Optimal Intertemporal Curative Drug Expenses: The Case of Hepatitis C in France

Pierre Dubois, Thierry Magnac

WORKING PAPERS

N° 1402

January 2023

"Optimal Intertemporal Curative Drug Expenses: The Case of Hepatitis C in France"

Pierre DUBOIS and Thierry MAGNAC

Toulouse School of Economics

Optimal Intertemporal Curative Drug Expenses: The Case of Hepatitis C in France

Pierre Dubois* Thierry Magnac†

January 2023‡

Abstract

Abstract: We study intertemporal trade-offs that health authorities (HAs) face when considering the control of an epidemy using innovative curative medical treatments. We set up a dynamically controlled Susceptibles-Infected-Recovered (SIR) model for an epidemy in which patients can be asymptomatic, and we analyze in a simple model, the optimality conditions of the sequence of cure expenses decided by HAs at the onset of the drug innovation. We show that analytical conclusions are ambiguous because of their dependence on parameter values. As an application, we focus on the case-study of Hepatitis C whose treatment underwent a major upheaval when curative drugs were introduced in 2014. We calibrate our controlled SIR model using French data and simulate optimal policies. We show that the optimal policy entails some front loading of the intertemporal budget compared to fixed annual ones. The analysis demonstrates how beneficial the intertemporal budgeting can be compared to non forward looking constant budget allocation.

Keywords: pharmacy, SIR model, controlled epidemy dynamics, optimal intertemporal policies, hepatitis C.

JEL codes: I12, I18

*Toulouse School of Economics, University of Toulouse, pierre.dubois@tse-fr.eu

†Toulouse School of Economics, University of Toulouse, thierry.magnac@tse-fr.eu

‡The authors thank Eric Baseilhac, Philippe de Donder, Catarina Goulão and Juilian Reif for helpful comments as well as participants to the TSE Workshop on "Innovation, regulation and organization in healthcare", Paris, June 27, 2022. The authors gratefully acknowledge funding from the Agence Nationale de la Recherche under grant ANR-17-EURE-0010 (Investissements d'Avenir program) and from the TSE-P Health Center whose lists of sponsors can be found at https://www.tse-fr.eu/health. The usual disclaimer applies.

1 Introduction

The prices of drugs that are negotiated by healthcare authorities (HAs hereafter) typically depend on the expected demand for the treatment and the value it brings for the particular medical condition being treated. In a world where chronic diseases are being treated by regular treatments, the trade-offs faced by healthcare authorities do not change much over time. However, new and innovative treatments can change these trade-offs, especially when they have the potential to cure chronic conditions. This can present a challenge for healthcare authorities when it comes to setting budgets, as the cost of treating a large number of patients may be out of reach. This problem is likely to become more frequent in the future as gene and cell therapies can bring drastic innovations in medical treatment, and potentially provide a cure to otherwise incurable diseases with long term chronic treatments.

These potentially valuable innovations shed light on the difficulty faced by health authorities to optimally allocate their budget intertemporally when a large stock of patients becomes curable. Even absent credit market imperfections, the health authority problem of optimal intertemporal allocation of an initial budget depends on the decreasing efficiency of treatment with the number of patients to be treated but also on the rate of transmission and infection in the untreated population in the case of communicable diseases. While a myopic budget allocation decision seems suboptimal, value based pricing which justifies high prices for pharmaceuticals with life saving curative values can challenge the short term "affordability" of health care budgets (Danzon, 2018).

The question on how to allocate an intertemporal budget when innovative curative medical treatments become available against a communicable disease has not been addressed in the literature even if the smoothing of payments over time based on performance may be useful (Danzon, 2018, Brennan and Wilson, 2014). The literature on pharmaceutical pricing and spending concentrates on the role of price regulation and price setting (Lakdawalla, 2018) in terms of access and incentives for innovation. Little is known about the intertemporal allocation of curative drug treatments when treatments affect future needs.

In this paper, we set up a Susceptibles-Infected-Recovered (SIR) model for an epidemy, and we analyze in a simple set-up, the optimality conditions of the sequence of cure expenses decided by healthcare authorities when the curative drug treatment appears in the market. In most European countries, bargaining over drug prices between health authorities and pharmaceutical firms are

annual without long-run commitment. However, long-run optimal planning could generate benefits for all parties (see Alvarez, Argente and Lippi, 2021 or Assenza, al., 2020, for a recent application to Covid).

These gains admittedly depend on diseases and drugs characteristics. We focus in this paper on the case of a grave sickness, Hepatitis C, whose treatment underwent a major upheaval when decisive curative drugs were introduced in 2014. Hepatitis C is informative because the management of the introduction on the market of therapeutic innovations illustrates well the intertemporal trade-off between expending money for treating patients with new drugs in the present or waiting and treating them in the future. These new drugs however were quite expensive, and set off the question of the optimal policies to be chosen over time to master the epidemy in a cost efficient way. HAs usually manage the budget impact of treating the accumulated patient stock by prioritizing patients at high risk and delaying treatment of stable patients as was done in France (Dessauce et al., 2019). Mouterde et al. (2016) describes how France restricted access to the new drugs called Direct Acting Antiviral agents (DAAs) based on a selection of patients depending on virus genotypes, disease stages and comorbidities in spite of all these treatments obtaining a European Union marketing authorization regardless of the patient's profile. Berdud et al. (2018) show how the in-class competition for DAAs had a positive impact on uptake and adoption of DAAs in the top-5 European countries.

The SIR model we consider is standard although it allows for undetected and asymptomatic infected patients, a quite common occurrence with Hepatitis C. Furthermore, we assume that the transmission rate is low so that the long-run equilibrium is disease-free as was the case for Hepatitis C in France after the 2000s. The inheritance of a stock of infected in 2010 had built up from the uncontrolled usage of syringes before the 2000s among drug-addicts, from unsafe blood transfusion, and from any contact, among medical professions, between blood of infected and susceptibles. Those causes of infection were at least partly under control in 2010.

We further assume that the new drug policy cures the disease with decreasing returns to scale, that is, an additional euro per patient is less and less likely to be effective on the rate at which patients are cured. It has various justifications given either by biological or economic reasons that we develop in the text. We also assume that the function describing the impact of new drugs remain constant over time or at least, this is what is anticipated by the HAs. We indeed mainly focus on what HAs decide at the onset of the new drug introduction on the market.

Our first contribution is to derive analytical results that characterize optimal policies using the calculus of variations in the dynamic problem. We show that indeed moving backward expenses in new drugs holding constant the intertemporal budget of HAs, reduces infection in the short-run although there are rebound effects of the epidemy in the medium run. This rebound effect seems particularly important in the case in which there are many asymptomatic patients who could not be administered the new treatment since they remain undetected.

Our second contribution is to simulate optimal policies using parameters that are calibrated to the epidemiologic and economic characteristics of Hepatitis C in France. We confirm the conclusions we set out above about the short-run gains as well as the rebound effects. The latter effect questions the intertemporal credibility of giving to HAs an endowment they are free to expend in the short run if additional resources can be renegotiated in the medium run.

Section 2 sets up the model, states our assumptions and describes how we calibrate parameters in our policy simulations. Section 3 develops analytical results and show that most are indicative, and remain generally inconclusive. Section 4 characterizes optimal policies obtained by simulations of a dynamically controlled SIR model and the last section concludes.

2 The set up

We start with setting up a Susceptible-Infected-Recovered (SIR) model with linear incidence (Hethcote, 2000) that allows for the existence of asymptomatic or undetected infected persons as is the case for some patients with Hepatitis C. We then turn to the description of an exogenous innovation process of drugs curing this disease with some probability. We discuss our main assumption that expenses in the *current* period are less and less effective when the treatment is scaled up. We end up presenting our specification for welfare as well as the calibrated values for parameters that we retain from the literature.

2.1 The SIR model

In a population whose size is independent of time and normalized to 1, we denote s_t the share of susceptibles, i_t is the share of identified infected referred to *infected*, and u_t the share of undetected infected referred to *undetected*. We set up the model in discrete time and write the laws of motion of shares as follows.

First, susceptibles can get infected in a way proportional to the infected rate in the population

or can die and be replaced by newborns:

$$\Delta s_{t+1} = -\beta(i_t + u_t)s_t + \nu(1 - s_t). \tag{1}$$

in which β is the strength of infection due to both infected groups, assumed equal across those groups, and ν is the natural death rate in this population.

Infection is first undetected and thus the share of undetected infected behaves as:

$$\Delta u_{t+1} = \beta(i_t + u_t)s_t - (\zeta + \nu)u_t, \tag{2}$$

in which ζ is the rate at which the undetected infected are identified. The share of detected infected thus behaves as:

$$\Delta i_{t+1} = \zeta u_t - (\rho_t + \nu)i_t, \tag{3}$$

in which the healing rate $\rho_t \leq 1 - \nu$, is the channel through which the health authorities aim at controlling the spread of infection by the administration of available drugs. We define more thoroughly below the impact of policies. Moreover, we do not report the evolution of the share of recovered individuals which is obtained by deduction, i.e. $1 - s_t - u_t - i_t$.

As an approximation, the mortality rate, ν, is assumed to be the same in all sub-populations because there exists a standard drug that does not fully cure the disease although it maintains patients in life (as was the case with interferon-based treatments before the arrival of direct-acting antivirals for Hepatitis C). The state variables are not only the SIR variables (s_t, i_t, u_t), but also the endowment of public funds aimed at financing a policy of expenses that affect the healing rate ρ_t. Given a specification of social welfare, the issue of optimal control can then be set up as the choice of an optimal policy among any infinite sequence of expenses over time.

In the following, we will assume that the only stable stationary equilibrium, without any intervention, is the disease-free one, $s_t = 1$, $u_t = i_t = 0$, because this seems a reasonable assumption for the epidemy of Hepatitis C at least in western European countries, and even if in-migrations which are not modelled here might delay the process. A sufficient condition is that $\beta < \nu$, as shown in Appendix A following Hethcote (2000). In addition, the domain of variation of the state variables is the set: $s_t \geq \nu$, $u_t \geq 0$, $i_t \geq 0$ and $s_t + i_t + u_t \leq 1$.

2.2 Policies and intertemporal budgets

The policy implemented by health authorities (HAs) is described by the average expenses per (detected) infected patient at period t, b_t, and full expenses are denoted $B_t = b_t i_t$. As i_t is

observed, setting policies in terms of b_t or B_t is equivalent. In this section, we do not include in b_t the incompressible costs of traditional drugs used to support the infected that health authorities expend, and we assume that b_t refers to expenses of the new generation of effective drugs only. Traditional drug expenses just add a passive element to b_t e.g. $b_t + c_0$, and have no impact on our analysis until the empirical simulation of Section 4. We also describe those expenses and their relationship with the costs of the new drugs in the descriptive Section 2.4.

Those average expenses, b_t, affect the healing rate of the infected through the remission rate, $\rho_t = \rho(b_t)$ only. [1]

Overall, this function satisfies the following characterization:

Assumption D(ecreasing Returns to Scale)

1. $\rho(0) = \rho^{(0)}$, $\rho(+\infty) = 1 - \nu$
2. $\rho(.)$ is C^1 and increasing,
3. $\rho(.)$ is concave.

In Assumption D.1, the natural remission rate is denoted $\rho^{(0)}$ and defines the healing rate in the case health authorities do not use new drugs. The upper limit of their intervention is given by the fraction of non-deceased, $1 - \nu$. Assumption D.2 posits the existence of new and effective drugs and their continuous and regular impact. Assumption D.3 implies that for any fixed full expenses B_t but different shares of infected, $i_t^{(1)} < i_t^{(2)}$, the effectiveness of average expenses, $b_t^{(1)} = \frac{B_t}{i_t^{(1)}} > \frac{B_t}{i_t^{(2)}} = b_t^{(2)}$, is larger in the second case, $\rho'(b_t^{(1)}) < \rho'(b_t^{(2)})$.

This can be justified by medical and economic reasons. The first medical reason is coming from the heterogeneity of treatment effects. As different genotypes of the virus react differently to the new drugs (Berdud et al. 2018), spending more and more on average makes the treatment less and less effective. Second, better targeting of heterogenous patients makes the treatment more effective but organizational costs of administering the new drugs are likely to be convex in the number of infected, since some are more difficult to approach or convince than others. Mouterde et al. (2016) explain the organizational constraints involved by the treatment of sick individuals. These medical reasons can be argued to be stable over time at least at the first order since the infected population is continuously renewed as undetected patients are detected.

[1] The function $\rho()$ is assumed time independent. Taking into account the expected drug innovation process by competing drug producers is left for future research. All trade-offs over time are here summarized by the marginal impacts of moving expenses across time periods.

Among economic reasons, the presence of multiple drugs on the market produced by different firms whose prices are bargained over with the health authorities leads to such a decreasing return function of drug usage. We leave for future work how this mechanism precisely works.

The intertemporal budget is given by a sequence of budgets $(B_1, ., B_t, ...)$ such that their sum discounted by the interest rate, r, is

$$\sum_{t=1}^{\infty} \frac{B_t}{(1+r)^t} = \sum_{t=1}^{\infty} \frac{b_t i_t}{(1+r)^t} \equiv A_1, \tag{4}$$

the total endowment received by the health authorities in the first period, and affected to drug expenses.[2] We do not take a stance on how A_1 is decided by the political authorities, and health authorities take it as a given. What interests us is the choice by the health authorities among *different* sequences of expenses e.g. between front-loaded, constant or back-loaded sequences among many others, which all have the same discounted total value equal to A_1.

2.3 Social welfare and optimal policy

In order to select a policy optimally, we have to evaluate whether a sequence $(b_1, ., b_t, ...)$ is *better* in some sense than another sequence $(\tilde{b}_1, ., \tilde{b}_t, ...)$ when $\sum_{t=1}^{\infty} \frac{b_t i_t}{(1+r)^t} = \sum_{t=1}^{\infty} \frac{\tilde{b}_t i_t}{(1+r)^t} = A_1$. This evaluation is derived taking as given a social welfare function, W, that depends on state variables (A_1, s_1, i_1, u_1) in the first period as well as on the sequence of a given policy followed over time, $b = (b_1, ., b_t, ...)$. We assume that social welfare, W, is additively separable over time in each period instantaneous welfare, $v(.)$, which depends only on the end-of-the-period share of infected, i_{t+1}:

$$W(A_t, s_t, i_t, u_t; b) = \sum_{\tau=t}^{\infty} \delta^{\tau-t} v(i_{\tau+1}). \tag{5}$$

In this expression, δ is the discount rate used by health authorities. We normalize $v(0) = 0$ and assume that $v(x)$ is decreasing and concave for $x \geq 0$. It expresses that HAs dislike infection and the more so, the higher it is, and the simplest example of such a specification is a quadratic function, $v(i_{t+1}) = -(i_{t+1})^2/2$.

The optimal policy is then obtained by solving the intertemporal program:

$$\begin{aligned} W^*(A_1, s_1, i_1, u_1) &= \max_b W(A_1, s_1, i_1, u_1; b) = \max_b \sum_{\tau=1}^{\infty} \delta^{\tau-1} v(i_{\tau+1}), \\ \text{subject to } \rho_t &= \rho(b_t),\ (1),\ (2),\ (3) \\ \text{and } A_{t+1} &= (1+r)A_t - b_t i_t, \text{ for all } t. \end{aligned}$$

[2] Let us mention again that traditional drugs supporting the infected are not yet counted in those expenses.

Because all objects are stationary, this is equivalent by the Bellman principle to solving:

$$\begin{aligned} W^*(A_1, s_1, i_1, u_1) &= \max_{b_1}(v(i_2) + \delta W^*(A_2, s_2, i_2, u_2)) \\ \text{subject to } \rho_1 &= \rho(b_1),\ (1),\ (2),\ (3) \text{ at time } t = 1, \\ \text{and } A_2 &= (1+r)A_1 - b_1 i_1. \end{aligned} \tag{6}$$

In this formulation, notice that all parameters are known by HAs and that for instance, the rate of undetected u_t is supposed to have been learned in the past.

Alternatives to this setting could first entertain the idea that HAs also care about the undetected, and not only the infected who were detected although it is not clear why the undetected, if affected by the disease, would not seek medical advice and be detected. The HAs however could internalize the fact that the undetected are going to induce more infections in the future. Second, it could be more realistic to assume that the infection rate β evolves over time in a probabilistic way, agents becoming aware of the danger of the disease, and thirdly that u_t is unknown with some prior distribution. We leave these alternatives for further research.

2.4 Descriptive statistics and calibrated parameters

We gathered various statistics from different sources that allow us to calibrate the values of parameters of interest using data from France. We start with parameters related to the epidemiological model, and next turn to the calibration of the efficiency of the new drugs.

Parameters of the SIR model A recent review of characteristics of the infection in Europe and the world can be found in Roudot-Thoraval (2021). Figures we extract from this paper are related to the French population aged 18-75 or 18-80. In 2004, the prevalence of anti-HCV antibodies was estimated at 0.53% with a confidence interval of [0.40-0.70] (Meffre et al., 2010) but the sero-prevalence was 0.84% (Roudot-Thoraval, 2021, p5). According to Roudot-Thoraval (2021), the respective estimates for 1994 are 1.1% for the anti-HCV prevalence and the sero-prevalence was 0.86%.[3] The HCV antibodies prevalence had decreased in 2011 to 0.42% with a 95% confidence interval of [0,33-0,53] and in 2016, was estimated at 0.30% with a confidence interval of [0.13-0.70] (Brouard et al., 2019). The decrease after 2004 was brought about by a much better control of the blood transfusion channel while transmissions through intravenous drug use and mother-to-infant

[3] A survey by Bruggmann et al. (2004) roughly reports the same numbers.

remained important (Roudot-Thoraval, 2021) while the decrease after 2014 was caused by the adoption of direct acting antiviral (DAA) drugs.

This is why our main scenario retains pre-2014 initial values of $u_0 + i_0 = 0.8\%$, and $s_0 = 98.8\%$ so that the recovered rate is 0.4% of the population. Furthermore, in Brouard et al., (2019), it was estimated that the share of people knowing their current infection was equal to 80.6% although with a large confidence interval [44.2-95.6]. In other surveys, this figure can be much lower (57% on Bruggman et al., 2014) and as low as 50% (Bottero et al., 2016). This is why we calibrate $\zeta = 0.03$ in order to obtain a ratio of $i_0/(u_0 + i_0)$ equal to roughly 60% and thus choose to have $u_0 = 0.3\%$ and $i_0 = 0.5\%$.

The incidence rate, and the strength of the infection β, are more difficult to nail down. As the prevalence is decreasing over the years before the introduction of the new drugs, we assume that the SIR model that we consider has a single stable equilibrium which is disease-free. It is shown in the Appendix, adapting Hethcote (2000) to our specific SIR model, that the condition on parameters is that $\beta < \nu$. Given that the reference population is above 19 years old and assuming that at 19, life expectancy is 60, it gives to ν a value approximately equal to $1/60 = 0.017$. If we assume that the newly infected are observed, the incidence rate in Western Europe is estimated in Hill et al. (2017, Table 1) to be equal to $\beta \simeq 35,440/2,364,430 = 0.015$ in agreement with the decrease between 1994 and 2011.[4] The fact that this is a combination of two transitions, from susceptibles to undetected and then to infected is not important in the absence of treatment of the infected since we can aggregate the two states in this case.

As for interest rates, we adopt the average value of long-run rates in 2014 which was around 2%[5].

Parameters of the efficiency of new drugs We calibrate parameters governing function $\rho(b)$ as follows:

$$\rho(b) = \rho^{(0)} + (1 - \nu - \rho^{(0)}) \left[1 - \exp(-\lambda b^{\alpha})\right], \tag{7}$$

which satisfies Assumption D when $\alpha \leq 1$, because it is concave, $\rho(0) = \rho^{(0)}$ and $\rho(\infty) = 1 - \nu$ but parameters λ and α should be calibrated.

We first retain a value for the natural remission rate of $\rho^{(0)} = 0.03$, slightly less than what

[4] This also agrees with rough figures gathered on French websites of 5,000 newly infected for a stock of 350,000 infectives.

[5] See https://data.oecd.org/fr/interest/taux-d-interet-a-long-terme.htm#indicator-chart.

Roudot-Thoraval (2021) reports for severe developments of the disease.[6] In this paper, the share of the treated by new drugs is roughly estimated to constitute 7% of the infected in 2014 and 19% in 2017 which means that strong restrictions are in place.

In the simulation exercises below, we also consider expenses related to traditional interferon treatments. We here start by assessing the costs of the new drugs (Direct-Acting Antivirals, DAAs) vs. the traditional ones. The share of infected that are not treated by new drugs are assumed to receive the interferon-based treatments that allow to maintain patients with chronic Hepatitis C alive. Denote c_{old} the per period cost of the latter treatment and $c_{new}(\rho_t)$ the cost of the innovative one, which is increasing and convex in ρ_t as assumed in Section 2.2. The full treatment per infected is the sum of the costs $c_{new}(\rho_t)$ for the treated with the new drug, $(\rho_t - \rho^{(0)})$, and of the costs, c_{old}, of the treated with the old one, $(1-\nu-\rho_t)$:

$$c_{new}(\rho_t)(\rho_t - \rho^{(0)}) + (1-\nu-\rho_t)c_{old} = (c_{new}(\rho_t) - c_{old})(\rho_t - \rho^{(0)}) + (1-\nu-\rho^{(0)})c_{old}.$$

In our model, the budget per infected patient treated with the new drug, b_t, is the additional cost over $(1-\nu-\rho^{(0)})c_{old}$, that is

$$b(\rho_t) = (c_{new}(\rho_t) - c_{old})(\rho_t - \rho^{(0)}).$$

Note that the traditional treatment, whose costs are $(1-\nu-\rho^{(0)})c_{old}$, does not affect the remission rate ρ_t.

Inverting equation (7) should match the last equation so that we obtain:

$$b(\rho_t) = \left[-\frac{1}{\lambda}\log(1 - \frac{\rho_t - \rho^{(0)}}{1-\nu-\rho^{(0)}})\right]^{1/\alpha} = (c_{new}(\rho_t) - c_{old})(\rho_t - \rho^{(0)}), \tag{8}$$

which delivers function $c_{new}(\rho_t)$:

$$c_{new}(\rho_t) - c_{old} = \frac{\left[-\frac{1}{\lambda}\log(1 - \frac{\rho_t - \rho^{(0)}}{1-\nu-\rho^{(0)}})\right]^{1/\alpha}}{\rho_t - \rho^{(0)}}. \tag{9}$$

We now calibrate $\left(c_{new}\left(\rho^{(0)}\right) - c_{old}\right)$ which leads to the calibration of λ given α. The ratio of differential cost and outcome in terms of quality of life which evaluated by health authorities writes $\frac{c_{new}-c_{old}}{QALY_{new}-QALY_{old}}$ (called in French, RDCR - "ratio différentiel coût-résultat"). In our model a cured patient is like a non-infected person, and thus enjoys a value of one QALY, while a sick

[6] When experimenting different values with simulation, we found that larger values are driving the epidemy more quickly to zero than what seems to be.

patient has a QALY evaluated at around 0.5 on average (HAS 2014, Avis d'Efficience Sovaldi (Sofosbuvir)) implying that $QALY_{new} - QALY_{old} = 0.5$. The ratio RDCR is around 20 000 euros per QALY for Sofosbuvir, the cheapest for most genotypes. This leads to an average differential cost of treatment of $c_{new}(\rho^{(0)}) - c_{old} = 20000 \times 0.5 = 10\ 000$ euros. By equation (9), we can derive when $\rho_t \to \rho^{(0)}$ and using that $\log(1-x) \sim -x$ when x is small:

$$(c_{new}(\rho^{(0)}) - c_{old})^{\alpha} = \frac{1}{\lambda} \frac{1}{1-\nu-\rho^{(0)}} (\rho_t - \rho^{(0)})^{1-\alpha},$$

that leads to:

$$\frac{1}{\lambda} = (1-\nu-\rho^{(0)}) * \frac{10000^{\alpha}}{(0.001)^{1-\alpha}},$$

using the previous calibrated parameters, and a small level of treatment $\rho_t - \rho^{(0)} = 0.001$.

Another important budget parameter is the cost of the old treatment. Indeed $b_t + \left(1-\nu-\rho^{(0)}\right) c_{old}$ is the average budget per infected that allows to cure $\rho(b_t)$ patients while treating other infected patients with the old treatment. According to Bronowicki et al. (2003) page 204, we can approximate c_{old} with 9,000 €. Given the other calibrated parameters of the epidemic, the budgetary discounted cost of the traditional treatment is slightly less than 1,000 € per inhabitant. The initial endowment that we consider is set to around 5,000 € and we shall assess the sensitivity of our results to some of those budget parameters.

3 Analytical results

Before turning to simulations that allow to go deeper in the understanding of the principles underlying the setting of optimal policies, we first provide some analytical results based on variational calculus. We first disentangle the effect of varying the sequence of budgets on the shares of susceptibles and infected. We then turn to the effects on welfare.

3.1 Controlling the infection

Fix a benchmark policy b and consider an alternative which is in the neighborhood of b, and such that the budget constraint (4) is satisfied. We investigate the consequences of a change of policy b into the alternative policy in terms of susceptibles, s_t, and infected, u_t and i_t. Formally, let $B_t = b_t i_t$ (resp. $B_t + dB$) and B_{t+1} (resp. $B_{t+1} - (1+r)dB$) implying the same intertemporal budget

$$B_t + \frac{B_{t+1}}{1+r} = B_t + dB + \frac{B_{t+1} - (1+r)dB}{1+r},$$

and consider the sequences $(B_1, ..., B_{t-1}, B_t, B_{t+1}, B_{t+2}...)$ and $(B_1, ..., B_{t-1}, B_t + dB, B_{t+1} - (1 + r)dB, B_{t+2}, ...)$ which are intertemporally equivalent in terms of endowments.

To study the differential effect of the two sequences we fix state (s_t, u_t, i_t). Histories diverge afterwards; and we denote dX the variation in a variable X with respect to the benchmark given by policy b holding fixed (A_t, s_t, u_t, i_t). Specifically, in terms of expenses per patient b_t we can write:

$$\begin{cases} dB_t = dB = & db_t i_t, \\ dB_{t+1} = -(1+r)dB = & db_{t+1} i_{t+1} + b_{t+1} di_{t+1}, \\ \text{For } k > 1,\ dB_{t+k} = 0 = & db_{t+k} i_{t+k} + b_{t+k} di_{t+k}. \end{cases}$$

so that:

$$\begin{cases} dB_t = dB = & db_t i_t, \\ db_{t+1} i_{t+1} = & -(1+r)dB - b_{t+1} di_{t+1}, \\ db_{t+k} i_{t+k} = & -b_{t+k} di_{t+k}. \end{cases} \tag{10}$$

We now look at the effect that this change has on infected in the short-run at $t+1$ and $t+2$ and next turn to the effects on other subpopulations at later periods.

The short-run effects Without loss of generality, suppose $dB > 0$. At period $t+1$, we have by equation (3) :

$$\begin{aligned} di_{t+1} &= -(d\rho_t) i_t, \\ &= -\rho_t' i_t db_t = -\rho_t' dB < 0. \end{aligned} \tag{11}$$

The share of infected decreases at period t when the budget is larger. Moreover, by equations (1) and (2), susceptibles and undetected are not affected, $du_{t+1} = ds_{t+1} = 0$ and therefore $u_{t+1} = u_t$ and $s_{t+1} = s_t$.

At period $t+2$, things are different. Differentiating exactly equation (3) gives:

$$di_{t+2} - di_{t+1} = -(d\rho_{t+1}) i_{t+1} - (\rho_{t+1} + \nu) di_{t+1},$$

which implies

$$\begin{aligned} di_{t+2} &= -\rho_{t+1}' db_{t+1} i_{t+1} + (1 - \rho_{t+1} - \nu) di_{t+1} \\ &= \rho_{t+1}' ((1+r)dB + b_{t+1} di_{t+1}) + (1 - \rho_{t+1} - \nu) di_{t+1} \\ &= (\rho_{t+1}'(1+r) - (b_{t+1}\rho_{t+1}' + 1 - \rho_{t+1} - \nu)\rho_t') dB \\ &= \left[(\rho_{t+1}' - \rho_t')(1+r) + (r + \nu + \rho_{t+1} - b_{t+1}\rho_{t+1}')\rho_t' \right] dB. \end{aligned} \tag{12}$$

using equations (10) and (11) between lines 1, 2 and 3.

Equation (12) delivers a clear interpretation. First, if $b_t \simeq b_{t+1}$, implying $\rho'_{t+1} \simeq \rho'_t$, we can show that there is a rebound effect of the share of infected at $t+2$:

$$di_{t+2} \simeq (r + \nu + \rho_{t+1} - b_{t+1}\rho'_{t+1})\rho'_t dB > 0,$$

and the infection is strengthened by moving expenses from the future to the present when expenses at the two periods are roughly equal. This has three sources. First, a positive interest rate makes expenses larger if earlier. Second, a later higher mortality affects the efficiency of the cure. The third term, $\rho_{t+1} - b_{t+1}\rho'_{t+1} > 0$ because $\rho(.)$ is concave and the larger the budget, the less efficient the cure.

This positive rebound effect is also true if $b_t > b_{t+1}$. This is not necessarily the case, however, if $b_t < b_{t+1}$ because $\rho'_{t+1} < \rho'_t$ and the rebound effect, di_{t+2}, could be negative. It is only in this case that moving expenses to the present is always favorable since a lower infected rate at $t+2$ is reinforced by the dynamic effects going through the infection of susceptibles.

Before looking at these dynamic effects, it is interesting to look into the impact under a different angle that makes the share of infected return to the benchmark path, $di_{t+2} = 0$, notwithstanding further impacts at $t+3$, and beyond. Considering equation (12), we get:

$$di_{t+2} = \rho'_{t+1}(1+r) - (1 - \nu - (\rho_{t+1} - b_{t+1}\rho'_{t+1}))\rho'_t, \tag{13}$$

thus

$$di_{t+2} = 0 \Longrightarrow \rho'_t = \rho'_{t+1}\frac{1+r}{1 - \nu - (\rho_{t+1} - b_{t+1}\rho'_{t+1})}. \tag{14}$$

By Assumption D, $\rho_{t+1} \leq 1 - \nu$, $\rho_{t+1} - b_{t+1}\rho'_{t+1}$ is positive and increasing from $\rho^{(0)}$ to a limit strictly lower than $1-\nu$, and the denominator is positive and lower than $1 - \nu - \rho^{(0)}$. A budgeting rule that would make $di_{t+2} = 0$ – so that infection returns to its previous level in period $t+2$ – sets ρ'_t to a larger value than ρ'_{t+1}, and therefore a b_t lower than b_{t+1}. Nonetheless, this rule neglects the dynamic returns to the change of policy, and further values di_{t+h} for $h > 2$ are remaining constant.

The infection channel The infection channel through infections delivers more complex dynamics. Equation (1) yields that the population is less infected because:

$$ds_{t+2} = -\beta(di_{t+1})s_t = \beta s_t \rho'_t dB > 0,$$

since $ds_{t+1} = 0$ as seen above. This also affects u_{t+2} by the opposite amount:

$$du_{t+2} = \beta(di_{t+1})s_t = -\beta s_t \rho'_t dB < 0.$$

Through this channel, the shock propagates further down. Indeed, we how have:

$$di_{t+3} - di_{t+2} = \zeta du_{t+2} - (\rho_{t+2} + \nu)di_{t+2} - d\rho_{t+2} i_{t+2}.$$

Recomposing, using the equations above, and line 3 in equation (10) yields:

$$\begin{aligned} di_{t+3} &= \zeta du_{t+2} + (1 - \rho_{t+2} - \nu)di_{t+2} + \rho'_{t+2} b_{t+2} di_{t+2}, \\ &= -\zeta\beta s_t \rho'_t dB + (1 - \rho_{t+2} - \nu + \rho'_{t+2} b_{t+2})di_{t+2}. \end{aligned}$$

Note that the first term is negative while the second term is positive if $di_{t+2} > 0$ under the conditions stated above and in particular if $\rho'_{t+1} \simeq \rho'_t$. If we set $di_{t+2} = 0$, as at the end of the previous development leading to equation (14) then there is an additional decrease of di_{t+3} because of the dynamic term in dB that affects u_{t+2}.

Nonetheless if we proceed in a similar way, and instead of setting di_{t+2} to zero, we set $di_{t+3} = 0$ to return to the original path at $t + 3$, we get:

$$di_{t+2} = \frac{\zeta\beta s_t \rho'_t}{1 - \nu - (\rho_{t+2} - \rho'_{t+2} b_{t+2})} dB > 0,$$

so that using equation (13):

$$(\rho'_{t+1}(1 + r) - (1 - \nu - (\rho_{t+1} - b_{t+1}\rho'_{t+1}))\rho'_t) = \frac{\zeta\beta s_t \rho'_t}{1 - \nu - (\rho_{t+2} - \rho'_{t+2} b_{t+2})}. \tag{15}$$

Denote

$$a(\rho_{t+k}) = 1 - \nu - (\rho_{t+k} - b_{t+k}\rho'_{t+k}) \in (0, 1 - \nu - \rho^{(0)}), \tag{16}$$

where b_{t+k} is such that $\rho_{t+k} = \rho(b_{t+k})$ and note that $a(\rho_{t+k})$ is decreasing with ρ_{t+k} because of the concavity of $\rho(.)$ and $a(0) = 1 - \nu - \rho^{(0)}$ and $a(\infty) \geq 0$.

Rewrite equation (15) as:

$$\rho'_{t+1}(1 + r) - a(\rho_{t+1})\rho'_t = \frac{\zeta\beta s_t \rho'_t}{a(\rho_{t+2})},$$

so that:

$$\rho'_t = \rho'_{t+1} \frac{1 + r}{a(\rho_{t+1}) + \frac{\zeta\beta s_t}{a(\rho_{t+2})}}.$$

Note in this case, the effect that we found at the end of the previous subsection is attenuated, depending on the value of b_{t+2} and thus ρ_{t+2}. It can even be the case that $a(\rho_{t+1}) + \frac{\zeta\beta s_t}{a(\rho_{t+2})} > 1 + r$ so that the recommendation at the end of the previous section, and derived from incomplete premises, that the optimal schedule of b_t is increasing with time can be no longer true.

Further dynamics We can continue down the line by writing:

$$ds_{t+3} = -\beta(di_{t+2} + du_{t+2})s_{t+2} - \beta(i_{t+2} + u_{t+2})ds_{t+2} - \nu ds_{t+2},$$

and this also affects u_{t+3}:

$$du_{t+3} = -\beta(di_{t+2} + du_{t+2})s_{t+2} - \beta(i_{t+2} + u_{t+2})ds_{t+2} - \zeta du_{t+2}.$$

In consequence,

$$\begin{aligned} di_{t+4} - di_{t+3} &= \zeta du_{t+3} - (\rho_{t+3} + \nu)di_{t+3} - d\rho_{t+3} i_{t+3}, \\ &= \zeta du_{t+3} - (\rho_{t+3} + \nu)di_{t+3} + \rho'_{t+3} b_{t+3} di_{t+3}, \end{aligned}$$

or:

$$\begin{aligned} di_{t+4} &= \zeta du_{t+3} - (\rho_{t+3} + \nu)di_{t+3} - d\rho_{t+3} i_{t+3}, \\ &= \zeta du_{t+3} + (1 - \nu - \rho_{t+3} + \rho'_{t+3} b_{t+3})di_{t+3}, \\ &= \zeta du_{t+3} + a(\rho_{t+3})di_{t+3}. \end{aligned}$$

and we can proceed as before. Thus, the structure of the optimal expenses over time depends on the parameters of the model in a very complex way, and we should build up realistic simulations to go further.

3.2 Welfare evaluation

Turning to welfare, as defined in equation (5), its variation is given by:

$$dW = \sum\nolimits_{\tau=t}^{\infty} \delta^{\tau-t} v'(i_{\tau+1}) di_{\tau+1}.$$

Given results in Section 3.1 and equation (16) defining function $a(.)$, we have:

$$\begin{aligned} di_{t+1} &= -\rho'_t dB, \\ di_{t+2} &= (\rho'_{t+1}(1+r) - a(\rho_{t+1})\rho'_t)dB, \\ di_{t+3} &= -\zeta\beta s_t \rho'_t dB + a(\rho_{t+2})di_{t+2}, \\ di_{t+4} &= \zeta du_{t+3} + a(\rho_{t+3})di_{t+3}, . \\ &... \end{aligned}$$

Even if dB is infinitesimal and rates (r, ζ, ν) are small, the attenuation of effects over time is slow if $a(\rho_{t+k})$ is not small.

Short run welfare gains In the following, we assume that $\delta = 1/(1+r)$ so that the interest rate reflects preferences for the present of health authorities.

Consider the impact in the short-run as defined by:

$$\begin{aligned} dW_{t,t+1} &= \sum_{\tau=t}^{t+1} \delta^{\tau-t} v'(i_{\tau+1}) di_{\tau+1}, \\ &= (-\rho'_t v'(i_{t+1}) + v'(i_{t+2})(\rho'_{t+1} - \delta a(\rho_{t+1})\rho'_t))dB. \end{aligned}$$

Assuming that the instantaneous utility is $v(i_{t+1}) = -\frac{(i_{t+1})^2}{2}$, we have $v'(i_{t+1}) = -i_{t+1}$ and:

$$\begin{aligned} dW_{t,t+1} &= (\rho'_t i_{t+1} - i_{t+2}(\rho'_{t+1} - \delta a(\rho_{t+1})\rho'_t))dB, \\ &= \left[\rho'_t(i_{t+1} - i_{t+2}) + i_{t+2}(\rho'_t - \rho'_{t+1}) + \delta i_{t+2} a(\rho_{t+1})\rho'_t\right] dB. \end{aligned}$$

We can distinguish different cases according to conditions $i_{t+1} \gtrless i_{t+2}$ and $\rho'_t \gtrless \rho'_{t+1}$ (e.g. $b_{t+1} \gtrless b_t$).

- If $i_{t+1} = i_{t+2}$: the observed infection is stable
  - if $b_t = b_{t+1}$ and thus $\rho'_t = \rho'_{t+1}$: there is a gain in short-term welfare if $dB > 0$ i.e. if we reallocate budget from the future to the present. This gain is due to the term $\delta a(\rho_{t+1})\rho'_t$ and is decreasing with the value of ρ_{t+1} since $a(\rho_{t+1})$ and ρ'_t are both decreasing.
  - $b_t > b_{t+1}$ and thus $\rho'_t < \rho'_{t+1}$: the previous gain is attenuated and disappears eventually
  - $b_t < b_{t+1}$ and thus $\rho'_t > \rho'_{t+1}$: the previous gain is amplified

  In conclusion, in this case, a solution $b_t > b_{t+1}$ is optimal in the short-run.

- If $i_{t+1} > i_{t+2}$: the observed infection is in a decreasing swing
  - $b_t = b_{t+1}$ and thus $\rho'_t = \rho'_{t+1}$: there is a gain in welfare if $dB > 0$ i.e. we reallocate budget from the future to the present. This gain is due to two terms $\rho'_t(i_{t+1} - i_{t+2})$ and $\delta a(\rho_{t+1})\rho'_t$.
  - $b_t > b_{t+1}$ and thus $\rho'_t < \rho'_{t+1}$: the previous gain is attenuated and disappears eventually
  - $b_t < b_{t+1}$ and thus $\rho'_t > \rho'_{t+1}$: the previous gain is amplified

  Here also, a solution $b_t > b_{t+1}$ is optimal in the short-run.

- If $i_{t+1} < i_{t+2}$: the observed infection is in an increasing swing

- $b_t = b_{t+1}$ and thus $\rho'_t = \rho'_{t+1}$: there is a gain in welfare if $dB > 0$ depending on the two terms $\rho'_t(i_{t+1} - i_{t+2})$ and $\delta a(\rho_{t+1})\rho'_t$.

It is the only case in which a solution $b_t > b_{t+1}$ might not be optimal in the short-run.

Medium-term welfare gains Consider the impact in the medium-run as defined by:

$$\begin{aligned} dW_{t,t+2} &= \sum_{\tau=t}^{t+2} \delta^{\tau-t} v'(i_{\tau+1}) di_{\tau+1}, \\ &= (-\rho'_t v'(i_{t+1}) + v'(i_{t+2})(\rho'_{t+1} - \delta a(\rho_{t+1})\rho'_t))dB, \\ &\quad + v'(i_{t+3})(-\zeta\beta s_t \rho'_t dB + a(\rho_{t+2}) di_{t+2}), \\ &= (-\rho'_t v'(i_{t+1}) + (v'(i_{t+2}) + v'(i_{t+3})a(\rho_{t+2}))(\rho'_{t+1} - \delta a(\rho_{t+1})\rho'_t))dB, \\ &\quad - v'(i_{t+3})\zeta\beta s_t \rho'_t dB. \end{aligned}$$

With $v'(i_{t+1}) = -i_{t+1}$ we get:

$$\begin{aligned} dW_{t,t+2} &= (\rho'_t i_{t+1} - (i_{t+2} + i_{t+3}a(\rho_{t+2}))(\rho'_{t+1} - \delta a(\rho_{t+1})\rho'_t))dB, \\ &\quad + i_{t+3}\zeta\beta s_t \rho'_t dB. \end{aligned}$$

The last term contributes as a positive effect due to the retroaction of a decrease in infection among susceptibles. There is however also a negative effect due to the decrease in treatment at period $t+1$ and contributing through the term $i_{t+3}a(\rho_{t+2})$. Overall, it seems intractable to assess the relative magnitude of these effects and we now turn to the formal description of optimal policies.

3.3 Optimal policies: Formal Characterization

We now characterize the conditions under which the sequence of expenses is optimal. In other words, we provide the first order condition derived from the Bellman equation that is necessary for optimality.

As described by equation (6), HAs maximize social welfare with respect to a sequence of expenses $b^{(t)}$:

$$\sum_{\tau=t}^{\infty} \delta^{\tau-t} v(i_{\tau+1}),$$

under the constraints of a SIR model, equations (1), (2) and (3) and a budget constraint:

$$A_{t+1} = (1+r)A_t - b_t i_t - c_{old}(1 - \nu - \rho^{(0)})i_t,$$

in which $c_{old}(1-\nu-\rho^{(0)})$ are the incompressible drug expenses. For simplicity, we assume that $\delta = \frac{1}{1+r}$ and we denote $x_t = (A_t, s_t, u_t, i_t)$ the vector of state variables and $x_{t+1} = F_t(x_t)$ their law of motion.

The Bellman equation writes as:

$$\begin{aligned} W_t(x_t) &= \max_{b_t} \{v(i_{t+1}) + \delta W_{t+1}(x_{t+1})\} \\ \text{s.t. } x_{t+1} &= F_t(x_t). \end{aligned} \tag{17}$$

If $W_{t+1}(x_{t+1})$ is a concave function in A_t, the necessary and sufficient first order condition of this program writes:

$$v'(i_{t+1})\frac{di_{t+1}}{db_t} + \delta \nabla W_{t+1}\frac{dx_{t+1}}{db_t} = 0,$$

as a function of the gradient $\nabla W_{t+1} = (\frac{\partial W_{t+1}}{\partial A_{t+1}}, \frac{\partial W_{t+1}}{\partial s_{t+1}}, ..)$ yielding:

$$-v'(i_{t+1})\rho'_t i_t + \delta\left[\frac{\partial W_{t+1}}{\partial A_{t+1}}(-i_t) + \frac{\partial W_{t+1}}{\partial i_{t+1}}(-\rho'_t i_t)\right] = 0,$$

since $\frac{ds_{t+1}}{db_t} = \frac{du_{t+1}}{db_t} = 0$. The first order condition thus writes:

$$-v'(i_{t+1})\rho'_t = \delta\left[\frac{\partial W_{t+1}}{\partial A_{t+1}} + \frac{\partial W_{t+1}}{\partial i_{t+1}}\rho'_t\right], \tag{18}$$

in which the LHS is positive. Assuming that $v'(i_{t+1}) = -i_{t+1}$ and rearranging yields:

$$(i_{t+1} - \delta\frac{\partial W_{t+1}}{\partial i_{t+1}})\rho'_t = \delta\frac{\partial W_{t+1}}{\partial A_{t+1}}.$$

This expresses that the sum of the marginal benefits of investing one additional euro per patient, in the current period, $i_{t+1}\rho'_t$, and in the future periods, $-\delta\frac{\partial W_{t+1}}{\partial i_{t+1}}\rho'_t$ (positive since $\frac{\partial W_{t+1}}{\partial i_{t+1}} < 0$) is equal to the marginal value of an additional euro next period.

Unfortunately, this equation, as well as the backward induction equations relating future and current marginal values of the state variables, do not lead to a tractable characterization through an Euler condition relating current and next period observables. The Euler equation involves four periods of observations and we let these developments for Appendix C. Furthermore, the second order condition is more involved and we will assume that it holds to ensure that the previous program has a unique solution. Further developments are relegated to Appendix C.

4 Counterfactual Empirical Analysis: Simulations

Because it is difficult to draw unambiguous formal conclusions from the dynamic model that we analyzed above despite its simplicity, we proceed now by simulation. We first capture the

diffusion of the epidemy by simulating the SIR model forward using the calibrated parameters that we presented in Section 2.4. In subsection 4.1, we describe the impact of a policy of expenses and illustrate the principle of variational calculus that underpinned Section 3.1. We then turn in subsection 4.2 to the simulation of the value function and the optimal policy.

4.1 Forward simulation: Controlling the epidemy

In those simulations, we mimic what has happened in 2014 for hepatitis C when a new generation of drugs, that was curing some patients for good, was introduced, while previous drugs were not leading to a significant remission rate before 2014.

Over a time span of 20 years, we simulate a trajectory such that a traditional drug (based on interferon based treatments), is used until $t = 5$, when a new drug family (DAAs) is introduced. Parameters are calibrated as presented in Section 2.4 and the original profile of the budget $(B_8, ., B_t, .)$ is increasing at rate r. Results are presented in Figure 1. The infection (observed and unobserved) is decreasing slowly over the time span. The policy intervention at $t = 5$ strongly affects the share of infected. Nonetheless, the evolution of undetected, and more generally non-susceptibles, is more muted because the impact is only indirect. This means that the channel of infection through undetected remains widely open.

We next analyze the effect of a change in the budget between time $t + 1$ and time t, when $t = 8$, keeping the total budget constant to illustrate the analytical results developed in Section 3.1. In this example presented in Figure 2, the reallocation is full so that the budget at time $t + 1$ is set to 0, and feeds in the budget at time t at the appropriate discounted value. In other words, the discounted value of the intertemporal budget remains constant. The impact at time t on the share of infected is strong and negative as expected while the rebound effect upwards at time $t + 1$ is also marked. Infection afterwards starts to slowly decrease, and asymptotically gets almost back to the value given by the initial budget sequence. The impact on the share of the undetected is similar with the difference that the decrease towards the asymptote is much slower than the infected. Overall, the effect on the share of non-susceptibles is as expected the exact opposite of the two previous impulse function responses.

The conclusion of this analysis is that front loading expenses is improving a lot the infection rate in the short run although a rebound effect exists in the medium run. This analysis remains partial since we are looking at changes in policies that are not what an optimal planner would

have chosen. To extend this variational analysis, we use simulations of the optimal policy and compare it to other policies.

4.2 Value functions and optimal policies

We now turn to the simulation of value functions and the derivation of optimal policies. We start by explaining how the simulation proceeds and then describe results of these simulations. Note that we now take into account the cost of curing patients who do not get access to the new drugs (DAAs) using traditional drugs. We assume that the cost per patient of this cure is constant and equal to $c_{old} = 9000$ € (see Section 2.4 for calibration details). The accumulation rule for the budget is now written as:

$$A_{t+1} = (1 + r)A_t - (b_t + c_{old})i_t. \tag{19}$$

Nonetheless, this modification to the decision program is not enough because of constraints $A_t \geq 0$ for all t since provisions should be made for expenses on traditional drugs after period t. Since the new drug cure is effective, it is enough to compute the provisions assuming that no expenses on new drugs are made after period t and that only the cost of traditional drugs matter. Denote those provisions:

$$\kappa_t(z_t) = c_{old} \sum\nolimits_{n=t}^{\infty} \frac{i_n}{(1 + r)^{n-t+1}}, \tag{20}$$

in which $z_t = (s_t, u_t, i_t)$, and replace the constraint $A_t \geq 0$ by the constraint $A_t \geq \kappa_t(z_t)$ for all t in the decision program.

Because the environment is asymptotically stationary, we adopt the strategy of iterating over the value function until it converges to the unique fixed point of the dynamic Bellman operator. Imposing that HAs prefer the present to the future e.g. $\delta(1 + r) < 1$ is a sufficient condition for the existence and uniqueness of the fixed point as the Bellman equation is a contraction.[7] The algorithm used is detailed in appendix B.

4.2.1 Results

Using those simulations, we compare three budgetary policies:

- the (very) conservative policy of spending the interest revenues of the endowment, e.g. for all t, $B_t = r(A_t - c_{old}i_t)$, subtracting the cost of the traditional treatment. This policy is

[7] Additional conditions are required for the contractive property when $\delta(1 + r) = 1$ (Stachursky, 2009).

called the *interest-only policy* in the following. Given that it necessarily covers the cure by traditional drugs, it yields a decreasing endowment that is however not fully used up.

- the optimal policy given the social welfare function we have posited, and called the *optimal policy* from now on.

- an ad-hoc *fixed budget policy* whose expenses are constant over time and such that after 40 periods the level of assets is the same as with the optimal policy. It is meant to replicate what HAs are currently able to do under strict one-period budgets.

Figure 3 reports the trajectories of assets under these three different policies. By construction, the interest-only policy is conservative and does not fully use up the endowment in contrast with the two other policies. Figure 3 shows that the optimal policy front-loads expenses with respect to the fixed budget policy. In other words, it is optimal to spend more in the first time periods while optimal expenses are much lower when approaching the 40-period horizon.

Front-loading is caused by the short-run effect that it has on the infected rate. The evolution of this infected rate is reported under the three different policies by Figure 4. The interest-only and the fixed budget policies are much less efficient at least until period 25 (fixed budget policy) or 33 (interest-only policy) in terms of lowering the rate of infected. All benefits of the optimal policy with respect to a fixed budget come before period 25 when the two curves of rates of infection cross. Costs of the optimal policy come afterwards. This means that the eradication of the disease is taking longer than the other policies in order to minimize the rate of infection in the short-run.

As optimality is geared towards infected, it is not surprising that the gains in terms of undetected patients are much less impressive as they appear in Figure 5. This is also true for susceptibles a graph that is not displayed here. Differences are minor in the former comparison while in the latter, the curve of susceptibles when the optimal policy is implemented is below the curve when the fixed budget policy is, and they intersect after period 40. Here also, the main gains appear at the beginning. Overall, these small effects on undetected means that the epidemy is continuously fueled in by new comers from this subpopulation, and this prevents a better control of the epidemy.

It illustrates what we have found in previous sections: it is optimal to front-load expenses because there are short-run advantages to do so. There exists however a rebound effect in the

medium-run which postpones the time of eradication of the disease and this rebound is due to the presence of undetected infected persons who cannot be treated.

4.2.2 Robustness

In Tables 1 to 5, we report sensitivity to changes in some parameters of interest: the preference for the present of HAs, δ, the differential cost of the new treatment with respect to the old one (as defined in equation (9)), the cost of the old treatment (c_{old}), the Weibull coefficient α of the $\rho(.)$ function affecting its concavity and its calibration (see equation (7)) and finally the initial level of the endowment, a_0.

In every Table, the baseline evaluation is reported in the middle column which is the same in all Tables. In rows, we report the evolution of the endowment over time (at period 5, 15 and 40) under the optimal policy when compared to the fixed budget and interest-only policy. We also report in these Tables the relative welfare and relative cost of the traditional treatment under the three policies of interest. We end up comparing the relative impacts of these policies in terms of infected and undetected rates in the population.

Table 1 compare these statistics when preference for the present of HAs increases: we set $\delta = 1/(1+r+i_\delta)$ in which i_δ varies between 0.005 and 0.07. As expected, it is optimal to increase the front-loading of expenses, and period-0 welfare also increases. This increase of early expenses has the expected negative impact on infected and undetected, especially in the short-run. It is to be noted that the relative cost of the traditional treatment is negative since less patients are treated with the old treatment. The effect of varying preferences for the present on these costs is small. This is also the case for the effects on the rate of undetected which is never above 10%.

The impact of an increasing differential cost of the new treatment on expenses is moderately negative as displayed in Table 2 in rows 1 to 6. Its impact on the difference between infected rates across policies, is however magnified when the new treatment is less costly. This is also at a much lower level for the rates of undetected. The overall effect on welfare at period-0 of the optimal policy is much higher as a result when differential costs are low. The effects of the costs of the old treatment as shown in Table 3 are more non-linear and no clear patterns arise. Table 4 displays the impact of a decreasing curvature of function $\rho(.)$. This decrease in the concavity increases slightly the front loading of policies and the effects on the infected and undetected rates

are becoming much bigger and slightly unstable[8] when the curvature decreases across different values of this parameter. The welfare of a front loading policy is also very much magnified when the curvature is smaller.

Finally, Table 5 displays results when the initial endowment of HAs varies. The pattern, with some variation, is that the optimal front loading is magnified when the endowment is low, probably because it is more difficult to attain a lower rate of infection with moderate expenses. There are non linear effects however for example in the welfare at period-0 and the impact on the various rates of infection.

All those results confirm what we have found in previous exercises, and show that those conclusions are robust to the variation in some important parameters of interest.

5 Conclusion

In the case of a disappearing epidemy like Hepatitis C, an equal budget policy is dominated by a front loaded policy in terms of welfare although it does not accelerate the full eradication of the disease. It is fair to say that this result is calibrated and is difficult to prove in a formal model. It depends on the main trade-off that we have been focussing on. Spending more today implies not only less infection tomorrow, and some dynamic externalities but also less effective cures.

There is much to be done to understand better the robustness of the optimal policy rules that we have derived in this paper. First, the rebound effect that we find sets the question of time inconsistencies of optimal policies since future budgets could be renegotiated in case of a rebound of the epidemy. Second, results should be also sensitive to the evolution with time of the trade-offs across periods that are summarized in this paper by the $\rho(.)$ function, and in particular, all trade-offs arising because of changing prices over time. Third, we set aside the issue of innovation in drug discovery which was of utmost importance in the Hepatitis C case. When innovation is expected by HAs or firms in the near future, it certainly becomes an important element in the debate since it involves a trade-off between differentially effective drugs over time. Danzon (2018) reminds us that HAs can delay treatment until competing treatments become available. Lothan et al. (2020) shows that the price of Hepatitis C treatments can also affect health authorities

[8] This is probably due to the various approximations we have used when setting up our solving algorithm. We use many tolerance parameters so as to solve the optimal program, the length of the period of interest, as well as quadratic approximations.

in terms of screening strategies in addition to treatment strategies. We leave these questions of bargaining and negotiations between companies and health authorities for further research.

REFERENCES

Alvarez, F., Argente, D., & Lippi, F. (2021). A simple planning problem for COVID-19 lockdown, testing, and tracing. *American Economic Review: Insights*, 3(3), 367-82.

Assenza, T., Collard, F., Dupaigne, M., Fève, P., Hellwig, C., Kankanamge, S., & Werquin, N. (2020). The hammer and the dance: Equilibrium and optimal policy during a pandemic crisis. TSE Working Paper, n. 20-1099, May 2020

Barber M. J., D. Gotham, G. Khwairakpam, A. Hill (2020) "Price of a hepatitis C cure: Cost of production and current prices for direct-acting antivirals in 50 countries", *Journal of Virus Eradication*, Volume 6, Issue 3, 100001

Berdud M., M. Garau, M. Neri, P. O'Neill, C. Sampson, A. Towse (2018) "R&D, Competition and Diffusion of Innovation in the EU: The Case of Hepatitis C", Research Paper 18/06 Office of Health Economics

Bottero, J., Brouard, C., Roudot-Thoraval, F., Deuffic-Burban, S., Hofliger, P., Abergel, A., & Yazdanpanah, Y. (2016). 2014 French guidelines for hepatitis B and C screening: a combined targeted and mass testing strategy of chronic viruses namely HBV, HCV and HIV. *Liver International*, 36(10), 1442-1449.

Brennan T.A., Wilson J.M. (2014), "The special case of gene therapy pricing". *Nat Biotechnol,* 2014;32:874–6.

Bronowicki J.P., J.P. Daurès, S. Deuffic-Burban, D. Dhumeaux, J. Izopet, et al. (2003) "Hépatite C : transmission nosocomiale, état de santé et devenir des personnes atteintes.", Institut national de la santé et de la recherche médicale (INSERM).

Brouard, C., Saboni, L., Gautier, A., Chevaliez, S., Rahib, D., Richard, J. B., & Lot, F. (2019). "HCV and HBV prevalence based on home blood self-sampling and screening history in the general population in 2016: contribution to the new French screening strategy", *BMC infectious diseases*, 19(1), 1-14.

Danzon PM. (2018) "Affordability Challenges to Value-Based Pricing: Mass Diseases, Orphan Diseases, and Cures". *Value in Health.* 21(3):252-257

Dessauce C., Semenzato, L. and Barthélémy P. (2019). «Les antiviraux à action directe (AAD) dans le traitement de l'hepatite C chronique : retour sur 4 ans de prise en charge par l'Assurance Maladie», Assurance Maladie. Points de repère n.52.

Ganne-Carrié, N.,and Bourlière, M. (2020), L'élimination des hépatites chroniques virales est-elle un objectif raisonnable à l'horizon 2030? *Bulletin Epidemiologique Hebdomadaire*, 602, Santé Publique France.

Haute Autorité de Santé, (2014) Avis d'Efficience Sovaldi (Sofosbuvir)

Han, X., (2019), "A Two-Step Estimator for Structural Models Using Approximation", WP https://sites.google.com/site/hanxintong/research

Hethcote, H. W. (2000), "The mathematics of infectious diseases", *SIAM review*, 42(4), 599-653.

Hill, A. M., Nath, S., & Simmons, B. (2017), "The road to elimination of hepatitis C: analysis of cures versus new infections in 91 countries", *Journal of virus eradication*, 3(3), 117-123.

Iyengar S., Tay-Teo K., Vogler S., Beyer P., Wiktor S., de Joncheere K., et al. (2016), "Prices, Costs, and Affordability of New Medicines for Hepatitis C in 30 Countries: An Economic Analysis", *PLoS Med* 13 (5)

Lakdawalla, D. N. (2018). "Economics of the Pharmaceutical Industry". *Journal of Economic Literature*, 56:397–449.

Lothan, R. and N. Gutman and D. Yamin (2020) "Country versus Pharmaceutical Company Interests for Hepatitis C Elimination", https://ssrn.com/abstract=3592319

Meffre, C., Le Strat, Y., Delarocque-Astagneau, E., Dubois, F., Antona, D., Lemasson, J. M., & Desenclos, J. C. (2010) "Prevalence of hepatitis B and hepatitis C virus infections in France in 2004: social factors are important predictors after adjusting for known risk factors" *Journal of medical virology*, 82(4), 546-555.

Mouterde A.L., F. Bocquet, I. Fusier, P. Paubel (2016) "Hepatitis C: how has France limited the expenses related to new treatments?", *Expert Review of Pharmacoeconomics & Outcomes Research*, 16:6, 655-657

Roudot-Thoraval F. (2021) "Epidemiology of hepatitis C virus infection" *Clinics and Research in Hepatology and Gastroenterology*, 45(3) doi: 10.1016/j.clinre.2020.101596.101596

Santé Publique (2021), https://www.santepubliquefrance.fr/maladies-et-traumatismes/hepatites-virales/hepatite-c/articles/prevalence-de-l-hepatite-c, Accessed July 20th 13:36

Santé Publique (2021b), https://www.santepubliquefrance.fr/les-actualites/2021/hepatites-b-et-c-donnees-epidemiologiques-2019

Stachurski, J. (2009). *Economic dynamics: theory and computation.* MIT Press.

A Stationary equilibria and stability

We follow Hethcote (2000).

In the absence of policy, $\rho_t = \rho_0$ or equivalently $b_t = 0$, we can derive the stationary equilibria by solving:

$$\begin{aligned} 0 &= rA_t \\ 0 &= -\beta(i_t + u_t)s_t + \nu(1 - s_t), \\ 0 &= \beta(i_t + u_t)s_t - (\zeta + \nu)u_t, \\ 0 &= \zeta u_t - (\rho_0 + \nu)i_t. \end{aligned}$$

Replacing the value of i_t from the fourth equation ($i_t = \frac{\zeta u_t}{\rho_0 + \nu}$) in the third one yields:

$$0 = u_t(\beta(\frac{\zeta}{\rho_0 + \nu} + 1)s_t - (\zeta + \nu)).$$

In consequence, the disease-free equilibrium is obtained by setting $u_t = 0$ which by the system above yields $i_t = A_t = 0$ and $s_t = 1$.

Another possible equilibrium assumes that $u_t \neq 0$. We then derive from the previous equation that:

$$\beta(1 + \frac{\zeta}{\rho_0 + \nu})s_t = \zeta + \nu \Longrightarrow s_t = s^* = \frac{(\zeta + \nu)(\rho_0 + \nu)}{\beta(\zeta + \rho_0 + \nu)}.$$

One condition of existence of the endemic equilibrium is therefore that $s^* < 1$ or $\beta \geq \beta^* = \frac{(\zeta+\nu)(\rho_0+\nu)}{\beta(\zeta+\rho_0+\nu)}$. Note that as $\frac{(\zeta+\nu)(\rho_0+\nu)}{\beta(\zeta+\rho_0+\nu)} \geq \nu$, equal when $\rho_0 = 0$, the condition $\beta \leq \nu$ for the absence of such an equilibrium as in the original development by Hethcote (2000).

If $s^* < 1$, we derive from the second, third and fourth system equations that the endemic equilibrium is given by:

$$\begin{aligned} (\zeta + \nu)u_t &= \nu(1 - s_t) = \nu(1 - s^*) \\ &\Longrightarrow u_t = \frac{\nu}{\zeta + \nu}(1 - s^*), i_t = \frac{\zeta\nu}{(\zeta + \nu)(\rho_0 + \nu)}(1 - s^*). \end{aligned}$$

and these equations satisfy the conditions on the state variables.

The stability condition of the disease-free equilibrium can be analyzed, (e.g. Heer and Maussner, 2009) using the Jacobian of the system of equations describing the SIR model (i.e. $z_{t+1} =$

$f(z_t)$) that is $J_0 = \nabla f$ evaluated at $z_t^{(0)} = (s_t^{(0)}, u_t^{(0)}, i_t^{(0)}) = (1, 0, 0)$.

$$J_0 = \begin{bmatrix} 1-\nu & -\beta & -\beta \\ 0 & 1+\beta-\zeta-\nu & \beta \\ 0 & \zeta & 1-\rho_0-\nu \end{bmatrix}.$$

Its eigenvalues are obtained by solving:

$$\begin{aligned} 0 &= \det(J_0 - \lambda I) = (1-\nu-\lambda)((1+\beta-\zeta-\nu-\lambda)(1-\rho_0-\nu-\lambda) - \beta\zeta) \\ &= (1-\nu-\lambda)\left[(1-\nu-\rho_0-\lambda)^2 + (\beta-\zeta+\rho_0)(1-\rho_0-\nu-\lambda) - \beta\zeta\right]. \end{aligned}$$

One root is $\lambda = 1-\nu$ and the other two are obtained by solving $(1-\rho_0-\nu-\lambda)^2 + (\beta-\zeta+\rho_0)(1-\rho_0-\nu-\lambda) - \beta\zeta = 0$ in $x = 1-\nu-\lambda$. The discriminant is equal to:

$$(\beta-\zeta+\rho_0)^2 + 4\beta\zeta > 0.$$

so that the roots are $x_\pm = \frac{-(\beta-\zeta+\rho_0) \pm \sqrt{(\beta-\zeta+\rho_0)^2+4\beta\zeta}}{2}$ and distinct. The roots of the original problem are thus $\lambda_\pm = 1-\rho_0-\nu+x_\pm$.

In the particular case of $\rho_0 = 0$, $x_\pm$ are $x_\pm = \frac{-(\beta-\zeta)\pm(\beta+\zeta)}{2}$ and thus equal to β or ζ. As non negative ν and ζ are such that $\nu+\zeta < 1$, the eigenvalues of J_0 have an absolute value less than one if and only if $\beta < \beta^* = \nu$ if $\rho_0 = 0$. This can be generalized to the case $\rho_0 > 0$. In sum, if $\beta < \beta^*$, the only stationary equilibrium is the disease-free equilibrium which is stable.

B Simulation Algorithm

Since the state space is formed by continuous variables, we should either discretize the state space or use a sieve space of functions. We do both by evaluating the value functions on a discrete grid of functions and using quadratic approximations (see e.g. Han, 2018). We use different steps that are described in more details below.

We start in Step 1 by computing the value function when HAs do not intervene in which case the epidemy develops according to the SIR model. In Step 2 we extend this setting to the case in which HAs can intervene in the current period only. Step 3 extends Step 2 by solving the first order condition of the Bellman equation when HAs spend their endowment in a finite number k of periods. Step 4 describes how we choose to stop the iteration process at step K when there is no change in the computed value function.

1. This is the benchmark step in which we evaluate the value function in the absence of intervention by HAs and approximate it by a quadratic function in the state space. For different values $z_0 = (s_0, u_0, i_0)$ on a grid of values, denoted $z_0^{(s)}$, covering the initial range of values of the processes we are interested in. We then forward simulate $\{z_t^{(s)}\}_{t\geq 1}$ using the SIR model, in *the absence of a policy* until T is sufficiently large and we evaluate $W_0^{(T)}(z_0^{(s)})$ as:

$$W_0^{(T)}(z_0^{(s)}) = \sum_{t=1}^{T} \delta^{t-1} v(i_t) = -\sum_{t=1}^{T} \delta^{t-1} \frac{(i_t)^2}{2} < 0.$$

We test the stability of $W_0^{(T)}(z_0^{(s)})$ with respect to T and choose T such that :

$$d(W_0^{(T)}(z_0^{(s)}), W_0^{(T+1)}(z_0^{(s)})) = \frac{1}{\#\{z_0^{(s)}\}} \Big(\sum_{z_0^{(s)}} \frac{(W_0^{(T)}(z_0^{(s)}) - W_0^{(T+1)}(z_0^{(s)}))^2}{\left|\frac{1}{\#\{z_0^{(s)}\}} \sum_{z_0^{(s)}} W_0^{(T)}(z_0^{(s)})\right|}\Big)^{1/2} < \varepsilon_{TOL}$$

with a sufficiently small tolerance level, ε_{TOL}. We chose this formulation to balance absolute and relative concerns and using the fact that the value function is always negative.

As this function is given only on the grid, $z_0^{(s)}$, we interpolate it by a quadratic function:

$$W_0^{(T)}(z_0^{(s)}) = c_0 + c_1' z_0^{(s)} + z_0^{(s)\prime} C_2 z_0^{(s)} + \varepsilon(z_0^{(s)}),$$

where c_0 is a constant, c_1 a vector of size 3, and C_2 a symmetric matrix of size 3. Denote $C^{(0)}$ the underlying vector of parameters whose estimates, $\hat{c}_0, \hat{c}_1$ and $\hat{C}_2$ are obtained by an OLS regression.[9] For any value z, we approximate $W_0^{(T)}(z)$ by

$$\hat{W}_0(z) = \hat{c}_0 + \hat{c}_1' z + z' \hat{C}_2 z.$$

We also at this step evaluate the cost of the traditional treatment for each value, $z_0^{(s)}$. Equation (20) yields:

$$\kappa(z_0^{(s)}) = c_{old} \sum_{t=0}^{\infty} \frac{i_t(z_0^{(s)})}{(1+r)^t},$$

when $i_t(z_0^{(s)})$ is the infection rate when HAs never use the new treatments.

2. We now extend the same grid of values, $z_0^{(s)}$, to allow for expenses for the new treatment by considering $x_0^{(s)} = (A_0^{(s)}, z_0^{(s)})$, in which $A_0^{(s)}$ is itself a grid of values for assets that health authorities are endowed with. This grid is bounded from below by 0 and bounded from

[9] Any approximating method can be used here. Sieves, as we do here with quadratic terms, might be preferable to kernel methods because the grid retained for the kernel might not be self-consistent.

above by the maximum value of assets. We won't consider any policy such that the level of assets increases at any period.

In a first step, we consider that the available endowment is fully expended by the new treatment for some and the traditional drug for others. We write that:

$$W_1^*(x_0) = W_1^*(A_0, z_0) = v(i_1) + \delta \hat{W}_0(z_1),$$

in which $\hat{W}_0(z_1)$ is the approximate value function derived at Step 1 and z_1 is the ex-post value of the state variables. The value function, $W_1^*(x_0)$, corresponds to the case when after time 1 there is no endowment left to spend to control the disease and the process is left to its own and converges to the unique stationary equilibrium of the eradication of the disease without intervention.

Note that under the budget rule in equation (19), and that assets are depleted in one period, $A_1 = \kappa_1(z_1)$, the provisions made for the traditional drug at period 1 and therefore:

$$\kappa_1(z_1) = (1+r)A_0 - b_0 i_0 - c_{old} i_0.$$

Using equation (20), we have that $\kappa_1(z_1) + c_{old} i_0 = (1+r)\kappa_0(z_0)$ so that b_0 is a function of $x_0 = (A_0, z_0)$:

$$b_0 = \frac{(1+r)(A_0 - \kappa_0(z_0))}{i_0},$$

and i_1 is obtained as a result (as well as the rest of z_1). As before, function $W_1^*(x_0)$ is evaluated only on a grid of points, $x_0^{(s)}$, and we approximate it by a quadratic function:

$$W_1^*(x_0^{(s)}) = c_0 + c_1' x_0^{(s)} + x_0^{(s)\prime} C_2 x_0^{(s)} + \varepsilon(x_0^{(s)}),$$

where, by using the same notation as before, c_0 is a constant, c_1 a vector of size 4, and C_2 a symmetric matrix of size 4. Denote $C^{(1)}$ the underlying vector of parameters whose estimates are obtained by an OLS regression, $\hat{C}^{(1)}$. Again we approximate $W_1^*(x_0)$ by its OLS predicted value $\hat{W}_1^*(x_0)$.

3. Building on Step 2 and $\hat{W}_1^*(x_0)$, we consider at the $k+1^{th}$ iteration step the same grid, $x_0^{(s)} = (A_0^{(s)}, z_0^{(s)})$, and solve:

$$V^{(k+1)}(x_0^{(s)}) = \max_{A_1^{(s)}} (v(i_1^{(s)}) + \delta \hat{W}_k^*(x_1^{(s)})),$$

under the laws of motion of the SIR model, $\hat{W}_k^*$ is the k^{th} step prediction of the value function and $A_1^{(s)} = (1+r)A_0^{(s)} - (b_0^{(s)} + c_{old})i_0^{(s)}$ in which $A_1^{(s)} \geq \kappa(z_1^{(s)})$. As in step 2, we can rewrite the equality and inequality constraint as:

$$(1+r)A_0^{(s)} - (b_0^{(s)} + c_{old})i_0^{(s)} \geq \kappa(z_1^{(s)}),$$

which leads to :

$$\begin{aligned}(b_0^{(s)} + c_{old})i_0^{(s)} &\leq (1+r)A_0^{(s)} - c_{old}i_0^{(s)} - \kappa(z_1^{(s)}) \\ &= (1+r)(A_0^{(s)} - \kappa(z_0^{(s)}))\end{aligned}$$

using equation (20). Next, we use either the maximization of the Bellman equation (17) or the first order condition (18) to solve this program under the last inequality constraint. This delivers the value function of a policy which uses up the full endowment in at most $k+1$ periods (given the provisions made for the traditional treatment).[10]

4. The previous iteration process can be described by the evolution over time of policy functions, value functions or parameters describing the quadratic approximation of the value functions $\hat{C}^{(k)}$. For the latter, we have:

$$\hat{C}^{(k+1)} = \Phi(\hat{C}^{(k)}),$$

in which Φ is described by the previous steps. Because $\delta(1+r) \leq 1$, $\Phi()$ is a contraction with modulus less or equal to $\delta(1+r)$. The Banach theorem implies that there is a single fixed point and that $\hat{C}^{(k)}$ converges to this fixed point say $\hat{C}$, (e.g. Stachurski, 2009). Therefore, value and optimal policy functions can be simulated.

[10] Alternatively, we could have backward simulation and *endogenous grids* that might solve partly for the issues of grid choices and quality of approximations. Set $x_1 = (A_1, z_1)$ and solve for b_0 using $v'(i_1) + \delta\frac{dV^{(k)}}{di_1} = 0$, see equation (18) as a function of $\rho'(b_0)$. Then solve for x_0 by inverting $x_{t+1} = f(x_t)$ (see Appendix C.5 in the Supplementary Appendix available upon request).

Online Supplementary Appendix

C Additional theoretical results

C.1 Second-order condition

The second order condition is more involved and we will assume that it holds to ensure that the previous program has a unique solution. Differentiating equation (18) (in the case $-v'(i_{t+1}) = i_{t+1}$) and dividing by i_t) yields an equivalent condition:

$$0 > \frac{\partial}{\partial b_t}\left(i_{t+1}\rho'_t - \delta\frac{\partial W_{t+1}}{\partial A_{t+1}} - \delta\frac{\partial W_{t+1}}{\partial i_{t+1}}\rho'_t\right) =$$

$$\begin{aligned}&\rho_t"(i_{t+1} - \delta\frac{\partial W_{t+1}}{\partial i_{t+1}}) + \delta\frac{\partial^2 W_{t+1}}{\partial (A_{t+1})^2}i_t\\ &+\delta\frac{\partial^2 W_{t+1}}{\partial A_{t+1}\partial i_{t+1}}\rho'_t i_t + \delta\frac{\partial^2 W_{t+1}}{\partial (i_{t+1})^2}(\rho'_t)^2 i_t.\end{aligned}$$

The first term is negative because of the concavity of ρ_t and $i_{t+1} - \delta\frac{\partial W_{t+1}}{\partial i_{t+1}} = \delta\frac{1}{\rho'_t}\frac{\partial W_{t+1}}{\partial A_{t+1}} > 0$ by equation (18) and the presumption that $\frac{\partial W_{t+1}}{\partial A_{t+1}} > 0$ since if not, the budget constraint would not be saturated. A sufficient condition for the other three terms to be negative is that W_{t+1} is concave in A_{t+1} and i_{t+1}, which would be the consequence of the concavity of ρ and $v(i_{t+1})$.

C.2 State equations

At the previous optimal value, consider the gradient ∇W_t defined by:

$$\nabla W_t = v'(i_{t+1})\frac{di_{t+1}}{dx_t^\top} + \delta\nabla W_{t+1}\frac{dx_{t+1}}{dx_t^\top}.$$

We get ∇W_{t+1} from:

$$\begin{aligned}\frac{dA_{t+1}}{dx_t^T} &= (1+r, 0, 0, -b_t),\\ \frac{ds_{t+1}}{dx_t^T} &= (0, 1-\beta(i_t+u_t)-\nu, -\beta s_t, -\beta s_t)\\ \frac{du_{t+1}}{dx_t^T} &= (0, \beta(i_t+u_t), 1+\beta s_t - (\zeta+\nu), \beta s_t)\\ \frac{di_{t+1}}{dx_t^T} &= (0, 0, \zeta, 1-\nu-\rho_t).\end{aligned}$$

We thus get:

$$\begin{aligned}
\frac{\partial W_t}{\partial A_t} &= \delta(1+r)\frac{\partial W_{t+1}}{\partial A_{t+1}}, \\
\frac{\partial W_t}{\partial s_t} &= \delta(1-\beta(i_t+u_t)-\nu)\frac{\partial W_{t+1}}{\partial s_{t+1}} + \delta\beta(i_t+u_t)\frac{\partial W_{t+1}}{\partial u_{t+1}}, \\
\frac{\partial W_t}{\partial u_t} &= -\zeta i_{t+1} - \delta\beta s_t \frac{\partial W_{t+1}}{\partial s_{t+1}} \\
&\quad +\delta(1+\beta s_t - (\zeta+\nu))\frac{\partial W_{t+1}}{\partial u_{t+1}} + \delta\zeta\frac{\partial W_{t+1}}{\partial i_{t+1}}, \\
\frac{\partial W_t}{\partial i_t} &= -i_{t+1}(1-\nu-\rho_t) - b_t\frac{\partial W_{t+1}}{\partial A_{t+1}} - \delta\beta s_t\frac{\partial W_{t+1}}{\partial s_{t+1}} \\
&\quad +\delta\beta s_t\frac{\partial W_{t+1}}{\partial u_{t+1}} + \delta(1-\nu-\rho_t)\frac{\partial W_{t+1}}{\partial i_{t+1}},
\end{aligned}$$

that can be written as:

$$\nabla W_t = R_t \nabla W_{t+1} \tag{21}$$

in which R_t is a 4×4 matrix that we assume to have full rank.[11]

C.3 Euler equations

The first order condition (18) writes:

$$i_{t+1} = \delta\left(\frac{1}{\rho_t'}\frac{\partial W_{t+1}}{\partial A_{t+1}} + \frac{\partial W_{t+1}}{\partial i_{t+1}}\right) = m_t \nabla W_{t+1}.$$

To write an Euler equation we have to "invert" this equation. This is possible if we consider $d_t = (i_{t-3}, i_{t-2}, i_{t-1}, i_t)$ and notice that:

$$i_{t-3} = m_{t-3}\nabla W_{t-2} = m_{t-3}R_{t-2}R_{t-1}R_t\nabla W_{t+1},$$

We can then write

$$d_t = M_t \nabla W_{t+1}$$

in which $M_t = \begin{pmatrix} m_t \\ m_{t-1}R_t \\ m_{t-2}R_{t-1}R_t \\ m_{t-3}R_{t-2}R_{t-1}R_t \end{pmatrix}$ is supposed to be full rank. This yields that

$$\nabla W_{t+1} = M_t^{-1} d_t,$$

[11] It certainly shows that if W_{t+1} is concave in A_{t+1} then W_t also is as a function of A_t. It is slightly more difficult to show that W_{t+1} is concave in i_{t+1}.

and therefore equation (21) yields

$$M_{t-1}^{-1} d_{t-1} = R_t M_t^{-1} d_t,$$

which is the Euler equation.

C.4 Preferences

Even if short-run preferences are known, it remains to set up long-run preferences encoded by the welfare function W_t.

One possible set of axioms on preferences relative to the vector (s_t, u_t, i_t, r_t), conditional on a fixed value of A_t that we can impose is the following:

1. If recovered persons are immune, then a change in s_t, $ds_t > 0$, compensated by an increase of r_t, $dr_t = -ds_t$ while $du_t = di_t = 0$ (because $s_t + u_t + i_t + r_t = 1$) affects welfare negatively. Writing:

$$\begin{aligned} dW_t &= \frac{\partial W_t}{\partial s_t} ds_t + \frac{\partial W_t}{\partial u_t} du_t + \frac{\partial W_t}{\partial i_t} di_t, \\ &= \frac{\partial W_t}{\partial s_t} ds_t < 0, \end{aligned}$$

   implies that $\frac{\partial W_t}{\partial s_t} < 0$.

2. Furthermore, it is always welfare-improving to have less infected than recovered. Fixing s_t, we get

$$dW_t = \frac{\partial W_t}{\partial u_t} du_t + \frac{\partial W_t}{\partial i_t} di_t,$$

   where $dr_t = -(du_t + di_t)$. This implies that $\frac{\partial W_t}{\partial u_t} < 0$ and $\frac{\partial W_t}{\partial i_t} < 0$.

3. It is also always welfare improving to have $ds_t = -di_t > 0$ at fixed u_t and r_t or to have $ds_t = -du_t > 0$ at fixed i_t and r_t so that:

$$\frac{\partial W_t}{\partial s_t} - \frac{\partial W_t}{\partial i_t} > 0, \frac{\partial W_t}{\partial s_t} - \frac{\partial W_t}{\partial u_t} > 0,$$

4. Finally, it is (weakly) welfare improving to have more identifiable infected than undetected infected since health policies do not reach the latter group. Keeping s_t, $r_t m$ constant and imposing $di_t = -du_t > 0$ should then be welfare improving. This means

$$\frac{\partial W_t}{\partial i_t} - \frac{\partial W_t}{\partial u_t} > 0.$$

These four axioms imply that:

$$0 > \frac{\partial W_t}{\partial s_t} > \frac{\partial W_t}{\partial i_t} > \frac{\partial W_t}{\partial u_t}.$$

For instance the welfare function

$$W_t = w_s(\frac{1-s_t}{3})^2 - w_u(u_t)^2 - w_i(i_t)^2,$$

in which $w_s < w_i < w_u$, satisfies the condition when $\frac{1-s_t}{3}, u_t$ and i_t are of the same orders of magnitude.

C.5 Inversion of $x_{t+1} = f(x_t)$

We set a value $x_{t+1} \in \mathcal{X}$ and to solve for x_t, we consider the four equations:

$$\begin{aligned}
\Delta A_{t+1} &= rA_t - b_t i_t \\
\Delta s_{t+1} &= -\beta(i_t + u_t)s_t + \nu(1 - s_t), \\
\Delta u_{t+1} &= \beta(i_t + u_t)s_t - (\zeta + \nu)u_t, \\
\Delta i_{t+1} &= \zeta u_t - (\rho_t + \nu)i_t.
\end{aligned}$$

as well as the first order condition (18) that presupposes that we know the derivatives of the value function at $t+1$ with respect to A_{t+1} and i_{t+1}.

First, by the latter, b_t and consequently ρ_t are derived.

Second, we consider the two last equations in which we set, $\sigma_t = u_t + i_t$ so as to obtain:

$$y_{t+1} = \begin{pmatrix} u_{t+1} \\ i_{t+1} \end{pmatrix} = \begin{pmatrix} 1-(\zeta+\nu) & 0 \\ \zeta & 1-(\rho_t+\nu) \end{pmatrix} y_t + \begin{pmatrix} \beta s_t \sigma_t \\ 0 \end{pmatrix},$$

which yields:

$$y_t = \frac{1}{(1-(\zeta+\nu))(1-(\rho_t+\nu))} \begin{pmatrix} 1-(\rho_t+\nu) & 0 \\ -\zeta & 1-(\zeta+\nu) \end{pmatrix} \left(y_{t+1} - \begin{pmatrix} \beta s_t \sigma_t \\ 0 \end{pmatrix} \right). \tag{22}$$

Condition: As both elements in y_t should be positive, we must have:

$$(1-(\rho_t+\nu))(u_{t+1} - \beta s_t \sigma_t) \geq 0, -\zeta(u_{t+1} - \beta s_t \sigma_t) + (1-(\zeta+\nu))i_{t+1} \geq 0.$$

Furthermore, imposing that $(1,1)y_t = u_t + i_t = \sigma_t$ yields :

$$\sigma_t = \begin{pmatrix} m & \frac{1}{1-(\rho_t+\nu)} \end{pmatrix} y_{t+1} - m\beta s_t \sigma_t, \tag{23}$$

in which $m = \frac{1-(\rho_t+\nu)-\zeta}{(1-(\zeta+\nu))(1-(\rho_t+\nu))}$.

Third, reconsidering the second equation of the system above yields:

$$s_{t+1} = s_t - \beta\sigma_t s_t + \nu(1 - s_t) = s_t(1 - \beta\sigma_t - \nu) + \nu, \tag{24}$$

and summing this equation multiplied by m with equation (23) to get rid of $s_t\sigma_t$ yields

$$c_{t+1} = \begin{pmatrix} m & \frac{1}{1-(\rho_t+\nu)} \end{pmatrix} y_{t+1} + m(s_{t+1} - \nu) = \sigma_t + m(1 - \nu)s_t. \tag{25}$$

From this equation, derive the expression for σ_t and plug it equation (24) to obtain:

$$s_t(1 - \beta c_{t+1} + \beta m(1 - \nu)s_t - \nu) + \nu - s_{t+1} = 0.$$

The discriminant writes,

$$\Delta = (1 - \beta c_{t+1} - \nu)^2 + 4\beta m(1 - \nu)(s_{t+1} - \nu),$$

so that because $\beta m(1 - \nu)(s_{t+1} - \nu) \geq 0$ we have two solutions:

$$\frac{-(1 - \beta c_{t+1} - \nu) \pm \sqrt{\Delta}}{2\beta m(1 - \nu)}.$$

Because $\beta m(1 - \nu) > 0$, we have only one solution such as $s_t > \nu$:

$$\frac{-(1 - \beta c_{t+1} - \nu) + \sqrt{\Delta}}{2\beta m(1 - \nu)}.$$

This delivers σ_t by equation (25) and (u_t, i_t) by equation (22). Finally, the first equation of the system identifies A_t.

Remark: A sanity check, in the absence of policy, $b = 0$, proves that $f^{-1}((0, 1, 0, 0)) = (0, 1, 0, 0)$ at the stationary and stable solution

Table 1: The impact of preferences for the present (δ)

$\delta = 1/(1+r+i_\delta)$	$i_\delta \longrightarrow$	0.0050	0.0213	0.0375	0.0537	0.0700
$\Delta_{opt/fix}a$	$t=5$	-6.97	-6.97	-12.6	-12.6	-13.3
	$t=15$	-28.6	-28.6	-32	-32	-32.1
	$t=40$	-2.07	-2.07	-1.76	-1.76	-1.74
$\Delta_{opt/ir}a$	$t=5$	-12.1	-12.1	-17.7	-17.7	-18.5
	$t=15$	-49.2	-49.2	-52.6	-52.6	-52.7
	$t=40$	-80.4	-80.4	-80.3	-80.3	-80.3
$\Delta^R_{opt/fix}w$		10.8	10.6	15.8	15.3	15.3
$\Delta^R_{opt/ir}w$		28	26.9	31.7	29.8	28.6
$\Delta^R_{opt/fix}c_{old}$		-3.58	-3.58	-4.66	-4.66	-4.76
$\Delta^R_{opt/ir}c_{old}$		-9.68	-9.68	-10.9	-10.9	-11
$\Delta^R_{opt/fix}i$	$t=5$	-5.81	-5.81	-10.3	-10.3	-10.7
	$t=15$	-32.1	-32.1	-33.9	-33.9	-33.5
	$t=40$	70.3	70.3	71.5	71.5	71.6
$\Delta^R_{opt/ir}i$	$t=5$	-11.6	-11.6	-16.4	-16.4	-16.8
	$t=15$	-68.1	-68.1	-70.5	-70.5	-70
	$t=40$	39.3	39.3	41.8	41.8	41.9
$\Delta^R_{opt/fix}u$	$t=5$	-0.0739	-0.0739	-0.303	-0.303	-0.332
	$t=15$	-1.72	-1.72	-2.27	-2.27	-2.31
	$t=40$	-0.486	-0.486	-0.475	-0.475	-0.475
$\Delta^R_{opt/ir}u$	$t=5$	-0.233	-0.233	-0.463	-0.463	-0.493
	$t=15$	-3.56	-3.56	-4.13	-4.13	-4.18
	$t=40$	-8.23	-8.23	-8.24	-8.24	-8.24

Note: Variation across i_δ values in which $\delta = 1/(1+r+i_\delta)$
(Rows 1-3 and 4-6) $\Delta_{opt/.}a$: Absolute difference between assets under the optimal policy (opt) vs the fixed budget (fix) or interest-only (ir) after $t = 5, 15, 40$ periods.
(Rows 7-10) $\Delta^R_{opt/.}w$, $\Delta^R_{opt/.}c_{old}$: Relative difference between welfare (resp. cost of old treatment) under the optimal policy (opt) vs the fixed budget (fix) or interest-only (ir).
(Rows 11-22) $\Delta^R_{opt/.}i$, $\Delta^R_{opt/.}u$: Relative difference between infected share (resp. undetected one) under the optimal policy (opt) vs the fixed budget (fix) or interest-only (ir) after $t = 5, 15, 40$ periods.

Table 2: The impact of the differential cost of the new treatment

$dcost$	$\rightarrow$	1000€	5750€	10500	15250€	20000€
$\Delta_{opt/fix}a$	$t=5$	-16.2	-12.4	-12.6	-12.7	-12.8
	$t=15$	-35.9	-31.1	-32	-31.5	-32.9
	$t=40$	-1.76	-1.61	-1.76	-1.94	-2.1
$\Delta_{opt/ir}a$	$t=5$	-22.2	-17.9	-17.7	-17.6	-17.6
	$t=15$	-59.4	-52.7	-52.6	-51.3	-52.1
	$t=40$	-90.8	-83.9	-80.3	-78	-76.2
$\Delta^R_{opt/fix}w$		30.5	18.6	15.8	13.8	12.5
$\Delta^R_{opt/ir}w$		55.2	37.7	31.7	27.5	24.7
$\Delta^R_{opt/fix}c_{old}$		-37.9	-10.7	-4.66	-2.03	-0.487
$\Delta^R_{opt/ir}c_{old}$		-39.2	-18.2	-10.9	-6.85	-4.25
$\Delta^R_{opt/fix}i$	$t=5$	-61.8	-14.4	-10.3	-8.42	-7.33
	$t=15$	-19.5	-55.5	-33.9	-24.8	-21.1
	$t=40$	76.9	78.6	71.5	63.3	57.1
$\Delta^R_{opt/ir}i$	$t=5$	-106	-23.8	-16.4	-13.1	-11.2
	$t=15$	-150	-129	-70.5	-49.8	-40.7
	$t=40$	72.5	59.7	41.8	32.2	28
$\Delta^R_{opt/fix}u$	$t=5$	-1	-0.384	-0.303	-0.261	-0.234
	$t=15$	-2.73	-2.61	-2.27	-2	-1.89
	$t=40$	-0.978	-0.997	-0.475	-0.132	0.234
$\Delta^R_{opt/ir}u$	$t=5$	-1.5	-0.606	-0.463	-0.39	-0.344
	$t=15$	-5.57	-5.03	-4.13	-3.54	-3.23
	$t=40$	-4.21	-8.25	-8.24	-7.51	-6.61

Notes: Variation across the differential cost of the new treatment $dcost$.

(Rows 1-3 and 4-6) $\Delta_{opt/.}a$: Absolute difference between assets under the optimal policy (opt) vs the fixed budget (fix) or interest-only (ir) after $t = 5, 15, 40$ periods.

(Rows 7-10) $\Delta^R_{opt/.}w$, $\Delta^R_{opt/.}c_{old}$: Relative difference between welfare (resp. cost of old treatment) under the optimal policy (opt) vs the fixed budget (fix) or interest-only (ir).

(Rows 11-22) $\Delta^R_{opt/.}i$, $\Delta^R_{opt/.}u$: Relative difference between infected share (resp. undetected one) under the optimal policy (opt) vs the fixed budget (fix) or interest-only (ir) after $t = 5, 15, 40$ periods.

Table 3: The impact of the cost of the old treatment

c_{old}	$\rightarrow$	1000€	5750€	10500	15250€	20000€
$\Delta_{opt/fix}a$	$t=5$	-6.42	-3.48	-12.6	-9.33	-16
	$t=15$	-19.1	-17.8	-32	-26.8	-39.8
	$t=40$	-3.46	-2.98	-1.76	-1.09	0.362
$\Delta_{opt/ir}a$	$t=5$	-13.1	-9.41	-17.7	-13.7	-19.6
	$t=15$	-45	-41	-52.6	-44.6	-55
	$t=40$	-98.2	-89.6	-80.3	-71.3	-61.5
$\Delta^R_{opt/fix}w$		7.88	5.83	15.8	13.3	20.7
$\Delta^R_{opt/ir}w$		25.1	21.5	31.7	27.4	33.9
$\Delta^R_{opt/fix}c_{old}$		0.0928	-1.16	-4.66	-6.18	-8.76
$\Delta^R_{opt/ir}c_{old}$		-7.33	-7.94	-10.9	-11.5	-13.2
$\Delta^R_{opt/fix}i$	$t=5$	-5.19	-2.96	-10.3	-8.02	-13.5
	$t=15$	-19.9	-19.7	-33.9	-29.9	-46.2
	$t=40$	68.9	67.5	71.5	67.6	66.8
$\Delta^R_{opt/ir}i$	$t=5$	-12.4	-9.32	-16.4	-13.2	-18
	$t=15$	-59.2	-55.7	-70.5	-61.6	-77.4
	$t=40$	35.2	32.9	41.8	34.8	34.7
$\Delta^R_{opt/fix}u$	$t=5$	-0.168	-0.101	-0.303	-0.247	-0.397
	$t=15$	-1.11	-0.927	-2.27	-1.93	-2.97
	$t=40$	-0.949	-0.775	-0.475	-0.652	-0.167
$\Delta^R_{opt/ir}u$	$t=5$	-0.366	-0.28	-0.463	-0.386	-0.514
	$t=15$	-3.28	-2.93	-4.13	-3.59	-4.44
	$t=40$	-9.27	-8.84	-8.24	-8.08	-7.15

Notes: Variation across the cost of the old treatment c_{old}.

(Rows 1-3 and 4-6) $\Delta_{opt/.}a$: Absolute difference between assets under the optimal policy (opt) vs the fixed budget (fix) or interest-only (ir) after $t = 5, 15, 40$ periods.

(Rows 7-10) $\Delta^R_{opt/.}w$, $\Delta^R_{opt/.}c_{old}$: Relative difference between welfare (resp. cost of old treatment) under the optimal policy (opt) vs the fixed budget (fix) or interest-only (ir).

(Rows 11-22) $\Delta^R_{opt/.}i$, $\Delta^R_{opt/.}u$: Relative difference between infected share (resp. undetected one) under the optimal policy (opt) vs the fixed budget (fix) or interest-only (ir) after $t = 5, 15, 40$ periods.

Table 4: The impact of the Weibull coefficient

	$\alpha \rightarrow$	0.3	0.4	0.5	0.6	0.7
$\Delta_{opt/fix}a$	$t=5$	-11.1	-10.6	-12.6	-15.9	-19.8
	$t=15$	-35	-35.2	-32	-34.8	-39.6
	$t=40$	-3.39	-2.93	-1.76	-1.19	-1.46
$\Delta_{opt/ir}a$	$t=5$	-14.6	-14.8	-17.7	-21.8	-25.9
	$t=15$	-50	-52.3	-52.6	-57.7	-64
	$t=40$	-65	-70.7	-80.3	-88.7	-92.9
$\Delta^R_{opt/fix}w$		1.39	4.99	15.8	36.2	48.6
$\Delta^R_{opt/ir}w$		3.07	10.6	31.7	67.3	86.5
$\Delta^R_{opt/fix}c_{old}$		0.511	0.636	-4.66	-29.8	-80.8
$\Delta^R_{opt/ir}c_{old}$		1.74	1.02	-10.9	-40.5	-78.7
$\Delta^R_{opt/fix}i$	$t=5$	-0.952	-2.84	-10.3	-49	-707
	$t=15$	-2.68	-9.26	-33.9	-83.3	-2.73
	$t=40$	11.2	29	71.5	85.4	53.5
$\Delta^R_{opt/ir}i$	$t=5$	-1.41	-4.43	-16.4	-79.3	-1604
	$t=15$	-4.44	-16	-70.5	-580	-7.11
	$t=40$	6.63	14.7	41.8	81.7	52.3
$\Delta^R_{opt/fix}u$	$t=5$	-0.0263	-0.0721	-0.303	-1.01	-2.1
	$t=15$	-0.322	-0.898	-2.27	-3.88	-2.64
	$t=40$	0.573	0.688	-0.475	-1.32	-2.01
$\Delta^R_{opt/ir}u$	$t=5$	-0.0415	-0.122	-0.463	-1.45	-3.07
	$t=15$	-0.516	-1.52	-4.13	-7.81	-5.09
	$t=40$	-0.597	-2.77	-8.24	-6.54	-4.46

Notes: Variation of the Weibull coefficient of the $\rho(.)$ function

(Rows 1-3 and 4-6) $\Delta_{opt/.}a$: Absolute difference between assets under the optimal policy (opt) vs the fixed budget (fix) or interest-only (ir) after $t = 5, 15, 40$ periods.

(Rows 7-10) $\Delta^R_{opt/.}w$, $\Delta^R_{opt/.}c_{old}$: Relative difference between welfare (resp. cost of old treatment) under the optimal policy (opt) vs the fixed budget (fix) or interest-only (ir).

(Rows 11-22) $\Delta^R_{opt/.}i$, $\Delta^R_{opt/.}u$: Relative difference between infected share (resp. undetected one) under the optimal policy (opt) vs the fixed budget (fix) or interest-only (ir) after $t = 5, 15, 40$ periods.

Table 5: The impact of the heatlth authority endowment

a_0	$\rightarrow$	2000€	3500€	5000€	6500€	8000€
$\Delta_{opt/fix}a$	$t=5$	-28.6	-20.6	-12.6	-4.62	-8.05
	$t=15$	-31.2	-51	-32	-5.96	-11.7
	$t=40$	-1.58	-1.49	-1.76	-2.82	-2.19
$\Delta_{opt/ir}a$	$t=5$	-29.4	-24.6	-17.7	-10.3	-14
	$t=15$	-36.4	-67.6	-52.6	-28.3	-34.8
	$t=40$	-32.6	-67.4	-80.3	-86.8	-88.6
$\Delta^R_{opt/fix}w$		14.7	20.8	15.8	6.21	11.2
$\Delta^R_{opt/ir}w$		18.3	33.5	31.7	22.5	29.4
$\Delta^R_{opt/fix}c_{old}$		-1.17	-1.14	-4.66	-5.29	-9.54
$\Delta^R_{opt/ir}c_{old}$		0.672	-4.77	-10.9	-12.5	-17.4
$\Delta^R_{opt/fix}i$	$t=5$	-13.5	-13.3	-10.3	-4.56	-8.13
	$t=15$	-8.34	-34.9	-33.9	-4.56	-11.9
	$t=40$	39.7	66.4	71.5	47.5	20.6
$\Delta^R_{opt/ir}i$	$t=5$	-14.1	-17.4	-16.4	-11.8	-16.8
	$t=15$	-13.1	-58.6	-70.5	-42.2	-61.1
	$t=40$	21.5	40	41.8	-8	-54.1
$\Delta^R_{opt/fix}u$	$t=5$	-0.497	-0.418	-0.303	-0.146	-0.312
	$t=15$	-2.44	-3.5	-2.27	-0.833	-1.39
	$t=40$	2.27	2.23	-0.475	-1.12	-1.33
$\Delta^R_{opt/ir}u$	$t=5$	-0.51	-0.526	-0.463	-0.341	-0.534
	$t=15$	-2.76	-4.86	-4.13	-2.98	-3.77
	$t=40$	-0.904	-4.44	-8.24	-8.96	-8.9

Notes: Variation across the health authority endowment, a_0, per population member.

(Rows 1-3 and 4-6) $\Delta_{opt/.}a$: Absolute difference between assets under the optimal policy (opt) vs the fixed budget (fix) or interest-only (ir) after $t = 5, 15, 40$ periods.

(Rows 7-10) $\Delta^R_{opt/.}w$, $\Delta^R_{opt/.}c_{old}$: Relative difference between welfare (resp. cost of old treatment) under the optimal policy (opt) vs the fixed budget (fix) or interest-only (ir).

(Rows 11-22) $\Delta^R_{opt/.}i$, $\Delta^R_{opt/.}u$: Relative difference between infected share (resp. undetected one) under the optimal policy (opt) vs the fixed budget (fix) or interest-only (ir) after $t = 5, 15, 40$ periods.

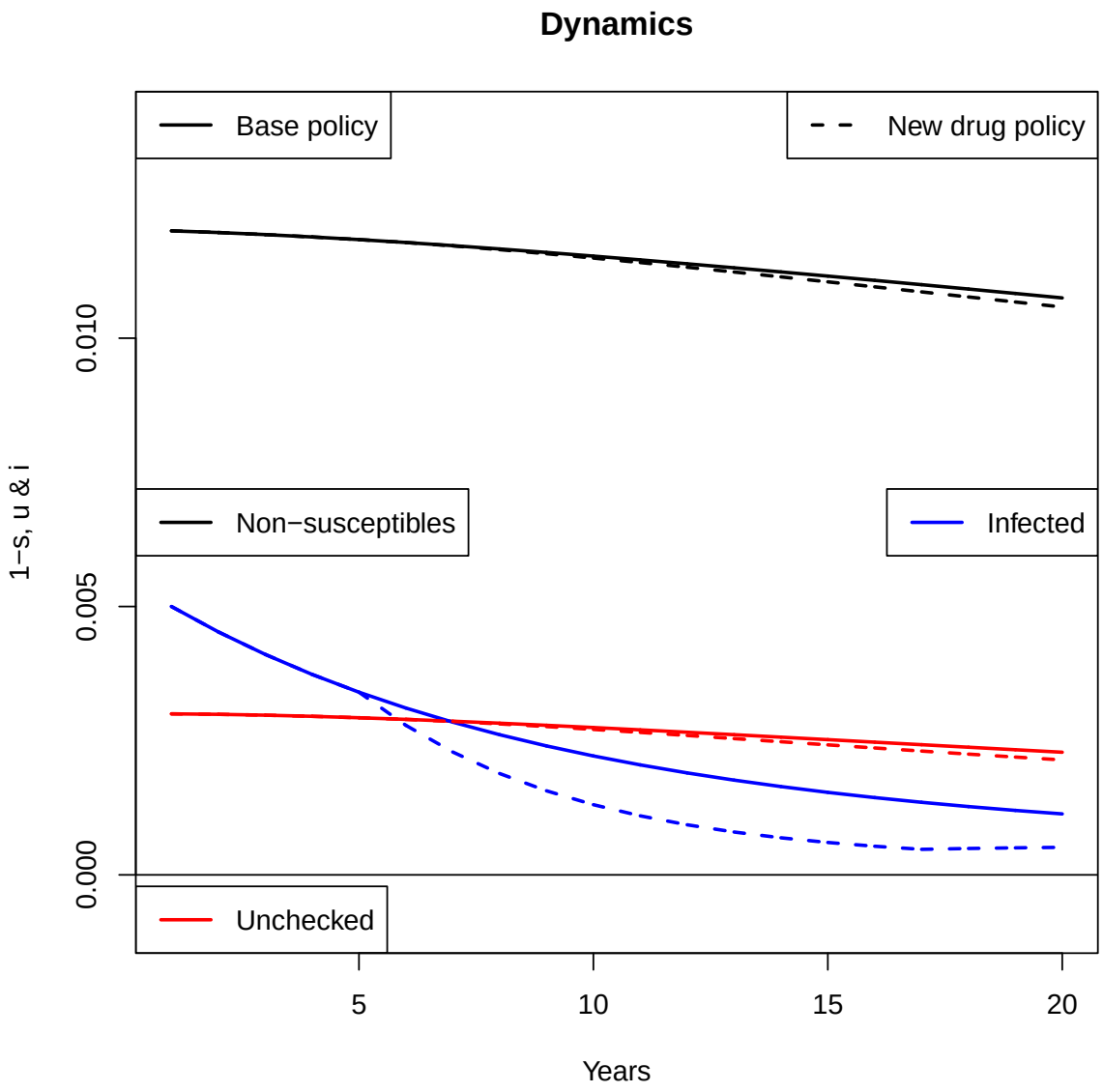
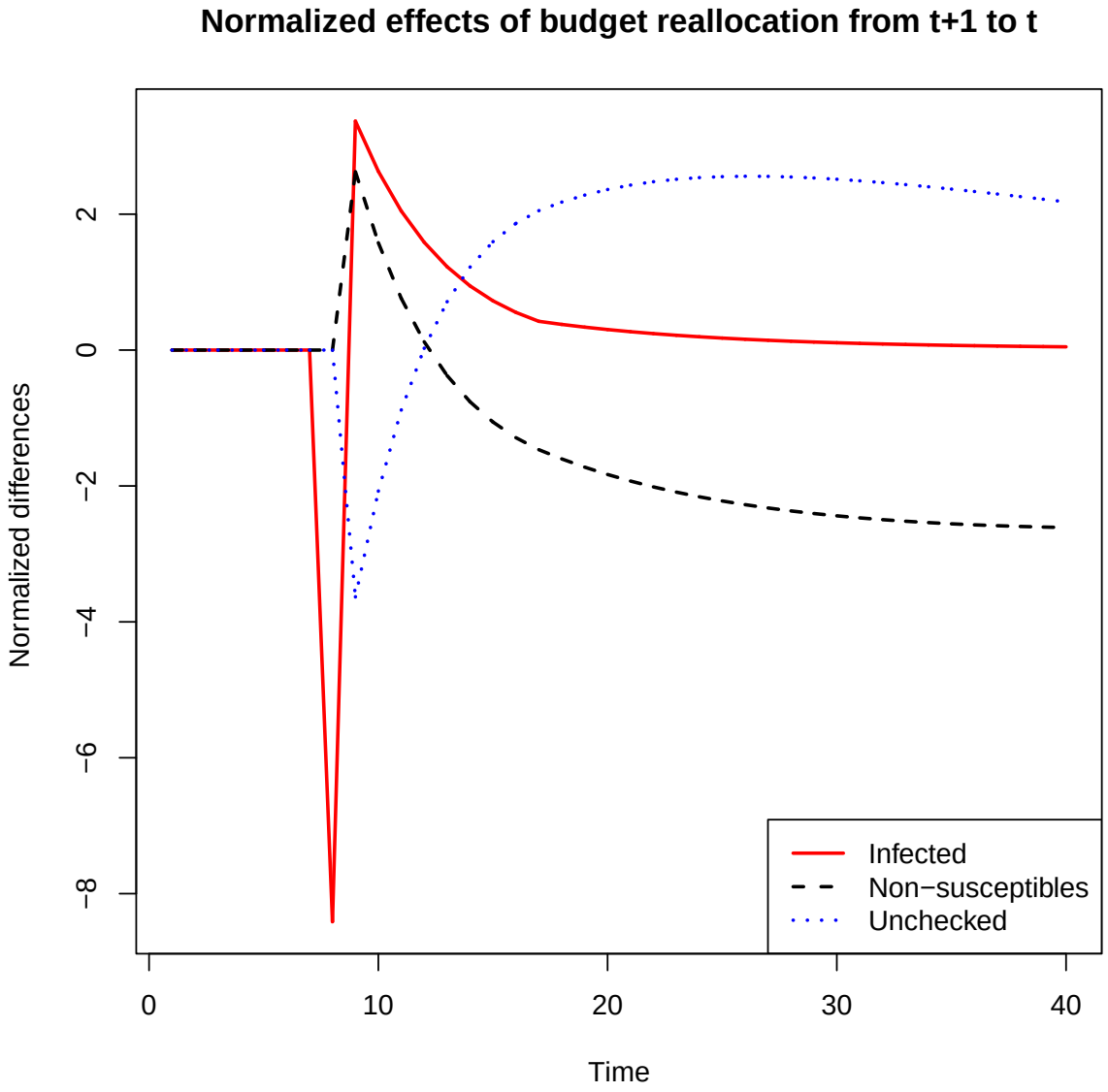
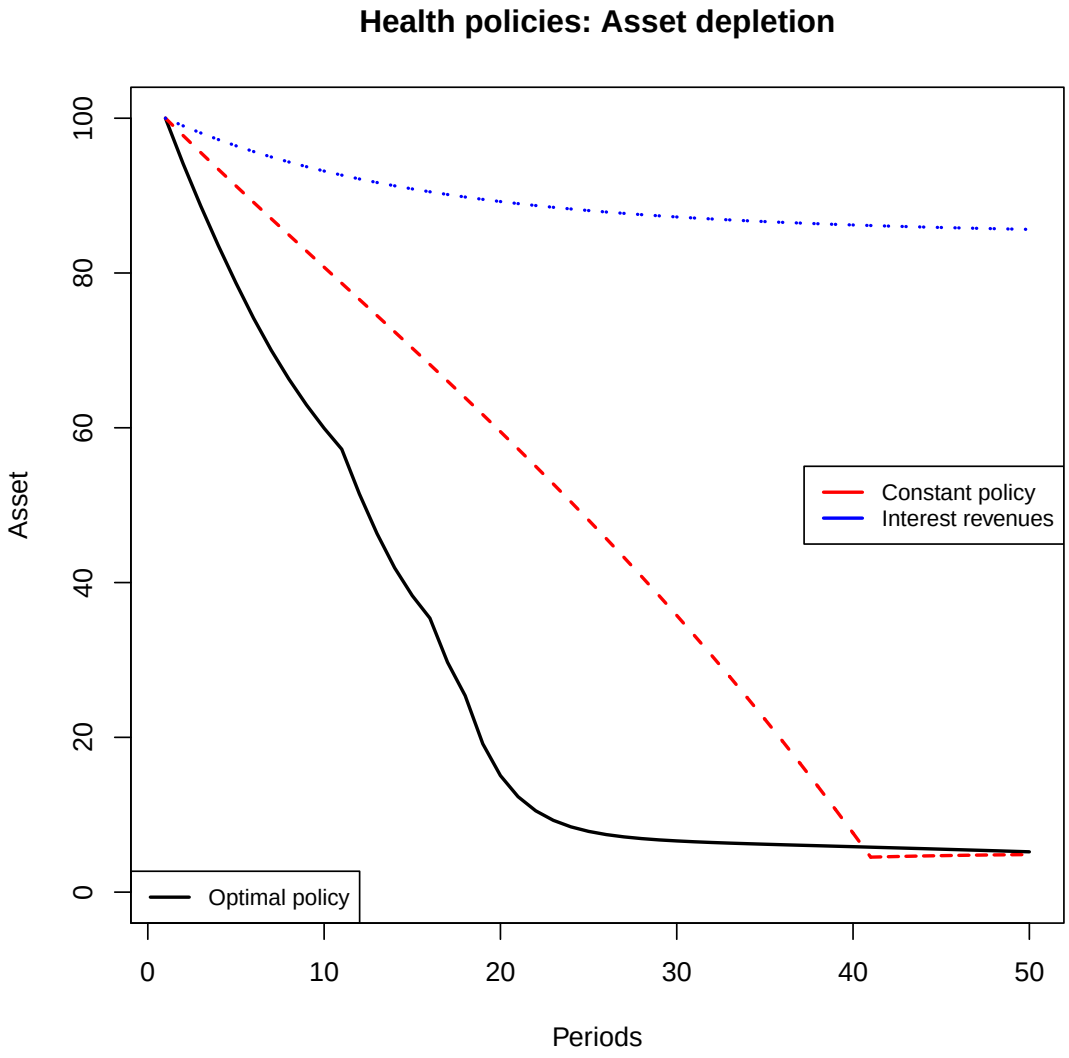


Figure 1: Infection and policy impact



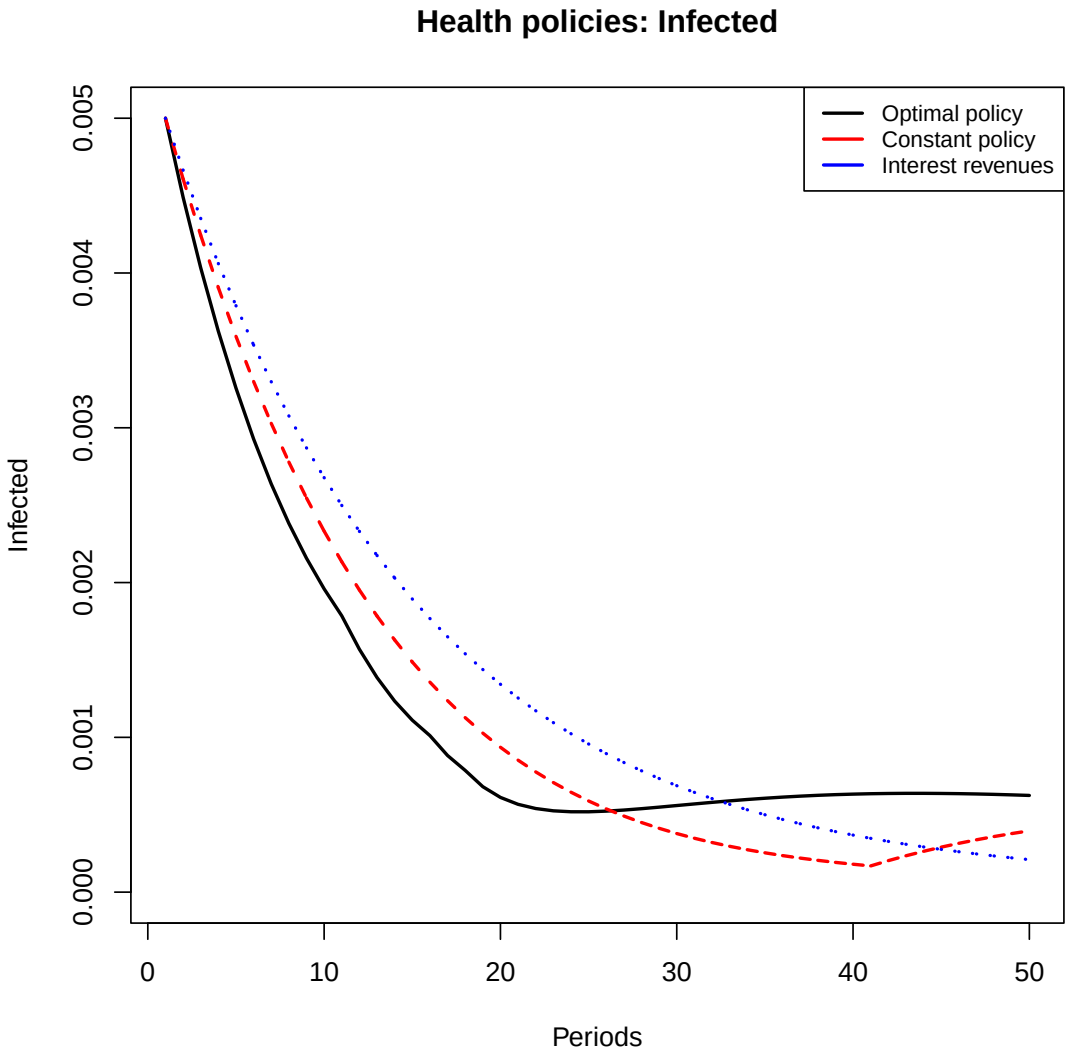
Note: The initial budget sequence is increasing at rate r. Budget at period 9 is fully reallocated at period 8. The impulse response functions have different scales which are not comparable since they vary widely across state variables.

Figure 2: Variational budget impact on state variables



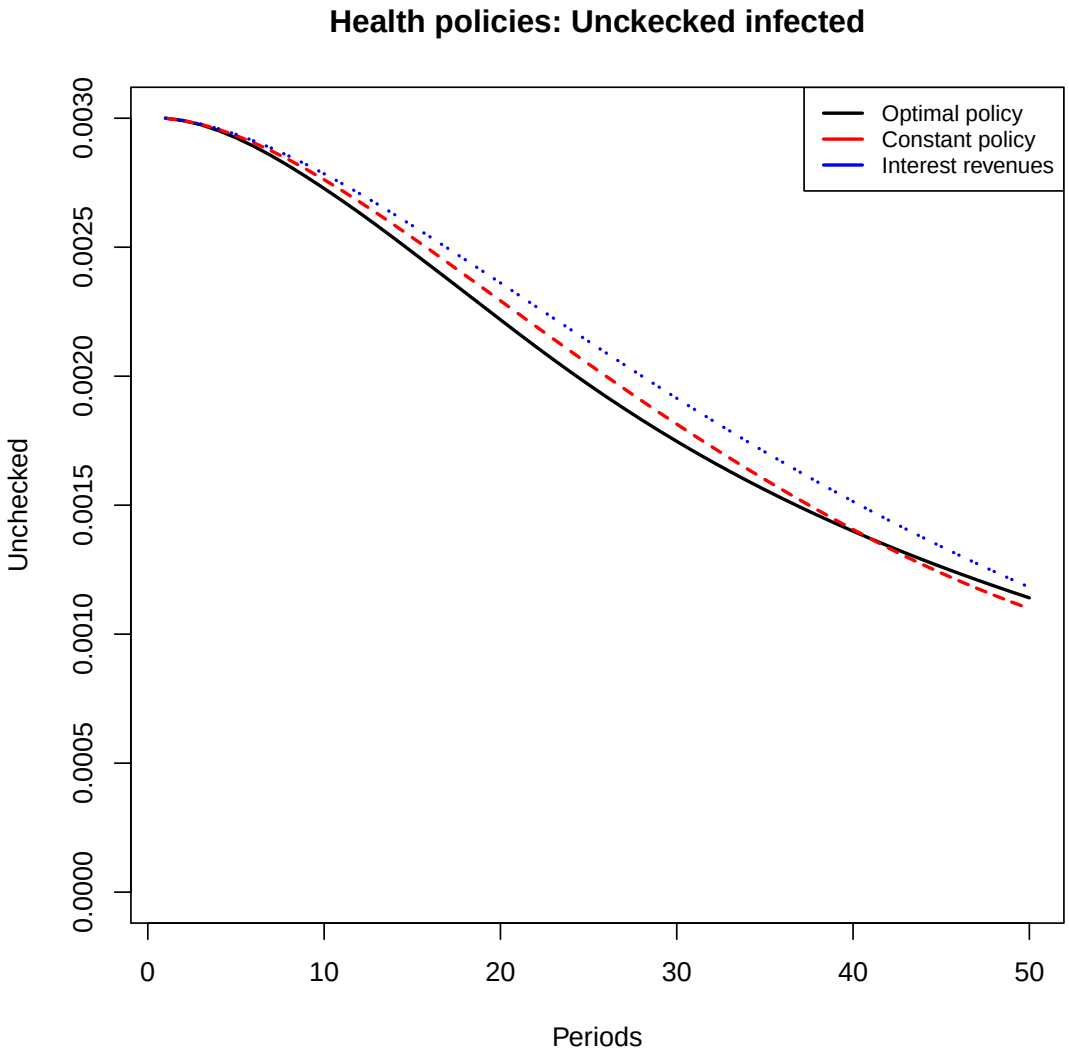
Note: Assets are normalized to 100 initially. Constant policy is the fixed budget policy and interest revenues is the one using interests only as defined in the text

Figure 3: Profile of asset decumulation over time



Note: Infected is the rate of infection in the population initialized at its calibrated value. Constant policy is the fixed budget policy and interest revenues is the one using interests only as defined in the text.

Figure 4: Profile of infected over time



Note: Unckecked infected is the rate of infection undetected in the population initialized at its calibrated value. Constant policy is the fixed budget policy and interest revenues is the one using interests only as defined in the text.

Figure 5: Profile of unchecked over time