INFECTIOUS DISEASE CONTROL AND HUMAN HEALTH INVESTMENT: LEARNING BY CONTROLLING

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Abstract

The paper endogenizes Ebola Virus Disease (EVD) incidence on economy allowing a two-way interaction between the economy and the disease dynamics to study health investment, disease control and learning associated with it. We show that the change in the steady state of economic variables is non-linear and can be non-monotonic. Disease control, and health capital investment increase with a decreasing discount rate as does the output share of disease control, although non-monotonically. While the disease-free steady state is parameter-free, a parameter-dependent steady state emerges from the endemic problem.

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1. Introduction

Infectious diseases affect both extensive and intensive labor supply, deplete health and physical capital and reduce market consumption opportunities (WHO, 2009). For example, during the 2014 Ebola Virus Disease (EVD) outbreak in West Africa, the annual growth of gross domestic product (GDP) of the most affected countries declines from 8.70% to 0% in Liberia, from 20.72% to -20.6% in Sierra Leone and from 3.94% to 3.81% in Guinea (World Bank, 2017). The disease also claimed 11,310 lives in those same countries (CDC, 2016). The damaging effect of diseases on economies, especially the most fragile ones, the recurrence and magnitude of disease shocks to human health justify the need to study infectious diseases' impact on economies in a more systematical way. However, by considering disease as exogenous shocks to economies, such modeling strategies might be insufficient and inconsistent in understanding the dynamics and effects of diseases on economies. In contrast, simultaneous modeling presents the advantage of accounting for both the direct and the indirect feedback effects of disease incidence

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on economic variables. The incidence of diseases negatively affects the labor force, while investments in health and disease control decrease the transmission of the disease and lead to an accumulation of human health capital (Goenka et al., 2014).

Previous studies have looked at the impact of infectious diseases on the economy. While their vast majority are devoted to empirical studies, theoretical research activity is also ongoing. Empirical studies investigate the question of whether there is any economic growth attached to investing in disease control and quantify the size and direction of the effect. Those studies rely on the steady states of the economic variables and the estimation of the effect of disease control on aggregates such as GDP per capita. Results show substantial positive effects (Bloom et al., 2009), positive moderate impacts to almost no impact (Ashraf et al., 2009), and even adverse effects (Acemoglu and Johnson, 2007; Young, 2005). As pointed out by Goenka and Liu (2012), those studies rest on the theoretical assumptions of a fixed saving rate and exogenous labor supply in the economy. However, labor supply and saving rate may be endogenous to the disease incidence. Therefore, an improved modeling approach that relies upon a simultaneous modeling of the dynamics of the economy and the explicit dynamics of the disease (Goenka et al., 2014) may highlight the level effects and economic fluctuations of the incidence of the disease on labor supply and health capital accumulation.

To untangle the two-way interaction between the disease transmission and the economy, and in order to improve the understanding of disease impacts on the economy at the macro level, we simultaneously model the disease and the economic dynamics. Applying a continuous-time neo-classical growth model, we endogenize the disease variable and

parameters[2], and account for the direct effects of the disease on the economy through the depletion of health capital and reduction of labor supply. Since the devastating Ebola Virus Disease outbreak in 2014 in West Africa, attention has increased to preparedness and responsiveness to epidemics and pandemics worldwide (WHO, 2016). Multiple donors, public health organizations, global humanitarian and non-for-profit organizations have set forth a score of actions and spent more than $500 million to control the disease (WHO, 2016). However, recently, a new EVD breaks out in central Africa and anyone could wonder whether any learned lessons from the 2014 outbreak could enhance the control of the ongoing outbreak. Learning-by-controlling, in a fashion analogous to the learning-by-doing applied to human capital in an endogenous growth model (Lucas, 1988; Romer, 1990), is a process that may reduce the cost of disease control and the likelihood or incidence of the next outbreak. Hence, the learning-by-controlling may enhance health capital accumulation.

Unlike previous studies (Geoffard and Philipson, 1996, Kremer1996; Gersovitz and Hammer, 2004), this paper endogenizes the effect of infectious diseases on the economy through human health capital and labor supply, taking the perspective of preparedness and responsiveness to diseases, as well as the learning in controlling the diseases. First, we consider a highly deadly disease with a non-waning immunity, i.e., the likelihood of relapse after recovery is zero, as it is with EVD (Berge et al., 2017). Second, after explicitly modeling the dynamics of EVD, we separate investment in general health expenditures from disease control as opposed to Grossman (1972) or Goenka et al., (2014). The two types of investment differ by their origination, purpose, and effect on the disease. For instance, during the EVD outbreak in West

[2] Most epidemiology models take those parameters as exogenous and utterly dependent on the biological system.

Africa in 2014, the response was mostly humanitarian and came from donors instead of local public health authorities. Since the year 2014 EVD outbreak, the share of external aid of the current health expenditures of the affected countries has increased dramatically. Two years before the outbreak, in 2012, the estimated share was 21.8 percent, 43.2 percent and 22 percent in Sierra Leone, Liberia, and Guinea, respectively. In the year 2015, at the end of the outbreak, the estimated share jumped to 52.6 percent , 70.9 percent and 24.9 percent in Sierra Leone, Liberia, and Guinea, respectively. These increases are higher than the 10-year average in the countries, suggesting an atypical investment of resource devoted to disease control, in the fashion that differs from the general health expenditures. Here, the response was mostly concerned with containing the disease than with rebuilding the health system. While medical expenditures may affect the disease indirectly through the extant health capital in the moment of the disease invasion, disease control directly affects the parameters such as the transmission rate of the disease. Third, we also allow for a learning process associated with the disease control such that accumulated control measures increase knowledge about the disease and reduce the damage potential of the disease. Also, the learning-by-controlling can result in decreased control costs and improved efficiency.

Empirical results show that the model has a disease-free steady state and an endemic steady state. As the discount rate changes, health capital, disease control, the fraction of susceptible and recovered, labor, medical expenditures, output, and consumption at steady state change monotonically in the disease-free model, while they exhibit a non-monotone behavior in the endemic model. The comparative statics on equilibrium outcomes show non-linearities in steady states, as opposed to the assumed linearity in reduced-form empirical studies that look at the effects of diseases on the economy. That highlights the role of endogenous change in health

expenditures and disease control. Learning-by-controlling, health capital, disease control investments, as well as consumption and output, in most part, increase for a low discount rate. Likewise, the output share of investments in disease control and general medical expenditures increases for a low discount rate.

Section Two lays out the model while section Three and Four solve the model and discuss the steady states. Section Five calibrates the model and section Six studies the effects of the varying discount rate on the steady states of the economic variables.

2. Model

We focus our modeling effort on medical expenditures in health, disease control and the learning attached to it. We consider a one-sector growth model where labor is supplied inelastically by the susceptible and the recovered from the disease. Goenka et al. (2014) have shown that elastic labor supply labor does not alter the dynamics of the model with disease incidence. There is a continuum of individuals of mass N_t, made of susceptible S_t, infected I_t and recovered R_t. We allow two categories of health investments: general health investment m_t, that can take the form of investment in health infrastructures and medical expenditures and only affect the disease dynamics indirectly; and disease control investments A_t spent as an emergency or humanitarian action which directly affect the incidence of the disease.

The model allows for learning e_t through disease control investments A_t. The learning-by-controlling reduces the cost of next disease control and the likelihood or incidence of the next outbreak. We can also illustrate learning-by-controlling by the differential responsiveness between two countries depending on their preparedness to the disease as well as on how weak and limited the infrastructure and capital are in each context. So that, starting with the same level of learning-by-controlling a country that has higher capital level could spend more resources in

disease control than a country where the capital level is limited; and thus, accumulates more knowledge about the disease. Although learning-by-controlling might be non-rivalrous, we do not account for any spillover effects in this model. Engaging disease control A_t and applying the existing knowledge e_t creates more learning-by-controlling. The learning-by-controlling process is a state variable which has a law of motion:

$$de_t/dt = E(A_t, e_t) \quad (1)$$

In equation (1), function $E(.)$ is increasing in control measures A_t and extant learning e_t. That is the learning-by-controlling increases for increasing disease control measures and extant learning.

The simultaneous model, formulated as a social planner's problem, results from a two-way interaction between health capital, the dynamics of the susceptible and the recovered and the disease incidence. A social planner formulation guarantees that the best possible outcome maximizes social welfare. For two reasons, individual choices to control the epidemiology parameters (Geoffard and Philipson, 1996; Kremer, 1996; Philipson, 2000) might be suboptimal. First, the externality in the disease transmission can lead to underspending of private disease control measures. Second, because of the contagion effects, private control may not be sufficient to curb the disease (Goenka et al., 2014). Furthermore, a social planner's formulation allows consumption for everyone in the economy, irrespective of their health condition and; thus, we do not need to keep track of individual health history over time. The social planner is endowed with an initial stock of health h_0, which depreciates at the rate δ, where $0 \leq \delta \leq 1$. The next sub-sections give a detailed account of the disease and the economic models.

2.1. Dynamics of SIR Epidemiology Model

The epidemiology model describes the dynamics of the disease. The dynamics of the disease are formed by a flow of mass between different compartments of the population depending on their

status regarding the disease and connected through the disease parameters. Consider a standard epidemiology model with three compartments: the susceptible S_t, the infected I_t, and the recovered R_t. This type of compartmental model has been used to study EVD (Berge et al., 2017). Figure 3.1 depicts the flow of the mass between the three compartments. Individuals are born healthy, without immunity to the disease, are susceptible to the disease, and upon infection become infective, i.e., can transmit the disease to others. They are equally likely to contract the disease, regardless of their age. There is a net birth rate b that does not depend on the disease dynamics (Hethcote, 2000). We denote by N the total population size at time zero so that $N = S_0 + I_0 + R_0$.

Three main parameters characterize the disease: the transmission, recovery and death rates α_t, γ_t and σ_t, respectively. The susceptible S_t are prone to the infection and do not have any acquired immunity against the pathogenic agent. They become infected with probability α_t (remain susceptible with probability $1 - \alpha_t$). The infected recovered with probability γ_t and die with probability σ_t. Therefore, the dynamics of the susceptible and infected compartments at time t are:

$$dS_t/dt = bN_t - \alpha_t I_t S_t/N_t$$

$$dI_t/dt = \alpha_t I_t S_t/N_t - (\gamma_t + \sigma_t)I_t$$

Individual's die at the *per capita* rate of σ_t. Since S_t, I_t and R_t are measured in the number of people; it follows that dS_t/dt, $d\,I_t/dt$ and dR_t/dt are the number of susceptible and infected per unit of time. The net birth rate b is the net number of people born per unit of time and σ_t the *per capita* death rate due to disease. $\alpha_t I_t$ is the force of the infection while $\alpha_t S_t$ is the number of susceptible who become infected per unit of time per infectious individual. S_t/N_t is the probability that a contact is with a susceptible. However, not all contacts with

susceptible necessarily lead to transmission. The transmission coefficient, α_t is the probability that contact results in transmission while $\alpha_t I_t S_t / N_t$ is the actual incidence of the disease, meaning the number of individuals who become infected per unit of time.

The infected, I_t, who form the prevalence of the disease, recover and move to the compartment of recovered at the rate γ_t. The recovered acquire immunity and cannot be re-infected by the same strain of the disease, which is consistent with the knowledge about the EVD (Berge et al., 2017). Hence, the dynamic of the recovered at time t is:

$$dR_t/dt = \gamma_t I_t$$

Putting the three differential equations together, we get the dynamics[3] of the disease as follow:

$$dS_t/dt = bN_t - \alpha_t I_t S_t / N_t$$

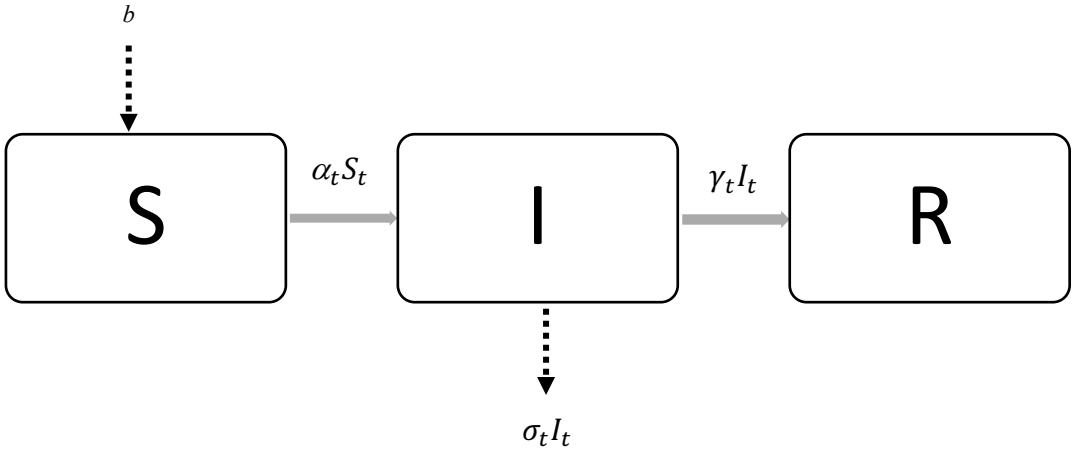
$$dI_t/dt = \alpha_t I_t S_t / N_t - (\gamma_t + \sigma_t) I_t$$

$$dR_t/dt = \gamma_t I_t$$

$$S_t, N_t > 0;\ \ I_t, R_t \geq 0\ \ \forall\, t;\ \ S_0, N_0 > 0;\ I_0, R_0 \geq 0;\ N = S_0 + I_0 + R_0$$

[3] The dynamics of the population N_t is such that $dN_t/dt = bN_t - \sigma_t I_t$. However, we do include those dynamics in our problem, since we are more interested in the steady states rather than the paths of the solutions to the problem.

Figure 3.1: Ebola SIR Compartment Disease Model



α_t = Transmission rate

γ_t= Recovery rate

σ_t = Death rate due to disease

S= Susceptible population

I= Infected population

R= Recovered population

To fit a general description of the disease without considering the size of the population of a specific country, we re-arrange the model as a frequency incidence model where every compartment is in terms of the proportion of the total population. As it appears in this model, the transmission rate α_t does not depend on the actual population but instead on the relative frequency of the infectives in the population. That allows us to abstract away from the usual mass incidence model where the dynamics of the disease depend on the number of infections. Anderson and May (1991) observe that the standard incidence model is common in epidemiology literature since the contact rate appears to be very weakly associated with the size of the population. We assume that newborns balance the dead people, such that the size of the population is constant, allowing us to isolate the frequency incidence model. To get the frequency incidence model, we divide each variable by the constant population N, such that:

$s_t = S_t/N\ ;\ i_t = I_t/N\ ;\ r_t = R_t/N.$

Therefore, the differential equation governing the disease is:

$$ds_t/dt = b - \alpha_t i_t s_t \tag{2}$$

$$di_t/dt = \alpha_t i_t s_t - (\gamma_t + \sigma_t) i_t \tag{3}$$

$$dr_t/dt = \gamma_t i_t \tag{4}$$

$$1 = s_t + i_t + r_t \quad (5)$$

Equations (2)-(5) can further be re-arranged as a two -dimensional system of equations below since the first two equations are independent of the third.

$ds_t/dt = b - \alpha_t i_t s_t$

$di_t/dt = \alpha_t i_t s_t - (\gamma_t + \sigma_t) i_t$

$r_t = 1 - s_t - i_t$

2.2. Equilibrium of the Epidemiology Model

The steady state of the SIR model hinges on equating equations (2)-(4) to zero and solving for the state variables. First, to get the sense of the maximum prevalence of the disease, it suffices to solve for the equation

$i_t = \alpha_t i_t s_t - (\gamma_t + \sigma_t) i_t = 0$. The prevalence first increases at time zero

$i_0 = \alpha_0 i_0 s_0 - (\gamma_0 + \sigma_0) i_0 > 0$, which gives a necessary and sufficient condition for an initial increase in the number of infected: $(\alpha_0 s_0)/(\gamma_0 + \sigma_0) > 1$. The maximum prevalence is attained when equation (3) equals zero, meaning $s = (\gamma + \sigma)/\alpha$. Equilibrium values are represented without time subscript. While the disease-free steady state is trivial since it occurs when there is no infection meaning $\tilde{s} + \tilde{r} = 1$, the equilibrium with disease relies on the behavior of the basic reproduction number $\mathcal{R}_0$ (Hethcote, 2000). Here, the subscript 0 is not a time subscript but just notational and follows the convention.

When $\mathcal{R}_0 = \alpha/(\gamma + \sigma) < 1$ and $i_0 > 0$, the number of infectives decreases to zero, and the disease dies out. The infectives decrease rapidly to zero, and the birth slowly increases the susceptible until eventually everyone is susceptible at disease-free equilibrium $\tilde{s} + \tilde{r} = 1$. When $\mathcal{R}_0 = \alpha/(\gamma + \sigma) > 1$, i_0 is small, s_0 is large with $\alpha/(\gamma + \sigma)\, s_0 > 1$, then s_t decreases and i_t increases up to a peak then decreases. The dynamics here are such that after the fraction of

the infections falls from the maximum prevalence and reaches the lower level, the birth of new susceptible increases gradually the fraction of susceptible until $\alpha/(\gamma+\sigma)\, s_t$ is large enough that another outbreak occurs such that

$\tilde{s} = (\gamma+\sigma)/\alpha\,,\ \tilde{\imath} = b/\gamma+\sigma\,,\ \tilde{r} = (\gamma+\sigma)(\alpha-\gamma-\sigma) - b\alpha/(\gamma+\sigma)\alpha$. The equilibrium characterizes an endemic disease, where the disease is present. Although, all susceptible have been infected and are now immune, not only new persons are born susceptible, but some individuals escape the disease, an observation made in practice and confirmed by the model (Martcheva, 2015).

2.3. Description of the Economy

The economy is a one-sector growth model, with a possibility of learning-by-controlling diseases, that endogenizes the dynamics of disease to allow a two-way interaction between disease and economic dynamics. There is a continuum of individuals of mass N_t. Labor supply in the economy comes from the susceptible s_t and recovered r_t. The infected are ill and cannot work. We consider that, in the absence of unemployment security the recovered will enter the labor force upon recovery. They, then, add to the susceptible to form the working population. The fraction of workers is bounded by one, such that $0 \leq\ s_t + r_t \leq 1\ \forall\ t$. The labor is indivisible and is supplied inelastically. At the time t, the labor supply inherits the dynamics of ds_t/dt and dr_t/dt as follows:

$$\begin{aligned} ds_t/dt &= b - \alpha_t(1-s_t-r_t)s_t \\ dr_t/dt &= \gamma_t(1-s_t-r_t) \end{aligned} \tag{6}$$

By endogenizing the transmission, recovery, and death rates, we consider that the transmission rate can be affected by disease control A_t, existing health capital h_t, and learning-by controlling the disease e_t, while recovery and death rates are function of health capital. The

transmission rate function is a monotone decreasing function of disease control, health capital and learning-by-controlling, i.e. higher disease control measures, higher learning-by-controlling and higher health capital reduce the transmission rate of the disease.

Furthermore, the transmission rate function $\alpha(.)$ satisfies the following conditions:

i. $\alpha(.)$: $\mathbb{R}_+^3 \rightarrow [0,1]$

ii. $\alpha(0,0,0) = 1$

iii. $\alpha(.)$ is such that $\alpha_1(A_t, h_t, e_t) \leq 0;\ \alpha_2(A_t, h_t, e_t) \leq 0;\ \alpha_3(A_t, h_t, e_t) \leq 0$ and $\alpha_{11}(A_t, h_t, e_t) \geq 0;\ \alpha_{22}(A_t, h_t, e_t) \geq 0;\ \alpha_{33}(A_t, h_t, e_t) \geq 0$. The transmission rate takes values such that:

$$0 \leq \alpha(A_t, h_t, e_t) \leq 1.$$

Similarly, the death rate $\sigma(.)$ is a function of health capital such that $\sigma'(h_t) \leq 0$ and $\sigma''(h_t) \geq 0$. The death rate is not a function of disease control[4]. The recovery rate $\gamma(.)$ is a function of health capital such that:

$\gamma'(h_t) \geq 0$ and $\gamma''(h_t) \leq 0$.

The main components of the economic model are detailed below.

Social welfare function: Households value consumption and inelastically supply labor. Incorporating an endogenous labor supply leaves the dynamics invariant under certain regularity conditions (Goenka et al., 2014). The lifetime discounted social welfare function is:

$$\int_{t=0}^{\infty} e^{-\theta t}\, u(c_t), \tag{7}$$

[4] The 2014 EVD outbreak has about 60% death rate. Although vaccine assays are ongoing and some with successful experimentation on humans in the ongoing outbreak in the East of DRC, they were not available in 2014 when the disease broke out, and EVD is still highly deadly.

Where c_t the consumption at time t, $u(.)$ is a utility function such that $u' \geq 0$, $u'' \leq 0$ and $\lim_{c_t \to 0} u' = \infty$; $\lim_{c_t \to \infty} u' = 0$. The discount rate θ is such that $\theta > 0 \ \forall \ t$. Both infected and non-infected consume.

Endowments: The representative consumer starts the course of its life with endowment h_0 of health capital. For simplicity and to reduce the dimensionality of the problem, there is no physical capital in the economy.

Single Production Sector: The production function $f(h_t)$ takes as input health capital h_t, capturing both extensive and intensive labor supply. The function $f(h_t)$ is the usual neo-classical technology, increasing in health capital, but the marginal product is decreasing.

Resources Constraint: General health investments m_t represent the investments that enter the health production function and are not related directly to disease control. They stand for medical expenditures to augment the health capital and are related to health conditions other than the disease under study. When an EVD breaks out, resources A_t are spent to control the disease. The output $Y_t = f(h_t)$ is used for consumption c_t, general medical expenditures m_t and disease control A_t.

$$c_t + m_t + A_t = f(h_t) \tag{8}$$

Law of Motion of Human Health Capital: Social planner carries to time t the precedent health capital reduced by how much the disease depletes health capital. The disease-related death rate $\sigma\ (h_t)$ captures the depletion effect of the outbreak on health capital through $\sigma\ (h_t)\ i_t = \sigma(h_t)\ (1 - s_t - r_t)$. We allow in the model the usual depreciation rate of health capital, whereby health capital depreciates with the time. Health capital is augmented through the health production function $g(m_t)$, that takes as inputs the general medical expenditures, and net births. Therefore, the law of motion of health capital is:

$$dh_t/dt = g(m_t) - [\delta + \sigma(h_t)(1 - s_t - r_t) - b]h_t, \quad (9)$$

where $g'(m_t) \geq 0$ and $g''(m_t) \leq 0$.

3. Social Planner's Problem

The social planner's problem is to choose consumption, medical expenditures and disease control investments as well as the next period health stock. The social planner chooses c_t, m_t and A_t in a manner that maximizes welfare to the economy :

$$\max_{c_t,\, m_t,\, A_t} \int_{t=0}^{\infty} e^{-\theta t} u(c_t) dt$$

subject to:

$$ds_t/dt = b - \alpha(A_t, h_t, e_t)(1 - s_t - r_t)s_t$$

$$dr_t/dt = \gamma(h_t)(1 - s_t - r_t)$$

$$dh_t/dt = g(m_t) - [\delta + \sigma(h_t)(1 - s_t - r_t) - b]h_t$$

$$c_t + A_t + m_t = f(h_t)$$

$$de_t/dt = E(A_t, e_t)$$

$$0 < s_t \leq 1 \quad (10)$$

$$0 \leq r_t \leq 1$$

$$m_t \geq 0;\ c_t > 0;\ A_t \geq 0;$$

$$e_0 \geq 0;\ h_0 > 0;\ s_0 > 0\ ;\ r_0 \geq 0$$

We formulate the problem in a constrained optimal control framework. The Lagrangian $\mathcal{L}(h_t, c_t, s_t, r_t, m_t, A_t, e_t, \boldsymbol{\lambda_t}, \mu_t)$, associated to the Hamiltonian, is a function of control variables, c_t, m_t, A_t, the state variables h_t, s_t, r_t, e_t, the co-state variables vector $\boldsymbol{\lambda_t}$ and Lagrangian multiplier μ_t. In terms of current Hamiltonian H, the Lagrangian is:

$$\mathcal{L}(h_t, c_t, r_t, s_t, m_t, A_t, e_t, \boldsymbol{\lambda}, \mu)$$
$$= u(c_t) + \lambda_{t1}[g(m_t) - [\delta + \sigma(h_t)(1 - s_t - r_t) - b]h_t]$$
$$+ \lambda_{t2}[b - \alpha(A_t, h_t, e_t)(1 - s_t - r_t)s_t] + \lambda_{t3}[\gamma(h_t)(1 - s_t - r_t)]$$
$$+ \lambda_{t4}[E(A_t, e_t)] + \mu_t[f(h_t) - c_t - A_t - m_t]$$

The problem is not concave since the law of motion of the susceptible is not concave due to the increasing returns of controlling diseases on the susceptible. Therefore, Mangasarian conditions do not apply. That is because the Hessian of the maximized Hamiltonian may not be negative definite since it is possible that[5] $\frac{\partial^2 H^*}{\partial^2 s_t} > 0$. Goenka et al. (2014) have nevertheless shown that there is an optimal solution to the problem, relying on the weak compactness of the feasible set, the weakly convergence of state variables and their associated derivatives, as well as the continuity of the state variables.

3.1. Dynamics Without Learning-by-Controlling

This baseline analysis abstracts away from learning-by-controlling in the economy. Let assume that the social planner does not learn from disease control. Such an analysis leaves out one state variable. Factors such as recurrence or duration of the disease can explain such a setting. For instance, a disease outbreak for which the country does not have a precedent, or a disease with a low likelihood to turning into an enduring epidemic reduces the opportunity to learning-by-controlling. The Lagrangian associated with the current Hamiltonian becomes:

$$\mathcal{L}(h_t, c_t, r_t, s_t, m_t, A_t, \boldsymbol{\lambda_t}, \mu_t)$$
$$= u(c_t) + \lambda_{t1}[g(m_t) - [\delta + \sigma(h_t)(1 - s_t - r_t) - b]h_t]$$
$$+ \lambda_{t2}[b - \alpha(A_t, h_t)(1 - s_t - r_t)s_t] + \lambda_{t3}[\gamma(h_t)(1 - s_t - r_t)]$$
$$+ \mu_t[f(h_t) - c_t - A_t - m_t]$$

The optimality and transversality conditions below define the solutions:

[5] Which rules out the Arrow sufficiency conditions.

$$c_t: u'(c_t) = \mu_t \tag{11}$$

$$m_t: \lambda_{t1} g'(m_t) = \mu_t \tag{12}$$

$$A_t: -\lambda_{t2}(1 - s_t - r_t)\alpha_1(A_t, h_t) = \mu_t \tag{13}$$

$$dh_t/dt: d\lambda_{t1}/dt = \lambda_{t1}\big[\theta + \delta - b + (1 - s_t - r_t)\big(h_t\sigma'(h_t) + \sigma(h_t)\big)\big] + \tag{14}$$

$$\lambda_{t2}[(1 - s_t - r_t)\alpha_2(A_t, h_t)\,] - \lambda_{t3}[(1 - s_t - r_t)\,\gamma'(h_t)] - \mu_{t1} f'(h_t)$$

$$ds_t/dt: d\lambda_{t2}/dt = -\lambda_{t1} h_t \sigma(h_t) + \lambda_{t2}[\theta + \alpha(A_t, h_t)(1 - \ s_t - r_t) - \alpha(A_t, h_t)s_t] + \tag{15}$$

$$\lambda_{t3}[\gamma(h_t)]$$

$$dr_t/dt: d\lambda_{t3}/dt = -\lambda_{t1} h_t \sigma(h_t) - \lambda_{t2}[\alpha(A_t, h_t)s_t] + \lambda_{t3}[\theta + \gamma(h_t)]$$

$$\lim_{t\to\infty} \lambda_{t1} e^{-\theta t}\, h_t = 0 \tag{16}$$

The co-state variables $\lambda_{t1},\ \lambda_{t2},$ and λ_{t3} represent the shadow values of health capital, the susceptible and recovered, respectively.

Equation (12) implies that the value of the marginal product of health expenditures equals the marginal utility of consumption. That is, the marginal benefit of health expenditures equals the cost of forgoing a unit of consumption. Similarly, combining equations (11) and (13), we have:

$$-\lambda_{t2}(1 - s_t - r_t)\alpha_1(A_t, h_t) = u'(c_t) \tag{17}$$

Equation (17) implies that the cumulative value of the marginal benefit of disease control on the fraction of the susceptible and recovered equals the marginal cost of a unit of consumption spent in controlling the disease. Therefore, the social planner will invest in disease control up to the point where the marginal benefit of disease control on the working population equals the marginal utility of consumption.

3.2. Dynamics with Learning-by-Controlling

In this situation, learning-by-controlling is accumulated through disease control. Having learning-by-controlling in the model alters the social planner's problem in a couple of aspects. First, there is another motive - if desirable for the society - to invest in disease control besides the curbing of the outbreak. The motive is to enhance the knowledge about the disease such that any future outbreak might be curbed effectively and at a lower cost. Learning-by-controlling enhances preparedness for disease outbreak and improves the incidence of the disease, which indirectly affects health capital accumulation. Second, although our model does not consider such a case, the learning-by-controlling could have spillover effects within the economy such that higher labor productivity could be achieved because of lower disease incidence. Third, learning-by-controlling adds one more dimension to the problem, increasing the mathematical complexity. We provide a numerical comparison of the dynamics of the economic variables in models with and without learning-by-controlling in a later section of this chapter.

The Lagrangian associated with the current Hamiltonian is:

$$\begin{aligned}\mathcal{L}(h_t, c_t, r_t, s_t, e_t\, m_t, A_t, \boldsymbol{\lambda_t}, \mu_t) \\ &= u(c_t) + \lambda_{t1}[g(m_t) - [\delta + \sigma(h_t)(1 - s_t - r_t) - b]h_t] \\ &+ \lambda_{t2}[b - \alpha(A_t, h_t, e_t)(1 - s_t - r_t)s_t] + \lambda_{t3}[\gamma(h_t)(1 - s_t - r_t)] \\ &+ \lambda_{t4}[E(A_t, e_t)] + \mu_t[f(h_t) - c_t - A_t - m_t]\end{aligned}$$

The optimality conditions are:

$$\begin{aligned}&c_t: u'(c_t) = \mu_t \\ &m_t: \lambda_{t1} g'(m_t) = \mu_t \\ &A_t: -\lambda_{t2}(1 - s_t - r_t)\alpha_1(A_t, h_t, e_t) + \lambda_{t4} E_1(A_t, e_t) = \mu_t \qquad (18)\end{aligned}$$

$$dh_t/dt: d\lambda_{t1}/dt = \lambda_{t1}\left[\theta + \delta - b + (1 - s_t - r_t)\left(h_t\sigma'(h_t) + \sigma(h_t)\right)\right] + \tag{19}$$

$$\lambda_{t2}[(1 - s_t - r_t)\alpha_2(A_t, h_t, e_t)] - \lambda_{t3}[(1 - s_t - r_t)\gamma'(h_t)] - \mu_t f'(h_t)$$

$$ds_t/dt: d\lambda_{t2}/dt = -\lambda_{t1}h_t\sigma(h_t) + \lambda_{t2}[\theta + \alpha(A_t, h_t, e_t)(1 - s_t - r_t) - \tag{20}$$

$$\alpha(A_t, h_t, e_t)s_t] + \lambda_{t3}[\gamma(h_t)]$$

$$dr_t/dt: d\lambda_{t3}/dt = -\lambda_{t1}h_t\sigma(h_t) - \lambda_{t2}[\alpha(A_t, h_t, e_t)s_t] + \lambda_{t3}[\theta + \gamma(h_t)] \tag{21}$$

$$de_t/dt: d\lambda_{t4}/dt = \lambda_{t2}[(1 - s_t - r_t)s_t\alpha_3(A_t, h_t, e_t)] + \lambda_{t4}[\theta - E_2(A_t, e_t)] \tag{22}$$

$$\lim_{t\to\infty} \lambda_{t1}e^{-\theta t}\, h_t = 0;\ \lim_{t\to\infty} \lambda_4 e^{-\theta t}\, e_t = 0 \tag{23}$$

Combining equations (11) and (18), we have:

$$-\lambda_{t2}(1 - s_t - r_t)\alpha_1(A_t, h_t, e_t) + \lambda_{t4}E_1(A_t, e_t) = u'(c_t) \tag{24}$$

Equation (24) implies that the cumulative value of the marginal benefit of disease control on labor force, through the susceptible and recovered, and on learning-by-controlling equals the marginal cost of a unit of consumption spent in controlling the disease. Therefore, the social planner will invest in disease control up to the point where the marginal benefit of disease control on the working population and on learning-by-controlling disease equals the marginal utility of consumption.

4. The Steady States of the Economy

To understand the equilibrium of the economy, we characterize the steady states of disease control, medical expenditures, health capital, learning-by-controlling, consumption, the fraction of the susceptible and recovered, and the resulting labor supply by the working population. The economy has a disease-free and an endemic steady state. Given the first order conditions of the optimization, there is always a disease-free steady state that does not depend on parameter values. Such a steady state occurs when the $\tilde{s} + \tilde{r} = 1$, the susceptible and recovered make the entire population, they are healthy and able to supply labor. Under the endemic condition, the

first order conditions lead to a steady state with parameters constraints. The sufficiency conditions are analogous to the one in Goenka et al. (2014) and are based on weak compactness of the feasible set.

The steady states of the economic variables in the social planner's problem derive from the system of non-linear equations (18)-(22), (11)-(12), (8)-(9), (6) and (1). Let the system of the equations be a matrix $[\boldsymbol{M}]$ (see supplementary material SM 3.1 for details). The steady states solve for:

$$[\boldsymbol{M}] = [\mathbf{0}] \tag{25}$$

The economy has a disease-free steady state characterized by $\tilde{s} + \tilde{r} = 1$; However, when $\tilde{s} + \tilde{r} = (\gamma + \sigma - b)/(\gamma + \sigma)$, meaning $\tilde{s} + \tilde{r} < 1$, the economy has an endemic steady state.

Case 1: $\tilde{s} + \tilde{r} = 1$. Consider the first order condition on disease control: $A = -\lambda_2(1 - s_t - r_t)\alpha_1(A_t, h_t, e_t) + \lambda_4 E_1(A_t, e_t) = \mu_t > 0$ implies that $\tilde{A} = 0$ since $\alpha_1(A_t, h_t, e_t)$ and $E_1(A_t, e_t)$ are finite. A disease-free steady state exists for all parameters values since $\tilde{s} + \tilde{r} = 1$ is parameter-free.

Case 2: An endemic steady state exists if and only if there exists γ, σ and $b > 0$ such that $\tilde{s} + \tilde{r} < 1$, and $(\tilde{s}, \tilde{r}, \tilde{h}, \tilde{e}, \tilde{m}, \tilde{A}, \tilde{c})$ is an endemic steady state solution to the dynamical system (25). There are non-negative $\tilde{s}, \tilde{r}, \tilde{h}, \tilde{e}, \tilde{m}, \tilde{A}$ and $\tilde{c}$ as solutions to the dynamical system (25) which characterizes the endemic steady state.

The economy has a unique disease-free steady state: the disease is completely eradicated; the working population is healthy and entirely able to supply labor. Furthermore, there is no need to invest in disease control. Subsequently, learning-by-controlling is limited to the existing knowledge $\bar{e}$. Such a steady state always exists and is parameter-free.

However, when the transmission rate among the susceptible is greater than zero, and the working population fraction is less than one, there is an endemic steady state. In that state, the disease is prevalent and depletes the health of the working population and their ability to work. That translates into fewer hours worked and reduced productivity for an hour worked. Furthermore, there is a non-negative investment in medical expenditures and disease control. Because of the investment in disease control, there is an added learning-by-controlling to the existing knowledge. The two types of steady states are exclusive.

The disease-free steady state has a closed form while the endemic steady state is more mathematical involved and cannot be solved analytically. In the next section, we provide more details using some functional forms to solve numerically by Newton Raphson method the endemic steady state.

5. Model Calibration

In this section, we calibrate the model using specific functional forms and parameters that describe both the economy and the disease. The calibration exercise concerns both the epidemiology model and the economy. We calibrate the epidemiology model using the transition matrix (see Table 3.1) which stands for the likelihood to move from one compartment to another. Disease parameters such as the recovery and death rate are drawn from literature and from the year 2014 outbreak of EVD in West Africa (WHO, 2016; Berge et al., 2017). There is a 2 percent chance to contract the disease once an individual is in contact with the pathogen. Thus, the susceptible stay prone to the disease with probability 0.98. There is one initial case from which the outbreak starts and spreads. Once infected, there is a 40 percent chance to remain infected and 60 percent to recover, which is consistent with the fatality rate of the disease (WHO, 2016). Once an individual recover there is a 99 percent chance that she will stay recovered and

unlikely to be infected by the same strain of EVD (Berge et al., 2017). We use the actual population size in Liberia (4.7 million), Sierra Leone (7million) and Guinea (12 million), the most affected countries by the 2014 EVD outbreak. The data on the disease such as the observed recovered population and disease control coverage rate are drawn from the humanitarian data exchange of the United Nations Office for the Coordination of Humanitarian Affairs (OCHA-HDX, 2018). The simulation uses a Markovian chain that takes in the initial conditions, time and the transition matrix probabilities (Table 3.1) to generate the state of the disease at period t.

Table 3.1: Transition Matrix of EVD SIR Model (in probability)

	S	**I**	**R**
S	0.98	0.02	0
I	0	0.40	0.60
R	0	0.01	0.99

The next exercise is to calibrate the economy and the dynamical model resulting from the two-way interaction between the economy and the disease. To characterize an example of steady state, we specify functional forms that meet the assumptions of the model. Let the preferences take the functional form $u(c_t) = log(c_t)$. The output and health production functions have the following functional forms:

$f(h_t) = \ h_t^{\varphi};\ g(m_t) = m_t^{\phi_1},$

where $0 < \phi_1 < 1;\ 0 < \varphi < 1$. The learning-by-controlling function is a-la-Romer with the existing knowledge e_t, the disease control investment A_t scaled by the effectiveness of the disease control ε. In a multiplicative form, $E(A_t, e_t) = \varepsilon A_t e_t$. Finally, let respectively the death, transmission, and recovery rates functions have the functional forms:

$\sigma(h_t) = e^{-\phi_2 h_t};\ \alpha(h_t, A_t, e_t) = e^{-\phi_3 h_t A_t e_t};\ \gamma(h_t) = -e^{-\phi_4 h_t},$

where $0 < \phi_2 < 1;\ 0 < \phi_3 < 1,\ 0 < \phi_4 < 1$.

A disease-free steady state satisfies the following conditions:

$$\tilde{s} + \tilde{r} = 1 \rightarrow \sigma(\tilde{h}) = \alpha(\tilde{h}, \tilde{A}, \tilde{e}) = \gamma(\tilde{h}) = 0;\ \tilde{A} = 0;\ dh_t/dt = de_t/dt = 0$$

$$dh_t/dt = 0 \rightarrow \tilde{m}^{\phi_1} = \delta\tilde{h} \tag{26}$$

From the conditions on the co-state variables, we have:

$$d\lambda_t/dt = 0 \rightarrow \frac{(\theta + \delta - b)}{\phi_1 \tilde{c}\tilde{m}^{\phi_1 - 1}} = \frac{\varphi\tilde{h}^{\varphi-1}}{\tilde{c}} \tag{27}$$

Plugging in the expression of $\tilde{h}$ in equation (27) we solve for $\tilde{m}$, $\tilde{h}$, $\tilde{y}$, and $\tilde{c}$ as:

$$\tilde{m} = \left[\frac{(\theta + \delta - b)}{\phi_1 \varphi \delta^{(1-\varphi)}}\right]^{\frac{1}{\varphi\phi_1 - 1}} \tag{28}$$

$$\tilde{h} = \left(\left[\frac{(\theta + \delta - b)}{\phi_1 \varphi \delta^{(1-\varphi)}}\right]^{\frac{\phi_1}{\varphi\phi_1 - 1}}\right) \Big/ \delta \tag{29}$$

$$\tilde{y} = \left(\left[\frac{(\theta + \delta - b)}{\phi_1 \varphi \delta^{(1-\varphi)}}\right]^{\frac{\varphi\phi_1}{\varphi\phi_1 - 1}}\right) \Big/ \delta^{\varphi} \tag{30}$$

$$\tilde{c} = \tilde{y} - \tilde{m} = \left(\left[\frac{(\theta + \delta - b)}{\phi_1 \varphi \delta^{(1-\varphi)}}\right]^{\frac{\varphi\phi_1}{\varphi\phi_1 - 1}}\right) \Big/ \delta^{\varphi} - \left(\left[\frac{(\theta + \delta - b)}{\phi_1 \varphi \delta^{(1-\varphi)}}\right]^{\frac{1}{\varphi\phi_1 - 1}}\right) \tag{31}$$

Equations (28)-(31) characterize the disease-free steady state for medical expenditures, health capital, output and consumption. The endemic steady state is solved numerically.

In the calibrated model, we use the following parameters: $\phi_1 = 0.2;\ \phi_2 = 0.5;\ \phi_3 = 0.5;\ \phi_4 = 0.5;\ \varphi = 0.36;\ \delta = 0.05$ and $b = 0.0482$. The choice of parameters follows the convention. If there is no existing convention to follow, especially for the parameters related to health production functions and disease dynamics, parameters are chosen such that the

assumptions imposed on the functions are met. The birth rate b is the average fertility rate in Liberia, Sierra Leone, and Guinea, during the period of the outbreak (World Bank, 2014).

Figures 3.2-3.4 illustrate the calibrated disease model outcomes and compare them to the actual data. In Figure 3.2 the observed recovered from EVD outbreak in 2014 are plotted against their simulated counterparts. Results show that the calibrated model matches well the data from the three countries. The calibration proves that the SIR model predicts in most part the dynamics of the diseases. Next, Figures 3.3-3.4 illustrate the simulated recovered with the actual disease control coverage rate. The disease control coverage rate is the ratio of the available or allocated humanitarian aid and the resources necessary to control the disease as estimated by the United Nations Office for the Coordination of Humanitarian Affairs (OCHA-HDX, 2018). For Liberia, Figure 3.3 shows the observed recovered and the disease control coverage rate while Figure 3.4 illustrates the simulated recovered and the disease control coverage rate. Results show that disease control coverage rate increases with time. For an increased disease control rate, the fraction of recovered increases. Therefore, there a proportional relationship between the disease control coverage rate and the number of recovered.

Figure 3.2: Dynamics of EVD Model: The Observed and Simulated Recovered

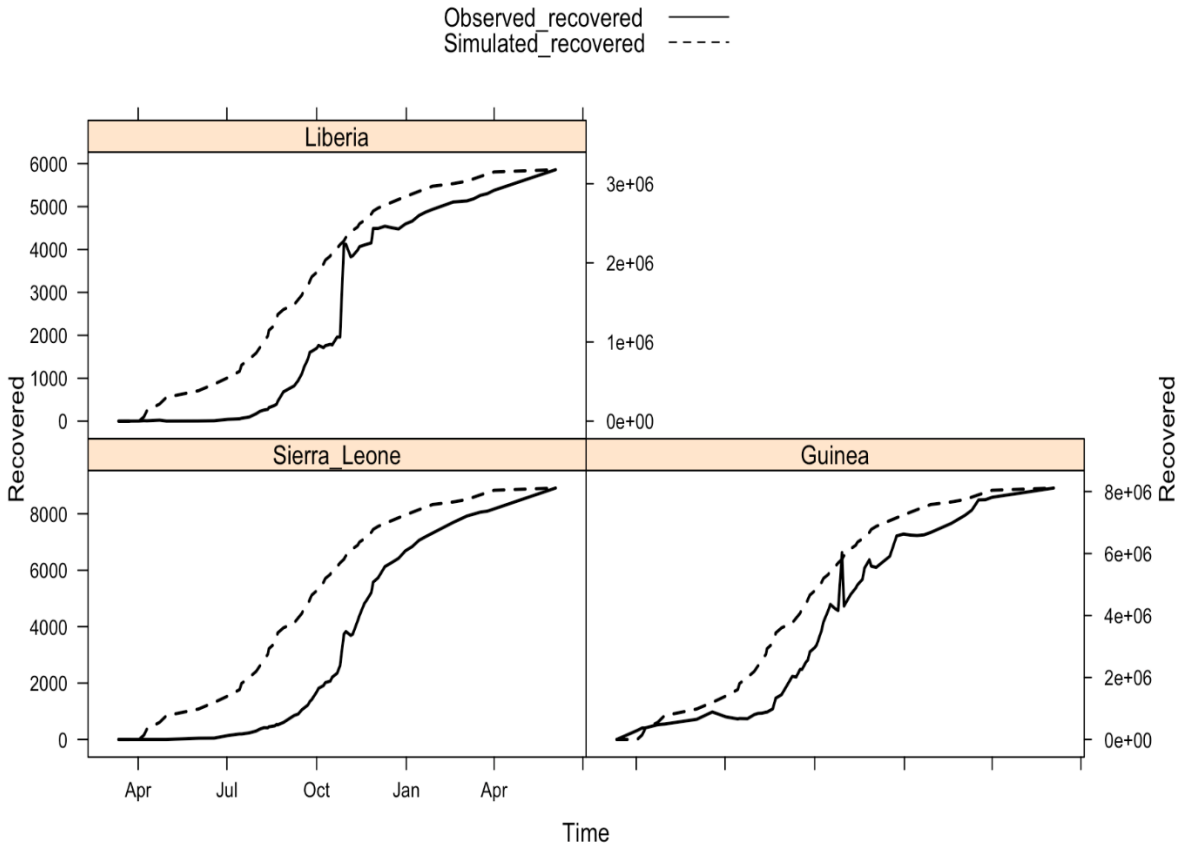


Figure 3.3: Disease Control Coverage Rate and The Observed Number of Recovered

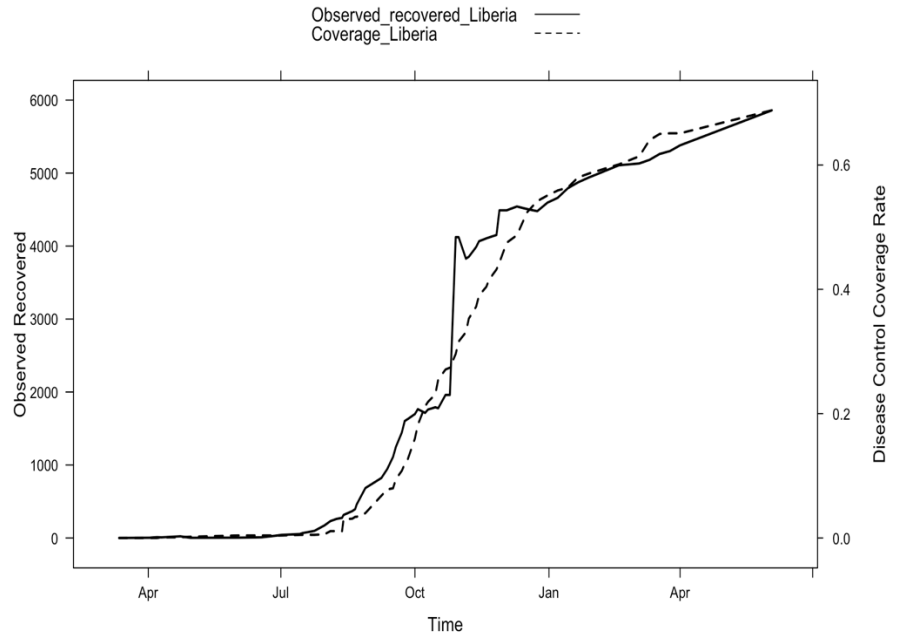
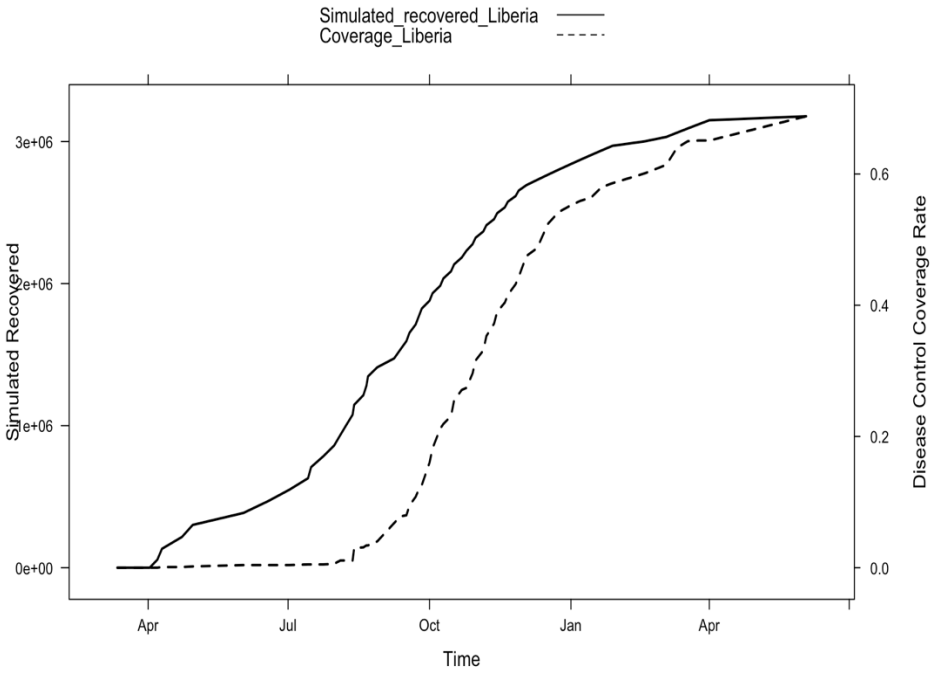


Figure 3.4: Disease Control Coverage Rate and The Simulated Number of Recovered



6. Effects of the Varying Discount Rate on the Steady States

To study the effects of an increasing average lifespan within the population on the dynamics of the economy with and without disease, we examine the comparative statics of the variables in equilibrium for varying discount rate. This analysis considers the discount rate as a measure of longevity within the population (Hall and Jones, 2007; Goenka et al., 2014). As the average lifespan increases among the population, people become more patient. That is to say that as the average lifespan increases within the population, people discount less time. From the analytical solutions of the disease-free scenario, in equations (28)-(31) and the numerical simulation of the endemicity, we plot in Figures 3.5a -3.5c the behavior of each economic variable as the discount rate increases, i.e., as people become less patient. Figures 3.5a refers to the scenario of the disease-free while Figures 3.5b-3.5c stand for the endemic situations with and without learning-by-controlling, respectively.

Dynamics for the disease-free scenario are illustrated in Figure 3.5 a. For the disease-free steady state, there is full employment, zero disease control and learning-by-controlling is equal to the existing knowledge, fixed at 0.24. For an increasing discount rate, meaning that people become less patient, health capital, general medical expenditures, consumption, and output decrease. However, the output share of the medical expenditures increases, but at a decreasing rate as the discount rate falls. The results are consistent with the findings in Goenka et al. (2014) and with the neo-classical growth model.

Figures 3.5b-3.5c refer to the dynamics of endemic scenario with and without learning-by-controlling. The dynamics of the economic variables at steady states are non-linear and non-monotonic. There is a non-linear relationship between the discount rate and the economic variables at steady state when the economy in under disease invasion. Economic variables such

as labor, health capital, medical expenditures, and disease control investment exhibit non-monotonicity for varying discount rate. Disease control expenses first increase before declining for the discount rate below 0.2. Learning-by-controlling, first declines before slightly increasing for the discount rate values less or equal to 0.1. General medical expenditures first increase and then decrease for the discount rate below 0.1.

The combined effect of disease control, and medical expenditures leads to an increase in health capital accumulation when the discount rate decreases, in most part. When people become more patient, they increase disease control, and general health expenditures. That translates into higher health capital accumulation, but not monotonically. These dynamics are consistent with and without learning-by-controlling except that medical expenditures first decline under a model without learning-by controlling and health capital accumulation declines starting from the discount rate of 0.2 under the model without learning-by controlling. With learning-by-controlling the output share of disease control, first decreases then increases for a low discount rate. It will eventually fall for low output values. However, without learning-by-controlling, the output share of disease control, first increases, then decreases; and returns to an increasing pattern for low output level and discount rate. The output share of medical expenditures first increases (decrease) in the model without learning-by-controlling, then decreases(increases) for low output level and discount rate.

For decreasing discount rate, the fraction of the recovered first decreases and then increases, without learning-by-controlling; stays unaffected before jumping under leaning-by-controlling for the discount rate below 0.1. Without learning-by controlling, the fraction of the susceptible first decreases before increasing for discount rate value below 0.2. With learning-by-controlling, the fraction of the susceptible increases, then starts falling when the fraction of the

recovered picks up for the discount rate below 0.1. The resulting dynamics in an increase of the labor supply as discount rate diminishes before decreasing for discount rate value below 0.1, in the model with learning-by-controlling. However, labor supply first decreases in the model without learning-by-controlling and then increases for the discount rate below 0.1. Since health capital is the source of effective labor, it follows that the labor supply increases for decreasing discount rate and below 0.1 in the model with learning-by-controlling and decreases in the model without learning-by-controlling. Nevertheless, the labor supply change is not monotone for all decreasing discount rate.

Figure 3.5a: Change in Economic Variable as the Discount Rate Changes (Disease-Free)

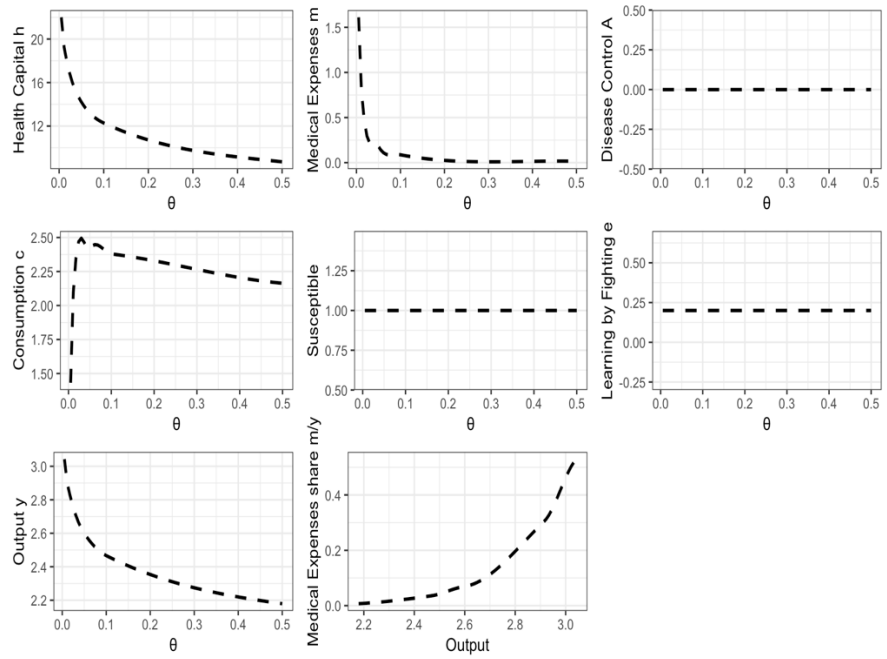


Figure 3.5b: Change in Economic Variable as the Discount Rate Changes (Endemic): Model With Learning-by-controlling

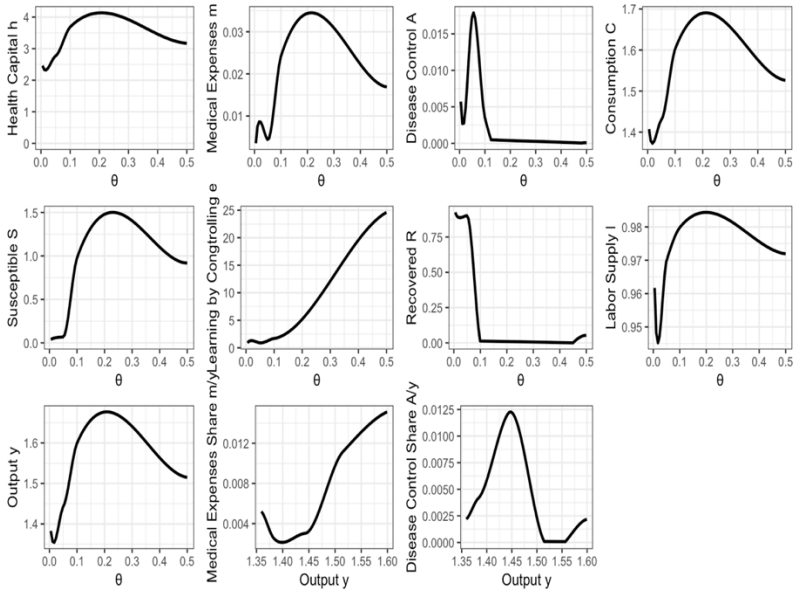
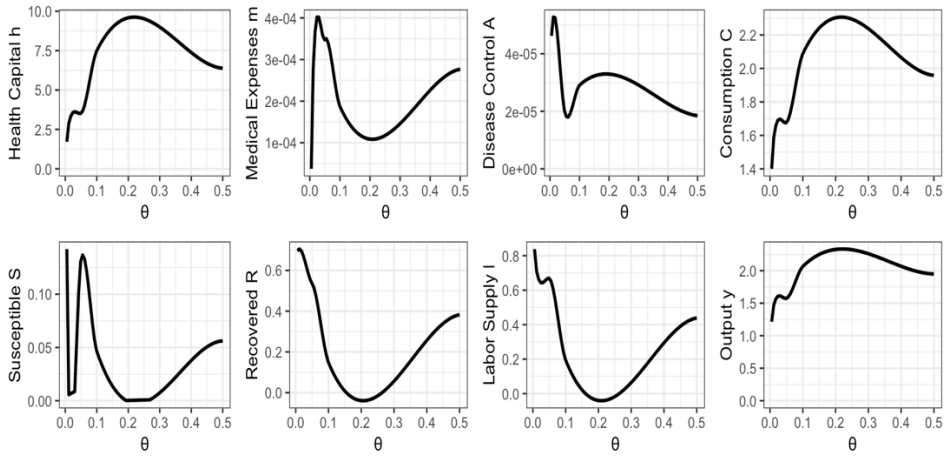


Figure 3.5c: Change in Economic Variable as the Discount Rate Changes

(Endemic): Model Without Learning-by-controlling



The comparative statics allow us to study the properties of the steady states of the economy for varying discount rate. Although it is common to assume linear change at equilibrium, findings indicate non-linearities in the change of steady states, highlighting the role of endogenous change in health expenditures and disease control, even in the context of permanent acquired immunity. Therefore, the non-linearity may be important in understanding the behavior of the model. As pointed by Goenka et al., (2014), the linearity assumption underlying the empirical works of the effects of diseases on economies (Acemoglu and Johnson, 2007; Ashraf et al., 2009; Bloom et al., 2009; Young, 2005) might not hold.

Beyond the possibility of studying the change of the output share of medical expenditures as discount rate varies, the model allows us to consider a change of the output share of disease control as discount rate changes. Precisely, the output share of disease control expenses increases as the lifespan increases in an economy. The findings in the literature that technological advance, economic growth, institutional change, and other factors are determinant in the health expenditures could gain in consistency and deeper understanding of the dynamics by also considering the effect of the discount rate on disease control. Therefore, the modeling of the dynamics of economics under an invasion of diseases may bring about insights on the impacts of the diseases on economies and guide disease preparedness, responsiveness and policies that address them. That is particularly relevant in the countries where infectious diseases make up an essential share of disease burden..

7. Conclusions

This paper develops a model that allows a two-way interaction between disease dynamics, where infected acquire permanent immunity, and economic dynamics that relies on a EVD SIR epidemiology model and a one-sector growth model. Although the disease dynamics make the

optimization problem non-concave, Goenka et al. (2014) have shown that certain optimality conditions are met, and local solutions exist. The paper contributes to the literature on simultaneous modeling of economic and epidemiology dynamics by 1) adding learning-by-controlling disease where acquired immunity does not wane and 2) clearly distinguishing between general health expenditures and disease control expenditures.

We calibrate both the epidemiology and the two-way interaction model (epidemiology and economic dynamics) and simulate results that allow an understanding of how the discount rate could affect the steady states of economic variables. Such an analysis is essential to understand whether and how factors, other than economic growth, technology, and institutional change could affect decisions to control a disease. In the economy, there is a disease-free steady state that is parameter-free and an endemic steady state that depends on parameter values. Disease control and health capital accumulation increase for decreasing discount rate. The pattern of learning-by-controlling, although increasing for a low discount rate, is non-linear and non-monotone.

Like learning-by-controlling, under the endemic scenario, labor, consumption, output share of disease control and medical expenditures, medical expenditures, output as well as the fraction of the susceptible and recovered exhibit non-linearities in equilibrium outcomes that shed light into the interactions between the dynamics of the economy and disease. The model, which can be extended to other diseases and categories of health investments and learning-by-controlling processes, improves diseases and economy dynamics simultaneous modeling that could bring about insights on impacts of the diseases on economies; thus, guiding disease preparedness and responsiveness and policies that address them in countries that are severely and recurrently embattled with infectious diseases.

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SUPPLEMENTARY MATERIAL

SM 3.1: System of Equations of Endemic Steady State

$$0 = \mu_t - u'(c_t)$$

$$0 = \mu_t - \lambda_{t1} g'(m_t)$$

$$0 = -\lambda_{t2}(1 - s_t - r_t)\alpha_1(A_t, h_t, e_t) + \lambda_{t4} E_1(A_t, e_t) - \mu_t$$

$$0 = \lambda_{t1}\left[\theta + \delta - b + (1 - s_t - r_t)\left(h_t \sigma'(h_t) + \sigma(h_t)\right)\right] + \lambda_{t2}[(1 - s_t - r_t)\alpha_2(A_t, h_t, e_t)] - \lambda_{t3}[(1 - s_t - r_t)\gamma'(h_t)] - \mu_t f'(h_t)$$

$$0 = -\lambda_{t1} h_t \sigma(h_t) + \lambda_{t2}[\theta + \alpha(A_t, h_t, e_t)(1 - s_t - r_t) - \alpha(A_t, h_t, e_t)s_t] + \lambda_{t3}[\gamma(h_t)]$$

$$0 = -\lambda_{t1} h_t \sigma(h_t) - \lambda_{t2}[\alpha(A_t, h_t, e_t)s_t] + \lambda_{t3}[\theta + \gamma(h_t)]$$

$$0 = \lambda_{t2}[(1 - s_t - r_t)s_t \alpha_3(A_t, h_t, e_t)] + \lambda_{t4}[\theta - E_2(A_t, e_t)]$$

$$0 = f(h_t) - (c_t + A_t + m_t)$$

$$0 = b - \alpha(A_t, h_t, e_t)(1 - s_t - r_t)s_t$$

$$0 = \gamma(h_t)(1 - s_t - r_t)$$

$$0 = g(m_t) - [\delta + \sigma(h_t)(1 - s_t - r_t) - b]h_t$$

$$0 = E(A_t, e_t)$$

$$0 \leq s_t \leq 1$$

$$0 \leq r_t \leq 1$$