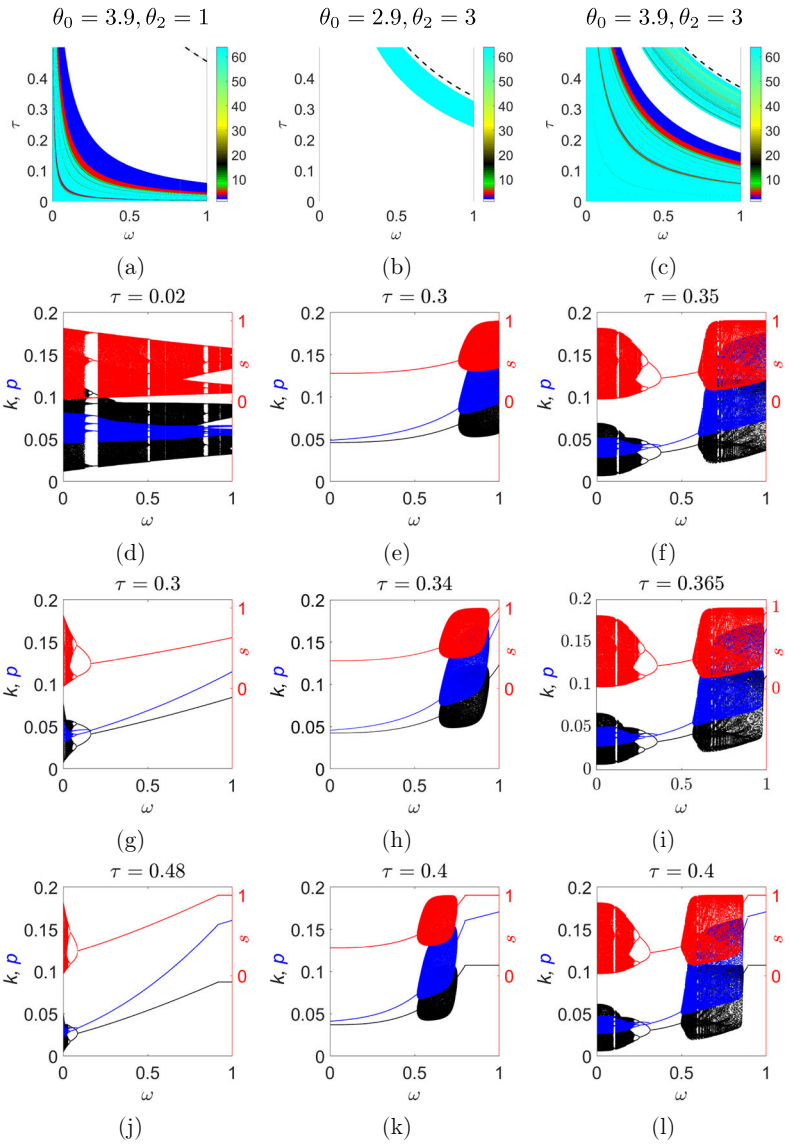

Fig. 1 Endogenous θ, exogenous A and β. *First row*: two-dimensional bifurcation diagrams in (ω, τ) plane for different values of θ_0 and θ_2. Attractors are shown in different colors according to their type, with *white* denoting convergence to a steady state, which is the disease-free one in the region above the *dashed line*. For different values of τ, the one-dimensional bifurcation diagrams, on varying ω, in each column are related to the two-dimensional bifurcation diagram in the first row. In each panel, the *black* and *blue bifurcation diagrams* correspond to variables k and p, respectively, and refer to the left vertical axis, while the *red bifurcation diagram* corresponds to variable s and refers to the right vertical axis

any $\omega \in [0, 1]$, while as τ increases, $\boldsymbol{\xi}^*$ becomes firstly unstable for large ω (panel (e)), then it is unstable for intermediate ω, with stability that is recovered for suitably large values of ω (panel (h)). Also, in this case, the disease-free steady state can become stable if τ and ω are sufficiently large (panel (k)). In all these cases, $\boldsymbol{\xi}^*$ loses/recovers stability through Neimark–Sacker bifurcations. We remark that the SIS equation is not a source of instability, as the contact rate is low. Each domain would be stable in isolation. However, coupling the three blocks can generate quasi-periodic dynamics.

The rationale can be explained by observing the quasi-periodic time series reported in Fig. 2. We report simulations for values of ω respectively before, within, and after the instability interval. In all reported simulations, the epidemic is initially at its maximum spread, which is reflected in a depressed capital level (low worker numbers) and a consequent reduction in pollution (low production). If ω is small, government healthcare expenditures can initially counteract epidemic diffusion, and the number of healthy agents starts increasing, reviving the economic course and, consequently, worsening the environmental situation. However, rising pollution worsens the contact rate. This effect is not fully offset by healthcare spending, so the epidemic recurs, albeit at a lower prevalence. This feedback loop repeats across s_t, k_t and p_t, but with progressively smaller swings. As a result, oscillations dampen, and trajectories converge to an endemic steady state with a non-negligible infection share, even though pollution remains low. In this setting, the negative effect of pollution on the epidemiological situation outweighs the positive effect of healthcare expenditure. If we increase ω, we obtain a scenario in which these two effects, on average, balance out, so we have persistent, large oscillations. When the epidemiological and economic situations are good, the large pollution level drives the scenario toward the opposite state. The epidemiological and economic domains reach a bottom, from which they emerge thanks to healthcare expenditures and improved environmental conditions. For larger ω, healthcare spending becomes sufficiently effective to offset the higher (persistent) pollution, and oscillations dampen, now settling to an endemic steady state characterized by a small fraction of infected people but a large pollution level.

In summary, endogenous fluctuations, whether periodic or chaotic, can originate in the epidemiological sphere and subsequently transmit to the economic and environmental spheres. Such oscillations, when chaotic and irregular, describe the typical "boom and bust" pattern of epidemics, and result in erratic behavior of capital, with consequent fluctuations in the amount of resources collected through taxation and

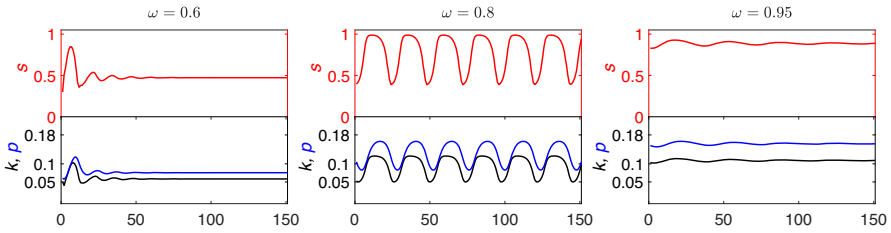

Fig. 2 Time series related to the simulation reported in Fig. 1 (h) for different values of ω. The *black* and *blue trajectories* correspond to variables k and p, respectively, and refer to the left vertical axis, while the *red trajectory* corresponds to variable s and refers to the right vertical axis

in their allocation for healthcare expenditure and environmental improvement. An appropriate policy that increases resources devoted to healthcare can help dampen these phenomena, providing stability to the endemic nature of the epidemic.

Quasi-periodic trajectories, on the other hand, arise from the interaction among the model's different spheres and are linked to policy choices that generate cyclic behavior, alternating phases of controlled epidemic/pollution spread with phases of controlled epidemic/degraded environmental quality. A different allocation between environmental and healthcare spending may stabilize the dynamics, but potentially at the cost of worse epidemiological outcomes or higher pollution.

In panel (c) of Fig. 1, we consider a setting for which both conditions in Eq. 13 can be violated, and what we observe can be thought of as the superposition of what happens in panels (a) and (c). Since θ_0 is large, the endemic steady state is unstable for the SIS model if the contact rate is θ_0. So $\boldsymbol{\xi}^*$ is unstable when $\tau = 0$ and for small taxation rates for any $\omega \in [0, 1]$, while as τ mildly increases, $\boldsymbol{\xi}^*$ can recover stability for large values of ω. If τ is further increased, we have the superimposition of a couple of destabilizing/stabilizing Neimark–Sacker bifurcations like those observed in the middle column of Fig. 1. In this case, $\boldsymbol{\xi}^*$ can have two (panel (f)) or three (panel (i)) stability changes (in addition to the final transcritical bifurcation), with different kinds of bifurcations occurring. If τ and ω are large enough, the disease-free steady state can recover stability (panel (l)).

We remark that, differently from the results in Cavalli et al. (2024c), taxation can have a destabilizing effect.

4.2 Endogenous θ and A

Now we take into account the effect of the epidemic spread on productivity, leaving β as the only exogenous term. In this case, it is immediate to see that conditions Eq. 13 simplify as follows.

Corollary 8 *If $\beta(s) \equiv \beta$ and $\gamma < \theta(\eta^*)$ conditions Eq. 13 become*

$$\begin{cases} E_\theta(\eta^*)\left(1+\frac{1}{1-a}E_A\left(\frac{\gamma}{\theta(\eta^*)}\right)\right) < \frac{(a+1)(2-\delta)[2-(\theta(\eta^*)-\gamma)]}{2(1-a)(\theta(\eta^*)-\gamma)}, \\ E_\theta(\eta^*)\left(1+\frac{1}{1-a}E_A\left(\frac{\gamma}{\theta(\eta^*)}\right)\right) > -\frac{(1-a+a\delta)[1-a+a(\theta(\eta^*)-\gamma)][\delta+(1-\delta)(\theta(\eta^*)-\gamma)]}{(\theta(\eta^*)-\gamma)(1-a)[1-a(1-\delta)(1-(\theta(\eta^*)-\gamma))]}, \\ E_\theta(\eta^*)\left(1+\frac{1}{1-a}E_A\left(\frac{\gamma}{\theta(\eta^*)}\right)\right) > \frac{(1+a)(1-\delta)+a-3-(1+a-\delta)(\theta(\eta^*)-\gamma)}{(1-a)(\theta(\eta^*)-\gamma)}. \end{cases} \tag{17}$$

Also in this case, the first condition in Eq. 17 is fulfilled when $\theta(\eta^*) - \gamma < 2$, and the comments after Corollary 5 are still valid. Since A has positive elasticity, ceteris paribus, it has a stabilizing effect. This is predictable and in line with the outcomes in Cavalli et al. (2024c), as it points to a more reactive effect of a decrease in the fraction of infected agents on productivity. The opposite occurs for the second condition in Eq. 17, with the elasticity of A having a destabilizing effect. If productivity reacts more quickly to a reduction in the spread of infection, this corresponds to an increase in pollution, and, as discussed in the previous section, this has a destabilizing effect. Now

we provide sufficient conditions on E_A to obtain a result similar to that in Proposition 6.

In what follows, we denote by $\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)'$ and $\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)''$ respectively the first and the second derivative of $E_A\left(\frac{\gamma}{\theta(\eta)}\right)$ with respect to η, i.e. $\frac{dE_A(\gamma/\theta(\eta))}{d\eta}$ and $\frac{d^2E_A(\gamma/\theta(\eta))}{d\eta^2}$.

Proposition 9 *If* $\theta'(\eta) \neq 0$, $E'_\theta(\eta) < 0$, $\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)' > 0$,

$$E''_\theta(\eta) > \left(\frac{\theta''(\eta)}{\theta'(\eta)} - \frac{2\theta'(\eta)}{\theta(\eta)-\gamma}\right)E'_\theta(\eta) - 2E'_\theta(\eta)\frac{\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)'}{E_A\left(\frac{\gamma}{\theta(\eta)}\right)} \tag{18}$$

and

$$\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)'' < \left(\frac{-2\theta'(\eta)}{\theta(\eta)-\gamma} + \frac{\theta''(\eta)}{\theta'(\eta)}\right)\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)', \tag{19}$$

we then have that the possible bifurcations are the same as those in Proposition 6.

For the numerical simulations, we introduce a function

$$A(s) = \max\left\{\min\left\{A_m + \frac{1-A_m}{A_1-A_0}(s-A_0), 1\right\}, A_m\right\}, \tag{20}$$

with $0 \leq A_0 < A_1 \leq 1$ and $A_m \in [0, 1)$. With function Eq. 20, the total factor productivity equals the smallest possible A_m for $s \leq A_0$, is linearly increasing on (A_0, A_1), and attains its maximum value 1 for $s \geq A_1$. We study under what conditions on the parameters of the function θ defined by Eq. 16 and on the restriction to (A_0, A_1) of the function A, the previous proposition can be applied.

Corollary 10 *Let* θ *be defined by Eqs. 16 and A by 20. For* $\eta \in \left(0, \min\left\{\frac{\delta\tau}{\alpha}, \theta^{-1}\{\gamma\}\right\}\right) \cap \left(\theta^{-1}\left(\frac{\gamma}{A_0}\right), \theta^{-1}\left(\frac{\gamma}{A_1}\right)\right)$ *and if*

$$\begin{cases} A_1A_m - A_0 > 0 \\ A_0 > \dfrac{1}{3} \end{cases} \tag{21}$$

conditions of Proposition 9 hold true, and so the possible bifurcations are the same as those in Proposition 6.

In Fig. 3 we report the results obtained by considering the same parameter settings used for Fig. 1, now with the endogenous total factor productivity function Eq. 20, for which we set $A_m = 0.5$, $A_0 = 0.35$, $A_1 = 0.75$. Comparing the two-dimensional bifurcation diagrams, the most evident difference is the presence of an additional region of instability in Fig. 3(a) with respect to Fig. 1(a), which highlights one (Fig. 3(g)) or two (Fig. 3(j)) Neimark–Sacker bifurcations. As noted after Corollary 8, endogenizing total factor productivity can lead to a violation of the second

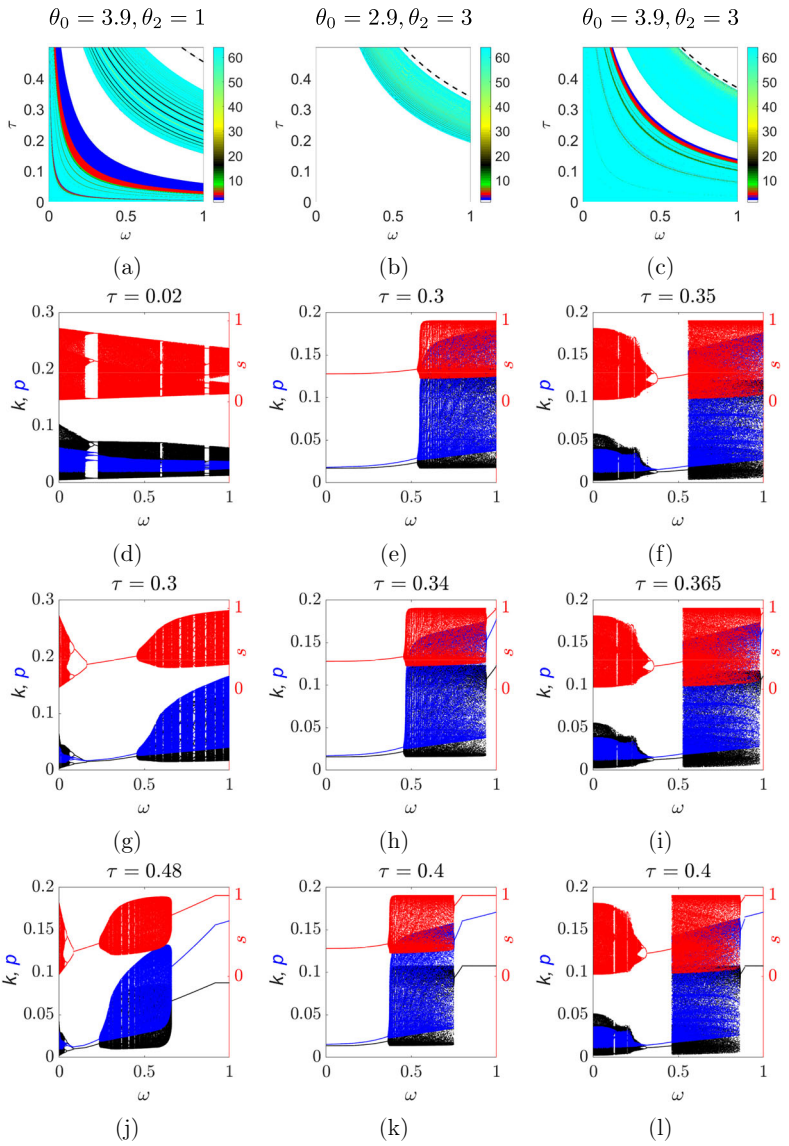

Fig. 3 Endogenous θ, and A, exogenous β. *First row*: two-dimensional bifurcation diagrams in (ω, τ) plane for different values of θ_0 and θ_2. Attractors are shown in different colors according to their type, with *white* denoting convergence to a steady state, which is the disease-free one in the region above the *dashed line*. For different values of τ, the one-dimensional bifurcation diagrams, on varying ω, in each column are related to the two-dimensional bifurcation diagram in the first row. In each panel, the *black* and *blue bifurcation diagrams* correspond to variables k and p, respectively, and refer to the left vertical axis, while the *red bifurcation diagram* corresponds to variable s and refers to the right vertical axis

stability condition in Eq. 17, which gives rise to the Neimark–Sacker bifurcation. Also in the other bifurcation diagrams of Fig. 3, we can see that the instability interval due to the Neimark–Sacker bifurcation is larger than in those corresponding to Fig. 1, with wider oscillations.

4.3 Endogenous θ, A and β

Now take into account all the possible sources of interaction, by also considering endogenous β. For the lack of analytical tractability, we avoid providing sufficient conditions under which the scenarios of Propositions 6 and 9 are guaranteed. We note that the probability to survive alters stability conditions through the elasticity of the saving propensity, which appears as a multiplicative factor of the elasticity of the contact rate. However, the extensive numerical investigations we performed seem to point out that its effect on stability is the weakest one when compared to those of θ and A. To show this, we consider

$$\beta(s) = s^{\alpha}, \tag{22}$$

in which $\alpha \in (0, 1)$ allows regulating the concavity of the probability to survive [15]. In Fig. 4 we report the simulations obtained by using the function Eq. 22 with $\alpha = 1/2$ and the same setting adopted for the scenarios reported in Fig. 3. As we can see, corresponding panels are almost identical.

We conclude this section by outlining some considerations relevant to policy design. First, there is a clear trade-off between investments in healthcare and environmental protection. Public spending that allocates most resources to pollution abatement proves ineffective at curbing fluctuations in the epidemic's evolution, especially at relatively low tax levels. Reallocating resources toward healthcare can stabilize the erratic dynamics of the disease, sustaining a regime characterized by endemic disease and low pollution levels. Disease eradication can be achieved only through substantial investments in healthcare, but this comes at the expense of environmental quality. In particular, a policy that is not sufficiently oriented toward healthcare may have the unintended effect of inducing quasi-periodic dynamics in pollution, which in turn become a source of recurrent worsening of the epidemic.

5 Model with public debt and subsidies

In this section, we test the robustness of the results for model Eq. 6 by accounting for subsidies and the government's ability to issue debt by introducing subsidies and allowing the government to issue debt. In Sections 3 and 4, we considered the limiting case in which infected adult agents cannot work and therefore have no income. Here, we show that our results carry over to a more realistic setting, in which the government devotes resources to subsidizing non-working agents. In what follows, we incorporate

[15] The analytical expression for β is actually not differentiable at $s = 0$, but this is not a problem since $s^* > 0$, so the analytical results for stability are still valid.

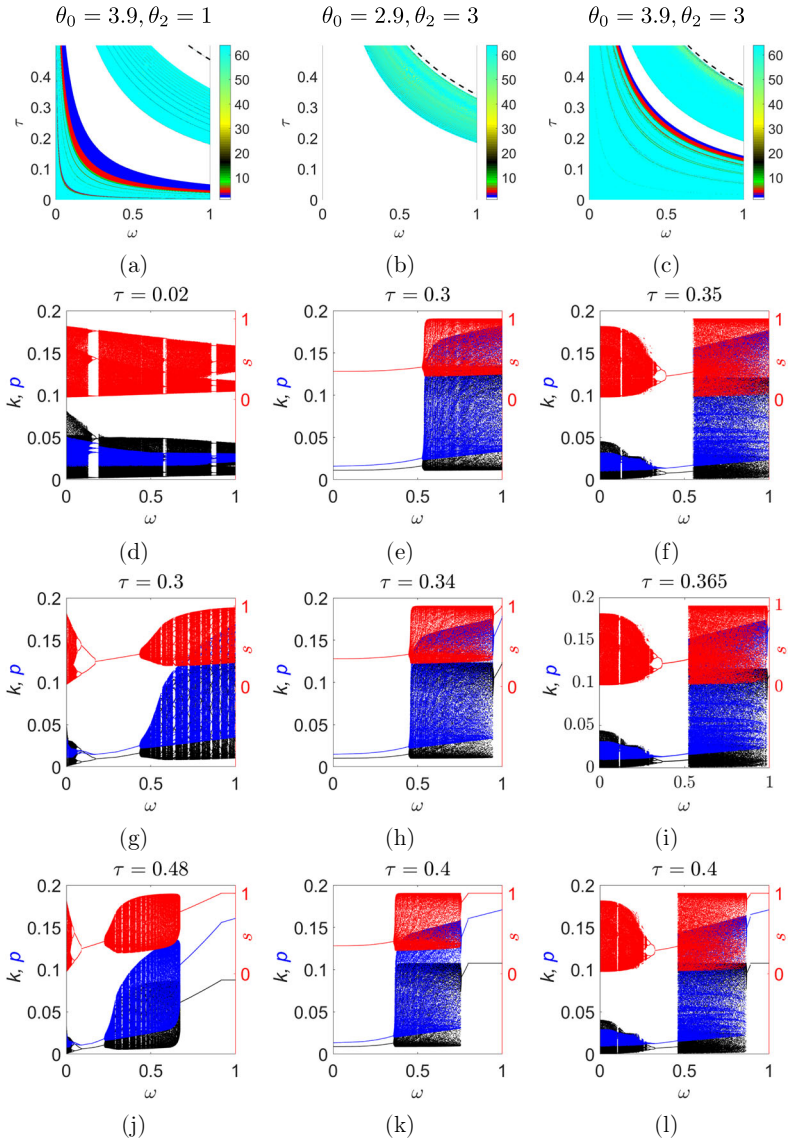

Fig. 4 Endogenous θ, A and β. *First row*: two-dimensional bifurcation diagrams in (ω, τ) plane for different values of θ_0 and θ_2. Attractors are shown in different colors according to their type, with *white* denoting convergence to a steady state, which is the disease-free one in the region above the *dashed line*. For different values of τ, the one-dimensional bifurcation diagrams, on varying ω, in each column are related to the two-dimensional bifurcation diagram in the first row. In each panel, the *black* and *blue bifurcation diagrams* correspond to variables k and p respectively, and refer to the left vertical axis, while the *red bifurcation diagram* corresponds to variable s and refers to the right vertical axis

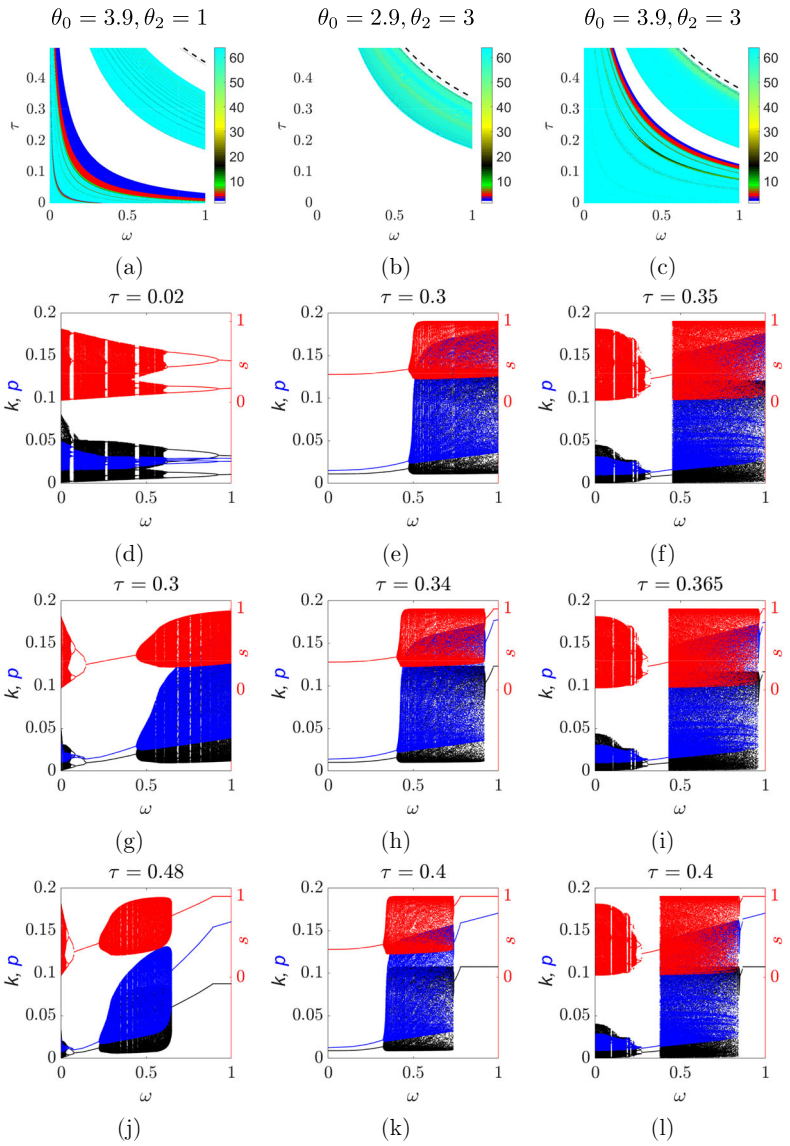

Fig. 5 Endogenous θ, A and β and subsidies. *First row*: two-dimensional bifurcation diagrams in (ω, τ) plane for different values of θ_0 and θ_2. Attractors are shown in different colors according to their type, with *white* denoting convergence to a steady state, which is the disease-free one in the region above the *dashed line*. For different values of τ, the one-dimensional bifurcation diagrams, on varying ω, in each column are related to the two-dimensional bifurcation diagram in the first row. In each panel, the *black* and *blue bifurcation diagrams* correspond to variables k and p, respectively, and refer to the left vertical axis, while the *red bifurcation diagram* corresponds to variable s and refers to the right vertical axis

subsidies and, in addition to tax revenues, we allow the government to issue debt to finance environmental and healthcare expenditures. In Fig. 5, we report the simulation obtained by introducing subsidies in the setting considered for Fig. 4. The additional parameters are $b = 0.02$, $\tau^{ex} = 0.018$ and $\tau^{end} = 0.05\tau$, which means that the endogenous amount of resources allocated to subsidies corresponds to 5% of overall taxation. As in Cavalli et al. (2024c), we remark that the values for these additional parameters are appropriately selected with respect to k_t. As we can see, the results reported in the corresponding panels of Figs. 4 and 5 are very similar, highlighting a mild overall effect on dynamics of the introduction of subsidies. These results support the validity of the analysis for the subsidy-free limiting case. Accordingly, the discussion in the previous sections also applies to the model with subsidies.

6 Conclusions

The study of interacting economic–epidemiological–environmental domains allowed for some interesting insights. Comparative statics show that, in some settings, trade-offs in resource allocation are unavoidable; regulators must therefore balance objectives to avoid excessively penalizing any one dimension. In addition to this, even if, for any reason, it is needed to devote more resources to either healthcare or the environment, it should be clear that this can be a source of instability. The exhibited level of complexity highlights the need for a precise dynamical investigation of the problem. In particular, overly simplified modeling for a single domain may yield misleading results in terms of effective policy interventions. Moreover, dynamical complexity can arise even if each domain, considered on its own, would not be a source of instabilities, and this points out the relevance of an approach based on integrated domains.

For this reason, in future research, we aim to generalize the interdependence between the domains, in particular by introducing a direct effect of environmental quality on productivity. The policy implications of the present model require a deeper analysis of the interaction dynamics and further development of the model, for instance by endogenizing ω so that it can adapt to the given context. Moreover, we aim to address the issue of firm uncertainty in estimating (marginal) total factor productivity, with the goal of proposing and studying suitable approximation methods. Other policy aspects, such as the need to allocate part of the resources to additional areas or to expand public debt while keeping it sustainable, may be the subject of interesting future developments.

Finally, it is worth mentioning that, in models that combine different spheres, it becomes necessary to reconsider, and possibly revise, how time scales are modeled across mechanisms. While a discrete time scale is appropriate for economic decisions, environmental and epidemiological processes typically evolve faster and may be better approximated in continuous time. This also requires reconsidering the information available to agents: for example, the survival probability β, which determines consumption/saving choices of the agents, depends on the epidemiological situation. However, the epidemic evolves within each life period, making it unsatisfactory to describe β as depending solely on the epidemiological state at the beginning of adulthood (s_t) or at retirement (s_{t+1}).

Appendix A Proofs

Proof of Proposition 1 Setting $s_t = s_{t+1} = s$, $k_t = k_{t+1} = k$ and $p_t = p_{t+1} = p$ in Eq. 6 we find

$$\begin{cases} (1-s)\left(\gamma - \theta\left(\frac{\omega g}{p}\right)s\right) = 0, \\ k = (1-\tau)(1-a)\frac{\beta(s)}{1+\beta(s)}A(s)k^a s^{1-a}, \\ p = \frac{\alpha-\lambda(1-\omega)\tau}{\delta}A(s)s^{1-a}k^a. \end{cases} \tag{A1}$$

where $g = \tau A(s)s^{1-a}k^a$. Solving the second equation with respect to k (which is assumed to be non-null), we find

$$k = \left(\frac{\beta(s)}{1+\beta(s)}(1-\tau)(1-a)A(s)\right)^{\frac{1}{1-a}} s \tag{A2}$$

Using the third equation in Eq. A1, we obtain

$$\frac{\omega g}{p} = \frac{\delta\omega\tau}{\alpha - \lambda(1-\omega)\tau}$$

which inserted in the first equation in Eq. A1 allows finding the two solutions $s^*_{df} = 1$ and $s^* = \frac{\gamma}{\theta\left(\frac{\delta\omega\tau}{\alpha-\lambda(1-\omega)\tau}\right)}$. This latter solution is admissible if $s^* < 1$, i.e. $\theta\left(\frac{\delta\omega\tau}{\alpha-\lambda(1-\omega)\tau}\right) > \gamma$, otherwise no endemic steady state is possible. Replacing s with either $s^*_{df} = 1$ or s^* in Eq. A2 and third equation in Eq. A1 allows concluding.

Proof of Proposition 2 Computing the partial derivatives of each component of $\boldsymbol{\xi}^*$ with respect to ω we find

$$\begin{aligned} \frac{\partial s^*}{\partial \omega} &= -\frac{\gamma(\alpha-\lambda\tau)}{\omega[\alpha-\lambda(1-\omega)\tau]\theta(\eta^*)}E_\theta(\eta^*) \\ \frac{\partial k^*}{\partial \omega} &= \frac{k^*}{s^*}\left[1 + \frac{1}{1-a}\left(E_{\frac{\beta}{1+\beta}}(s^*) + E_A(s^*)\right)\right]\frac{\partial s^*}{\partial \omega} \\ \frac{\partial p^*}{\partial \omega} &= \frac{A(s^*)}{\delta}(s^*)^{1-a}(k^*)^a\left[\lambda\tau - \frac{\alpha-\lambda\tau}{\omega}E_\theta(\eta^*)\left(1\right.\right. \\ &\quad \left.\left. + \frac{a}{1-a}E_{\frac{\beta}{1+\beta}}(s^*) + \frac{1}{1-a}E_A(s^*)\right)\right] \\ &= \frac{A(s^*)}{\delta}(s^*)^{1-a}(k^*)^a\left[\lambda\tau - \frac{\alpha-\lambda\tau}{\omega}E_\theta(\eta^*)E_{g^*}(s^*)\right] \end{aligned}$$

Recalling that $E_\theta(g) < 0$ for $g > 0$ and both $E_{\frac{\beta}{1+\beta}}$ and E_A are strictly positive, we can conclude.

Proof of Proposition 3 and 4 We begin with the study of stability for the endemic steady state. We compute each element of the Jacobian matrix of M evaluated at $\boldsymbol{\xi}^*$. We have

$$\begin{aligned} J_{11} &= \frac{\partial M_1}{\partial s}(s,k,p) \\ &= -\theta(\eta)(1-s) - \gamma + s\left[\theta(\eta) - \frac{\omega\tau k^a s^{1-a}}{p}\theta'(\eta)\left(A'(s) + \frac{A(s)}{s}(1-a)\right)(1-s)\right] + 1 \end{aligned}$$

from which

$$J_{11} = -\theta(\eta)(1-s) - \gamma + s\theta(\eta) - \eta\theta'(\eta)\left(s\frac{A'(s)}{A(s)} + 1 - a\right)(1-s) + 1$$

Evaluating it at $\boldsymbol{\xi}^*$, at which we have $s^* = \gamma/\theta(\eta^*)$, we find

$$J_{11}^* = (\gamma - \theta(\eta^*))\left[1 + E_\theta(\eta^*)\left(1 - a + E_A(s^*)\right)\right] + 1$$

We have

$$J_{12} = \frac{\partial M_1}{\partial k}(s,k,p) = -\frac{a\omega\tau A(s)}{p}s(1-s)\theta'(\eta)\left(\frac{s}{k}\right)^{1-a} = -\frac{a\eta\theta'(\eta)s(1-s)}{k}$$

so

$$J_{12}^* = \frac{as^*E_\theta(\eta^*)(\gamma - \theta(\eta^*))}{k^*}$$

We have

$$J_{13} = \frac{\partial M_1}{\partial p}(s,k,p) = s(1-s)\theta'(\eta)\frac{\omega\tau A(s)s^{1-a}k^a}{p^2} = \frac{\eta\theta'(\eta)s(1-s)}{p}$$

so

$$J_{13}^* = -\frac{E_\theta(\eta^*)s^*(\gamma - \theta(\eta^*))}{p^*}$$

Let us consider the second equation of Eq. 6. We have

$$J_{21} = \frac{\partial M_2(s,k,p)}{\partial s} = \frac{M_2}{s}E_{M_2}(s)$$

where

$$E_{M_2}(s) = E_{\beta/(1+\beta)}(s) + E_A(s) + 1 - a$$

$$J_{21} = \frac{\partial M_2(s,k,p)}{\partial s} = \frac{M_2(s,k,p)}{s}\left(E_{\frac{\beta}{1+\beta}}(s) + E_A(s) + 1 - a\right)$$

so

$$J_{21}^* = \frac{k^*}{s^*}\left(E_{\frac{\beta}{1+\beta}}(s^*) + E_A(s^*) + 1 - a\right)$$

We have

$$J_{22} = \frac{\partial M_2(s,k,p)}{\partial k} = a(1-\tau)(1-a)\frac{\beta(s)}{1+\beta(s)}A(s)k^{a-1}s^{1-a} = \frac{aM_2(s,k,p)}{k}$$

and hence $J_{22}^* = a$. Moreover, $J_{23} = J_{23}^* = 0$. Noting that

$$(\alpha - \lambda(1-\omega)\tau)A(s)k^a s^{-a} = \frac{M_3(s,k,p) - (1-\delta)p}{s}$$

we have

$$\begin{aligned} J_{31} &= \frac{\partial M_3(s,k,p)}{\partial s} = (\alpha - \lambda(1-\omega)\tau)A(s)\left(\frac{k}{s}\right)^a\left[s\frac{A'(s)}{A(s)} + 1 - a\right] \\ &= \left(\frac{M_3 - (1-\delta)p}{s}\left[s\frac{A'(s)}{A(s)} + 1 - a\right]\right) \end{aligned}$$

from which

$$J_{31}^* = \frac{\delta p^*}{s^*}(E_A(s^*) + 1 - a)$$

Finally, we have

$$J_{32} = \frac{\partial M_3(s,k,p)}{\partial k} = a(\alpha - \lambda(1-\omega)\tau)A(s)k^{a-1}s^{1-a} = a\frac{M_3 - (1-\delta)p}{k}$$

so

$$J_{32}^* = a\delta\frac{p^*}{k^*}$$

and

$$J_{33} = J_{33}^* = \frac{\partial M_3}{\partial p}(\boldsymbol{\xi}^*) = 1 - \delta$$

The resulting Jacobian matrix is then

$$J^* = \begin{pmatrix} (\gamma - \theta(\eta^*))[1 + E_\theta(\eta^*)(E_A(s^*) + 1 - a)] + 1 & \frac{as^*E_\theta(\eta^*)(\gamma-\theta(\eta^*))}{k^*} & -\frac{E_\theta(\eta^*)s^*(\gamma-\theta(\eta^*))}{p^*} \\ \frac{k^*}{s^*}\left[E_{\frac{\beta}{1+\beta}}(s^*) + E_A(s^*) + 1 - a\right] & a & 0 \\ \frac{\delta p^*}{s^*}(E_A(s^*) + 1 - a) & a\delta\frac{p^*}{k^*} & 1 - \delta \end{pmatrix}$$

As reported in Lines et al. (2020), stability is guaranteed by

$$\begin{cases} 1 + m(J^*) + \mathrm{tr}(J^*) + \det(J^*) > 0 \\ 1 + m(J^*) - \mathrm{tr}(J^*) - \det(J^*) > 0 \\ 1 - (\det(J^*))^2 - m(J^*) + \mathrm{tr}(J^*)\det(J^*) > 0 \\ 3 - m(J^*) > 0 \end{cases}$$

where $m(J^*)$ denotes the sum of principal minors of order two of the Jacobian. We stress that the first and the third conditions are respectively related to the possible emergence of a flip and Neimark–Sacker bifurcation. Since

$$\begin{aligned}
\mathrm{tr}(J^*) =& a - \delta + 2 + (\gamma - \theta(\eta^*))[1 + E_\theta(\eta^*)(E_A(s^*) + 1 - a)] \\
\det(J^*) =& a\left[1 - \delta + (\gamma - \theta(\eta^*))\left(1 - \delta - E_\theta(\eta^*)E_{\frac{\beta}{1+\beta}}(s^*)\right)\right] \\
m(J^*) =& (1 + a)(1 - \delta) + a + (\gamma - \theta(\eta^*))\left[1 + a - \delta + E_\theta(\eta^*)\left(-aE_{\frac{\beta}{1+\beta}}(s^*)\right.\right. \\
& \left.\left. + E_A(s^*) + 1 - a\right)\right]
\end{aligned}$$

we find that the second condition becomes $\delta(\theta(\eta^*) - \gamma)(1 - a) > 0$ and hence it is always fulfilled.

Using the expression of E_{g^*} defined by Eq. 12 and rearranging terms in the first, third, and fourth stability conditions, we find the three conditions in Eq. 13.

We conclude with the study of stability for the disease-free steady state. Recalling the expressions of J_{ij} and evaluating them at $\boldsymbol{\xi}^*_{df} = (1, p^*_{df}, k^*_{df})$ we find that J^*_{df} is a lower triangular matrix in which the diagonal elements, providing its eigenvalues, are

$$J^*_{11} = \theta(\eta^*) - \gamma + 1, \quad J^*_{22} = a \in (0, 1), \quad J^*_{33} = 1 - \delta \in (0, 1).$$

Since $\theta(\eta^*) - \gamma < 0$, $J^*_{11} < 1$ and since, thanks to $\theta(\eta^*) - \gamma > -1$, we have $J^*_{11} > 0$, and this concludes the proof.

Proof of Proposition 6 Since we focus on possible bifurcations, we take into account only the first two conditions in Eq. 14. We recall that $\eta^*(\omega)$ is increasing and $\alpha > \lambda \geq \lambda\tau$ thanks to Assumption 1. Moreover, we recall that the existence of the endemic steady state requires $\gamma/\theta(\eta^*) < 1$. If this is true for any $\omega \in [0, 1]$, we can choose $\omega_{tr} = 1$, otherwise, ω_{tr} corresponds to the value of ω at which $\gamma/\theta(\eta^*) = 1$. In both cases, stability of the endemic steady state must be studied on $[0, \omega_{tr})$.

The remainder of the proof proceeds as follows: we take into account the two stability conditions in Eq. 14 and we find the set on which they are not fulfilled, whose ending points identify possible bifurcation values.

Since

$$\left(\frac{2 - (\theta(\eta) - \gamma)}{(\theta(\eta) - \gamma)}\right)' = -\frac{2\theta'(\eta)}{(\theta(\eta) - \gamma)^2} > 0 \tag{A3}$$

the right-hand side of the first condition in Eq. 14 is strictly increasing while, thanks to $E'_\theta(\eta) < 0$, its left hand side is strictly decreasing, and hence the inequality is either always fulfilled or there exists a unique η_f such that it is true for $\eta > \eta_f$. Consequently, if the inequality is always fulfilled, we do not have any value of ω to remove from the stability set, and we can choose $\omega_f < 0$ so that $[0, \omega_f]$ is empty. Conversely, solving $\frac{\delta\omega\tau}{\alpha - \lambda(1-\omega)\tau} > \eta_f$, we obtain the inequality

$$\tau(\delta - \lambda\eta_f)\omega > (\alpha - \lambda\tau)\eta_f.$$

If $\delta - \lambda\eta_f > 0$, we obtain the stability interval $\omega > \omega_f = \frac{\eta_f(\alpha-\lambda\tau)}{\tau(\delta-\lambda\eta_f)}$, and the first condition in Eq. 14 is false on $[0, \omega_f]$. If instead $\delta - \lambda\eta_f \leq 0$, then the inequality is not fulfilled and we may pose $\omega_f = 1$. This provides the conclusions about the flip bifurcation threshold.

Now we focus on the second condition in Eq. 14. The right-hand side can be rewritten as

$$\frac{\delta}{1-a+a\delta+a(1-\delta)(\theta(\eta)-\gamma)} - \frac{\delta}{\theta(\eta)-\gamma} - \frac{1-a+a\delta}{1-a}$$

Let us introduce $f : \left[0, \min\left\{\frac{\delta\tau}{\alpha}, \theta^{-1}\{\gamma\}\right\}\right] \to \mathbb{R}, \eta \mapsto f(\eta)$ defined by

$$f(\eta) = E_\theta(\eta) - \left(\frac{\delta}{1-a+a\delta+a(1-\delta)(\theta(\eta)-\gamma)} - \frac{\delta}{\theta(\eta)-\gamma} - \frac{1-a+a\delta}{1-a}\right)$$

whose domain takes into account all the possible and feasible values of η^* for $\omega \in [0, 1]$.

We have

$$\frac{(\theta(\eta)-\gamma)^2 f'(\eta)}{\theta'(\eta)} = (\theta(\eta)-\gamma)^2 \frac{E'_\theta(\eta)}{\theta'(\eta)} - \delta + \frac{a\delta(1-\delta)(\theta(\eta)-\gamma)^2}{[1-a+a\delta+a(1-\delta)(\theta(\eta)-\gamma)]^2} \tag{A4}$$

Note that

$$\left(\frac{(\theta(\eta)-\gamma)^2}{[1-a+a\delta+a(1-\delta)(\theta(\eta)-\gamma)]^2}\right)' = \frac{2(1-a+a\delta)(\theta(\eta)-\gamma)}{[1-a+a\delta+a(1-\delta)(\theta(\eta)-\gamma)]^3}\theta'(\eta) < 0 \tag{A5}$$

and

$$\left((\theta(\eta)-\gamma)^2 \frac{E'_\theta(\eta)}{\theta'(\eta)}\right)' = 2(\theta(\eta)-\gamma)E'_\theta(\eta) + (\theta(\eta)-\gamma)^2 \frac{E''_\theta(\eta)\theta'(\eta) - E'_\theta(\eta)\theta''(\eta)}{(\theta'(\eta))^2} \tag{A6}$$

is negative thanks to Eq. 15. This allows concluding that the function $(\theta(\eta) - \gamma)^2 f'(\eta)/\theta'(\eta)$ is strictly decreasing and can have at most one zero. Consequently, f can have at most one critical point, too, which guarantees that $f(\eta) = 0$ can have at most two solutions.

Different situations can occur, but they must be characterized by a positive f on a right neighborhood of $\eta = 0$, since, noting that $E_\theta(0) = 0$, we have

$$f(0) = \frac{(1-a+a\delta)[1-a+a(\theta(0)-\gamma)][\delta+(1-\delta)(\theta(0)-\gamma)]}{(\theta(0)-\gamma)(1-a)[1-a(1-\delta)(1-(\theta(0)-\gamma))]} > 0$$

A first situation is that in which we may have $f(\eta) > 0$ for any η, in which the second condition in Eq. 14 is always fulfilled and we can choose $\omega_{1,ns} > \omega_{2,ns}$ so that $[\omega_{1,ns}, \omega_{2,ns}]$ is empty.

In a second situation, there is just a unique solution to $f(\eta) = 0$ and $f(\eta)$ is positive on $0 < \eta < \eta_{1,ns}$. In this case, setting $\omega_{1,ns}$ as the value for which $\frac{\delta\omega\tau}{\alpha-\lambda\tau+\lambda\omega\tau} = \eta_{1,ns}$ and $\omega_{2,ns} = \omega_{tr}$, we have that the second condition in Eq. 14 is not fulfilled for $\omega \in [\omega_{1,ns}, \omega_{tr}]$. In the last possible situation we have two solutions to $f(\eta) = 0$, and $f(\eta)$ is positive for $\eta < \eta_{1,ns}$ and $\eta > \eta_{2,ns}$ and negative otherwise. Setting $\omega_{1,ns}$ and $\omega_{2,ns}$ as the values for which $\frac{\delta\omega\tau}{\alpha-\lambda\tau+\lambda\omega\tau}$ is equal to $\eta_{1,ns}$ and $\eta_{2,ns}$, respectively, we have that the second condition in Eq. 14 is not fulfilled for $\omega \in [\omega_{1,ns}, \omega_{2,ns}] \cap [0, \omega_{tr}]$. This provides the conclusions about the Neimark–Sacker bifurcation thresholds. □

Proof of Corollary 7 We have $E_\theta(\eta) = -\theta_1\theta_2\eta^{\theta_2}$ and $E'_\theta(\eta) = -\theta_1\theta_2^2\eta^{\theta_2-1} < 0$. Setting $z = \theta_0 e^{-\theta_1\eta^{\theta_2}}$, inequality Eq. 15 corresponds to

$$\theta_1\theta_2^2\eta^{\theta_2-2}(1-\theta_2) > -\theta_1\theta_2^2\eta^{\theta_2-1}\frac{(\theta_2-1)(z-\gamma)+\theta_1\theta_2\eta^{\theta_2}z+\gamma\theta_1\theta_2\eta^{\theta_2}}{\eta(z-\gamma)} \tag{A7}$$

Noting that for $\eta \in \left(0, \min\left\{\frac{\delta\tau}{\alpha}, \theta^{-1}\{\gamma\}\right\}\right)$ we have $z - \gamma > 0$, the previous inequality is equivalent to

$$\theta_1\theta_2\eta^{\theta_2}(z+\gamma) > 0 \tag{A8}$$

and hence it is true. □

Proof of Proposition 9 We stress once more that since we study bifurcations, we take into account only the first two conditions in Eq. 17. We start by noting that the right-hand sides in both conditions in Eqs. 14 and 17 are the same, so we focus on the left-hand sides.

Since $E_\theta(\eta) < 0$, $E_A\left(\frac{\gamma}{\theta(\eta)}\right) > 0$, $E'_\theta(\eta) < 0$ and $\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)' > 0$, we have

$$\left(E_\theta(\eta)\left(1+\frac{1}{1-a}E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)\right)' = E'_\theta(\eta)\left(1+\frac{1}{1-a}E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)+\frac{E_\theta(\eta)}{1-a}\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)' < 0.$$

The left-hand side of the first condition in Eq. 17 is then decreasing and, recalling Eq. A3, the same conclusions of Proposition 6 related to the first stability condition hold also in this situation.

Let us introduce $\tilde{f} : \left[0, \min\left\{\frac{\delta\tau}{\alpha}, \theta^{-1}\{\gamma\}\right\}\right] \to \mathbb{R}$, $\eta \mapsto \tilde{f}(\eta)$ defined by

$$\tilde{f}(\eta) = E_\theta(\eta)\left(1+\frac{1}{1-a}E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right) - \left(\frac{\delta}{1-a+a\delta+a(1-\delta)(\theta(\eta)-\gamma)} - \frac{\delta}{\theta(\eta)-\gamma} - \frac{1-a+a\delta}{1-a}\right)$$

whose domain takes into account all the possible and feasible values of η^* for $\omega \in [0, 1]$. Following the proof of Proposition 6, we want to show that $\left(\frac{(\theta(\eta)-\gamma)^2\tilde{f}'(\eta)}{\theta'(\eta)}\right)'$ is negative, in order to have the same conclusions about the second stability condition.

Recalling Eqs. A4 and A5, this is guaranteed if

$$\left(\frac{(\theta(\eta)-\gamma)^2\left(E_\theta(\eta)\left(1+\frac{1}{1-a}E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)\right)'}{\theta'(\eta)}\right)' < 0$$

We have

$$\left(\frac{(\theta(\eta)-\gamma)^2\left(E_\theta(\eta)\left(1+\frac{1}{1-a}E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)\right)'}{\theta'(\eta)}\right)' = \left(\frac{(\theta(\eta)-\gamma)^2(E_\theta(\eta))'}{\theta'(\eta)}\right)' + \frac{1}{1-a}\left(\frac{(\theta(\eta)-\gamma)^2\left(E_\theta(\eta)E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)'}{\theta'(\eta)}\right)'$$

in which, recalling Eq. A6, the former addend is negative under condition Eq. 15, which is implied by condition Eq. 18 since $E'_\theta(\eta) < 0$, $\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)' > 0$ and $E_A\left(\frac{\gamma}{\theta(\eta)}\right) > 0$. Concerning the latter addend, we have

$$\begin{aligned}\left(\frac{(\theta(\eta)-\gamma)^2\left(E_\theta(\eta)E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)'}{\theta'(\eta)}\right)' &= \left(\frac{(\theta(\eta)-\gamma)^2\left(E'_\theta(\eta)E_A\left(\frac{\gamma}{\theta(\eta)}\right)+E_\theta(\eta)\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)'\right)}{\theta'(\eta)}\right)' \\ &= \left(\frac{(\theta(\eta)-\gamma)^2}{\theta'(\eta)}\right)' E'_\theta(\eta)E_A\left(\frac{\gamma}{\theta(\eta)}\right)+\left(\frac{(\theta(\eta)-\gamma)^2}{\theta'(\eta)}\right)' E_\theta(\eta)\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)' \\ &\quad+\frac{(\theta(\eta)-\gamma)^2}{\theta'(\eta)}E_\theta(\eta)\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)''+\frac{(\theta(\eta)-\gamma)^2}{\theta'(\eta)}E'_\theta(\eta)\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)' \\ &\quad+\frac{(\theta(\eta)-\gamma)^2}{\theta'(\eta)}E''_\theta(\eta)E_A\left(\frac{\gamma}{\theta(\eta)}\right)+\frac{(\theta(\eta)-\gamma)^2}{\theta'(\eta)}E'_\theta(\eta)\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)'\end{aligned}$$

Considering the second and third addends in the previous expression (i.e., those having factor $E_\theta(\eta)$), we have

$$\left(\frac{(\theta(\eta)-\gamma)^2}{\theta'(\eta)}\right)' E_\theta(\eta)\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)' + \frac{(\theta(\eta)-\gamma)^2}{\theta'(\eta)}E_\theta(\eta)\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)'' < 0$$

thanks to the bound on $\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)''$ in Eq. 19. Considering all the remaining addends, we have

$$\left(\frac{(\theta(\eta)-\gamma)^2}{\theta'(\eta)}\right)' E'_\theta(\eta)E_A\left(\frac{\gamma}{\theta(\eta)}\right)+\frac{(\theta(\eta)-\gamma)^2}{\theta'(\eta)}\left[2E'_\theta(\eta)\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)'+E''_\theta(\eta)E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right] < 0$$

thanks to the lower bound on $E''_\theta(\eta)$ in Eq. 18. This allows concluding the proof as in Proposition 6. □

Proof of Corollary 10 We have already shown in the proof of Corollary 7 that $E_\theta(\eta) < 0$. We have

$$E_A(s) = \frac{(1-A_m)s}{(1-A_m)s + A_1A_m - A_0}$$

and hence, setting $z = \theta_0 e^{-\theta_1\eta^{\theta_2}}$,

$$\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)' = \frac{\eta^{\theta_2-1}\gamma\theta_1\theta_2 z(A_1A_m - A_0)(1-A_m)}{[\gamma(1-A_m)+(A_1A_m-A_0)z]^2}$$

which is positive thanks to the first condition in Eq. 21.

Let us focus on condition Eq. 18. Recalling Eq. A7 and using

$$-2E'_\theta(\eta)\frac{\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)'}{E_A\left(\frac{\gamma}{\theta(\eta)}\right)} = \frac{2\eta^{2\theta_2-2}\theta_1^2\theta_2^3 z(A_1A_m - A_0)}{\gamma(1-A_m)+z(A_1A_m-A_0)}$$

condition Eq. 18 can be rephrased into

$$\frac{-(A_1A_m - A_0)z^2 + (3A_1A_m - 3A_0 + 1 - A_m)\gamma z + (1-A_m)\gamma^2}{(z-\gamma)[\gamma(1-A_m)+(A_1A_m-A_0)z]} > 0$$

Since $z > \gamma$, the sign of the left-hand side depends on that of $-(A_1A_m - A_0)z^2 + (3A_1A_m - 3A_0 + 1 - A_m)z\gamma + (1-A_m)\gamma^2$. We study its positivity for $z \in (\gamma, \gamma/A_0)$, which guarantees that condition Eq. 18 is fulfilled for $\eta \in \left(0, \min\left\{\frac{\delta\tau}{\alpha}, \theta^{-1}\{\gamma\}\right\}\right) \cap \left(\theta^{-1}\left(\frac{\gamma}{A_0}\right), \theta^{-1}\left(\frac{\gamma}{A_1}\right)\right)$. The concave polynomial $-(A_1A_m - A_0)z^2 + (3A_1A_m - 3A_0 + 1 - A_m)\gamma z + (1-A_m)\gamma^2$ of degree two in z is positive for $z = \gamma$, while at $z = \gamma/A_0$ positivity requires

$$-A_0^2(A_m+2) + A_0(2 - A_m + 3A_1A_m) - A_1A_m > 0. \tag{A9}$$

The previous condition is fulfilled for $A_0 \in \left(\frac{1}{3}, A_1\right)$. In fact, the left-hand side is a concave parabola in variable A_0. Since for $A_0 = \frac{1}{3}$ we have that

$$-\frac{1}{9}(A_m+2) + \frac{1}{3}(2 - A_m + 3A_1A_m) - A_1A_m = -\frac{4}{9}A_m + \frac{4}{9} \geq 0$$

since $A_m \leq 1$ and for $A_0 = A_1$ we have that

$$(1-A_m)(A_1 - A_1^2) \geq 0$$

this gives Eq. A9.

This guarantees that condition Eq. 18 holds true.

Let us focus on condition Eq. 19. We have

$$\left(E_A\left(\frac{\gamma}{\theta(\eta)}\right)\right)'' = \frac{(A_1A_m - A_0)(1-A_m)\gamma\theta_1\theta_2\eta^{\theta_2-2}z}{[\gamma(1-A_m)+(A_1A_m-A_0)z]^3}[\gamma(1-A_m)(\theta_2 - 1 - \theta_1\theta_2\eta^{\theta_2}) + (A_1A_m - A_0)z(\theta_2 - 1 + \theta_1\theta_2\eta^{\theta_2})]$$

so condition Eq. 19 can be rephrased as

$$-\frac{2\eta^{2\theta_2}\gamma^2\theta_1^2\theta_2^2 z^2(A_1A_m - A_0)(1-A_m)(A_1A_m - A_0 + 1 - A_m)}{\eta^2(z-\gamma)[\gamma(1-A_m)+z(A_1A_m-A_0)]^3} < 0$$

which is true thanks to the first condition in Eq. 21.

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Declarations

Competing interests The authors declare no competing interests.

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