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Infectious disease and endogenous cycles: lockdown hits two birds with one stone

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Abstract

This paper develops a competitive Ramsey-Cass-Koopmans framework in which an infectious disease evolves according to a simple SIS model. It aims at examining how the lockdown affects infectious disease persistence, individual welfare, and economic dynamics. In contrast to the existing literature, two types of infectives are introduced: (1) symptomatics and (2) asymptomatics. While the former is assumed to be too ill to work, the latter supply their labour and spread the disease. The government imposes a lockdown as an instrument to control the disease spread. In the long run, when the contamination rate of the disease is relatively high and the share of asymptomatics is low enough, the lockdown is welfare improving regardless of the degree of household empathy toward infectives. Moreover, a stable limit cycle can emerge near the endemic steady-state, through a Hopf bifurcation, when the share of infectives increases sufficiently the marginal utility of consumption. Particularly, we prove that it is possible to tune the lockdown to simultaneously obtain the limit cycle disappearance and the disease eradication (Bogdanov-Takens bifurcation). In this sense, the lockdown allows *hitting two birds with one stone*.

JEL codes: C61, E13, I18, O41.

Keywords: Bogdanov-Takens bifurcation, Hopf bifurcation, Lockdown, Ramsey model, SIS model.

1 Introduction

As early as the Old Testament, isolation measures are mentioned: Moses recommended 40 days of isolation in order to purify oneself after contact with lepers (Leviticus, XIII). Similarly, in Ancient Egypt, Ramses II would have isolated lepers in the desert in order to prevent the spread of the disease (Kilwein, 1995).

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These isolation measures are found in the 6th century during the bubonic plague epidemic (also known as Justinian's plague, after the Emperor Justinian who had it, did not perish and was immune to it). It appeared in Arabia in 541 and reached Constantinople in 542, killing up to 40% of the city. Emperor Justinian took advantage of this epidemic to set up sanitary isolation procedures. Although it had no significant effect on the disease and its spread, the Constantinople Quarantine is considered as one of the first quarantine measures. Isolation of patients developed throughout the Middle Ages in order to fight leprosy, and then spread to other epidemics such as the plague.

The notion of quarantine as we know it today appeared during the Black Death epidemic in the 14th century. At this time, the aim was to isolate any person or goods contaminated. Two kinds of public health control were put in place: municipal quarantine and the isolation of victims (Hays, 1938). The 40-day isolation period that gave the name quarantine was first introduced in the port of Venice in 1377. In addition to the maritime quarantine, the Italian health authorities also introduced reactive quarantine, which consists of the isolation of an infected person at home as well as those with whom they have been in contact (Byrne, 2008). Sometimes, the measures were much more extreme: houses were walled up with the inhabitants inside regardless of their condition (Ziegler, 1982). In 1423, to facilitate the application of quarantine, the Venice Senate created the first place of sanitary isolation on the island of Lazaretto Vecchio. This lazaret system was then used throughout Europe (1467, Genoa; 1476 and 1526, Marseille; 1569, Nantes; 1596, Le Havre; 1622, Toulon; 1831, Bordeaux). For almost three centuries, the quarantine system was applied in Europe and, despite sometimes heavy human losses, proved to be relatively effective (Drews 2013) against the plague (London in 1665) and subsequently against many epidemics (syphilis in northern Europe in 1492; yellow fever in Philadelphia in 1793; cholera in New York in 1832).

If the current Covid epidemic has nothing in common in terms of mortality with its ancestor, the plague, it is nevertheless privileged to have succeeded in confining half of humanity and causing an unprecedented economic and social crisis (Sardon, 2020). Its economic recession for 2020 has been estimated to 6.2% for European Union (UE-27)[1]. Concerning mortality, Meyerowitz-Katz and Merone (2020) report that the infection fatality rate for the Covid-19 is about 0.68%. It is important to note that the "infection fatality rate" differs from the usual "case fatality rate" since the former includes asymptomatic while the latter does not (Meyerowitz-Katz and Merone, 2020).

Considering simultaneously the very high economic impact of lockdown and the low reported infection fatality rate, it appears that another important element has to be taken into account to understand the social acceptance of lockdown regarding the Covid-19 pandemic. In a recent study, Grignoli and al. (2021) point out the role of empathic dispositions to accept restrictions of personal freedom during the Covid-19 pandemic. From this point on, one of the main objectives of our paper is to study conditions for social acceptance of lock-

[1] EUROSTAT - https://ec.europa.eu/eurostat/databrowser/view/tec00115/default/table?lang=fr

down in the long run in a market economy in which empathy toward infectives is considered. Then, we follow the important stream of literature developed during the Covid-19 pandemic by embedding an epidemiological model into an economic growth model. More precisely, we develop a competitive Ramsey-Cass-Koopmans framework in which an infectious disease evolves according to a simple SIS model, and examine how the lockdown affects infectious disease persistence, individual welfare, and economic dynamics.

The first attempt to introduce a disease spread model into an economic growth model dates back to Goenka and Liu (2012) to the best of our knowledge. The epidemiological block considered in their paper is known in epidemiology as the SIS model, which consists of dividing the population into two groups: Susceptibles and Infectives. At each period, a susceptible agent can contract the disease when an encounter with an infective occurs, while an infective can recover from the disease and then get back to the susceptible group. That is, the SIS model only describes an infectious disease that does not confer immunity after recovery. Following Hethcote (1976), the SIS model may model the spread of bacterial diseases like meningitis or protozoan diseases as malaria or sleeping sickness. However, the macroeconomic literature on the Covid-19 pandemic has been widely focused on another epidemiological block: the SIR model.[2] The main difference between the SIS and the SIR model is that, in the SIR model, when an infective agent recovers from the disease, she obtains a permanent immunity and can never contract the disease again. In other words, in the long run, the infectious disease always disappears in the SIR model while the disease can persist in the SIS model.

In a recent contribution, Alvarez et al. (2020) have precisely considered a dynamic general equilibrium model in which an infectious disease evolves according to a SIR model. They focus on the central planner solution: the Government chooses the time-path of lockdown to minimize the value of fatalities and the output cost generated by the disease and the lockdown itself. Because the epidemiological block renders the optimization program non-convex, Alvarez et al. (2020) provide only a numerical solution using data from the Covid-19 pandemic. They point out, in particular, that it is optimal to introduce a severe lockdown at the very beginning of the pandemic and then loosen the lockdown gradually. Eventually, the disease disappears in the long run due to the SIR hypothesis.

Despite this appealing conclusion, the successive waves of Covid-19 contamination worldwide warn us about findings on the immunity gained from recovering and then on the use of the SIR hypothesis. In another recent paper, Bosi et al. (2021) reconsider the optimal lockdown level in a dynamic general equilibrium model with three main differences regarding Alvarez et al. (2020): (1) focusing on the SIS hypothesis rather than the SIR one, (2) overcoming the convexity issue by proposing not to study lockdown as a dynamic variable but as a static one and (3) introducing an empathic representative household since her

[2] See for example Acemoglu et al. (2020), Alvarez et al. (2020), Atkeson (2020) or Eichenbaum and Rebelo (2020).

preferences are negatively affected by the share of infectives. Interestingly, Bosi et al. (2021) point out that it is never optimal to introduce a lockdown to control the disease without empathy. Moreover, they observe that when it is optimal to introduce a lockdown (i.e., when the representative household is sufficiently empathic/altruistic), it is not always optimal to eradicate the disease.

It should be noted that both Alvarez et al. (2020) and Bosi et al. (2021) are mainly concerned by the Covid-19 pandemic. Still, they ignore an essential feature in their models, namely the existence of asymptomatic agents, who are infective individuals without symptoms. To figure out how important asymptomatic agents are for the transmission of the Covid-19, it is interesting to refer to Russell et al. (2020). Indeed, they report that by 20 February 2020, among 619 passengers of the ship called "Diamond Princess" positive to the Covid-19, there are 318 asymptomatics, representing around 51% of infective individuals.

The present paper proposes to complete the existing literature by considering explicitly asymptomatic infectives. More precisely, we consider a dynamic general equilibrium model where a disease evolves according to the SIS hypothesis without vital dynamics (i.e., the disease is not lethal) and where preferences are negatively affected by the share of infectives (empathic households). The Government is supposed to intervene by imposing a lockdown to reduce the disease spread. Our main goal is to examine how the lockdown affects infectious disease evolution and economic dynamics. Moreover, while the representative household considers the lockdown level as given, we address the question of her social acceptance by discussing conditions under which a more stringent lockdown is welfare improving at the steady-state.

As in Alvarez et al. (2020) and Bosi et al. (2021), we assume that the Government imposes the lockdown to control the disease evolution. However, the two main differences between our paper and these studies should be underlined. First, we consider that infectives are divided into two groups: symptomatics and asymptomatics. While symptomatics are assumed to be too ill to work, asymptomatics have not the consciousness of their illness. The latter supply their labour, and spread the disease. To take into account the fact that contamination occurs at work, we introduce two assumptions: (1) a susceptible can contract the disease only through an encounter with an asymptomatic infective and (2) contamination rate is a positive function of production intensity. Second, while Bosi et al. (2021) and Alvarez et al. (2020) consider the planner solution, we instead focus on the market solution. More precisely, the disease is viewed as a pure externality by the representative household, and the lockdown is not a control variable. This modeling can overcome the convexity issue pointed out by Bosi et al. (2021) or Alvarez et al. (2020). Our tractable model and numerical simulation allow us to analyze the economic effects of lockdown explicitly. Our results can be described as follows:

First, it is shown that a more stringent lockdown may be welfare improving at the steady-state in two configurations: (1) when the share of asymptomatics is sufficiently low and the contamination rate is relativyly high, (2) when the representative household is sufficiently empathic. While this last configuration was expected because it is closely related to the result pointed out by Bosi et al.

(2021), the former configuration is more surprising. Indeed, since the disease is not lethal, the only economic cost results in labour loss due to symptomatic infectives or lockdown. The lockdown has then two distinct effects on labour supply (and then on consumption): (1) a positive health effect (the lockdown prevents the disease spread and then promotes labour supply by reducing infectives) and (2) a negative direct effect by avoiding household to supply their labour. If the share of asymptomatics is very low, the economic cost due to the disease is very high because an important share of infectives is too ill to supply their labour. In this case, the positive health effect dominates the negative direct effect, which implies that a more stringent lockdown increases the labour supply (and then consumption), meaning a welfare improvement regardless of the degree of the household's empathy.

Our second result is related to the dynamics around the endemic steady-state. It is shown that a stable limit cycle can emerge through a Hopf bifurcation when the share of infective increases the marginal utility of consumption. In this case, a higher share of infectives at a given period is followed by a drop in the next period. Finally, the close analysis of the dynamics shows that by adjusting the degree of lockdown appropriately depending on the recovery rate of the disease, its contamination rate, and the share of asymptomatics among ill agents, the limit cycle can collapse through a Bogdanov-Takens bifurcation. In this case, the limit cycle disappears, but the disease is also eradicated: by stabilizing the economy and eradicating the disease at the same time, a convenient degree of lockdown allows to *hit two birds with one stone*.

The rest of the paper is organized as follows. Section 2 presents the general model. The steady-state is studied in section 3, while section 4 explores the local dynamics. Section 5 concludes, and technical proofs are presented in Appendix.

2 Model

2.1 Disease

We consider an economy where there is an infectious disease evolving according to a SIS model. More precisely, the population N is split into two groups: susceptibles (S) and infectives (I). Among infectives, a share $a \in (0,1)$ is assumed to be asymptomatic while $(1-a)$ represents the share of ill individuals unable to work because of severe symptoms. Susceptibles and asymptomatic infectives supply their labour inelastically. We consider that the disease is contracted at work. To prevent the disease spread, the Government introduces a lockdown: a share $\lambda \in (0,1)$ of the labour force is locked down. That is, the effective labour supply $\hat{L}$ is composed of susceptibles (S) and asymptomatic infectives (aI), who are not locked down:

$$\hat{L} = (1-\lambda)(S + aI) \tag{1}$$

Introducing asymptomatics implies that some ill households can work, which contrasts with the existing literature mixing epidemiology and economics where

ill households are always assumed to be too sick to work[3].

To simplify the exposition, the population is supposed to remain constant over time ($\dot{N} = 0$) with no birth and no death. Since only agents who are not locked down can contract the disease, the susceptible group (S) and infective group (I) evolve according to:

$$\dot{S} = -\beta(1-\lambda)\frac{aI}{S+aI}S + \gamma I \tag{2}$$

$$\dot{I} = \beta\frac{aI}{S+aI}(1-\lambda)S - \gamma I \tag{3}$$

where $\beta > 0$ is the contamination rate while $\gamma > 0$ represents the recovery rate. Let us denote by $s = S/N$, and $i = I/N$ respectively the share of susceptibles and infectives in the population. It follows that $i = 1 - s$ while $\dot{s} = \dot{S}/N$. Focusing on (2) and dividing its both side by N, we obtain:

$$\dot{s} = \xi(s) \equiv (1-s)\left[\gamma - \frac{\beta(1-\lambda)a}{(1-a)s+a}s\right] \tag{4}$$

From (4) we recover the main feature of the SIS model (Hethcote, 1976), that is, the possible coexistence of two steady-states, namely, a disease-free one,

$$s = 1 \equiv s_1$$

and an endemic one,

$$s = \frac{a\gamma}{\beta(1-\lambda)a - \gamma(1-a)} \equiv s^*$$

Considering s^*, there is no guarantee that $s^* \in (0,1)$. If it is not the case, the only steady-state is the disease-free one. Conversely, if $s^* \in (0,1)$, the disease-free steady-state coexists with the endemic one. Following Hethcote (2000), in epidemiology, the so-called R_0 index captures the capacity of an infectious disease to invade a population and then, to persist in the long run. This is the case when $R_0 > 1$. In our epidemiological context:

$$R_0 \equiv \frac{a\beta(1-\lambda)}{\gamma} \tag{5}$$

The following proposition sums up the main characteristics of the SIS model considered here.

Proposition 1 *(SIS dynamics)*

1) If $R_0 < 1$, the dynamics of the disease characterized by (4) possess a unique steady-state (disease-free, i.e. s_1), which is locally stable.

[3] The interested reader can refer to Caulkins et al. (2021), Bosi et al. (2021), La Torre et al. (2021), among others.

2) If $R_0 > 1$, the dynamics of the disease characterized by (4) possess two steady-states: (1) a disease-free one (i.e. s_1), which is locally unstable and (2) an endemic one (i.e. s^*), which is locally stable.

Proof. See the Appendix. ■

Obviously, when introducing both lockdown and asymptomatic infectives, we recover the usual conclusion of the SIS model (Hethcote, 2000): the infectious disease invades a population and persists in the long run if and only if $R_0 > 1$.

Furthermore, as discussed before, contamination is assumed to occur at work. That is, we consider that the contamination rate β is directly affected by the production intensity $y = Y/\hat{L}$ such that $\beta'(y) > 0$.[4] The following assumption sums up its properties.

Assumption 1 $\beta'(y) > 0$ *and* $\beta(0) = 0$.

Assumption 1 is compatible with the fact that contamination occurs at work: the higher the production, the higher is the contamination. When the economy collapses, i.e., $y = 0$, there are no contacts, then no contamination. This situation naturally implies that $\beta(0) = 0$. For further reference, let us introduce π, the first order elasticity of β with respect to y:

$$\pi \equiv \frac{y\beta'(y)}{\beta(y)} > 0.$$

2.2 Households

Since both symptomatics and locked down households are not able to work, we assume a perfect social security system (as in Bosi and Desmarchelier, 2018), which implies that the total labour income earned by the effective labour force is distributed equally to every household so that:

$$w\hat{L} = \omega N$$

where w is the wage rate and ω is the representative household's income whatever her situation: susceptible or infected.

Considering that h represents the representative household's wealth and $(r-\delta)h$ depicts the net interest of h, with $r \geq 0$ and $\delta \in (0,1)$ representing respectively the real interest rate and the capital depreciation rate. The household's budget constraint is then given by:

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

where $c \geq 0$ represents the consumption. From the expression of effective labour force (1), it follows that the household's income is given by:

$$\omega = w(1-\lambda)[(1-a)s + a]$$

[4] The reader can remark that a more stringent lockdown lowers the effective labour supply (also the production level) and hence the contamination rate since $\beta'(y) > 0$.

The household's budget constraint then becomes:

$$\dot{h} = (r - \delta) h + w (1 - \lambda) [(1 - a) s + a] - c \tag{6}$$

As in Bosi et al. (2021), it is assumed that the household's utility u is positively affected by consumption and negatively by the share of infective i. The marginal disutility of the share of infectives ($u_i < 0$) depicts how empathic is the representative household. The following assumption describes properties of $u(c, i)$.

Assumption 2 $u_c > 0$, $u_i < 0$, $u_{cc} < 0$, $u_{ci} \lessgtr 0$ *and usual limit conditions* $\lim_{c \to 0} u_c = +\infty$ *and* $\lim_{c \to +\infty} u_c = 0$.

At this step of the reasoning, the sign of the cross-derivative u_{ci} remains free. Indeed, both signs can be justified theoretically. On the one hand, a higher share of infectives can depress the representative household and lowers the marginal utility of consumption (i.e. $u_{ci} < 0$). On the other hand, since a higher share of infectives reduces utility, the representative household can compensate this utility loss by a higher consumption level (implying $u_{ci} > 0$).[5] Before going further, let us introduce the first and second-order elasticities:

$$\varepsilon_c \equiv \frac{c u_c}{u} > 0 \text{ and } \varepsilon_i \equiv \frac{i u_i}{u} < 0$$

and:

$$\varepsilon_{cc} \equiv \frac{c u_{cc}}{u_c} < 0 \text{ and } \varepsilon_{ci} \equiv \frac{i u_{ci}}{u_c} \lessgtr 0$$

Notice that $-1/\varepsilon_{cc}$ is the intertemporal elasticity of substitution in consumption.

The representative household chooses the consumption path which maximizes the discounted intertemporal utility,

$$\int_0^{+\infty} e^{-\theta t} u(c, i)\, dt \tag{7}$$

with respect to the budget constraint (6), taking as given the initial wealth level h_0. Parameter $\theta > 0$ represents the discount rate and captures the household's impatience. Note also that differently from other previous studies such as Alvarez et al. (2020) and Bosi et al. (2021), we investigate the competitive market equilibrium. The infectious disease is then treated as a pure externality and the lockdown level is not a control variable.

Proposition 2 *(Household's optimization program) The first-order conditions of the representative household's program are given by a static relation linking the Lagrangian multiplier μ, consumption and infective share*

$$\mu = u_c(c, i), \tag{8}$$

a dynamic Euler equation

$$\dot{\mu} = (\delta + \theta - r) \mu, \tag{9}$$

[5] Such an ambiguity of externalitites on household's preferences are usual in environmental economics (Michel and Rotillon, 1995).

and the budget constraint (6) jointly with the transversality condition $\lim_{t\to+\infty} e^{-\theta t}\mu h = 0$.

Proof. See the Appendix. ■

Applying the implicit function theorem on the static relation (8) reveals that consumption is a function of μ and i. That is, $c = c(i, \mu)$ such that:

$$\frac{\mu}{c}\frac{\partial c}{\partial \mu} = \frac{1}{\varepsilon_{cc}} < 0$$

and:

$$\frac{i}{c}\frac{\partial c}{\partial i} = -\frac{\varepsilon_{ci}}{\varepsilon_{cc}} \lesseqgtr 0 \tag{10}$$

We remark that if health (captured by s or i) and consumption are complements ($u_{ci} < 0$[6]), then a higher share of infectives implies a drop in consumption (indeed, $\varepsilon_{ci} < 0$). Conversely, if health and consumption are substitutes ($u_{ci} > 0$[7]), then a higher share of infectives implies an increase in consumption (indeed, $\varepsilon_{ci} > 0$).

2.3 Firms

The production sector consists of a representative firm who behaves competitively. Both capital K and labour L are used to produce a quantity Y of a composite good that can be consumed or saved. The technology is depicted by an aggregate production function $Y = F(K, L)$. The following assumption sums up properties of the production function F.

Assumption 3 *$F : \mathbb{R}^2_+ \to \mathbb{R}$ is C^1, homogenous of degree one, strictly increasing and concave. Standard Inada conditions hold.*

The representative firm chooses the amount of capital (K) and labour (L) which maximize its profit $Y - rK - wL$ taking as given prices (r and w). Assumption 3 ensures that this program is well-defined. As usual, the first order conditions give:

$$r \equiv r(k) = f'(k) \tag{11}$$

$$w \equiv w(k) = f(k) - kf'(k) \tag{12}$$

where $k \equiv K/L$ and $y = f(k) \equiv F(k, 1) = Y/L$.

For further reference, we introduce two well-known elasticities: the share of capital income in the total income (α) and the capital-labour elasticity of substitution (σ). More precisely:

$$\alpha \equiv \frac{kf'(k)}{f(k)} \text{ and } \sigma \equiv -\frac{f'(k)\left[f(k) - kf'(k)\right]}{kf(k)f''(k)}$$

[6] Recall that $s = 1 - i$, that is, $u_{ci} < 0$ is equivalent to $u_{cs} > 0$.

[7] Recall that $s = 1 - i$, that is, $u_{ci} > 0$ is equivalent to $u_{cs} < 0$.

As usual, elasticities of factor prices with respect to capital intensity are fully expressed in terms of α and σ:

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

2.4 Equilibrium

At the equilibrium, labour demand is just equal to labour supply ($L = \hat{L}$):

$$L = (1 - \lambda)(S + aI) \tag{13}$$

Let $l \equiv L/N$, it follows:

$$l = (1 - \lambda)[(1 - a)s + a] \tag{14}$$

and then:

$$\dot{l} = (1 - \lambda)(1 - a)\dot{s} \tag{15}$$

Considering (14), (4) and Assumption 1, we can rewritte equation (15) as follows:

$$\dot{l} = (1 - \lambda - l)\left(\gamma - \beta(f(k))\frac{a(1-\lambda)}{1-a}\frac{l - a(1-\lambda)}{l}\right) \tag{16}$$

Moreover, at the equilibrium, aggregate demand for capital is equal to aggregate supply, namely, $K = Nh$ and then, $h = kl$. That is, $\dot{h} = \dot{l}k + \dot{k}l$. Considering jointly (6), (8), (11), (12) and (14), it follows that:

$$\dot{k} = [r(k) - \delta]k + w(k) - \frac{c(\mu, i)}{l} - \frac{\dot{l}}{l}k \tag{17}$$

Definition 1 *(Dynamical system) Equilibrium dynamics are represented by the following system:*

$$\dot{\mu} \equiv f_1(\mu, k, l) = \mu(\theta + \delta - r(k)) \tag{18}$$

$$\dot{k} \equiv f_2(\mu, k, l) = (r(k) - \delta)k + w(k) - \frac{c(i, \mu)}{l} - \frac{\dot{l}}{l}k \tag{19}$$

$$\dot{l} \equiv f_3(\mu, k, l) = (1 - \lambda - l)\left(\gamma - \beta(f(k))\frac{a(1-\lambda)}{1-a}\frac{l - a(1-\lambda)}{l}\right) \tag{20}$$

Note that to obtain this system, we simply consider (9) jointly with (11) as well as (16) and (17). Finally, considering (14) and the rate of infectives $i = 1 - s$, we obtain:

$$i = \frac{1 - \lambda - l}{(1 - \lambda)(1 - a)} \equiv i(l) \tag{21}$$

3 Steady-State

3.1 Existence and multiplicity

At the steady-state, $\dot{\mu} = \dot{k} = \dot{l} = 0$, Assumption 3 ensures the invertibility of $r(k)$. That is, considering (18), it follows that there always exists a unique capital level k^* at the steady-state such that:

$$k^* = r^{-1}(\theta + \delta) > 0 \tag{22}$$

Since $k = k^*$, equation (20) gives us the following relationship at the steady-state:

$$(1 - \lambda - l)\left(\gamma - \beta(f(k^*))\frac{a(1-\lambda)}{1-a}\frac{l - a(1-\lambda)}{l}\right) = 0 \tag{23}$$

From this last equation, we recover one of the main feature of the SIS model: the possible coexistence of two steady-states. Indeed, equation (23) is verified if:

$$\begin{aligned} 1 - \lambda - l &= 0 \Longleftrightarrow l = l_1 \\ \text{and/or } \gamma - \beta(f(k^*))\frac{a(1-\lambda)}{1-a}\frac{l - a(1-\lambda)}{l} &= 0 \Longleftrightarrow l = l^* \end{aligned}$$

with:

$$l_1 = 1 - \lambda \in (0, 1) \tag{24}$$

$$l^* = \frac{a(1-\lambda)R_0}{R_0 - 1 + a} \tag{25}$$

where R_0 is given by (5) depending on β with $\beta = \beta(f(k^*))$. l_1 represents the level of l at the so-called disease-free steady-state. If $0 < l^* < l_1$, then, l^* is also a steady-state (the so-called endemic steady-state). However, if $l^* < 0$ or if $l^* > l_1$, then the disease-free steady-state is the unique one.

From equation (19), we can obtain two different levels of consumption at the steady-state corresponding to l_1 and l^*:

$$c_1 = ([r(k^*) - \delta]k^* + w(k^*))l_1 > 0 \tag{26}$$

and

$$c^* = ([r(k^*) - \delta]k^* + w(k^*))l^* > 0 \tag{27}$$

Finally, considering (8), we obtain the two different levels of μ at the steady-state, corresponding to the two different levels of consumption and labour described before:

$$\begin{aligned} \mu_{_1} &= u_c(c_1, i_1) \\ \mu^* &= u_c(c^*, i^*) \end{aligned}$$

where, following (21):

$$i_1 = \frac{1-\lambda-l_1}{(1-\lambda)(1-a)} \text{ and } i^* = \frac{1-\lambda-l^*}{(1-\lambda)(1-a)}$$

From the previous discussion, it follows that two steady-states can coexist: (1) a disease-free one, namely $\left(k^*, l_1, \mu_{_1}\right)$ and (2) an endemic one, namely (k^*, l^*, μ^*). The latter exists if and only if $0 < l^* < l_1$. The following proposition discusses conditions under which this economy possesses a unique or multiple steady-states.

Proposition 3 *(Steady-states) Let Assumptions 1, 2 and 3 hold:*

1) If $R_0 < 1$, then, the disease-free steady-state $\left(k^*, l_1, \mu_{_1}\right)$ is the unique steady-state.

2) If $R_0 > 1$, then the disease-free steady-state $\left(k^*, l_1, \mu_{_1}\right)$ coexists with an endemic steady-state (k^*, l^*, μ^*).

3) If $R_0 = 1$, $l_1 = l^*$, then the two steady-states collide.

Proof. Simply consider (25) and remark that $0 < l^* < l_1$ if and only if $R_0 > 1$. ■

Propositions 1 and 3 give the same conclusion concerning the steady-state: $R_0 > 1$ implies the existence of an endemic steady-state, i.e., a steady-state where the infectious disease persists. The Ramsey-Cass-Koopmans structure does not modify the epidemiological conclusion.

The next section aims to discuss how the lockdown λ affects the macroeconomic variables as well as the household's welfare in the long run. Since R_0 is a function of λ, it appears convenient to interpret Proposition 3 in terms the share of asymptomatics a. Let us introduce:

$$\hat{a} \equiv \frac{\gamma}{(1-\lambda)\beta}$$

Corollary 1 *If $a > \hat{a}$ $(a < \hat{a})$, then $l^* < l_1$ $(l^* > l_1)$ while if $a = \hat{a}$, then $l^* = l_1$.*

Proof. We simply consider Proposition 3 and remark that $R_0 > 1$ $(R_0 < 1)$ if and only if $a > \hat{a}$ $(a < \hat{a})$ while $R_0 = 1$ if and only if $a = \hat{a}$. ■

Assumption 4 $\gamma < (1-\lambda)\beta$.

Assumption 4 ensures that $\hat{a} < 1$. Given Assumption 4, the existence of an endemic steady-state implies that $a > \hat{a}$. In other words, when the share of asymptomatics a is higher than the threshold $\hat{a}$, the disease persists in the long run. This condition on a is not surprising since the contamination occurs exclusively because of asymptomatic workers (see (2)), and hence, the share of asymptomatics should be high enough to allow the infectious disease to persist in the long run.

3.2 Comparative statics and welfare

In this section, we analyze how the lockdown impacts the macroeconomic variables at the steady-state. We also examine the conditions under which the lockdown is welfare improving.

Proposition 4 *(Comparative statics)*

1) At both disease-free and endemic steady-states:

$$\frac{\lambda}{k^*}\frac{\partial k^*}{\partial \lambda} = 0 \tag{28}$$

2) At the disease-free steady-state:

$$\frac{\lambda}{l_1}\frac{\partial l_1}{\partial \lambda} = \frac{\lambda}{c_1}\frac{\partial c_1}{\partial \lambda} = -\frac{\lambda}{1-\lambda} < 0 \tag{29}$$

$$\frac{\lambda}{i_1}\frac{\partial i_1}{\partial \lambda} = 0 \tag{30}$$

3) At the endemic steady-state, if Assumption 4 holds:

$$\frac{\lambda}{l^*}\frac{\partial l^*}{\partial \lambda} = \frac{\lambda}{c^*}\frac{\partial c^*}{\partial \lambda} = \frac{\lambda}{1-\lambda}\frac{2\gamma(1-a)-\beta a(1-\lambda)}{(1-\lambda)\beta a-\gamma(1-a)} \lessgtr 0 \tag{31}$$

$$\frac{\lambda}{i^*}\frac{\partial i^*}{\partial \lambda} = \frac{a\beta}{(1-\lambda)\beta a-\gamma(1-a)}\frac{a\gamma\lambda}{\gamma-a\beta(1-\lambda)} < 0 \tag{32}$$

Proof. See the Appendix. ■

Results given in this Proposition are intuitive. Since there is no disease at the disease-free steady-state, a lockdown always affects the economy negatively. It reduces both the labour supply and the consumption level but does not affect the share of infectives. At the endemic steady-state, a more stringent lockdown reduces contacts between agents, which reduces the disease spreading and then the share of infected agents in the long run. Concerning the labour supply at the endemic steady-state, the next proposition clarifies its effect.

Let:

$$\tilde{a} \equiv \frac{2\gamma}{\beta(1-\lambda)+2\gamma}$$

We note that $\tilde{a} > \hat{a}$ implies $\hat{a} < \frac{1}{2}$,that is $\gamma < \frac{(1-\lambda)\beta}{2}$.

Proposition 5 *(Comparative statics continued) Let the economy be at the endemic steady-state (Assumption 4 holds jointly with $a > \hat{a}$).*

1) If the threshold $\hat{a}$ is relatively low $\left(\hat{a} < \frac{1}{2}\right)$ so that $\gamma < \frac{(1-\lambda)\beta}{2}$, then:

(a) $\tilde{a} > a > \hat{a}$ *implies that:*

$$\frac{\lambda}{l^*}\frac{\partial l^*}{\partial \lambda} = \frac{\lambda}{c^*}\frac{\partial c^*}{\partial \lambda} > 0$$

(b) $a > \tilde{a} > \hat{a}$ *implies that:*

$$\frac{\lambda}{l^*}\frac{\partial l^*}{\partial \lambda} = \frac{\lambda}{c^*}\frac{\partial c^*}{\partial \lambda} < 0$$

2) If the threshold $\hat{a}$ *is relatively high* $\left(\frac{1}{2} < \hat{a} < 1\right)$ *so that* $\frac{(1-\lambda)\beta}{2} < \gamma < (1-\lambda)\beta$ *, then:*

$$\frac{\lambda}{l^*}\frac{\partial l^*}{\partial \lambda} = \frac{\lambda}{c^*}\frac{\partial c^*}{\partial \lambda} < 0$$

Proof. See the Appendix. ■

Proposition 5 can be explained as follows. A more stringent lockdown (i.e., a higher value of λ) has two opposite effects on labour supply (and then on consumption) : (1) a direct negative effect since locked down households are not allowed to work and (2) an indirect positive health effect, which increases the share of healthy households and hence, increases the labour supply. It should be noticed that since the infectious disease is not lethal, its only economic cost results in its effects on labour supply (when ill households are symptomatic and too sick to work). When the share of asymptomatics a is relatively high, the negative effect outweighs the positive one. In the extreme situation where all ill households are asymptomatic (i.e. $a = 1$), the infectious disease does not affect labour supply (no economic cost) since every household can work as if there was no disease. In such a situation, a higher rate of lockdown will reduce the labour supply. In other words, in this case, the positive health effect disappears and the negative direct effect fully explain the negative impact of a more stringent lockdown on labour supply. On the opposite, if the share of asymptomatics a is very low, the economic cost of the disease is magnified and hence, the positive health effect dominates the negative effect, and explains the positive effect of lockdown on the labour supply.

In a sense, the share of asymptomatics captures the severity of the infectious disease and hence, the magnitude of the negative externality: if a is very high (low), the economic impact of the negative externality generated by the disease is very low (high) and then, a more stringent lockdown decreases (increases) the labour supply and the household consumption.

Let W be the intertemporal welfare evaluated at the endemic steady-state:

$$W \equiv \int_0^{+\infty} e^{-\theta t} u\left(c^*, i^*\right) dt = \frac{u\left(c^*, i^*\right)}{\theta}$$

We obtain:

$$\frac{\lambda W'(\lambda)}{W} = \varepsilon_c \frac{\lambda}{c^*}\frac{\partial c^*}{\partial \lambda} + \varepsilon_i \frac{\lambda}{i^*}\frac{\partial i^*}{\partial \lambda} \tag{33}$$

The following proposition discusses the conditions under which a more stringent lockdown is welfare improving.

Proposition 6 *(Welfare and lockdown) The lockdown is welfare improving if and only if:*

$$\frac{\frac{\lambda}{c^*}\frac{\partial c^*}{\partial \lambda}}{\frac{\lambda}{i^*}\frac{\partial i^*}{\partial \lambda}} < -\frac{\varepsilon_i}{\varepsilon_c} \tag{34}$$

Proof. Simply consider (33). ■

Focusing on Proposition (5), condition (34) is always verified in the case (1a). In other words, when the contamination rate β is relatively high and the share of asymptomatics a is low enough, the lockdown is always welfare improving . A higher lockdown has a double benefits: it increases both consumption and health. In such a situation, it is optimal to set the lockdown such that the disease is eradicated (i.e. $\lambda = 1 - l_1 = 1 - [\gamma/(a\beta)]$ giving $i = 0$). This result clearly contrasts with the existing literature. Indeed, without considering the presence of asymptomatics among ill households, Bosi et al. (2021) obtain a similar result if and only if the representative household is sufficiently empathic/altruistic toward the infectives while, the case (1a) in Proposition (5) is obtained regardless of the degree of household's empathy.

Furthermore, if the representative household is very concerned by the health situation in the economy ($\varepsilon_i \to -\infty$), condition (34) is also always verified and then, the lockdown has to be set in order to eradicate the infection disease (i.e. $\lambda = 1 - l_1 = 1 - [\gamma/(a\beta)]$). Considering relation (34), the Government has to increase the lockdown since the representative household overvalues health situation in the economy with respect to consumption. This result means that it appears crucial to have a clear picture of households' preferences when a Government introduces a lockdown. If agents are not empathic enough, the Government can face a social mistrust by introducing a lockdown or to impose a more stringent lockdown.

4 Local dynamics

Within this section, local dynamics are explored in two ways. First, we focus on an analytical approach to discuss general conditions for which complex dynamics, like limit cycles, can emerge and give economic insights. Second, we propose a numerical exploration to complete our analytical results. For brevity convenience, we only focus on the endemic steady-state (i.e. Assumption 4 holds, $a > \hat{a}$ and then $l = l^* < l_1$).

4.1 Analytical approach: bifurcation and endogenous cycles

To capture the dynamics around the endemic steady-state, we apply the methodology developed by Bosi and Desmarchelier (2019). The general idea is to compute the Jacobian matrix J of the system (18)-(19)-(20), evaluated at the endemic steady-state, and to exploit the fact that T, S and D, respectively the trace, the sum of principle minors of order two and the determinant of J are

functions of the three eigenvalues of J, namely λ_1, λ_2 and λ_3. More precisely, $T = \lambda_1 + \lambda_2 + \lambda_3$, $S = \lambda_1\lambda_2 + \lambda_2\lambda_3 + \lambda_1\lambda_3$ and $D = \lambda_1\lambda_2\lambda_3$.

The Jacobian matrix J, evaluated at the endemic steady-state, is given by:

$$\begin{aligned} J &\equiv \begin{bmatrix} \frac{\partial f_1}{\partial \mu} & \frac{\partial f_1}{\partial k} & \frac{\partial f_1}{\partial l} \\ \frac{\partial f_2}{\partial \mu} & \frac{\partial f_2}{\partial k} & \frac{\partial f_2}{\partial l} \\ \frac{\partial f_3}{\partial \mu} & \frac{\partial f_3}{\partial k} & \frac{\partial f_3}{\partial l} \end{bmatrix} \\ &= \begin{bmatrix} 0 & \frac{\mu}{k}\Delta & 0 \\ -\frac{k}{\mu}\Phi\frac{1}{\varepsilon_{cc}} & \theta + \alpha\pi\gamma\Psi & \frac{k}{l}\left(\Phi\left(1 - \frac{\varepsilon_{ci}}{\varepsilon_{cc}}\frac{1}{\Psi}\right) + \beta\Psi\Omega\right) \\ 0 & -\alpha\pi\gamma\frac{l}{k}\Psi & -\beta\Psi\Omega \end{bmatrix} \end{aligned}$$

with:

$$\begin{aligned} \Delta &\equiv \frac{(\theta+\delta)(1-\alpha)}{\sigma} > 0 \\ \Phi &\equiv \frac{\theta + (1-\alpha)\delta}{\alpha} > 0 \\ \Psi &\equiv \frac{(l_1 - l)}{l} \geq 0 \\ \Omega &\equiv \frac{a^2 l_1^2}{(1-a)\, l} > 0 \end{aligned} \tag{35}$$

It follows that:

$$T = \theta + \alpha\pi\gamma\Psi - \beta\Psi\Omega \lesseqgtr 0 \tag{36}$$

$$S = -\beta\Psi\Omega(\theta + \alpha\pi\gamma\Psi) - \alpha\pi\gamma\Psi\left(\Phi\left(\frac{\varepsilon_{ci}}{\varepsilon_{cc}}\frac{1}{\Psi} - 1\right) - \beta\Psi\Omega\right) + \Phi\frac{1}{\varepsilon_{cc}}\Delta \tag{37}$$

$$D = -\beta\frac{\Phi}{\varepsilon_{cc}}\Psi\Omega\Delta \geq 0 \tag{38}$$

Lemma 1 *If $a > \hat{a}$, $D > 0$ while $D = 0$ if and only if $a = \hat{a}$.*

Proof. Simply consider Corollary 1 and focus on (35) and (38). ■

Lemma 2 *Since $a > \hat{a}$, there is no room for local indeterminacy.*

Proof. Following Lemma 1. If $D > 0$, it follows that there is always, at least, one unstable eigenvalue. ■

The impossibility of local indeterminacy implies that the endemic steady-state is locally saddle-path stable or locally unstable.

Proposition 7 *(Transcritical bifurcation) A transcritical bifurcation occurs if and only if $a = \hat{a}$.*

Proof. Following Bosi and Desmarchelier (2019, Proposition 2), a transcritical bifurcation occurs if and only if $D = 0$. If $a = \hat{a}$, $l^* = l_1$ implying $\Psi = D = 0$ (see Lemma 1). ■

Proposition 7 describes what happen when $D = 0$. Such a configuration can give rise to three main bifurcation scenarios depending upon the number of steady-state[8]. When two steady-states collide and disappear, a saddle-node bifurcation occurs. When two steady-states collide and exchange their stability properties, a transcritical bifurcation appears. When there are three steady-states exchanging their stability properties, a pitchfork bifurcation occurs. Considering Corollary 1, it is clear that a transcritical bifurcation occurs in this economy when $a = \hat{a}$ (i.e. $D = 0$).

We are now interested in the possible existence of endogenous cycles. Before going further, let:

$$\varepsilon_{ci}^{H} = \varepsilon_{cc}\left(\Psi + \frac{\beta\Omega}{\Phi}\left[\Psi^2 + \frac{1}{\alpha\pi\gamma}\left(\frac{\Psi\frac{\Phi}{\varepsilon_{cc}}\Delta}{\theta + \alpha\pi\gamma\Psi - \beta\Psi\Omega} + \frac{\Phi\Delta}{\varepsilon_{cc}\beta\Omega} - \Psi\left(\theta + \alpha\pi\gamma\Psi\right)\right)\right]\right)$$

and:

$$\pi^{H} \equiv \frac{\beta\Psi\Omega - \theta}{\alpha\gamma\Psi}$$

Proposition 8 *(Hopf bifurcation) Assume that $a > \hat{a}$ jointly with $\pi > \pi^H$. A limit cycle arises near the endemic steady-state, through a Hopf bifurcation, if and only if $\varepsilon_{ci} = \varepsilon_{ci}^H$.*

Proof. See the Appendix. ■

Considering jointly Proposition 8 and Lemma 2, it appears that the stable manifold is two-dimensional on one side of the Hopf bifurcation and is of dimension zero on the other side. That is, the steady-state looses (gains) its saddle-path stability through the Hopf bifurcation.

To interpret the occurrence of a Hopf bifurcation, it is necessary to know the sign of ε_{ci}^H. As discussed in the proof of Proposition 8 , $S > 0$ when $\varepsilon_{ci} = \varepsilon_{ci}^H$. Focusing on (37), $S > 0$ if and only if $\varepsilon_{ci} > 0$. We then conclude that $\varepsilon_{ci}^H > 0$. Having this in mind, we are now able to interpret the occurrence of the Hopf bifurcation. Assume that the economy is at the endemic steady-state at time t (i.e. $a > \hat{a}$) and assume an exogenous rise in the share of infectives i. Because $\varepsilon_{ci} > 0$, rise implies an increase in consumption and a decrease in saving, which means a drop in capital level and a lower production level. A lower production level results in a lower contamination rate (see Assumption 1 and remark that $\pi > \pi^H$ at the Hopf bifurcation) implying a lower level of infectives at time $t+1$ (see equation (3)). That is, a higher share of infectives is followed by a drop in the next period generating endogenous cycles.

The existence of a Hopf bifurcation deserves two more comments. First, we already know from Proposition 7 that a transcritical bifurcation can occur. What's happen when the conditions for a Hopf bifurcation's occurence meet the

[8] See Bosi and Desmarchelier (2019), among others.

conditions for a transcritical bifurcation's occurence? Second, there exists two types of Hopf bifurcation. A supercritical Hopf bifurcation gives birth to an attractive (stable) limit cycle, while a subcritical Hopf bifurcation gives birth to a repulsive (unstable) limit cycle. To discriminate between those two configurations, we have to study the sign of the first Lyapunov coefficient evaluated at the Hopf bifurcation point. While the stability of the limit cycle will be discussed numerically in the next subsection, the following proposition discusses the simultaneous occurrence of the transcritical and Hopf bifurcations.

Proposition 9 *(Bogdanov-Takens bifurcation) If $a = \hat{a}$ jointly with $\varepsilon_{ci} = \frac{\Delta}{\alpha\pi\gamma}$, then, the limit cycle, surrounding the endemic steady-state, shrinks and disappears while the two steady-states collide. A Bogdanov-Takens bifurcation occurs.*

Proof. See the Appendix. ■

It is interesting to remark that $\varepsilon_{ci}^{H} = \frac{\Delta}{\alpha\pi\gamma}$ when $a = \hat{a}$ (i.e. $\Psi = 0$). Geometrically, a variation of a implies a variation of Ψ and then a variation of ε_{ci}^{H}. That is, a decrease of a from 1 to $\hat{a}$ describes a curve in the $\left(\Psi, \varepsilon_{ci}^{H}\right)$-plane. The Bogdanov-Takens bifurcation is the point of this curve such that $\Psi = 0$ (i.e. $a = \hat{a}$). The existence of a Bogdanov-Takens bifurcation is very interesting, specially in terms of economic policy. Indeed, we can interpret $a = \hat{a}$ in terms of lockdown parameter:

$$a = \hat{a} \Longleftrightarrow \lambda = 1 - \frac{\gamma}{a\beta} \equiv \hat{\lambda}$$

Let us consider the case where the economy is at the endemic steady-state (i.e. $a > \hat{a}$ or equivalently $\lambda < \hat{\lambda}$) such that preferences imply the occurrence of endogenous cycles ($\varepsilon_{ci} = \varepsilon_{ci}^{H}$). Proposition 9 shows that it is possible for the Government to tune the lockdown in order to eradicate the disease as well as to stabilize the economy, that is, to *hit two birds with one stone*. Indeed, by setting $\lambda = \hat{\lambda}$, since $\varepsilon_{ci} = \varepsilon_{ci}^{H}$, a Bogdanov-Takens bifurcation occurs, the limit cycle shrinks and disappears while the endemic steady-state collide with the disease-free one ensuring the disease eradication.

4.2 Numerical exploration

As discussed previously, the Hopf bifurcation can be superciritical giving birth to a stable limit cycle or subcritical giving birth to an unstable limit cycle. Determining whether the limit cycle arising through the Hopf bifurcation studied in Proposition 8 is crucial. Indeed, the likelihood for the economic equilibrium to follow a limit cycle is extremely low if the limit cycle is unstable and conversely, is very high when the limit cycle is stable. To discriminate between those two cases, we have to study the sign of the first Lyapunov coefficient at the Hopf bifurcation point. A convenient way to proceed is to evaluate the first Lyapunov coefficient L_1 numerically by using the MATCONT package for Matlab. To that purpose, we consider the following usual functional form for

the utility function:

$$u(c,i) = \frac{\left(ci^{-\eta}\right)^{1-\varepsilon}}{1-\varepsilon} \tag{39}$$

with $\varepsilon > 0$ and $\eta > 0$. Considering (39), it follows that:

$$\varepsilon_{cc} = -\varepsilon < 0 \text{ and } \varepsilon_{ci} \equiv \eta(\varepsilon - 1) \lesseqgtr 0$$

Clearly, $\varepsilon_{ci} > 0\,(< 0)$ if and only if $\varepsilon > 1\,(< 1)$. In addition, using (8), we obtain explicitly the consumption demand as a function of μ and i:

$$c = \mu^{-\frac{1}{\varepsilon}} i^{\eta\left(\frac{\varepsilon-1}{\varepsilon}\right)}$$

Moreover, let:

$$\beta(y) = \beta_0 y^{\pi}$$

with $\pi \geq 0$ and $\beta_0 \in (0,1)$. β_0 captures the contamination rate when $\pi = 0$. Finally, we use a Cobb-Douglas production function:

$$f(k) = Ak^{\alpha}$$

where $A > 0$ and $\alpha \in (0,1)$. We can remark that the share of capital income in the total income is equal to α and the capital-labour elasticity of substitution $\sigma = 1$.

Let us consider the following calibration:

Table 1

Parameter	A	α	θ	δ	β_0	γ	π	a	λ	ε
Value	1	0.3	0.025	0.1	0.8	0.17	0.5	0.2	0.1	2

Parameters A, α, θ and δ are set at their usual quarterly values. Parameter ε is set to ensure that $\varepsilon_{ci} > 0$ (see discussion right after Proposition 8) while γ, π, β_0 and a are set in order to verify Assumption 4, $a > \hat{a}$ and $\pi > \pi^H$. Those parameters' values imply the following endemic steady-state:

$$\begin{aligned} k^* &= 3.492\,7 \\ l^* &= 0.829\,09 \\ c^* &= 0.916\,99 \\ i^* &= 0.098486 \end{aligned}$$

This leads to $R_0 = 1.021\,9 > 1$. Considering Table 1, we obtain:

$$\eta^H = \frac{\varepsilon_{ci}^H}{\varepsilon - 1} = 3.883\,0$$

At $\eta = \eta^H$, a Hopf bifurcation occurs. At this point:

$$\mu^* = 1.467\,4 \times 10^{-4}$$

Eigenvalues are given by:

$$\begin{aligned} \lambda_1 &= 0.0491253i \\ \lambda_2 &= -0.0491253i \\ \lambda_3 &= 0.0231487 \end{aligned}$$

λ_1 and λ_2 are two complex numbers with a zero real part which implies the emergence of limit cycle, through a Hopf bifurcation, around the endemic steady-state. The first Lyapunov coefficient L_1 is automatically reported by MATCONT at the Hopf bifurcation:

$$L_1 = -8.42896 * 10^{-3} < 0$$

Since $L_1 < 0$, it follows that the Hopf bifurcation is supercritical which means that the limit cycle studied in Proposition 8 is stable. That is, the likelihood for the economic equilibrium to follow the limit cycle near the endemic steady-state is extremely high (when conditions depicted in Proposition 8 are verified). Figure 1 gives a representation of this stable limit cycle.

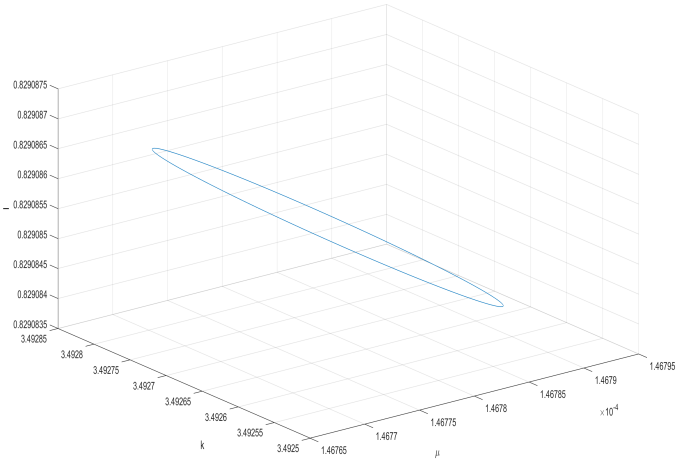

Figure 1: The stable limit cycle

5 Conclusion

Within this paper, we have developed a framework at the crossroad of economics and epidemiology to study how the lockdown affects the infectious disease persistence, the household's welfare and the economic dynamics. Taking into account

the fact that infectives can be asymptomatics, we have found that the lockdown can be welfare improving in the long run if the share of asymptomatics is sufficiently low and the contamination rate of the disease is relatively high. This result contrasts with the existing literature because it is obtained regardless of the household's empathy toward infectives. Moreover, the study of the local dynamics around the endemic steady-state has revealed that a stable limit cycle can appear through a Hopf bifurcation if the share of infectives increases sufficiently the marginal utility of consumption. In particular, we prove that it is possible to tune the lockdown to obtain simultaneously the limit cycle disappearance and the disease eradication (Bogdanov-Takens bifurcation). In this sense, the lockdown allows to *hit two birds with one stone*.

6 Appendix

Proof of Proposition 1

First of all, remark that:

$$s^* = \frac{a}{a + R_0 - 1}$$

Clearly, $s^* \in (0,1)$ if and only if $R_0 > 1$. Moreover:

$$\begin{aligned} \xi'(1) &= \gamma(R_0 - 1) \\ \xi'(s^*) &= \frac{\gamma}{s^*}\left(\frac{1 - R_0}{R_0}\right) \end{aligned}$$

It follows that $\xi'(1) < 0\,(>0)$ if and only if $R_0 < 1\,(>1)$. Finally, since $s^* \in (0,1)$, that is $R_0 > 1$, $\xi'(s^*) < 0$. ■

Proof of Proposition 2

To solve the maximization of (7) subject to (6), we apply the Pontryagin's maximum principle. In particular, we follow Seierstad and Sydsaeter (1987, Theorem 12, p. 234) or Acemoglu (2009, Theorem 7.13, p.254). The current value Hamiltonian is given by:

$$H = u(c,i) + \mu\left[(r - \delta)h + w(1 - \lambda)((1 - a)s + a) - c\right]$$

where μ is the Lagrangian multiplier. First-order conditions are given by:

$$\begin{aligned} \frac{\partial H}{\partial c} &= u_c(c,i) - \mu = 0 \\ \frac{\partial H}{\partial h} &= \mu(r - \delta) = \theta\mu - \dot{\mu} \\ \frac{\partial H}{\partial \mu} &= (r - \delta)h + w(1 - \lambda)((1 - a)s + a) - c = \dot{h} \end{aligned}$$

jointly with the transversality condition $\lim_{t \to +\infty} e^{-\theta t}\mu h = 0$. ■

Proof of Proposition 4

We differentiate (22), (24), (25), (26) and (27) and consider (21). Moreover, remark that $a > \hat{a}$ ensures that $(1-\lambda)\beta a - \gamma(1-a) > 0$ and $\gamma - a\beta(1-\lambda) < 0$. ■

Proof of Proposition 5

$a > \hat{a}$ ensures that $l^* < l_1$ while assumption 4 ensures that $\hat{a} < 1$. Moreover, $a > \hat{a}$ ensures that $(1-\lambda)\beta a - \gamma(1-a) > 0$ while $a < \tilde{a}\,(> \tilde{a})$ ensures that $2\gamma(1-a) - \beta a(1-\lambda) > 0\,(< 0)$. Finally, remark that $\tilde{a} > \hat{a}\,(< \hat{a})$ if and only if $\gamma < \frac{(1-\lambda)\beta}{2}\left(> \frac{(1-\lambda)\beta}{2}\right)$. ■

Proof of Proposition 8

Following Bosi and Desmarchelier (2019, Proposition 4), a Hopf bifurcation occurs if and only if $D = ST$ with $S > 0$. Here, a necessary (but not sufficient) condition for which this configuration holds is $T > 0$ (indeed, since $a > \hat{a}$, we know that $D > 0$, see Lemma 1). Interestingly $T > 0$ if and only if $\pi > \pi^H$. Moreover, $D = ST$ if and only if $\varepsilon_{ci} = \varepsilon_{ci}^H$. At this point, since $D > 0$ and $T > 0$, this ensures that $S > 0$. The last proposition follows. ■

Proof of Proposition 9

Following Bosi and Desmarchelier (2019, Proposition 6), a Bogdanov-Takens bifurcation occurs if and only if $D = S = 0$. Following Lemma 1, $D = 0$ if and only if $a = \hat{a}$. Moreover, when $a = \hat{a}$, $S = 0$ if and only $\varepsilon_{ci} = \frac{\Delta}{\alpha\pi\gamma}$. ■

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