



# FAERE

French Association  
of Environmental and Resource Economists

## Working papers

### Pollution and infectious diseases

Stefano Bosi - David Desmarchelier

WP 2016.22

Suggested citation:

S. Bosi , D. Desmarchelier (2016). Pollution and infectious diseases.  
*FAERE Working Paper, 2016.22.*

ISSN number: 2274-5556

[www.faere.fr](http://www.faere.fr)

# Pollution and infectious diseases\*

Stefano BOSI<sup>†</sup>      David DESMARCHELIER<sup>‡</sup>

May 16, 2016

## Abstract

Recent empirical contributions highlight the negative impact of pollution on labor supply. This relationship is explained by two mechanisms: (1) pollution modifies agents' work-leisure trade-off as it deteriorates their working conditions (incentive effect); (2) a polluted environment is likely to generate more frequent epidemic outbreaks and to affect agents' immune systems (health effect). Bosi *et al.* (2015) explore the aggregate consequences of the incentive effect and show that it can generate endogenous fluctuations of the economic activity. The present paper rather focuses on the health effect as we study a Ramsey model augmented with the spread of infectious disease. We find that industrial pollution may generate limit cycles around an endemic steady state. More precisely, the economic system may undergo a transcritical bifurcation followed by two Hopf bifurcations near this steady state.

**Keywords:** Pollution, SIS model, Ramsey model, Hopf bifurcation, transcritical bifurcation.

**JEL categories:** D9, Q5, I1.

---

\*We would like to thank Julien Arino for helpful comments about the SIS model. This research has been conducted as part of the project LABEX MME-DII (ANR11-LBX-0023-01).

<sup>†</sup>EPEE, University of Evry, Evry, France. Email: sbosi@univ-evry.fr.

<sup>‡</sup>Corresponding author: ECONOMIX, University of Paris X, Nanterre, France. Email: david.desmarchelier@gmail.com.

# 1 Introduction

Recent empirical contributions highlight the negative effect of pollution on labor supply (Graff Zivin and Neidell (2014), Carson *et al.* (2011), Hanna and Oliva (2014)). By studying a database on industrial activities in Mexico City, Hanna and Oliva (2014) find that a one-percent increase in air pollution reduces the number of worked hours by 0.61 percent. On a theoretical ground, two mechanisms contribute to explain this negative impact: (1) an incentive effect through which pollution affects people's work-leisure trade-off by deteriorating their working conditions (Bosi *et al.* (2015)), and (2) a health effect, through which pollution weakens agents' immune systems and increases the likelihood of epidemic outbreaks (Caren (1981)). Consequences of the health effect on economic activities are potentially important, as illness is recognized as one of the main causes of work absenteeism (Akazawa *et al.* (2003)).

In their study of the incentive effect, Bosi *et al.* (2015) use a discrete time Ramsey model in which pollution and labor are non-separable variables of the utility function. In this model, periodic cycles may arise around the steady state through a flip bifurcation when pollution increases labor disutility. Departing from this result, we propose to address the issue of endogenous cycles when the aforementioned health effect operates. Intuition indeed suggests that if pollution affects agents' immune systems, then labor supply should decrease, which, in turn, should lead to a smaller production and so on. This potential rationale for macroeconomic volatility is grounded on a solid body of medical evidence on the effect of pollution immune systems (Caren (1981), Bauer *et al.* (2012)).

In mathematical epidemiology, disease spreading is usually represented by a dynamic system describing the evolution of healthy and unhealthy populations (Hethcote (2009)). The most fundamental of these models is the SIS model (Susceptible-Infected-Susceptible). It explains the spread of an endemic disease for which recovery does not confer immunity: individuals move from the susceptible class ( $S$ ) to the infective one ( $I$ ), and then go back to the susceptible class ( $S$ ). SIS dynamics notably succeed in describing the spread of gonorrhea, chagas disease or Rocky Mountain spotted fever (Hethcote and Van den Driessche (2000)).

Inter-disciplinary contributions, mixing models of disease spreading with microeconomic foundations often held interesting and counter-intuitive results. For instance, a conventional view in mathematical epidemiology suggests that a higher ratio of HIV infected individuals implies more new infections in following periods, while Geoffard and Philipson (1996), by allowing agents to perform microeconomic trade-offs, rather argue that this larger ratio could in fact generate a drop in new infections, as it provides strong incentive for condom adoption. Delfino and Simmons (2000) study the evolution of infected and healthy individuals in a Lotka-Volterra system, augmented with parameters functions of economic variables. They show that, in these conditions, multiple steady state arise. Gersovitz and Hammer (2004) focuses on a dynamic cost-benefit analysis between public prevention and therapeutic efforts, and they compare the centralized and the decentralized solution. They recommend government to levy

taxes for maximizing social welfare.

One of the first attempts to introduce infectious diseases in capital accumulation models is provided by Goenka and Liu (2012). They embed a SIS model in a discrete-time Ramsey economy with endogenous labor supply. While labor force is exclusively composed by healthy individuals, these latter tune their labor supply via a consumption-leisure arbitrage. When studying the aggregate dynamics of the model, they find that periodic cycles and chaotic dynamics arise for highly infectious diseases. Goenka *et al.* (2014) develops a continuous-time version of this model, augmented to take in account optimal health expenditures. In this version of the model, labor supply is inelastic, and therefore, the aggregate labor supply inherits the dynamics of the susceptible class. Goenka *et al.* (2014) assume that spending on public health affects the parameters of infection, namely the probability being infected and the time of recovery. The study also focuses only on the social planner solution. Through a local analysis, Goenka *et al.* (2014) exclude the possibility of endogenous cycles.

We challenge this latter conclusion by considering an economy in which pollution deteriorates the household's immune system. Our model is similar to the one used by Goenka *et al.* (2014) in the sense that it is a continuous-time Ramsey economy where only healthy individuals work. However, we focus on a competitive economy rather than on a planned one. In our model, pollution is an externality generated by production. It increases both the probability of being sick and the time of recovery. The economy exhibits one or two steady states: a disease free and an endemic one. When studying the local dynamics around these steady states, we find two limit cycles (stable and unstable) near the endemic steady state when pollution becomes excessive. Thus, contrary to Goenka *et al.* (2014), we find similarities between the health effect and the incentive effect (Bosi *et al.* (2015)), as both situations generate endogenous macroeconomic cycles around the steady state.

The rest of the paper is organized as follows. We introduce the model and we derive the dynamic system in Sections 2 and 3. In Section 4, we compute the steady state and we formulate the conditions for its existence and uniqueness. Section 5 provides general conditions for local bifurcations and indeterminacy of a three-dimensional system. We also consider the dynamics around the disease-free and around the endemic steady states. We then propose a numerical illustration in Section 6. Section 7 concludes.

## 2 Fundamentals

We consider a continuous-time Ramsey economy with an endemic disease. As in Goenka *et al.* (2014), the labor supply consists only of healthy people but a pollution externality, coming from production, impairs the household's immune system.

## 2.1 Disease

Epidemiologists use the SIS model to study the spread of endemic diseases. Population ( $N$ ) is divided in two classes: susceptible ( $S$ ) and infective ( $I$ ) with  $S + I = N$ . The proportion of susceptible and infective are given by  $s = S/N$  and  $i = I/N$ .  $\beta > 0$  denotes the average number of adequate contacts (sufficient to transmit the disease) of an infective per unit of time and  $S/N$  the probability to face a susceptible during a contact. Thus,  $\beta S/N$  is the average number of adequate contacts with susceptibles of one infective per unit of time, while the number of new infectives per unit of time is given by  $\beta IS/N$ . An infective is seek during a period of time after which he recovers and becomes a new susceptible ( $\gamma = -\dot{I}/I$  is the recovery rate in absence of new contamination, a sort of exponential decay rate from infection). Indeed, the SIS model postulates that the infection does not confer immunity. In the following, for the sake of simplicity, we will omit the time argument  $t$ .

The evolution of  $S$  and  $I$  over time is simply given by:

$$\dot{S} = -\beta \frac{I}{N} S + \gamma I \quad (1)$$

$$\dot{I} = \beta \frac{I}{N} S - \gamma I \quad (2)$$

In an oversimplified world with no births, no deaths, no migrations, the population remains constant over time. Therefore,  $N = S + I$  gives  $\dot{S} + \dot{I} = 0$  and equation (1) becomes:

$$\dot{s} = (1 - s)(\gamma - \beta s) \quad (3)$$

As in Goenka *et al.* (2014), we assume that the labor force ( $L$ ) consists only of healthy people:  $L = S$ . Since  $l = L/N \leq 1$ ,  $l$  inherits the dynamics of  $s$ :

$$\dot{l} = (1 - l)(\gamma - \beta l) \quad (4)$$

We can see that (4) exhibits two steady state:  $l = 1$  and  $l = \gamma/\beta$  with  $\gamma < \beta$ . The first one is called *disease-free* because the disease disappears while the other is called *endemic* because the disease persists. As seen above, some medical evidence highlights the negative effects of pollution ( $P$ ) on the immune system (Caren (1981), Bauer *et al.* (2012)) and supports the theoretical assumption of  $\beta$  and  $\gamma$  and as increasing and decreasing functions of  $P$  respectively.

**Assumption 1** *The function  $\beta(P) : \mathbb{R}_+ \rightarrow \mathbb{R}_+$ , is  $C^2$  with  $\beta'(P) > 0$ ,  $\lim_{P \rightarrow 0} \beta(P) = 0$  and  $\lim_{P \rightarrow +\infty} \beta(P) = +\infty$ .  $\gamma(P) : \mathbb{R}_+ \rightarrow \mathbb{R}_+$  is also  $C^2$  with  $\gamma'(P) < 0$ ,  $\lim_{P \rightarrow 0} \gamma(P) = +\infty$  and  $\lim_{P \rightarrow +\infty} \gamma(P) = 0$ .*

Pollution is a negative externality. Theorists are used to introduce externalities in the fundamentals (production or utility functions). The impact of pollution on preferences has been largely considered in economic literature (Heal (1982), Itaya (2008), Fernandez et al. (2012), Bosi *et al.* (2015) among others).

The channel we focus on is different: pollution affects the economy by rendering diseases more infective and reducing labor supply in turn. The drop in labor supply is not a straight result of the consumption-leisure arbitrage as in Bosi *et al.* (2015), but comes from an incapacity to work due to the infectious disease. We aim at understanding how the sensitivity of the immune system to pollution (captured by the shapes of  $\beta$  and  $\gamma$  with respect to  $P$ ) promotes the occurrence of economic fluctuations through the channel of labor supply.

## 2.2 Preferences

The household earns a capital income  $rh$  and a labor income  $\omega$ , where  $r$  and  $h$  denote respectively the real interest rate and the individual wealth at time  $t$ . Income is consumed and saved/invested according to the budget constraint:

$$\dot{h} \leq (r - \delta)h + \omega - c \quad (5)$$

In this model, healthy people work while sick people don't. However, for simplicity, we assume a perfect social security, that is a full unemployment insurance in the case of illness. Healthy and sick agents earn the same labor income  $\omega$ .  $L$  healthy people supply one unit of labor at a wage  $w$ . Under a balanced-budget rule for social security, we obtain  $\omega N = wL$ . Therefore,  $\omega = wl$ .

Gross investments include the capital depreciation at the rate  $\delta$ . For simplicity, the population of consumers-workers is normalized to unity:  $N = 1$ . Such a normalization implies  $L = Nl = l$ ,  $K = Nh = h$  and  $h = K/N = kl$ .

The household's preferences have standard properties.

**Assumption 2** *Preferences are rationalized by a  $C^2$  felicity function  $u : \mathbb{R}_+ \rightarrow \mathbb{R}_+$  with  $u'(c) > 0$ ,  $u''(c) < 0$ ,  $\lim_{c \rightarrow 0} u'(c) = +\infty$  and  $\lim_{c \rightarrow +\infty} u'(c) = 0$ .*

The illness lowers labor supply and the individual income in turn. The fundamental link between labor supply and pollution is represented by equation 4 with Assumption 1. The agent maximizes the intertemporal utility function  $\int_0^\infty e^{-\rho t} u(c) dt$  under the budget constraint (5), where  $\rho > 0$  is the rate of time preference. Setting the Hamiltonian  $H = e^{-\rho t} u(c) + \mu [(r - \delta)h + \omega - c]$  and deriving the first-order conditions  $\partial H / \partial c = 0$ ,  $\partial H / \partial h = -\dot{\mu}$  and  $\partial H / \partial \mu = \dot{h}$ , we get

$$\dot{h} = (r - \delta)h + \omega - c \quad (6)$$

$$\dot{c} = \varepsilon(c)(r - \delta - \rho)c \quad (7)$$

where  $\varepsilon(c) \equiv -u'(c) / [cu''(c)]$  is the elasticity of intertemporal substitution, jointly with the transversality condition  $\lim_{t \rightarrow +\infty} \mu h = \lim_{t \rightarrow +\infty} e^{-\rho t} u'(c) h = 0$ .

By definition, a central planner internalizes the infectious diseases. Conversely, in a market economy, households take pollution as given. In mathematical terms, pollution is no longer a maximization argument and the model

becomes more tractable because the joint concavity of Hamiltonian with respect to consumption, wealth and pollution is no longer required.<sup>1</sup>

Equation (7), where pollution plays no role, is the core of any Ramsey model. On the one hand, pollution does not change the household's preferences and, on the other hand, agents do not internalize the externality. However, even if pollution does not affect directly the Euler equation (7), that is the intertemporal trade-off between consumption and saving, it promotes infectious disease, lowers labor supply (equation (4)) and income, and, eventually, wealth accumulation through the budget constraint (6).

### 2.3 The representative firm

Firms share the same technology and take prices as given. Under constant returns to scale, their program is equivalent to that of an aggregate representative firm. This firm maximizes the profit  $F(K, L) - rK - wL$  taking as given the real interest rate  $r$  and the real wage  $w$ , where  $Y \equiv F(K, L)$  is the aggregate production function and  $K$  and  $L$  are the aggregate demands for capital and labor at time  $t$ .

**Assumption 3** *The production function  $F : \mathbb{R}_+^2 \rightarrow \mathbb{R}_+$  is  $C^1$ , constant returns to scale, strictly increasing in both the arguments and concave. Standard Inada conditions hold.*

Profit maximization is correctly defined under Assumption 3 and yields the first-order conditions:

$$r = f'(k) \equiv r(k) \text{ and } w = f(k) - kf'(k) \equiv w(k)$$

where  $k \equiv K/L$  denotes the capital intensity and  $f(k) \equiv F(k, 1)$  the average productivity. The second-order conditions are also satisfied because of the concavity.

We introduce the capital share in total income  $\alpha$  and the elasticity of capital-labor substitution  $\sigma$ :

$$\alpha(k) \equiv \frac{kf'(k)}{f(k)} \text{ and } \sigma(k) = \alpha(k) \frac{w(k)}{kw'(k)}$$

In addition, price elasticities depend on them:

$$\frac{kr'(k)}{r(k)} = -\frac{1 - \alpha(k)}{\sigma(k)} \text{ and } \frac{kw'(k)}{w(k)} = \frac{\alpha(k)}{\sigma(k)}$$

### 2.4 Pollution

The aggregate level of pollution  $P$  is a pure externality coming from industrial activity, namely  $Y$ . For simplicity, we assume a linear process  $P = aY$ . The

---

<sup>1</sup>Goenka *et al.* (2014) consider the central planner's solution in a Ramsey model with SIS and provide necessary and sufficient conditions for utility maximization.

lack of persistence means that pollution is a flow as in Itaya (2008) or Fernandez *et al.* (2012) among others.  $a > 0$  captures the effect of production on the environment.  $a$  is expected to play an important role in the stability properties of our economy because, for any production level, a larger environmental impact of industrial activities ( $a$ ) implies a higher pollution level. From Assumption 1, this entails more infective diseases, lower labor supply and production in turn and, at the end, business cycles.

### 3 Equilibrium

Labor supply and demand are equal in equilibrium:  $L = l$ . Because of the constant returns to scale (Assumption 3), the pollution process writes

$$P = alf(k) \quad (8)$$

Under Assumption 1, equations (4) and (8) yield

$$\dot{l} = (1 - l) [\gamma(alf(k)) - \beta(alf(k))l] \quad (9)$$

Therefore, dynamics are determined by (6), (7), (9) and the transversality condition. We observe that  $h = kl$  and, therefore,  $\dot{h}/h = \dot{k}/k + \dot{l}/l$ . Replacing this equality and noticing that  $r(k)k + w(k) = f(k)$ , we obtain a three-dimensional dynamic system

$$\begin{aligned} \dot{k} &= f_1(k, l, c) \equiv f(k) - \delta k - c/l - g(k, l)k \\ \dot{l} &= f_2(k, l, c) \equiv g(k, l)l \\ \dot{c} &= f_3(k, l, c) \equiv \varepsilon(c)[r(k) - \delta - \rho]c \end{aligned} \quad (10)$$

where

$$g(k, l) \equiv [\gamma(alf(k)) - \beta(alf(k))l](1 - l)/l \quad (11)$$

Therefore, the introduction of a disease affecting labor supply adds a third dimension to the basic Ramsey model. Conversely, system (10) simplifies without the epidemiological block: indeed,  $l = 1$  implies  $g(k, l) = 0$  and  $\dot{l} = 0$ . Thus, the second equation in (10) becomes superfluous and we recover exactly a two-dimensional Ramsey model.

Our model is close to Goenka *et al.* (2014) in terms of fundamentals. However, they focus on the central planner's solution and obtain a six-dimensional dynamic system. The central planner decides not only the allocations, but also the optimal pollution level. Our approach (market economy) turns out to be simpler in mathematical terms: pollution is an aggregate externality and, thus, no longer argument of utility maximization. In our case, dynamics are determined by a three-dimensional dynamic system (10). Differently from them, this lower dimensionality allows us to carry out a complete analysis of local bifurcations.

Under strict concavity, the central planner's program yields a unique solution (equilibrium path). Conversely, in a market economy, concavity (Assumption 2

and 3) ensures the individual maximization to be well-defined without excluding equilibrium multiplicity. In this respect, in the stability analysis of market dynamics, it is important to distinguish between backward and forward-looking variables and check the possibility of (local) equilibrium indeterminacy (arising when the dimension of the stable manifold exceeds the number of predetermined variables). System 10, involves two predetermined variables ( $k$  and  $l$ ) and one jump variable ( $c$ ). In the sequel, this distinction will allow us to prove the equilibrium uniqueness in the sense of local determinacy.

## 4 Steady state

In this section, we study the existence and multiplicity of stationary solutions of system (10). At the steady state,  $\dot{k} = \dot{l} = \dot{c} = 0$ , that is:

$$\begin{aligned} r(k) &= \rho + \delta \\ g(k, l) &= 0 \\ c &= [f(k) - \delta k] l \end{aligned} \tag{12}$$

The first equation is the standard Modified Golden Rule (MGR). The existence of a unique solution  $k > 0$  of MGR is ensured by Assumption 3. Replacing  $k$  in the second equation, we get  $l$ . Eventually, knowing  $(k, l)$ , we obtain  $c$  from the third equation. Since  $k$  is unique, the existence and uniqueness of a stationary solution depends upon the number of pairs  $(c, l)$  satisfying the second and the third equation of (12). More precisely, the following characterization holds.

**Proposition 1** *Let Assumptions 1, 2 and 3 hold. The stationary level of capital is unique and given by  $k^* = r^{-1}(\rho + \delta)$ . Call  $l^*$  the solution of  $\gamma(af(k^*)) = \beta(af(k^*))l$ . This solution exists, is unique and positive.*

- (1) *If  $0 < l^* < 1$ , there are two steady states:  
 $(k, l, c) = (k^*, 1, f(k^*) - \delta k^*)$  (disease-free),  
 $(k, l, c) = (k^*, l^*, [f(k^*) - \delta k^*] l^*)$  (endemic).*
- (2) *If  $l^* \geq 1$ , the steady state is unique (disease-free).*

**Proof.** See the Appendix. ■

The last proposition shows that system (10) exhibits two steady states:  $l^*$  and  $l = 1$ , but,  $l^*$  is admissible from an economic point of view only when  $\gamma \leq \beta$ .

To give an intuition for the loss of uniqueness of the disease-free steady state, we consider an economy with low pollution. In this case, the recovery rate ( $\gamma$ ) is large while the probability to become sick after a physical contact with an infected individual ( $\beta$ ) is low (Assumption 1). By consequence, the number of agent who recover from the disease is possibly higher than the number of people becoming ill and the disease will end up being eradicated in the long run. Formally,  $\gamma > \beta$  implies  $l^* > 1$  and, hence, the uniqueness of the disease-free steady state. Now, assume an increase in the pollution level and, thus, an increase in the probability to become ill after a infectious contact ( $\beta$ ), and a

decrease in the recovery rate ( $\gamma$ ). Since the recovery rate remains higher than the probability to become ill ( $\gamma > \beta$ ), the disease will vanish in the long run and the disease-free will remain the only admissible steady state. But, if the recovery rate becomes lower than the probability to become ill ( $\gamma \leq \beta$ ), then the number of new infected individuals exceeds the number of individuals who recover from the disease. In such a situation, the disease will persist in the long run and two steady states will coexist: the endemic and the disease-free regime.

The critical pollution level below which the disease-free steady state becomes unique and above which it coexists with the endemic one, makes the recovery rate equal to the probability of becoming sick ( $\gamma = \beta$ ), that is the number of new infected individuals equal to the number of individuals who recover from the disease. In this critical case, the disease is not eradicated in the long run, but it does not change the labor supply ( $l^* = 1$ ): the endemic and the disease-free steady state coincide. At this critical level, we expect the occurrence of a transcritical bifurcation with an exchange of stability properties between the steady states. This conjecture will be proved in the next section.

Three forces drive this mechanism: (1) the environmental impact of production ( $a$ ) because it raises the pollution level, (2) the sensitivity of the probability to become ill to pollution ( $\beta'(P)$ ) because it increases the number of new infected individuals and (3) the sensitivity of the recovery rate to pollution ( $\gamma'(P)$ ) because it augments the number of individuals who recover from the disease. Since these forces play a role in the multiplicity of steady states, we have to study their effects on the dynamics around any steady state. The rest of the paper is devoted to local dynamics.

## 5 Bifurcations

Let the elasticity of intertemporal substitution be constant:  $\varepsilon(c) = \varepsilon$ . In order to study the local dynamics, we linearize the three-dimensional dynamic system (10) around the steady state and we obtain a Jacobian matrix:

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial k} & \frac{\partial f_1}{\partial l} & \frac{\partial f_1}{\partial c} \\ \frac{\partial f_2}{\partial k} & \frac{\partial f_2}{\partial l} & \frac{\partial f_2}{\partial c} \\ \frac{\partial f_3}{\partial k} & \frac{\partial f_3}{\partial l} & \frac{\partial f_3}{\partial c} \end{bmatrix} = \begin{bmatrix} \rho - k^* g_k & \frac{c}{l^{*2}} - k^* g_l & -\frac{1}{l^*} \\ l^* g_k & l^* g_l & 0 \\ -\varepsilon(\delta + \rho) \frac{1-\alpha}{\sigma} \frac{c^*}{k^*} & 0 & 0 \end{bmatrix} \quad (13)$$

where  $\alpha = \alpha(k^*)$ ,  $\sigma = \sigma(k^*)$  and

$$g_k \equiv \frac{\partial g}{\partial k}(k^*, l^*) = a(\rho + \delta)(1 - l^*)(\gamma' - \beta' l^*) \leq 0$$

$$g_l \equiv \frac{\partial g}{\partial l}(k^*, l^*) = [(\gamma' - \beta' l^*) a f - \beta] \frac{1 - l^*}{l^*} - \frac{1}{l^*} \frac{g}{1 - l^*}$$

because  $\beta' > 0$  and  $\gamma' < 0$  (see Assumption 1).

The full characterization of local bifurcations of a three-dimensional system is complicated. A convenient methodology focuses on the characteristic polynomial whose coefficients depend on the determinant ( $D$ ), the sum of minors of

order two ( $S$ ) and the trace ( $T$ ) of matrix (13):

$$D = -(1 - \alpha)(\rho + \delta)l^*g_l \frac{\varepsilon}{\sigma} \frac{c^*}{k^*l^*} \quad (14)$$

$$S = \rho l^* g_l - \left[ k^* g_k + (1 - \alpha)(\rho + \delta) \frac{\varepsilon}{\sigma} \right] \frac{c^*}{k^* l^*} \quad (15)$$

$$T = \rho - k^* g_k + l^* g_l \quad (16)$$

where

$$\frac{c^*}{k^* l^*} = \frac{\rho + \delta}{\alpha} - \delta$$

In continuous time, a local bifurcation generically arises when the real part of an eigenvalue  $\lambda(p)$  of the Jacobian matrix crosses zero in response to a change of parameter  $p$ . Denoting by  $p^*$  the critical parameter value of bifurcation, we get generically two cases.

(1) When a real eigenvalue crosses zero:  $\lambda(p^*) = 0$ , the system undergoes a saddle-node bifurcation (either an elementary saddle-node or a transcritical or a pitchfork bifurcation) depending upon the number of steady states. According to Proposition (1), system (10) always exhibits two steady states, meaning that  $\lambda(p^*) = 0$  entails the occurrence of a transcritical bifurcation.

(2) When the real part of two complex and conjugate eigenvalues  $\lambda(p) = q(p) \pm ih(p)$  crosses zero, the system undergoes a Hopf bifurcation. More precisely, in this case, we require  $q(p^*) = 0$  and  $h(p) \neq 0$  in a neighborhood of  $p^*$  (see Bosi and Ragot (2011, p. 76)).

System (10) is three-dimensional with two predetermined variables ( $k$  and  $l$ ) and one jump variable ( $c$ ). Thus, multiple equilibria (local indeterminacy) arise when the three eigenvalues of the Jacobian matrix (13) evaluated at the steady state have negative real parts: either  $\lambda_1, \lambda_2, \lambda_3 < 0$  or  $\text{Re } \lambda_1, \text{Re } \lambda_2 < 0$  and  $\lambda_3 < 0$ .

The methodology to analyze local bifurcation in the case of three-dimensional dynamic system is difficult and quite specific. It deserves a short presentation (next three subsections).

## 5.1 Transcritical bifurcation

A transcritical bifurcation is typically associated to an interchange of stability properties between two steady states and occurs when a real eigenvalue crosses zero, say  $\lambda_3 = 0$ .

Focus on the Jacobian matrix  $J$  and consider the determinant, the sum of minors of order two and the trace:

$$\begin{aligned} D &= \lambda_1 \lambda_2 \lambda_3 \\ S &= \lambda_1 \lambda_2 + \lambda_1 \lambda_3 + \lambda_2 \lambda_3 \\ T &= \lambda_1 + \lambda_2 + \lambda_3 \end{aligned}$$

**Proposition 2** (*transcritical characterization*) *In our model, a transcritical bifurcation generically arises if and only if  $D = 0$ . In the transcritical bifurcation value  $p^* = p_S$ , we have*

$$\lambda_1(p_S) = \frac{T(p_S)}{2} - \sqrt{\left[\frac{T(p_S)}{2}\right]^2 - S(p_S)} \quad (17)$$

$$\lambda_2(p_S) = \frac{T(p_S)}{2} + \sqrt{\left[\frac{T(p_S)}{2}\right]^2 - S(p_S)} \quad (18)$$

*These eigenvalues are nonreal if and only if  $T(p_S)^2 < 4S(p_S)$ .*

**Proof.** See the Appendix. ■

We observe that conditions (17) and (18) refer in general to a saddle-node bifurcation (either elementary saddle-node or transcritical or pitchfork). However, in our context, because of the two coalescing steady states, we have, generically, a transcritical bifurcation.

## 5.2 Hopf bifurcation

A Hopf bifurcation occurs when the real part of two complex and conjugate eigenvalues  $\lambda(p) = q(p) \pm ih(p)$  crosses zero. More precisely, we require  $q(0) = 0$  and  $h(p) \neq 0$  in a neighborhood of  $p = 0$ , where  $p = 0$  is the normalized bifurcation value of parameter (see Bosi and Ragot (2011)).

**Proposition 3** (*Hopf characterization*) *In the case of a three-dimensional system, a Hopf bifurcation generically arises if and only if  $D = ST$  and  $S > 0$ .*

**Proof.** See the Appendix. ■

## 5.3 Local indeterminacy

In our economy, there are two predetermined variables ( $k$  and  $l$ ) and a jump variable ( $c$ ). As seen above, indeterminacy requires the three eigenvalues with negative real parts: either  $\lambda_1, \lambda_2, \lambda_3 < 0$  or  $\text{Re } \lambda_1, \text{Re } \lambda_2 < 0$  and  $\lambda_3 < 0$ .

**Proposition 4** (*local indeterminacy*) *In the case of system (10), if all the eigenvalues are real, the equilibrium is locally indeterminate if and only if  $D, T < 0$  and  $S > 0$ .*

**Proof.** See the Appendix. ■

Focus first on Proposition 2 and notice that  $\lambda_1(p_S)$  and  $\lambda_2(p_S)$  may be real or nonreal. If they are real  $\text{Re } \lambda_1(p_S) = \lambda_1(p_S)$  and  $\text{Re } \lambda_2(p_S) = \lambda_2(p_S)$ .

**Proposition 5** (*local indeterminacy through a transcritical bifurcation*) *Let  $p_S$  be the transcritical bifurcation value of a parameter  $p$  such that  $D(p_S) = 0$ . The equilibrium is generically locally indeterminate in a (left or right) neighborhood of  $p_S$  if and only if  $\text{Re } \lambda_1(p_S), \text{Re } \lambda_2(p_S) < 0$ , where  $\lambda_1(p_S)$  and  $\lambda_2(p_S)$  are given by (17) and (18).*

**Proof.** See the Appendix. ■

**Corollary 6** *In our model, local indeterminacy generically occurs through a transcritical bifurcation at  $p = p_S$  if and only if  $D(p_S) = 0$ ,  $S(p_S) > 0$  and  $T(p_S) < 0$ .*

**Proof.** See the Appendix. ■

Focus now on the possibility of local indeterminacy through a Hopf bifurcation.

Notice that, unfortunately, Proposition 4 is of little use because it is difficult to know whether the eigenvalues are real. In the nonreal case, the necessary condition of Proposition 4 still holds. Indeed, indeterminacy ( $\text{Re } \lambda_1 = \text{Re } \lambda_2 < 0$  and  $\lambda_3 < 0$ ) implies

$$\begin{aligned} D &= \lambda_1 \lambda_2 \lambda_3 = \left[ (\text{Re } \lambda_1)^2 + (\text{Im } \lambda_1)^2 \right] \lambda_3 < 0 \\ S &= \lambda_1 \lambda_2 + (\lambda_1 + \lambda_2) \lambda_3 = (\text{Re } \lambda_1)^2 + (\text{Im } \lambda_1)^2 + 2 \text{Re } \lambda_1 \lambda_3 > 0 \\ T &= \lambda_1 + \lambda_2 + \lambda_3 = 2 \text{Re } \lambda_1 + \lambda_3 < 0 \end{aligned}$$

However, the sufficient condition fails: even if

$$D = \lambda_1 \lambda_2 \lambda_3 = \left[ (\text{Re } \lambda_1)^2 + (\text{Im } \lambda_1)^2 \right] \lambda_3 < 0$$

still implies  $\lambda_3 < 0$ , conditions  $D, T < 0$  and  $S > 0$  don't rule out the unpleasant case  $\text{Re } \lambda_1 = \text{Re } \lambda_2 > 0$ .

We provide instead another sufficient condition for local indeterminacy, that is more restrictive.

**Proposition 7** (*local indeterminacy through a Hopf bifurcation*) *Let  $p_H$  the Hopf bifurcation value of a parameter  $p$  such that  $D(p_H) = S(p_H)T(p_H)$  and  $S(p_H) > 0$ . If  $D(p_H) < 0$ , the equilibrium is locally indeterminate for some value of  $p$  around  $p_H$ .*

**Proof.** See the Appendix. ■

Since system (10) has two steady states, we will address the stability issue separately for each one in the next two sections.

## 5.4 Dynamics around the disease-free steady state

Let us apply the general methodology presented above to the disease-free steady state. The disease-free steady state is characterized by  $l = 1$ . In addition,  $\eta = \eta(P) \equiv \beta(P) - \gamma(P)$  may be positive or negative.

We observe that  $g(k^*, 1) = g_k(k^*, 1) = 0$  and  $g_l(k^*, 1) = \beta - \gamma$ . The determinant (14), the sum of minors of order two (15) and the trace (16) become

$$D = -\eta(1 - \alpha)(\delta + \rho) \frac{\varepsilon c^*}{\sigma k^*} \quad (19)$$

$$S = \rho\eta - (1 - \alpha)(\delta + \rho) \frac{\varepsilon c^*}{\sigma k^*} \quad (20)$$

$$T = \eta + \rho \quad (21)$$

where  $\alpha = \alpha(k^*)$ ,  $\sigma = \sigma(k^*)$  and

$$\frac{c^*}{k^*} = \frac{\rho + \delta}{\alpha} - \delta$$

In Section 4, three aspects were pointed out as the driving forces for dynamics: (1) the environmental impact of production ( $a$ ), (2) the sensitivity of the probability to become sick to pollution ( $\beta'(P)$ ) and (3) the sensitivity of recovery rate to pollution ( $\gamma'(P)$ ). In terms of economic meaning and mathematical tractability,  $a$  seems the more convenient parameter to study the bifurcations.

#### 5.4.1 Transcritical bifurcation

**Proposition 8** *In our model, a transcritical bifurcation generically occurs when  $\eta = 0$ . All the eigenvalues are real with*

$$\lambda_1(p_S) = \frac{\rho}{2} - \sqrt{\frac{\rho^2}{4} + (1 - \alpha)(\delta + \rho) \frac{\varepsilon c^*}{\sigma k^*}} < 0 \quad (22)$$

$$\lambda_2(p_S) = \frac{\rho}{2} + \sqrt{\frac{\rho^2}{4} + (1 - \alpha)(\delta + \rho) \frac{\varepsilon c^*}{\sigma k^*}} > \frac{\rho}{2} > 0 \quad (23)$$

when  $\lambda_3$  crosses zero.

**Proof.** We apply Proposition 2. A transcritical bifurcation generically arises if and only if  $D = 0$ , that is  $\eta = 0$ . In addition,  $S = -(1 - \alpha)(\delta + \rho)\varepsilon c^*/(\sigma k^*)$  and  $T = \rho$ . Eigenvalues (17) and (18) become real and equal to (22) and (23). ■

When the transcritical bifurcation occurs  $\lambda_1 < 0 < \lambda_2$  and  $\lambda_3 = 0$ . Moreover,  $\lambda_3 < 0$  (because  $D > 0$ ) if and only if  $\eta < 0$ . Therefore, the disease-free steady state from stable saddle point (with a two-dimensional stable manifold) becomes an unstable saddle point (with a two-dimensional unstable manifold) when  $\eta$  goes through zero from below.

We observe that  $\eta$  is endogenous. However, under Assumption 1, equation (8) implies the existence of a critical parameter value  $a_T$ , a bifurcation point in terms of a fundamental parameter.

**Corollary 9** *There exists a unique transcritical bifurcation point  $a = a_T > 0$  such that  $\eta = 0$ .*

The occurrence of a transcritical bifurcation is not surprising. According to Section 4,  $\eta = 0$  implies that the number of new infected individuals is just equal to the number of individuals that recover from the disease. That is, even if the disease persists, it is not enough infective to modify the level of labor supply ( $l^* = 1$ ), that is, when  $a = a_T$ , the disease-free and the endemic steady state coalesce and exchange their stability properties.

### 5.4.2 Hopf bifurcation

In the case of disease-free steady state, the Hopf bifurcation makes no sense from an economic point of view because a limit cycle around  $l = 1$  implies meaningless values  $l > 1$ . In addition, Proposition 3 rules out any limit cycle also from a mathematical point of view even if the restriction  $l > 1$  is not taken into account.

**Proposition 10** *There is no room for limit cycles.*

**Proof.** Assume that a Hopf bifurcation occurs. Proposition 3 implies  $S > 0$ , that is  $\eta > 0$ , which implies in turn  $T > 0$  (see 21) and  $D < 0$  (see 19), that is  $D \neq ST$ , a contradiction. ■

### 5.4.3 Local determinacy

**Proposition 11** *There is no room for local indeterminacy.*

**Proof.** When a transcritical bifurcation occurs  $\eta = 0$ ,  $D = 0$ ,  $S < 0$  and  $T > 0$ . Corollary 6 applies. ■

Noticing that two variables are predetermined and one eigenvalue is always negative (Proposition 8), we reach the same conclusion.

## 5.5 Dynamics around the endemic steady state

Let us analyze the dynamics around the endemic steady state using the general methodology presented above (subsections 5.1 to 5.3). The following lemma allows us to carry out the bifurcation analysis.

**Lemma 12** *The determinant, the sum of minors of order two and the trace of matrix (13) around the endemic steady state become:*

$$D = \eta(1 + \theta)(1 - \alpha)(\rho + \delta) \frac{\varepsilon}{\sigma} \frac{c^*}{k^* l^*} > 0 \quad (24)$$

$$S = -\rho\eta(1 + \theta) + \left[ \alpha\eta\theta - (1 - \alpha)(\rho + \delta) \frac{\varepsilon}{\sigma} \right] \frac{c^*}{k^* l^*} \quad (25)$$

$$T = \rho - \eta - \eta\theta(1 - \alpha) \quad (26)$$

where  $c^*/(k^* l^*) = (\rho + \delta)/\alpha - \delta$ .

**Proof.** Consider (14), (15) and (16). Endemic means  $l^* \in (0, 1)$  and, then,  $l^* = \gamma/\beta$ . Thus,  $k^* g_k = -\alpha\eta\theta < 0$  and  $l^* g_l = -\eta(1 + \theta) < 0$ , where  $\eta = \eta(P) \equiv \beta(P) - \gamma(P) > 0$  and

$$\theta = \theta(P) \equiv \frac{P\beta'(P)}{\beta(P)} - \frac{P\gamma'(P)}{\gamma(P)} > 0$$

because  $\beta - \gamma = \beta(1 - l^*) > 0$ ,  $\beta' > 0$  and  $\gamma' < 0$ . ■

### 5.5.1 Transcritical bifurcation

**Corollary 13** *A transcritical bifurcation occurs at  $\eta = 0$*

**Proof.** Simply apply Proposition 2. ■

Both the steady states (endemic and disease-free) are concerned by this bifurcation. Indeed, they coalesce at  $\eta = 0$  and the eigenvalues become the same for both.

### 5.5.2 Hopf bifurcation

**Proposition 14** *Let  $l^*(a)$  be solution of equation*

$$\gamma(al^*f(k^*)) = \beta(al^*f(k^*))l^* \quad (27)$$

*The Hopf bifurcation value  $a_H$  is solution of equation*

$$\eta(al^*(a)f(k^*)) = \frac{2m\rho}{1 - m\alpha\theta \pm \sqrt{(1 + m\alpha\theta)^2 - 4m(1 + \theta)}} \quad (28)$$

where

$$m \equiv \frac{\frac{\varepsilon}{\sigma} \frac{1-\alpha}{\alpha} \frac{\rho+\delta}{\rho}}{\theta - \frac{\rho(1+\theta)}{\rho+(1-\alpha)\delta}} \quad (29)$$

with

$$1 - m\alpha\theta > 0 \text{ and } (1 + m\alpha\theta)^2 - 4m(1 + \theta) > 0 \quad (30)$$

**Proof.** See the Appendix. ■

The reader may question whether the set of values of fundamental parameters compatible with this bifurcation is nonempty. We observe that inequalities (30) are satisfied by values of  $m$  in a neighborhood of 0. In terms of fundamentals parameters, this happens, for instance, when the income effects are dominant ( $\varepsilon$  is sufficiently low) or input are substitutable ( $\sigma$  is sufficiently high). Of course, these conditions are sufficient and don't preclude other parameter configurations compatible with the occurrence of Hopf bifurcations.

We will introduce isoelastic fundamentals (technology and preferences with constant elasticities) to provide numerical simulations. We will fix a sufficiently low elasticity of intertemporal substitution to capture the case of dominant income effects.

Let us interpret the emergence of a limit cycle through a Hopf bifurcation around the endemic steady state. Consider the economy in this steady state at time  $t$  and an exogenous increase in the pollution stock. Assumption 1 entails a lower recovery rate ( $\gamma$ ) and a higher probability to become ill after a physical contact with an infected individual ( $\beta$ ). Eventually, the higher pollution level results in lower labor supply and household's income. According to 7, under a large income effect (keep  $\varepsilon$  close to 0), the household reduces her saving to smooth consumption over time. Capital accumulation slows down and the lower

capital level at  $t + 1$  results in a lower production pollution level at  $t + 1$ . Thus, a drop follows the rise in pollution and an endogenous fluctuation takes place.

The main rationale for business cycles rests on the negative effect of pollution on labor supply. In a recent contribution, Bosi *et al.* (2015) have also pointed out the negative relation between pollution and labor supply as a source of cycles, but the mechanism at work looks very different. Indeed, in their model, pollution affects labor supply through the incentive channel: more precisely, pollution raises the marginal disutility of labor supply by worsening working conditions and induces agents to substitute working time with leisure. Conversely, in our paper, pollution has no direct effect on households' preferences (Assumption 2), but drops labor supply through its health effects (Assumption 1). According to medical evidences (Caren (1981), Bauer *et al.* (2012)), pollution raises the infectivity of an endemic disease by reducing the recovery rate and augmenting the probability of becoming sick after a physical contact with an infected individual (Assumption 1). Since an infected individual is less able or unable to work, a higher pollution level results in a lower labor supply. We call this mechanism the health channel. Proposition 14 enriches the existing literature on environmental business cycles by proposing a new mechanism, the health channel, as a possible rationale for endogenous cycles.

### 5.5.3 Local determinacy

**Corollary 15** *One eigenvalue is positive and the endemic steady state is locally determinate.*

**Proof.** We observe that  $D > 0$ . This implies that there exists one positive real eigenvalue. Indeed, if all the eigenvalues are real and negative,  $D = \lambda_1 \lambda_2 \lambda_3 < 0$ , a contradiction. If one eigenvalue is real and negative, and the other two are nonreal, we obtain  $D = \lambda_1 \lambda_2 \lambda_3 = \left[ (\text{Re } \lambda_1)^2 + (\text{Im } \lambda_1)^2 \right] \lambda_3 < 0$ , a contradiction. Locally indeterminacy requires that all the eigenvalues have negative real parts. ■

**Corollary 16** *The endemic steady state is saddle-path stable if  $S > 0$  and  $T < 0$ , that is if*

$$\eta \left( \alpha \theta - \rho (1 + \theta) \frac{k^* l^*}{c^*} \right) > (1 - \alpha) (\rho + \delta) \frac{\varepsilon}{\sigma}$$

$$\eta [1 + (1 - \alpha) \theta] > \rho$$

where

$$\frac{c^*}{k^* l^*} = \frac{\rho + \delta}{\alpha} - \delta$$

**Proof.** Because one eigenvalue (say  $\lambda_3$ ) is positive and two variables are pre-determined ( $k$  and  $l$ ), saddle-path stability holds if and only if the other two eigenvalues have negative real parts, that is  $\lambda_1 \lambda_2 > 0$  and  $\lambda_1 + \lambda_2 < 0$ . A

sufficient condition (but not necessary) is

$$\begin{aligned} S &= \lambda_1 \lambda_2 + (\lambda_1 + \lambda_2) \lambda_3 > 0 \\ T &= \lambda_1 + \lambda_2 + \lambda_3 < 0 \end{aligned}$$

■

We observe that, in the case of a Hopf bifurcation, the steady state is characterized by  $\lambda_3 > 0$  and a change of sign of  $\operatorname{Re} \lambda_1 = \operatorname{Re} \lambda_2$ . In the case of a supercritical Hopf, the steady state from a saddle point (with unstable manifold of dimension one and central manifold of dimension two) becomes a source and a stable limit cycle arises around. In this case, the equilibrium is locally unique and converges to this stable limit cycle.

## 6 Simulations

Proposition 14 provides a critical value for the occurrence of limit cycles. The present section illustrates this proposition through a graphical example of limit cycle based on a calibrated simulation.<sup>2</sup>

Notice that  $\alpha = \alpha(k^*)$ ,  $\sigma = \sigma(k^*)$ ,  $\varepsilon = \varepsilon(c)$ ,  $\eta = \eta(P)$ ,  $\theta = \theta(P)$ . This means that  $\alpha$  and  $\sigma$  depend on each other. Fortunately, the Cobb-Douglas case makes them independent and computation feasible. To simplify, we assume  $\sigma = 1$  (Cobb-Douglas) and constant elasticities for  $u$ ,  $\beta$  and  $\gamma$ :

$$\varepsilon, \varepsilon_\beta \equiv \frac{P\beta'(P)}{\beta(P)} > 0 \text{ and } \varepsilon_\gamma \equiv -\frac{P\gamma'(P)}{\gamma(P)} > 0$$

which implies in turn a constant  $\theta = \varepsilon_\beta + \varepsilon_\gamma > 0$ . However,  $\eta$  remains endogenous and depends on  $P$ .

$$\begin{aligned} \beta(P) &\equiv BP^{\varepsilon_\beta} \text{ and } \gamma(P) \equiv GP^{-\varepsilon_\gamma} \\ \eta(P) &= BP^{\varepsilon_\beta} - GP^{-\varepsilon_\gamma} \end{aligned}$$

Let us solve equation (27)

$$G [al^* f(k^*)]^{-\varepsilon_\gamma} = B [al^* f(k^*)]^{\varepsilon_\beta} l^*$$

to obtain

$$l^*(a) = \left(\frac{G}{B}\right)^{\frac{1}{1+\theta}} [af(k^*)]^{-\frac{\theta}{1+\theta}}$$

where  $k^* = r^{-1}(\rho + \delta)$ .

The transcritical critical value corresponds to  $\eta(P) = 0$ , that is to  $BP^{\varepsilon_\beta} = GP^{-\varepsilon_\gamma}$ . Let, for simplicity,  $B = G$ . We get  $P = 1$ . Since  $P = alf(k)$ , the transcritical critical value solves  $al^* f(k^*) = 1$ , that is

$$a [af(k^*)]^{-\frac{\theta}{1+\theta}} f(k^*) = 1 \tag{31}$$

---

<sup>2</sup>The numerical exercise is done by using the MATCONT package (version 5p4) for MATLAB.

The solution of equation

$$z(a) \equiv \left( [af(k^*)]^{\frac{1}{1+\theta}} \right)^{\varepsilon_\beta} - \left( [af(k^*)]^{\frac{1}{1+\theta}} \right)^{-\varepsilon_\gamma} \\ = \frac{2m\rho}{1 - m\alpha\theta \pm \sqrt{(1 + m\alpha\theta)^2 - 4m(1 + \theta)}} > 0 \quad (32)$$

gives us the Hopf bifurcation value  $a_H$ .

Notice that the RHS does not depend on  $a$  and that  $z(0) = -\infty$ ,  $z(+\infty) = +\infty$  and  $z'(a) > 0$ . Thus,  $z$  crosses the RHS for a positive value  $a_H$ .

Let  $f(k) = Ak^\alpha$ . We find explicitly

$$k^* = \left( \frac{\alpha A}{\rho + \delta} \right)^{\frac{1}{1-\alpha}} \quad \text{and} \quad f(k^*) = A \left( \frac{\alpha A}{\rho + \delta} \right)^{\frac{\alpha}{1-\alpha}}$$

Equation (32) writes now

$$\left( \left[ aA \left( \frac{\alpha A}{\rho + \delta} \right)^{\frac{\alpha}{1-\alpha}} \right]^{\frac{1}{1+\theta}} \right)^{\varepsilon_\beta} - \left( \left[ aA \left( \frac{\alpha A}{\rho + \delta} \right)^{\frac{\alpha}{1-\alpha}} \right]^{\frac{1}{1+\theta}} \right)^{-\varepsilon_\gamma} \\ = \frac{2m\rho}{1 - m\alpha\theta \pm \sqrt{(1 + m\alpha\theta)^2 - 4m(1 + \theta)}} \quad (33)$$

under two restrictions:

$$(1 + m\alpha\theta)^2 - 4m(1 + \theta) > 0 \\ 1 - m\alpha\theta > 0$$

Consider the following calibration:

Parameters	$A$	$\alpha$	$\delta$	$\rho$	$\varepsilon$	$\varepsilon_\beta$	$\varepsilon_\gamma$
Value	1	0.33	0.025	0.01	0.01	1	1

(34)

$\alpha$ ,  $\delta$  and  $\rho$  take quarterly values. Parameters  $\varepsilon$ ,  $\varepsilon_\beta$  and  $\varepsilon_\gamma$  fit the restrictions in Proposition 14.

Solving (31), we get the transcritical critical value:

$$a_T = 0.33117$$

According to (32), Hopf bifurcations occur at

$$a_+ \approx 0.33178 \quad (35)$$

$$a_- \approx 0.33257 \quad (36)$$

MATCONT can detect a local bifurcation when the bifurcation parameter varies in a convenient range. In our case, we consider an interval around the critical values and we verify that MATCONT finds in this range the same bifurcation

values we have obtained with (32).<sup>3</sup> Figure 1 is generated by MATCONT and shows all the pollution steady state values ( $P$ ) in the interval  $(0.331, 0.333) \ni a$ .

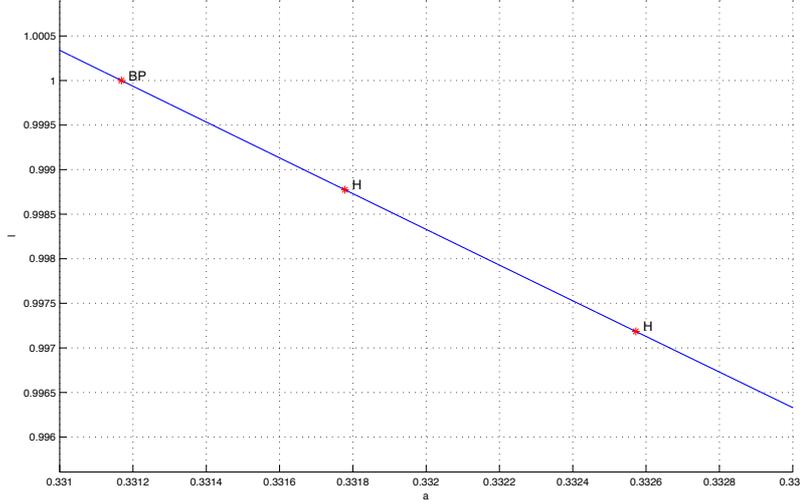


Figure 1:  $P$  when  $a \in (0.331, 0.333)$ .

In Figure 1,  $BP$  and  $H$  denote respectively the occurrence of a transcritical bifurcation (branch point) and a Hopf bifurcation. The following table summarizes the results obtained with MATCONT:

Bifurcation type	value of $a$	First Lyapunov coeff.	Steady state	Eigenvalues
Transcritical ( $BP$ )	0.33117		$k^* = 28.470616$ $l^* = 1$ $c^* = 2.307845$	$\lambda_1 = -0.00163391$ $\lambda_2 = 0$ $\lambda_3 = 0.0116339$
Hopf ( $H$ )	0.33178	$1.453221 * 10^{-3}$	$k^* = 28.470616$ $l^* = 0.998775$ $c^* = 2.305017$	$\lambda_1 = 0.00713083$ $\lambda_2 = -0.00313139i$ $\lambda_3 = 0.00313139i$
Hopf ( $H$ )	0.33257	$-6.234565 * 10^{-3}$	$k^* = 28.470616$ $l^* = 0.997185$ $c^* = 2.301348$	$\lambda_1 = 0.00340303$ $\lambda_2 = -0.00687335i$ $\lambda_3 = 0.00687335i$

We observe that the two Hopf boundaries evaluated by MATCONT are very close to ours (see (35) and (36)). We know that  $s \equiv S/N = L/N \equiv l$  (Section 2.1). Focus on the first Hopf bifurcation in the above table: healthy people represent around 99.88% of the whole population. This share slightly lowers to 99.72% in the case of the second Hopf bifurcation. In epidemiological literature, gonorrhoea is a typical infectious disease correctly represented through a SIS

<sup>3</sup>MATCONT uses the original nonlinear system (10) instead of the Jacobian matrix.

dynamics (Hethcote and Van den Driessche (2000)). Interestingly, the share of infected individuals at the first Hopf bifurcation point (that is 0.12%) is close to what is empirically observed. For instance, in the U.S., the Centers for Disease Control and Prevention (U.S. Department of Health and Human Services) report in their 2015 survey that the national gonorrhea rate is about 0.11% in 2014.

The first Lyapunov coefficient associated with the Hopf boundary provides informations about the stability of the associated limit cycle. In particular, the first Hopf bifurcation ( $a = 0.331778$ ) is subcritical and gives rise to an unstable limit cycle (see Figure 2) while the second Hopf bifurcation ( $a = 0.332572$ ) is supercritical and generates a stable limit cycle (see Figure 3).

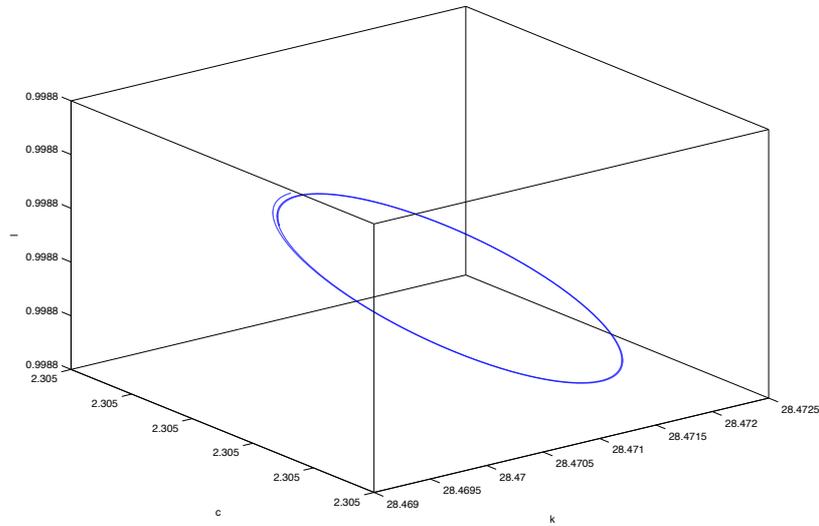


Figure 2: The unstable limite cycle.

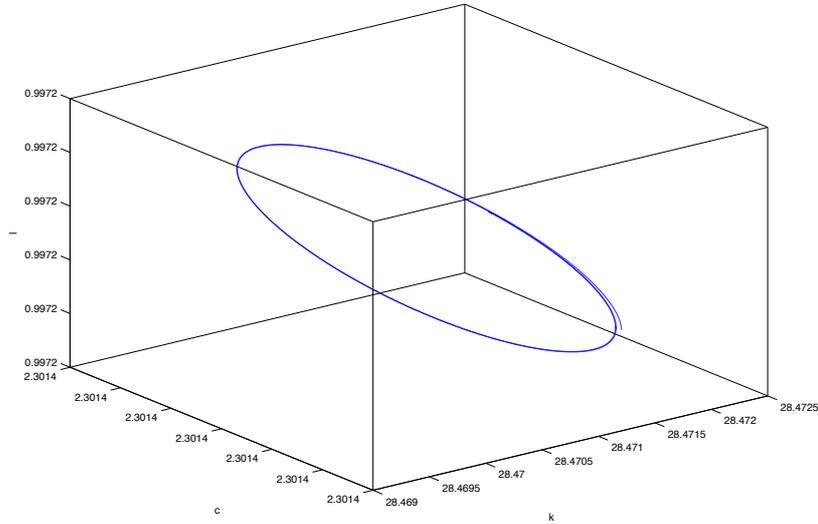


Figure 3: The stable limit cycle

The issue of equilibrium uniqueness (determinacy) in the case of a three-dimensional system with two predetermined variables deserves some additional comment.

Focus first on the transcritical bifurcation, characterized by the exchange of stability of two steady states. Assume, without loss of generality, a bifurcation such that the endemic saddle point becomes a source, while the disease-free steady state experiences the converse. More precisely, the stable manifold around the endemic (disease-free) steady state loses (gains) a dimension passing from two to one (from one to two).

In a three-dimensional space, two predetermined variables ( $k$  and  $l$ ) fix a line. The intersection of this line with a two-dimensional stable manifold is generically unique and identifies a unique starting point, that is a unique equilibrium path converging to the steady state and lying on the two-dimensional stable manifold around.

Before (after) the transcritical bifurcation, the unique equilibrium lies on the two-dimensional stable manifold converging to the endemic (disease-free) steady state. In both the cases, the equilibrium remains unique.

Consider now the Hopf bifurcations around the endemic steady state.

The first one is subcritical. Assume that, without loss of generality, this bifurcation generates a limit cycle on the two-dimensional center manifold around the endemic steady state. If  $k$  and  $l$  are in a neighborhood of the endemic steady state, they fix a line in a neighborhood of the limit cycle and a unique intersection of this line with the center manifold. This intersection determines a unique starting point and a unique equilibrium trajectory. If the intersection is inside the limit cycle, the equilibrium path converges to the endemic steady state following a spiral. If it is outside, the trajectory diverges from the limit cycle and

the equilibrium may fail to exist (because of the transversality condition or the non-negativity constraints) or converge to the disease-free steady state. In the last case, the equilibrium exists, but it remains unique because of the unique starting point and there is no room for global indeterminacy. However, the local analysis does not allow us to conclude about equilibrium existence outside the limit cycle.

The second Hopf bifurcation is supercritical. Applying the same arguments, we find that a starting point inside (outside) the limit cycle determines an equilibrium trajectory converging to the cycle following an internal (external) spiral. In both the cases, the equilibrium exists and it is unique because the starting point is unique, that is the intersection of the line determined by  $k$  and  $l$ , and the center manifold in a neighborhood of the limit cycle.

## 7 Conclusion

In this paper, we addressed convergence issues for an economy in which pollution lowers the labor supply through a so-called health effect. In this purpose, we considered an augmented Ramsey model with the spread of an infectious disease. This spreading mechanism takes the form of a SIS model in which pollution increases both the probability of being infected and the time of recovery.

We provide general conditions under which the disease-free steady state is either the only admissible regime or coexists with an endemic one. We then demonstrate that the economic system may undergo a transcritical bifurcation followed by two Hopf bifurcations (unstable and stable limit cycles) near the endemic steady state. Such a situation emerges when the industrial pollution becomes excessive. It follows that, the health channel by which pollution reduces labor supply, an empirically grounded impact, is likely to generate endogenous cycles of economic activities. In this respect, the health channel represents an alternative rationale for cycles to the incentive channel considered by Bosi *et al.* (2015).

## 8 Appendix

### Proof of Proposition 1

Define  $g(l) \equiv \gamma(alf(k^*))$  and  $b(l) \equiv \beta(alf(k^*))l$ . Under Assumption 3,  $g(0) = +\infty$ ,  $g(+\infty) = 0$ ,  $g'(l) < 0$ ,  $b(0) = 0$ ,  $b(+\infty) = +\infty$ ,  $b'(l) > 0$ . Continuity and strict monotonicity on  $\mathbb{R}_+$ , and the limit conditions imply that the intersection of the graphs of  $g$  and  $b$  exists and is unique. In addition, the corresponding abscissa  $l^*$  is positive. ■

### Proof of Proposition 2

Generically,  $\lambda_3 = 0$  if and only if  $D = 0$ . In this case,  $S = \lambda_1\lambda_2$  and  $T = \lambda_1 + \lambda_2$ . Solving this system of two equations for  $\lambda_1$  and  $\lambda_2$ , we get (17) and (18). ■

### Proof of Proposition 3

*Necessity* In a three-dimensional dynamic system, we require at the bifurcation value:  $\lambda_1 = i\psi = -\lambda_2$  with no generic restriction on  $\lambda_3$  (see Bosi and Ragot (2011) or Kuznetsov (1998) among others). The characteristic polynomial of  $J$  is given by:  $P(\lambda) = (\lambda - \lambda_1)(\lambda - \lambda_2)(\lambda - \lambda_3) = \lambda^3 - T\lambda^2 + S\lambda - D$ . Using  $\lambda_1 = i\psi = -\lambda_2$ , we find  $D = \psi^2\lambda_3$ ,  $S = \psi^2$ ,  $T = \lambda_3$ . Thus,  $D = ST$  and  $S > 0$ .

*Sufficiency* In the case of a three-dimensional system, one eigenvalue is always real, the others two are either real or nonreal and conjugated. Let us show that, if  $D = ST$  and  $S > 0$ , these eigenvalues are nonreal with zero real part and, hence, a Hopf bifurcation generically occurs.

We observe that  $D = ST$  implies

$$\lambda_1\lambda_2\lambda_3 = (\lambda_1\lambda_2 + \lambda_1\lambda_3 + \lambda_2\lambda_3)(\lambda_1 + \lambda_2 + \lambda_3)$$

or, equivalently,

$$(\lambda_1 + \lambda_2) [\lambda_3^2 + (\lambda_1 + \lambda_2)\lambda_3 + \lambda_1\lambda_2] = 0 \quad (37)$$

This equation holds if and only if  $\lambda_1 + \lambda_2 = 0$  or  $\lambda_3^2 + (\lambda_1 + \lambda_2)\lambda_3 + \lambda_1\lambda_2 = 0$ . Solving this second-degree equation for  $\lambda_3$ , we find  $\lambda_3 = -\lambda_1$  or  $-\lambda_2$ . Thus, (37) holds if and only if  $\lambda_1 + \lambda_2 = 0$  or  $\lambda_1 + \lambda_3 = 0$  or  $\lambda_2 + \lambda_3 = 0$ . Without loss of generality, let  $\lambda_1 + \lambda_2 = 0$  with, generically,  $\lambda_3 \neq 0$  a real eigenvalue. Since  $S > 0$ , we have also  $\lambda_1 = -\lambda_2 \neq 0$ . We obtain  $T = \lambda_3 \neq 0$  and  $S = D/T = \lambda_1\lambda_2 = -\lambda_1^2 > 0$ . This is possible only if  $\lambda_1$  is nonreal. If  $\lambda_1$  is nonreal,  $\lambda_2$  is conjugated, and, since  $\lambda_1 = -\lambda_2$ , they have a zero real part. ■

#### **Proof of Proposition 4**

*Necessity* In the real case, we obtain  $D = \lambda_1\lambda_2\lambda_3 < 0$ ,  $S = \lambda_1\lambda_2 + \lambda_1\lambda_3 + \lambda_2\lambda_3 > 0$  and  $T = \lambda_1 + \lambda_2 + \lambda_3 < 0$ .

*Sufficiency* We want to prove that, if  $D, T < 0$  and  $S > 0$ , then  $\lambda_1, \lambda_2, \lambda_3 < 0$ . Notice that  $D < 0$  implies  $\lambda_1, \lambda_2, \lambda_3 \neq 0$ .

$D < 0$  implies that at least one eigenvalue is negative. Let, without loss of generality,  $\lambda_3 < 0$ . Since  $\lambda_3 < 0$  and  $D = \lambda_1\lambda_2\lambda_3 < 0$ , we have  $\lambda_1\lambda_2 > 0$ . Thus, there are two subcases: (1)  $\lambda_1, \lambda_2 < 0$ , (2)  $\lambda_1, \lambda_2 > 0$ . If  $\lambda_1, \lambda_2 > 0$ ,  $T < 0$  implies  $\lambda_3 < -(\lambda_1 + \lambda_2)$  and, hence,

$$S = \lambda_1\lambda_2 + (\lambda_1 + \lambda_2)\lambda_3 < \lambda_1\lambda_2 - (\lambda_1 + \lambda_2)^2 = -\lambda_1^2 - \lambda_2^2 - \lambda_1\lambda_2 < 0$$

a contradiction. Then,  $\lambda_1, \lambda_2 < 0$ . ■

#### **Proof of Proposition 5**

$D(p_S) = 0$  if and only if  $\lambda_3(p_S) = 0$  without loss of generality.

*Necessity* If the equilibrium is locally indeterminate in a (left or right) neighborhood of  $p_S$ , then there exists  $\varepsilon > 0$  such that  $\text{Re } \lambda_1(p), \text{Re } \lambda_2(p), \lambda_3(p_S) < 0$  for any  $p \in (p_S - \varepsilon, p_S)$  or for any  $p \in (p_S, p_S + \varepsilon)$ , and, hence, generically,  $\text{Re } \lambda_1(p_S), \text{Re } \lambda_2(p_S) < 0$  and  $\lambda_3(p_S) = 0$ .

*Sufficiency* If  $\text{Re } \lambda_1(p_S), \text{Re } \lambda_2(p_S) < 0$  and  $\lambda_3(p_S) = 0$ , then there exists  $\varepsilon > 0$  such that  $\text{Re } \lambda_1(p), \text{Re } \lambda_2(p), \lambda_3(p_S) < 0$  (local indeterminacy) for any  $p \in (p_S - \varepsilon, p_S)$  or for any  $p \in (p_S, p_S + \varepsilon)$ . ■

#### **Proof of Corollary 6**

*Necessity* If local indeterminacy occurs through a saddle-node bifurcation at  $p = p_S$ , that is  $\text{Re } \lambda_1(p_S), \text{Re } \lambda_2(p_S) < 0$  and  $\lambda_3(p_S) = 0$  (Proposition 5), then, in the real case,  $D(p_S) = \lambda_1(p_S) \lambda_2(p_S) \lambda_3(p_S) = 0$ ,  $S(p_S) = \lambda_1(p_S) \lambda_2(p_S) > 0$  and  $T(p_S) = \lambda_1(p_S) + \lambda_2(p_S) < 0$ , and, in the nonreal case,  $D(p_S) = \lambda_1(p_S) \lambda_2(p_S) \lambda_3(p_S) = 0$ ,  $S(p_S) = \lambda_1(p_S) \lambda_2(p_S) = [\text{Re } \lambda_1(p_S)]^2 + [\text{Im } \lambda_1(p_S)]^2 > 0$  and  $T(p_S) = \lambda_1(p_S) + \lambda_2(p_S) = 2 \text{Re } \lambda_1(p_S) < 0$ .

*Sufficiency* Conversely, if  $D(p_S) = 0$ ,  $S(p_S) > 0$  and  $T(p_S) < 0$ , then  $D(p_S) = \lambda_1(p_S) \lambda_2(p_S) \lambda_3(p_S) = 0$  implies without loss of generality  $\lambda_3(p_S) = 0$ ,  $S(p_S) = \lambda_1(p_S) \lambda_2(p_S)$  and  $T(p_S) = \lambda_1(p_S) + \lambda_2(p_S)$ . If  $\lambda_1(p_S)$  and  $\lambda_2(p_S)$  are real,  $S(p_S) > 0$  and  $T(p_S) < 0$  implies  $\lambda_1(p_S), \lambda_2(p_S) < 0$ , while, if  $\lambda_1(p_S)$  and  $\lambda_2(p_S)$  are nonreal  $T(p_S) = 2 \text{Re } \lambda_1(p_S) < 0$ , so that  $\text{Re } \lambda_1(p_S) = \text{Re } \lambda_2(p_S) < 0$ . Thus, in both the cases,  $\text{Re } \lambda_1(p_S), \text{Re } \lambda_2(p_S) < 0$  and  $\lambda_3(p_S) = 0$ , and Proposition 5 implies local indeterminacy through a saddle-node bifurcation at  $p = p_S$ .

#### Proof of Proposition 7

From Proposition 3, we have  $\text{Re } \lambda_1(p_H) = \text{Re } \lambda_2(p_H) = 0$ . Therefore,  $D(p_H) = [\text{Im } \lambda_1(p_H)]^2 \lambda_3(p_H) < 0$  and  $\lambda_3(p_H) < 0$ . Thus, there exists  $\varepsilon > 0$  such that, generically, we have  $\text{Re } \lambda_1(p), \text{Re } \lambda_2(p), \lambda_3(p) < 0$  (local indeterminacy) for any  $p \in (p_H - \varepsilon, p_H)$  or, alternatively, for any  $p \in (p_H, p_H + \varepsilon)$ .

■

#### Proof of Proposition 14

A Hopf bifurcation generically arises if and only if  $D = ST$  and  $S > 0$  (Proposition 3). Replacing (24), (25) and (26) in  $D = ST$ , we find

$$\frac{\rho + \alpha\eta\theta - \eta(1 + \theta)}{\rho + \alpha\eta\theta} = \frac{(1 - \alpha)(\rho + \delta) \frac{\varepsilon}{\sigma}}{\eta [\alpha\theta - \rho(1 + \theta) \frac{k^*l^*}{c^*}]} \quad (38)$$

while  $S > 0$  is equivalent to  $\eta/\rho > m > 0$ .

Let  $x \equiv \rho/\eta$ . Equation (38) writes:

$$\frac{x - 1 - (1 - \alpha)\theta}{x + \alpha\theta} = mx$$

that is  $mx^2 - (1 - m\alpha\theta)x + 1 + (1 - \alpha)\theta = 0$ .

Since we are interested in positive solutions  $x > 0$ , we require  $1 - m\alpha\theta > 0$ .

$$x_{\pm} = \frac{1 - m\alpha\theta \pm \sqrt{(1 - m\alpha\theta)^2 - 4m[1 + (1 - \alpha)\theta]}}{2m}$$

or, equivalently,

$$x_{\pm} = \frac{1 - m\alpha\theta \pm \sqrt{(1 + m\alpha\theta)^2 - 4m(1 + \theta)}}{2m}$$

The solutions are real if and only if  $(1 + m\alpha\theta)^2 - 4m(1 + \theta) > 0$ . In this case, we have  $0 < x_- < x_+$ . Solving for  $\eta$ , we get

$$\eta_{\pm} = \frac{\rho}{x_{\pm}} = \frac{2m\rho}{1 - m\alpha\theta \pm \sqrt{(1 + m\alpha\theta)^2 - 4m(1 + \theta)}}$$

The Hopf bifurcation value  $a_H$  is obtained solving the equation (28) with inequality  $\eta(a_H l^*(a_H) f(k^*)) > m\rho > 0$  (equivalent to  $S > 0$ ).

Since  $0 < x_- < x_+$ , we have also  $\eta_- > \eta_+ > 0$ . It is easy to check that  $\eta_+ > m\rho$ , then  $\eta_- > \eta_+ > m\rho > 0$ . ■

## References

- [1] Akazawa, M., J. Sindelar, and D. Paltiel (2003), "Economic costs of influenza-related work absenteeism," *Value in Health* 6, 107-115.
- [2] Bauer, R., D. Diaz-Sanchez, and D. Jaspers (2012), "Effects of air pollutants on innate immunity: The role of toll-like receptors and nucleotide-binding oligomerization domain-like receptors," *The Journal of Allergy and Clinical Immunology* 129, 14-24.
- [3] Bosi, S., D. Desmarchelier, and L. Ragot (2015), "Pollution effects on labor supply and growth," *International Journal of Economic Theory* 11, 371-388.
- [4] Bosi, S., and L. Ragot (2011). *Introduction to discrete-time dynamics*. CLUEB, Bologna.
- [5] Calderon-Garciduenas, L., A. Solt, C. Henriquez-Roldan, R. Torres-Jardon, B. Nuse, L. Herritt, R. Villarreal-Calderon, N. Osnaya, I. Stone, R. Garcia, D. Brooks, A. Gonzales-Maciel, R. Reynoso-Robles, R. Delgado-Chavez, and W. Reed (2008), "Long-term air pollution exposure is associated with neuroinflammation, an altered innate immune response, disruption of the blood-brain barrier, ultrafine particulate deposition, and accumulation of amyloid  $\beta$ -42 and  $\alpha$ -synuclein in children and young adults," *Toxicologic Pathology* 36, 289-310.
- [6] Caren, L. (1981), "Environmental pollutants: effects on the immune system and resistance to infectious disease," *Bioscience* 31, 582-586.
- [7] Carson, R.T., P. Koundouri and C. Nauges (2011), "Arsenic mitigation in Bangladesh: A household labor market approach," *American Journal of Agricultural Economics* 93, 407-414.
- [8] Centers for Disease Control and Prevention (2015), *Sexually Transmitted Disease Surveillance 2014*. U.S. Department of Health and Human Services, Atlanta.
- [9] Delfino, D., and P. Simmons (2000), "Positive and normative issues of economic growth with infectious disease," *The University of York Discussion Papers in Economics* 2000/48.
- [10] Fernandez, E., R. Pérez and J. Ruiz (2012), "The environmental Kuznets curve and equilibrium indeterminacy," *Journal of Economic Dynamics & Control* 36, 1700-1717.

- [11] Geoffard, P.Y., and T. Philipson (1996), "Rational epidemics and their public control," *International Economic Review* 37, 603-624.
- [12] Gersovitz, M., and J. Hammer (2004), "The economical control of infectious diseases," *The Economic Journal* 114, 1-27.
- [13] Goenka, A., and L. Liu (2012), "Infectious diseases and endogenous fluctuations," *Economic Theory* 50, 125-149.
- [14] Goenka, A., L. Liu and M. Nguyen (2014), "Infectious diseases and economic growth," *Journal of Mathematical Economics* 50, 34-53.
- [15] Graff Zivin, J., and M. Neidell (2014), "Temperature and the allocation of time: Implications for climate change," *Journal of Labor Economics* 32, 1-26.
- [16] Hanna, R., and P. Oliva (2014), "The effect of pollution on labor supply: Evidence from a natural experiment in Mexico City," *Journal of Public Economics* (forthcoming).
- [17] Heal, G (1982), "The use of common property resources," In *Explorations in Natural Resource Economics*, The Johns Hopkins University Press for Resources for the Future, Baltimore.
- [18] Hethcote, H. W (2009), "The basic epidemiology models: models, expressions for  $R_0$ , parameter estimation, and applications," In *Mathematical Understanding of Infectious Disease Dynamics*, Lecture Notes Series, Institute for Mathematical Sciences, National University of Singapore, volume 16, chapter 1.
- [19] Hethcote, H. W., and P. Van den Driessche (2000), "Two SIS epidemiologic models with delays," *Journal of Mathematical Biology* 40, 3-26.
- [20] Itaya, J.I. (2008), "Can environmental taxation stimulate growth? The role of indeterminacy in endogenous growth models with environmental externalities," *Journal of Economic Dynamics & Control* 32, 1156-1180.
- [21] Kuznetsov, Y. (1998), *Elements of Applied Bifurcation Theory*. Applied Mathematical Sciences, volume 112, Springer.