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

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Abstract

Abstract: We study intertemporal tradeoffs that health authorities face when considering the control of an epidemic using innovative curative medical treatments. We set up a dynamically controlled susceptible-infected-recovered (SIR) model for an epidemic in which patients can be asymptomatic, and we analyze the optimality conditions of the sequence of cure expenses decided by health authorities at the onset of the drug innovation process. 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, the treatment for which 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. The analysis demonstrates how beneficial intertemporal budgeting can be compared to non-forward-looking constant budget allocation.

Keywords: pharmaceuticals, SIR model, controlled epidemic dynamics, optimal intertemporal policies, hepatitis C.

JEL codes: I12, I18

1   Introduction

Negotiated drug prices by healthcare authorities (HAs) are typically contingent on projected treatment demand and the therapeutic value for specific medical conditions. In a world where chronic ailments are managed with regular therapies, HA's dilemmas remain fairly consistent over time. However, novel and innovative treatments can disrupt these tradeoffs, particularly when they have the potential to outright cure chronic diseases. This can present a challenge for HAs when setting budgets, as the cost of treating a large number of patients may be prohibitive. This issue is likely to intensify as gene and cell therapies promise revolutionary advancements in healthcare, potentially providing cures for previously incurable chronic diseases that require long-term treatment.

These potentially groundbreaking 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 its budget depends on the likely 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 lifesaving 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 for a communicable disease become available 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, Hlávka et al., 2020). Hlávka et al. (2020) investigate the impact of intertemporal sequencing of treatment. They analyze the sequencing of current-period budgets expended for curing congestive heart failure (CHF) for Medicare beneficiaries in 2009-2014 in the US. They compare the status quo sequence equalizing budget per period to deferred payment schemes varying by the level of downpayments. They show, as we do, that patients would benefit from deferred payments. The main difference from our setting is their use of detailed micro data, while we use calibrated macro data and the fact that CHF is a non-contagious disease so that a susceptible-infected-recovered (SIR) model is not useful. Neither do the authors attempt to

derive the optimal sequence of treatment as we do by using welfare evaluations. 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 establish an SIR model for an epidemic, and in a simple setup, we analyze the optimality conditions of the sequence of cure expenses decided by HAs when a 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 et al., 2020, for a recent application to Covid).

These gains admittedly depend on disease and drug characteristics. We focus in this paper on the case of a grave illness, hepatitis C, whose treatment underwent a major upheaval when decisive curative drugs were introduced in 2014. The case of hepatitis C is informative because the management of the market introduction of therapeutic innovations well illustrates the intertemporal tradeoff between expending money on treating patients with new drugs in the present or waiting and treating them in the future. These new drugs however were quite expensive and gave rise to the question of the optimal policies to be chosen over time to master the epidemic in a cost-efficient way. Health authorities 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 despite 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 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 professionals, between the blood of infected and susceptible

persons. 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 increasingly 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 health authorities. We mainly focus on what health authorities decide at the onset of the introduction of a new drug on the market, and we leave to future research the additional tradeoff they may face when they know the available health technology will improve in the future, for example because they have good knowledge of forthcoming novel therapies. This obviously adds an additional aspect to the intertemporal tradeoff that depends on the decision-maker's information about the future. In the case of hepatitis C, the effect of anticipated future entries of new drugs could add complications that are left to future research. Here, we address what the optimal tradeoff is in 2014 between sequences of dynamic expenses while holding fixed expectations about the future – in particular innovative new drugs.

Our first contribution is to derive analytical results that characterize optimal policies using the calculus of variations in the dynamic problem. We show that moving backward expenses in new drugs while holding constant the intertemporal budget of health authorities reduces infection in the short run although there are rebound effects of the epidemic 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 epidemiological 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 awarding health authorities an endowment that they are free to expend in the short run if additional resources can be renegotiated in the medium run. Indeed, the optimal management of an intertemporal budget allocation decided ex ante for the treatment of a disease needs the ability to commit to a hard budget constraint that may be difficult to comply when it means spending less per patient in the future than in the past. This is however the lesson from this optimal allocation, which is optimal given an intertemporal hard budget constraint.

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 shows 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 the paper.

2   The setup

We start by setting up an 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 next turn to the description of the impact of an exogenous innovation process for drugs curing this disease. We discuss our main assumption that new drug-related expenses are increasingly less effective in the current period when the treatment is scaled up. We then present our specification for welfare and the calibrated values for parameters that we retain from the literature on hepatitis C in France, a typical Western European country for these matters.

2.1   The SIR model

In a population whose size is independent of time and normalized to 1, we denote the following state variables: s_t, the share of susceptibles; i_t, the share of identified infected referred to as infected; and u_t, the share of undetected infected referred to undetected. We set up the model in discrete time and write the transitions between states as displayed in Figure 1. We omit the share of recovered individuals because it is obtained by deduction from other shares, i.e., $1 - s_t - u_t - i_t$. We assume that recovered cannot become infected again by the disease, which is a simplification given that once a patient is cured, the average risk of recurrence is less than one percent (Simmons et al. 2016) and that the mortality rate, ν, is the same in all subpopulations. There exists a traditional drug that does not fully cure the disease although it keeps patients alive – as was the case with interferon-based treatments before the arrival of DAAs for hepatitis C, called the new drug in the following.

Other restrictions are presented after writing the dynamic equations of the SIR model displayed in Figure 1.

First, as susceptibles can become infected in a way proportional to the sum of infected and undetected rates in the population or can die and be replaced by newborns, the change in susceptibles

over time satisfies:

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

in which β is the strength of infection due to both infected groups, assumed to be equal across those groups, and ν is the natural death rate in this population. As shown in Figure 1, newborns replacing one-by-one deceased individuals are supposed to belong to susceptibles, and this explains the presence of the entry rate, ν, in this equation.

Infection is first undetected, and thus, the share of *undetected* 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 and become detected infected. The share of *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 attempt to control the spread of infection by the administration of drugs that lead to recovery. In the absence of new drugs and using traditional treatments only, we define below a lower bound $\rho^{(0)}$ for the healing rate ρ_t. By assumption, natural remission, $\rho^{(0)}$, or the channel to recovery, is not open to the undetected (e.g., Figure 1) whose only transition possibility is restricted to the (detected) infected. This assumption simplifies our setting and has no qualitative consequences for our conclusions since natural remission for undetected cannot be controlled by the HAs, or observed by the econometrician, and furthermore remains fixed in our developments.

The state variables are not only the SIR variables (s_t, i_t, u_t) but also the endowment of public funds at time 1 and denoted A_1, aimed at financing a policy of new drug 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 sequences of expenses over time that have a finite interest-rate-discounted sum equal to A_1. The endowment decision of the intertemporal budget A_1 is supposed to be done ex ante according to the public policy priorities and overall budget.

In the following, we will assume that the only stable stationary equilibrium, without any intervention, is the disease-free equilibrium, with $u_t = i_t = 0$ and $s_t = 1$, because this seems a reasonable assumption for the epidemic of hepatitis C at least in western European countries (Roudot-Thoraval, 2021) even if in-migrations that are not modeled here might delay the process.

A sufficient condition for this stable stationary equilibrium to be disease free 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 $s_t \geq \nu$ (e.g., newborns are susceptibles), $u_t \geq 0$, $i_t \geq 0$ and $s_t + i_t + u_t \leq 1$.

2.2 Policies and intertemporal budgets

The policy implemented by health authorities is described by the expenses on new and traditional drugs in period t that are denoted B_t. We compute these expenses according to the decomposition of the (detected) infected subpopulation in four groups according to their "types" and report it in Table 1:

Table 1: Expenses for the infected sub-population

Types	Share	Cost per patient	Total Cost	Destination
Natural remission	$\rho^{(0)}$	0	0	Recovery
Treated: new drugs	$\rho_t - \rho^{(0)}$	$c_{new}(\rho_t)$	$c_{new}(\rho_t)(\rho_t - \rho^{(0)})$	Recovery
Treated: traditional	$1 - \nu - \rho_t$	c_{old}	$c_{old}(1 - \nu - \rho_t)$	Infected
Deceased	ν	0	0	Deceased

in which c_{old} is the per period cost of the traditional treatment and $c_{new}(\rho_t) > c_{old}$ is the cost of the more expensive innovative treatment. The cost of the latter is increasing with the exit rate, ρ_t, in a way described below. In Table 1, we used the convention that types are known at the beginning of the period and that costs for natural remission and deceased are set to zero. This means that costs are generated by the treatments using the new and traditional drugs. The treatment cost for each infected is:

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

We now isolate each of these two components of the cost, the first being in the hands of the HAs while the second remains fixed.

2.2.1 Policy variables

Define b_t, the additional budget expended on the new drug per infected patient, as:

$$b_t(\rho_t) = \left(c_{new}\left(\rho_t\right) - c_{old}\right)\left(\rho_t - \rho^{(0)}\right), \tag{4}$$

as well as the traditional drug cost of a treated patient

$$c_0 = (1 - \nu - \rho^{(0)})c_{old}. \tag{5}$$

Therefore, average expenses per (detected) infected patient in period t are equal to $b_t + c_0$, and as i_t is observed, full expenses B_t are:

$$B_t = (b_t + c_0)i_t.$$

Note that setting policies of the HAs in terms of b_t or B_t is equivalent since c_0 and i_t are given. The new drug budget variable, b_t, affects the healing rate of the infected through the remission rate, ρ_t.

The interpretation of Equation (4) is straightforward. When $\rho_t = \rho^{(0)}$, new drugs are not used and $b_t(\rho^{(0)}) = 0$. When positive, $b_t(\rho_t)$ is increasing and convex with ρ_t as $c_{new}(\rho_t)$ is increasing. Under these conditions, we can invert Equation (4) to obtain $\rho_t = \rho(b_t)$, which turns out to be a more natural and convenient mapping to work with since it maps expenses, b_t, a policy in the hands of the HAs, into a remission rate ρ_t.[1] This is the function that we now specify.

2.2.2 Effectiveness of the new drug treatment

We specify function $\rho(b_t)$ defined above as satisfying the following characterization:

Assumption D(ecreasing Returns to Scale)

1. $\rho(0) = \rho^{(0)}$, $\rho(+\infty) = 1 - \nu$

2. $\rho(.)$ is increasing with a continuous derivative,

3. $\rho(.)$ is concave.

In Assumption D.1, the natural remission rate is denoted $\rho^{(0)}$ and defines the healing rate in the case where 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, $b_t^{(1)} + c_0 = \frac{B_t}{i_t^{(1)}} > \frac{B_t}{i_t^{(2)}} = b_t^{(2)} + c_0$, is larger in the second case, $\rho'(b_t^{(1)}) < \rho'(b_t^{(2)})$.

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[1] Functions $c_{new}()$ and $\rho()$ are assumed to be time independent. Taking into account the expected drug innovation process in the future by competing drug producers is left for future research. All tradeoffs over time are here summarized by the impacts of moving expenses across time periods conditional on a fixed new drug treatment efficiency.

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 increasingly more on average makes the treatment increasingly less effective. Second, better targeting of heterogenous patients makes the treatment more effective, but the 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.

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 to future work how this mechanism precisely works.

The intertemporal budget A_1 or total endowment received by the health authorities in the first period can be spent in a sequence of drug expense budgets $(B_1, ., B_t, ...)$ such that their discounted sum using the interest rate r, is

$$A_1 \equiv \sum_{t=1}^{\infty} \frac{B_t}{(1+r)^t} = \sum_{t=1}^{\infty} \frac{(b_t + c_0)i_t}{(1+r)^t}, \tag{6}$$

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 of a sequence of new drug expenses, b_t, among *different* sequences, e.g., front loaded, constant or backloaded sequences among many others, which all have the same discounted value equal to A_1.

2.3 Social welfare and optimal policy

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 both lead to the same total intertemporal budget $\sum_{t=1}^{\infty} \frac{(b_t+c_0)i_t}{(1+r)^t} = \sum_{t=1}^{\infty} \frac{(\tilde{b}_t+c_0)i_t}{(1+r)^t} = A_1$. This evaluation is derived from a specific social welfare function, W, that depends on state variables (A_1, s_1, i_1, u_1) in the first period and on the sequence of a given policy followed over time, $b = (b_1, ., b_t, ...)$. We assume here that social welfare, W, is additively separable over time in each-period instantaneous welfare, $v(.)$, which depends only on the end-of-period share of infected, i_{t+1}:

$$W(A_t, s_t, i_t, u_t; b) = \sum_{\tau=t}^{\infty} \delta^{\tau-t} v(i_{\tau+1}). \tag{7}$$

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 larger the infection, the larger the marginal dislike. The simplest example of such a specification is a quadratic function, $v(i_{t+1}) = -(i_{t+1})^2/2$.

We then look for the optimal policy on the budget sequences assuming that the initial endowment is larger than what would be needed to treat all detected patients with the traditional drug and that at each period in the future, the remaining intertemporal budget is large enough to treat all with the old drug. It is indeed likely that it would not be acceptable to spend too much and then not have enough budget to treat patients at least with the old technology. We thus introduce this constraint, which is equivalent to assuming the welfare cost of no treatment is almost infinite. Thus, we have additional constraints on A_t because HAs are willing to make provisions for expenses on traditional drugs in each period. Since the new drug cure is effective and more expensive than the traditional drug, it is enough to compute the provisions for traditional drugs assuming that no expenses on new drugs are made after period t and that only the cost of traditional drugs matter. Denote those provisions for all t:

$$\kappa_t(s_t, i_t, u_t) = c_0 \sum_{n=t}^{\infty} \frac{i_n}{(1+r)^{n-t+1}}, \tag{8}$$

in which c_0 is given by Equation (5). Since our conditions lead to the eradication of the disease, this function is always well defined. Furthermore, we will assume in the following that the HA's initial endowment is high enough that it covers at least the traditional drug provisions and $A_1 \geq \kappa_1(s_1, i_1, u_1)$.

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

$$
\begin{aligned}
W^*(A_1, s_1, i_1, u_1) &\equiv \max_b W(A_1, s_1, i_1, u_1; b) = \max_b \sum_{\tau=1}^{\infty} \delta^{\tau-1} v(i_{\tau+1}), \\
\text{subject to } \rho_t &= \rho(b_t), \ (1), (2), (3), \ A_{t+1} \geq \kappa_{t+1}(s_{t+1}, i_{t+1}, u_{t+1}), \\
\text{and } A_{t+1} &= (1+r)A_t - (b_t + c_0)i_t, \text{ for all } t.
\end{aligned}
$$

Because all objects are stationary, by the Bellman principle, this is equivalent to solving for any $A_1 \geq \kappa_1(s_1, i_1, u_1)$:

$$
\begin{aligned}
W^*(A_1, s_1, i_1, u_1) &= \max_{b_1}(v(i_2) + \delta W^*(A_2, s_2, i_2, u_2)) \tag{9} \\
\text{subject to } \rho_1 &= \rho(b_1), \ (1), (2), (3) \text{ at time } t = 1, \\
\text{and } A_2 &= (1+r)A_1 - (b_1 + c_0)i_1 \geq \kappa_2(s_2, i_2, u_2).
\end{aligned}
$$

In this formulation, note that all parameters are known by HAs and that for instance, the rate of undetected u_t is supposed to have been learned or estimated in the past, which is a reasonable hypothesis given the evaluations of these rates described in next section.

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. 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 third that u_t is unknown with some prior distribution. We leave these alternatives to further research.

2.4 Descriptive statistics and calibrated parameters

We gathered various statistics from different sources that allow the values of parameters of interest to be calibrated 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 infection by hepatitis C in Europe and the world can be found in Roudot-Thoraval (2021). The figures we extract from this paper are related to the French population aged 18 to 75 or 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 seroprevalence was 0.84% (Roudot-Thoraval, 2021, p5). According to Roudot-Thoraval (2021), the respective estimates for 1994 are 1.1% for anti-HCV prevalence and 0.86% for seroprevalence.[2] HCV antibody 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 considerably better control of the blood transfusion channel while transmissions through intravenous drug use and mother-to-infant remained important (Roudot-Thoraval, 2021). The decrease after 2014 was caused by the adoption of 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 aware of their current infection was equal to 80.6%, although

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

with a large confidence interval [44.2-95.6]. In other surveys, this figure can be much lower (57% in Bruggman et al., 2014) and as low as 50% (Bottero et al., 2016). This is why we calibrate $\zeta = 0.03$ 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 calibrate. 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 that 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 18 years old and assuming that at 18, life expectancy is 60, it gives 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.[3] The fact that this is a combination of two transitions, from susceptible 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.

For interest rates, we adopt the average value of long-run rates in 2014, which was approximately 2%.[4]

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

$$\rho(b) = \rho^{(0)} + (1 - \nu - \rho^{(0)}) \left[1 - \exp(-\lambda b^\alpha)\right], \tag{10}$$

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

We first retain a value for the natural remission rate of $\rho^{(0)} = 0.03$, slightly less than what Roudot-Thoraval (2021) reports for severe developments of the disease.[5] We also retain a value for α at approximately 0.5, although uncertainty is pervasive so that we assess results with respect to it below.

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

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

[5] When experimenting with different values in simulation, we found that larger values are driving the epidemic more quickly to zero than what can be observed in the data.

For calibrating λ, we use more detailed information on the costs of the new drugs (DAAs) and the traditional interferon treatment. Inverting Equation (10) should match Equation (4) so that we obtain:

$$b(\rho_t) = \left[-\frac{1}{\lambda} \log(1 - \frac{\rho_t - \rho^{(0)}}{1 - \nu - \rho^{(0)}}) \right]^{1/\alpha} = (c_{new}(\rho_t) - c_{old})(\rho_t - \rho^{(0)}), \qquad (11)$$

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

$$c_{new}(\rho_t) - c_{old} = \frac{\left[-\frac{1}{\lambda} \log(1 - \frac{\rho_t - \rho^{(0)}}{1 - \nu - \rho^{(0)}}) \right]^{1/\alpha}}{\rho_t - \rho^{(0)}}. \qquad (12)$$

We now calibrate $\left(c_{new}(\rho^{(0)}) - c_{old} \right)$, the difference between the marginal cost of the new drug when no one is treated and the constant marginal cost of the old treatment. This leads to the calibration of λ given α. Our main source is the measured ratio of differential cost and outcome in terms of quality of life evaluated by health authorities which 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 patient has a QALY evaluated at approximately 0.5 on average (HAS 2014, Avis d'Efficience Sovaldi (Sofosbuvir)) implying that $QALY_{new} - QALY_{old} = 0.5$. The ratio RDCR is approximately 21,000 euros per QALY for Sofosbuvir, the cheapest for most genotypes. This leads to an average differential cost of treatment of $c_{new}(\rho^{(0)}) - c_{old} = 21,000 \times 0.5 = 10,500$ euros. By Equation (12), we can derive that 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:

$$\frac{1}{\lambda} = (1 - \nu - \rho^{(0)}) * \frac{10500^\alpha}{(0.001)^{1-\alpha}},$$

using the previous calibrated parameters and a small level of treatment $\rho_t - \rho^{(0)} = 0.001$ or 20% of the infected. This small level of treatment corresponds roughly to the estimated share of treated by new drugs by Roudot-Thoraval (2021) (7% of the infected in 2014 and 19% in 2017).

Another important budget parameter is the cost of the old treatment. Indeed, the value of $b_t + \left(1 - \nu - \rho^{(0)} \right) c_{old}$ is the average budget per infected that allows one 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} using a value of approximately 10,000€, say 10,500 €. 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 approximately 5,000 € per inhabitant, and we assess the sensitivity of our results to some of those budget parameters.

Our calibrated parameters are reported in the following Table 2.

Table 2: Calibrated parameter values

Name	Parameter	Value
Infection rate	β	0.015
Mortality rate	ν	0.0125
Detection rate	ζ	0.03
Remission rate	ρ_0	0.1
Interest rate	r	0.02
Discount rate	δ	$1/(1.04)$
Susceptibles: $t = 0$	s_0	0.988
Undetected: $t = 0$	u_0	0.003
Infected: $t = 0$	i_0	0.005
New drug: marginal cost	$c_{new}(\rho_0)$	21,000€
Old drug: marginal cost	c_{old}	10,500€
Efficiency parameter	α	0.7

3 Analytical results

Before turning to simulations that allow us to better understand 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 sequence, and consider an alternative policy in its neighborhood in the sense that policies differ slightly at periods t and $t+1$ only and are such that the budget constraint (6) is satisfied. Formally, let $B_t = (b_t + c_0)i_t$ (respectively $B_t + dB$) and B_{t+1} (respectively $B_{t+1} - (1+r)dB$) deliver the same intertemporal budget equation:

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

The two sequences $(B_1, ..., B_{t-1}, B_t, B_{t+1}, B_{t+2}...)$ and $(B_1, ..., B_{t-1}, B_t+dB, B_{t+1}-(1+r)dB, B_{t+2}, ...)$ are intertemporally equivalent in terms of endowments, and we compare their consequences in terms of susceptibles, s_{t+k}, and infected, u_{t+k}, and i_{t+k} for any future period after t $(k > 0)$ when we fix state variables, (s_t, u_t, i_t).

Denote dX the variation in a variable X with respect to the benchmark (e.g., dB) given that (s_t, u_t, i_t) is fixed. Specifically, in terms of new drug expenses per patient b_t, we can write:

$$\begin{cases} dB_t = dB = & db_t i_t, \\ dB_{t+1} = -(1+r)dB = & db_{t+1}i_{t+1} + (b_{t+1}+c_0)di_{t+1}, \\ \text{For } k > 1, \ dB_{t+k} = 0 = & db_{t+k}i_{t+k} + (b_{t+k}+c_0)di_{t+k}. \end{cases}$$

so that:

$$\begin{cases} dB_t = dB = & db_t i_t, \\ db_{t+1}i_{t+1} = & -(1+r)dB - (b_{t+1}+c_0)di_{t+1}, \\ db_{t+k}i_{t+k} = & -(b_{t+k}+c_0)di_{t+k}. \end{cases} \tag{13}$$

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

The short-run effects Without loss of generality, suppose that $dB > 0$, so that we front load expenses backwards in time. In period $t+1$, we have by Equation (3) :

$$\begin{aligned} di_{t+1} &= -(d\rho_t)i_t, \tag{14} \\ &= -\rho'_t i_t db_t = -\rho'_t dB < 0. \end{aligned}$$

As expected, the share of infected decreases in period t when the budget expended on new drugs is larger and the magnitude of the effect is proportional to the marginal effectiveness (ρ'_t) of new drugs. 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 $s_{t+1} = s_t$.

In period $t+2$, things become more interesting because the impact on infected is affected by the budget decrease in period $t+1$. The full derivation of the impact is summarized as:

Lemma 1 *Define for any $b \geq 0$:*

$$a(b) = 1 - \nu - (\rho(b) - (b+c_0)\rho'(b)), \tag{15}$$

By Assumption D, $a(b)$ is nonnegative and decreasing in b. Furthermore,

$$di_{t+2} = (\rho'_{t+1}(1+r) - a(b_{t+1})\rho'_t)dB. \tag{16}$$

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Proof. See Section B.1. ∎

The interpretation of Equation (16) is as follows. First, when expenses in the two periods are equal ($b_t = b_{t+1}$), so that $\rho'_{t+1} = \rho'_t$, there could be a rebound effect of the share of infected at $t + 2$ (e.g., $\frac{di_{t+2}}{dB} > 0$) depending on whether $1 + r > a(b_{t+1})$ that is, if

$$r + \nu + (\rho(b_{t+1}) - b_{t+1}\rho'(b_{t+1})) - c_0\rho'(b_{t+1}) > 0. \tag{17}$$

If $c_0 = 0$, the rebound effect is always positive, because $\rho_{t+1} - b_{t+1}\rho'_{t+1} > 0$ (see Proof of Lemma 1). The existence and size of the rebound effect thus depends on how small the cost parameter of the traditional treatment c_0 is and on how effective ($\rho'(b_{t+1})$) the new drug treatment is. The size of the rebound effect has other sources. A positive interest rate makes the rebound effect larger since shifting budget from the future ($t + 1$) to the present (t) leads to comparatively lower expenditure in the present due to foregone interest. The other term of Equation (17), $\rho_{t+1} - b_{t+1}\rho'_{t+1} > 0$, is related to the curvature of function $\rho(.)$ and the more curved it is, the larger the rebound effect.

This discussion holds true if $b_t > b_{t+1}$ since $\rho'_t < \rho'_{t+1}$. 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 become negative even if $c_0 = 0$.

Before investigating longer term dynamic effects, it is interesting to consider the impact of these changes from 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 (16), we obtain:

$$di_{t+2} = 0 \implies \rho'_t = \rho'_{t+1}\frac{1 + r}{a(b_{t+1})}. \tag{18}$$

A budgeting rule that would make $di_{t+2} = 0$ sets ρ'_t to a value larger than ρ'_{t+1} and, therefore, a b_t lower than b_{t+1} when $a(b_{t+1}) < 1 + r$. Nonetheless, this rule neglects the dynamic returns to the change of policy, through the lower infection rate at period t that affects susceptibles and undetected at period t.

The infection channel Another channel operates through infections, and its presence 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:

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

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

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

that by recomposing, using the equations above, and line 3 in Equation (13) yields:

$$
\begin{aligned}
di_{t+3} &= \zeta du_{t+2} + (1 - \rho_{t+2} - \nu)di_{t+2} + \rho'_{t+2}(b_{t+2} + c_0)di_{t+2}, \\
&= -\zeta\beta s_t\rho'_t dB + (1 - \rho_{t+2} - \nu + \rho'_{t+2}(b_{t+2} + c_0))di_{t+2}, \\
&= -\zeta\beta s_t\rho'_t dB + a(b_{t+2})di_{t+2},
\end{aligned}
$$

Note that the first term is negative while the second term is positive or negative since $a(b_{t+2}) > 0$, and di_{t+2} is positive or negative depending on our discussion after Lemma 1.

If we proceed in a similar way to what leads to Equation (18), 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 obtain:

$$di_{t+2} = \frac{\zeta\beta s_t\rho'_t}{a(b_{t+2})}dB > 0.$$

so that using Equation (16):

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

and:

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

Compared to Equation (18), this means that it is more likely that $a(b_{t+1}) + \frac{\zeta\beta s_t}{a(\rho_{t+2})} > 1 + r$ since the second term is positive and that affects the optimal schedule of b_t.

We can continue with further dynamics, although the conclusion is that the effect of moving expenses over time depends on the parameters of the model in a complex way. We now turn to the evaluation of social welfare by HAs that would lead to the choice of an optimal solution.

3.2 Welfare evaluation

As welfare is defined in Equation (7), its variation with respect to detected at any period is given by:

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

Given the results in Section 3.1 and Equation (15) defining function $a(.)$, we have derived above that:

$$di_{t+1} = -\rho'_t dB,$$
$$di_{t+2} = (\rho'_{t+1}(1+r) - a(b_{t+1})\rho'_t) dB,$$
$$di_{t+3} = -\zeta\beta s_t \rho'_t dB + a(b_{t+2}) di_{t+2}.$$

We can thus examine short- and medium-run welfare gains and losses.

Short-run welfare gains For simplicity, 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 (two periods) as defined by:

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

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:

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

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 $\delta a(b_{t+1})\rho'_t$ and is decreasing with the value of ρ_{t+1} since $a(b_{t+1})$ and ρ'_t are both decreasing.

  - If $b_t > b_{t+1}$ and thus $\rho'_t < \rho'_{t+1}$:, the previous gain is attenuated and disappears eventually.

  - If $b_t < b_{t+1}$ 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.

  – If $b_t = b_{t+1}$ 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 $\delta a(b_{t+1})\rho'_t$.

  – If $b_t > b_{t+1}$ and thus $\rho'_t < \rho'_{t+1}$:, the previous gain is attenuated and disappears eventually

  – If $b_t < b_{t+1}$ 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.

  – If $b_t = b_{t+1}$ 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 $\delta a(b_{t+1})\rho'_t$.

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

Thus, when considering short-term effects only, having a front loaded budget is always optimal unless the infection is in an increasing swing, in which case it may be better to spend more in the future. However, medium- and long-term welfare effects may be important and change this conclusion.

Medium-term welfare gains   Consider the impact in the medium run (3 periods) as defined by:

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

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

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

The last term contributes 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 in period $t+1$ and contributing through the term $i_{t+3}a(b_{t+2})$.

Overall, it seems intractable to assess the relative magnitude of these effects. This conclusion also applies to the construction of optimal policies (see Dubois and Magnac, 2023). This is why we now turn to simulations in a realistic context that evaluate the optimal expense policy when new drugs against hepatitis C were introduced in 2014. Specifically, we can go beyond the case $\delta = 1/(1+r)$ analyzed in this Section by allowing for more realistically calibrated $\delta < 1/(1+r)$ (see Table 2).

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 now proceed by simulation. We first capture the diffusion of the epidemic 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 an expense policy and illustrate the principle of variational calculus that underpinned Section 3. We then turn in Subsection 4.2 to the simulation of different policies including the optimal policy.

4.1   Forward simulation: Controlling the epidemic

In these simulations, we attempt to model what happened in 2014 for hepatitis C in France when a new generation of drugs, which permanently cured some patients, was introduced, while previous drugs were not leading to remission before 2014.

We start with a simple example in Figure 2 using the calibrated parameters for hepatitis C presented in Section 2.4. We first simulate the evolution of shares of non-susceptibles $1 - s_t$, infected and undetected in the SIR model over a time span of 20 years in the absence of the new drugs but using traditional drugs (based on interferon-based treatments). As displayed by the solid line in Figure 2, the shares of infected (in blue), undetected (in red) and of non-susceptibles (in black) all slowly decrease over time, signaling the predicted eradication of the disease in the long run (Appendix A and Roudot-Thoraval, 2021). Second, we assume that the new DAA drug treatment curing hepatitis C is introduced from period 5 until period 17 (both arbitrarily) using a fixed budget sequence for the new drug $(b_5 i_5, \cdots, b_{17} i_{17})$ increasing at rate r so that within-period

budgets are constant in period 0-euros, and we simulate the structural break in the epidemic at time 5 as displayed by the dotted curves in Figure 2. As expected, the negative impact on the share of infected (in blue) is strong and lasting. For undetected and non-susceptibles, the impact is much less significant because it is indirect only and operates through the strength of the infection of susceptibles.

For concreteness, we next analyze the effect of a change in the budget between time $t+1$ and time t, when $t = 8$ (arbitrarily), holding the total endowment constant so that we can illustrate the analytical results developed in Section 3.1. In this example, the reallocation is small so that the budget at time $t = 8$ is increased by 10%. As presented in Figure 3 following the approach of impulse function responses in macroeconomics, the impact at time $t = 8$ on the share of infected (solid curve) is strong and negative as expected, while the rebound effect shifting upwards at time $t + 1$ is also marked (e.g., Lemma 1). Infection afterwards slowly decreases and asymptotically almost returns 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 toward the asymptote is much slower than the infected. Overall, the effect on the share of susceptibles is a combination of the two previous impulse response functions.

The conclusion of this analysis is that front loading expenses substantially improves the infection rate in the short run, although a rebound effect exists in the medium run. This analysis remains partial since we consider 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.

The accumulation rule for the budget is written as:

$$A_{t+1} = (1 + r)A_t - (b_t + c_0)i_t, \tag{19}$$

and the decision variable is b_t. Provisions should be made however to cover the baseline costs of the traditional drug at any time t as in Equation (8):

$$\kappa_t(z_t) = c_0 \sum_{n=t}^{\infty} \frac{i_n}{(1 + r)^{n-t+1}}, \tag{20}$$

in which $z_t = (s_t, i_t, u_t)$, and the constraint $A_t \geq \kappa_t(z_t)$ for all t appears in the decision program described by Bellman equation (9) and having four state variables (A_t, s_t, i_t, u_t).

Because the environment is asymptotically stationary, we adopt the strategy of iterating over the value function (taking into account optimal policy functions to accelerate the procedure when the optimal policy is used) 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.[6] Nonetheless, we need to first compute the baseline costs in Equation (20), and this is done by simulating the SIR model with various initial conditions. The second step is a fully standard value and policy function iterative process as given by Equation (9). The algorithm used is detailed in Appendix C.

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, $B_t = r(A_t - c_0 i_t)$, subtracting the cost of the traditional treatment. This policy is 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 expended.

- The optimal policy given by the social welfare function we have posited, called the *optimal policy* henceforth.

- An ad hoc *fixed budget annual 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.

We analyze the impact of the optimal policy in contrast with the fixed annual budget policy – that neutralizes the monetary endowment effect at period 40 – as well as in contrast with the interest-only policy. We proceed by displaying results graphically in Figures 5 and 6 as well as presenting numerical results in Table 3. Figure 4 reports the trajectories of assets under these three different policies. By construction, the interest-only policy is conservative and does not fully

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

consume the endowment in contrast with the two other policies. Figure 4 shows that the optimal policy front loads expenses with respect to the fixed annual budget policy. In other words, it is optimal to spend more in the early time periods while optimal expenses are much lower when approaching the 40-period horizon.

Optimal 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 scenarios in Figure 5. The interest-only and the fixed annual budget policies have a lower effect than the optimal policy on infection at least until period 25 for the fixed budget policy or 33 for the interest-only policy. 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.

Table 3: Comparisons between optimal policy vs. fixed budget and interest only policies

Statistics	Optimal vs. Fixed Budget	Optimal vs. Interest payments
$\Delta^R Welfare$	13.5%	28.7%
$\Delta^R C_{old}$	-4.6%	-12.8%
$\Delta i_{[1:40]}$	-0.332	-1.44
$\Delta^R i_5$	-8.42%	-14.7%
$\Delta^R i_{15}$	-34.2%	-73.4%
$\Delta^R i_{40}$	74.5%	46.2%
$\Delta^R u_5$	-0.224%	-0.391%
$\Delta^R u_{15}$	-2.08%	-4.03%
$\Delta^R u_{40}$	-0.446%	-8.31%

Notes: $\Delta^R Welfare$: Relative welfare improvement of the optimal policy; $\Delta^R C_{old}$: Relative improvement of costs expended on the traditional drug of the optimal policy; $\Delta i_{[1:40]}$: absolute cumulative improvement of the share of infected over the first 40 periods; $\Delta^R i_t$: relative improvement of the share of infected at period t; $\Delta^R u_t$: relative improvement of the share of undetected at period t.

Table 3 (first row) reports a relative improvement in welfare of 13.5% (respectively 28.7%) with respect to the fixed annual budget suboptimal policy (the interest-only policy). The decrease in costs paid for the traditional drug treatment (second row) of -4.6% (-12.8%) contributes to this improved welfare. Overall, the cumulative improvement in terms of shares of infected is equal to -0.33% (-1.44%) in Table 3 (row 3) that equalizes expenses between the optimal and fixed annual budget policies over the first 40 periods. This Figure is to be compared with an initial infection rate of 0.5% for the infected and 0.3% for the undetected.

The infection rate is lower by 8.42% (14.7%) in period 5 and lower by 34.2% (73.4%) in period 15. Costs of the optimal policy come in the longer run since the total endowment is fixed. This means that the eradication of the disease when using the optimal policy takes longer than the other policies to minimize the rate of infection in the short run. At period 40, this materializes in a strong rebound effect as an increase in the infection rate of 74.5% (46.2%) although at levels that are considerably smaller. This might involve the absence of credibility of the hard budget constraint.

As optimality is geared toward detected infected, it is not surprising that the gains in terms of undetected patients are substantially less impressive as reported in Figure 6 and Table 1 (last three rows). This is also true for non-susceptibles in a graph that is not displayed here. For undetected also, the main gains appear at the beginning. The relative decrease in the share of undetected is -0.2% when compared to the fixed annual budget policy (-0.4 for the interest-only policy) at period 5, is equal to -2.1% (-4%) at period 15 and to -0.45% (-8.3%) at period 40. Overall, these small effects on the undetected lead to the important conclusion that the epidemic is continuously fueled by newcomers from this subpopulation, and this prevents satisfactory control of the epidemic.

It illustrates well and complements what we found in previous sections: it is optimal to front load expenses because there are short-run advantages to do so. However, there exists a rebound effect in the medium run that 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 Comparative statics and robustness

In Dubois and Magnac (2023), we reported the complete analysis of the sensitivity to changes in a few parameters of interest: the preference for the present of HAs, δ, the differential cost of the new treatment with respect to the old treatment (as defined in Equation (12)), the cost of the old treatment (c_{old}), the Weibull coefficient α of the $\rho(.)$ function affecting its concavity and its calibration (see Equation (10)) and finally the initial level of the endowment, a_0. This analysis delivers simulated comparative statics of the outcomes as well as robustness.

These exercises confirm the robustness of our results with small variations across the values of most of these parameters and in particular parameters describing the preference for the present δ and the initial endowment a_0 of the HAs. The results are nonetheless quite sensitive to the Weibull parameter, which confirms our prior that the returns to scale of the new drug treatment

are important although this parameter is difficult to calibrate.

Also important are the parameters describing the costs of traditional drugs and the differential cost of the new drugs. These parameters have the opposite impact on the welfare improvements yielded by the optimal policy vs. the fixed annual budget and interest-only policies. An increasing cost of the traditional drugs leads to welfare improvements because the treatment by new drugs brings larger benefits. This is the contrary when the differential costs of the new drugs are larger since improvements in infection rates are more expensive to obtain.

5   Conclusion

Our research's primary contributions are twofold. First, we derive analytical results characterizing optimal policies using the calculus of variations, revealing that reallocating expenses early toward new drugs while maintaining the intertemporal budget reduces short-term infections, despite medium-term rebound effects. Second, we simulate optimal policies based on parameters calibrated to the epidemiological and economic characteristics of hepatitis C in France, affirming our earlier findings about short-term gains and rebound effects. These results underscore the challenge of adhering to an intertemporal hard budget constraint when allocating resources for disease treatment, emphasizing the need for commitment to long-term budget planning.

Specifically, in the case of a disappearing epidemic such as hepatitis C, an equal annual 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 difficult to prove in a formal model. It depends on the main tradeoff that we have been focussing on.

There is much to be done to better understand the robustness of the optimal policy rules that we 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 epidemic. Second, the results should be also sensitive to the evolution over time of the tradeoffs across periods that are summarized in this paper by the $\rho(.)$ function, and in particular, all tradeoffs arising because of changing prices over time. Third, we set aside the issue of innovation in drug discovery that 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 tradeoff between differentially effective drugs over time. Danzon (2018) reminds us that HAs can delay treatment until competing treatments become available. Lothan et

al. (2022) shows that the price of hepatitis C treatments can also affect health authorities in terms of screening strategies in addition to treatment strategies. Finally, we also leave the questions of bargaining and negotiations between companies and health authorities for further research, as we take as given the cost of novel treatments.

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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 equation 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:

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

One condition of existence of the endemic equilibrium is therefore that $s^* < 1$ or $\beta \geq \beta^* = \frac{(\zeta + \nu)(\rho_0 + \nu)}{(\zeta + \rho_0 + \nu)}$. 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:

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

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 $\rho_0 = 0$, solutions are $x_\pm = \frac{-(\beta - \zeta) \pm (\beta + \zeta)}{2}$ and thus equal to β or ζ. As nonnegative 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 of $\rho_0 > 0$. In sum, if $\beta < \beta^*$, the only stationary equilibrium is the disease-free equilibrium, which is stable.

B Proofs

B.1 Proof of Lemma 1

For any $b \geq 0$, by Assumption D, ρ is smooth, increasing and concave. By the definition (15) of $a(b)$, $a'(b) = (b + c_0)\rho''(b) < 0$ and $a(b)$ is decreasing. Furthermore, as $\rho(\infty) = 1 - \nu$, $a(\infty) \geq 0$, we thus have $a(b) \geq 0$. Moreover, $a(b) \leq a(0) = 1 - \nu - \rho^{(0)} + c_0\rho'(0)$, which is positive by Assumption D.

Second, exactly differentiating Equation (3) yields:

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

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because $du_{t+1} = 0$. This yields:

$$
\begin{aligned}
di_{t+2} &= -\rho'_{t+1}db_{t+1}i_{t+1} + (1 - \rho_{t+1} - \nu)di_{t+1} \\
&= \rho'_{t+1}((1 + r)dB + (b_{t+1} + c_0)di_{t+1}) + (1 - \rho_{t+1} - \nu)di_{t+1} \\
&= (\rho'_{t+1}(1 + r)dB + ((b_{t+1} + c_0)\rho'_{t+1} + 1 - \rho_{t+1} - \nu)di_{t+1} \\
&= (\rho'_{t+1}(1 + r) - a(b_{t+1})\rho'_t)dB.
\end{aligned}
$$

using Equation (13) between the first and second lines and Equation (14) in the third line.

C 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 greater detail below.

We start in Step 1 by computing the value function when HAs do not intervene in which case the epidemic 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:

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

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

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

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:

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

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.[7] For any value z, we approximate $W_0^{(T)}(z)$ by

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

In this step, we also 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 above by the maximum value of assets. We will not consider any policy such that the level of assets increases in 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.

---

[7] 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.

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 in 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:

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

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 kth 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, using the Bellman equation (9) leads to the optimal policy under the last inequality constraint. This delivers the value function that uses up the full endowment in at most $k+1$ periods (given the provisions made for the traditional treatment).

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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 than 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.
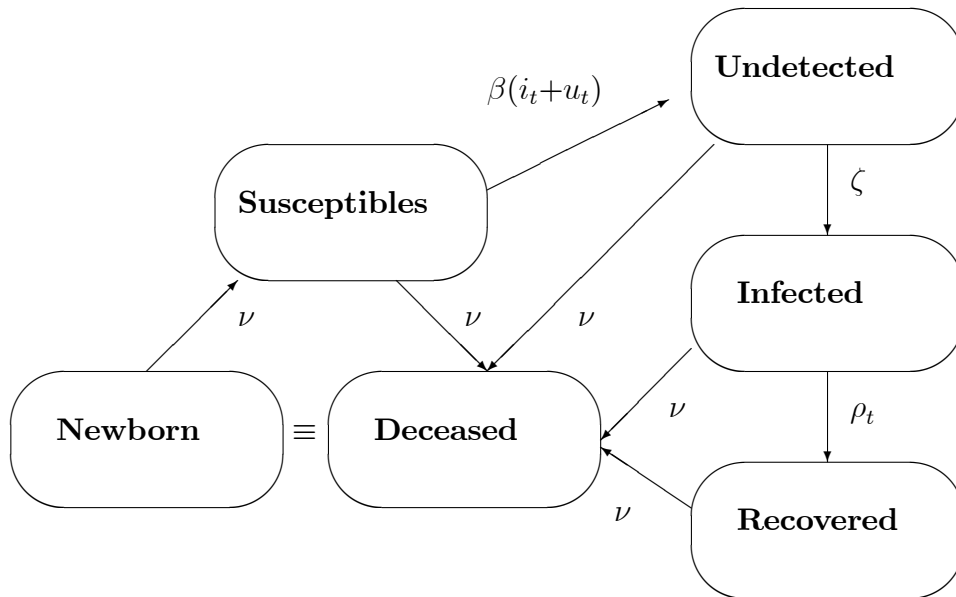
Note: Transition rates between states Susceptibles, Undetected, Infected and Recovered as well as Deceased are given along arrows of the corresponding transitions. By assumption, birth rates are equal to death rates.

Figure 1: Parameters of SIR transitions
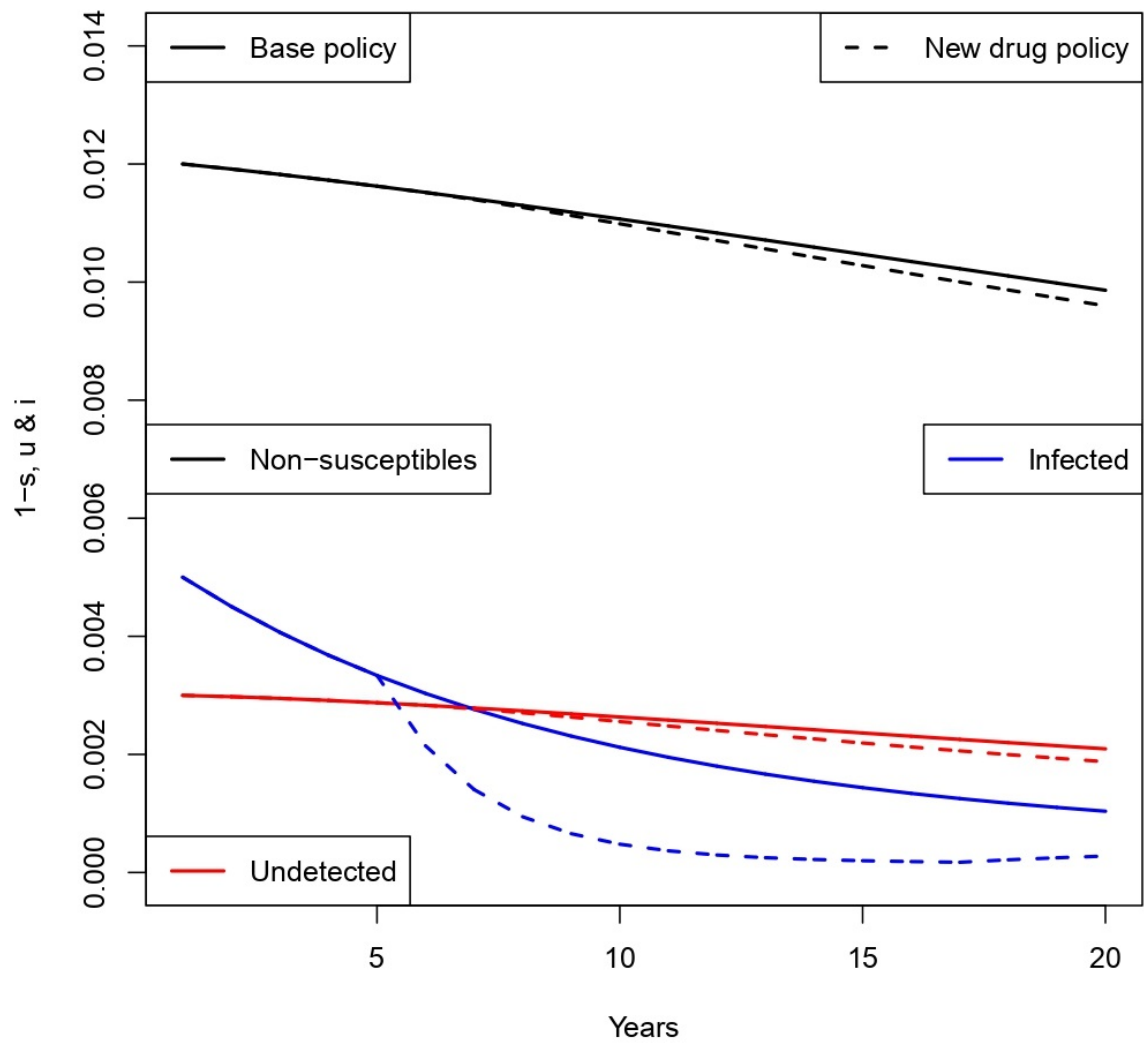
Note: 1-s = non susceptibles, i = infected, u = undetected. Reading: The share of infected at time 0 is 0.5. The new drug policy for a fraction of infected is in place between periods 5 and 17

Figure 2: Infection and policy impact
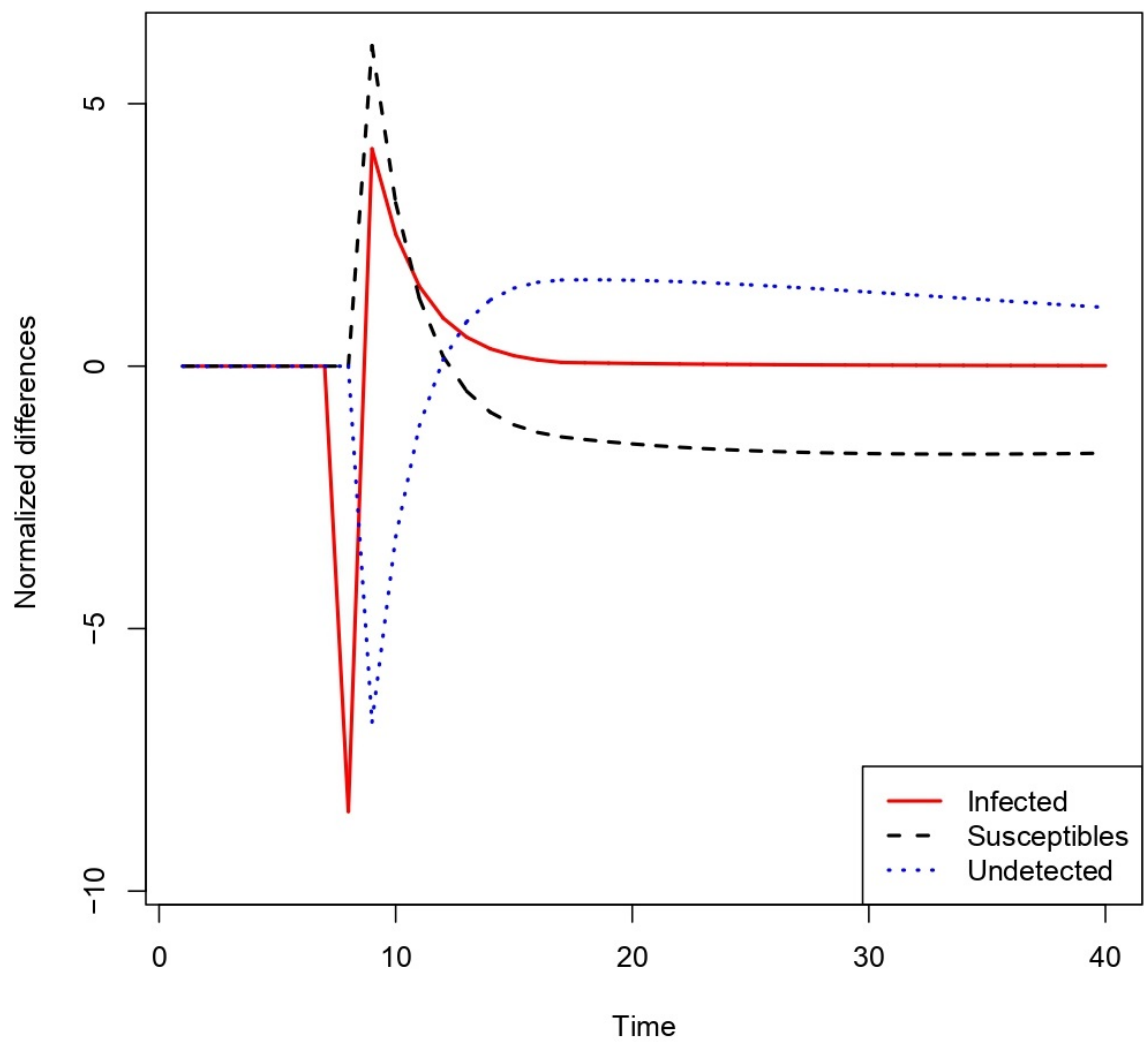
Note: The initial budget sequence is increasing at rate r. Some budget at period 9 is reallocated at period 8. The impulse response functions have different scales which are normalized in such a way that their standard deviations are equal to 1 over time. Scales are not comparable since they vary widely across state variables.
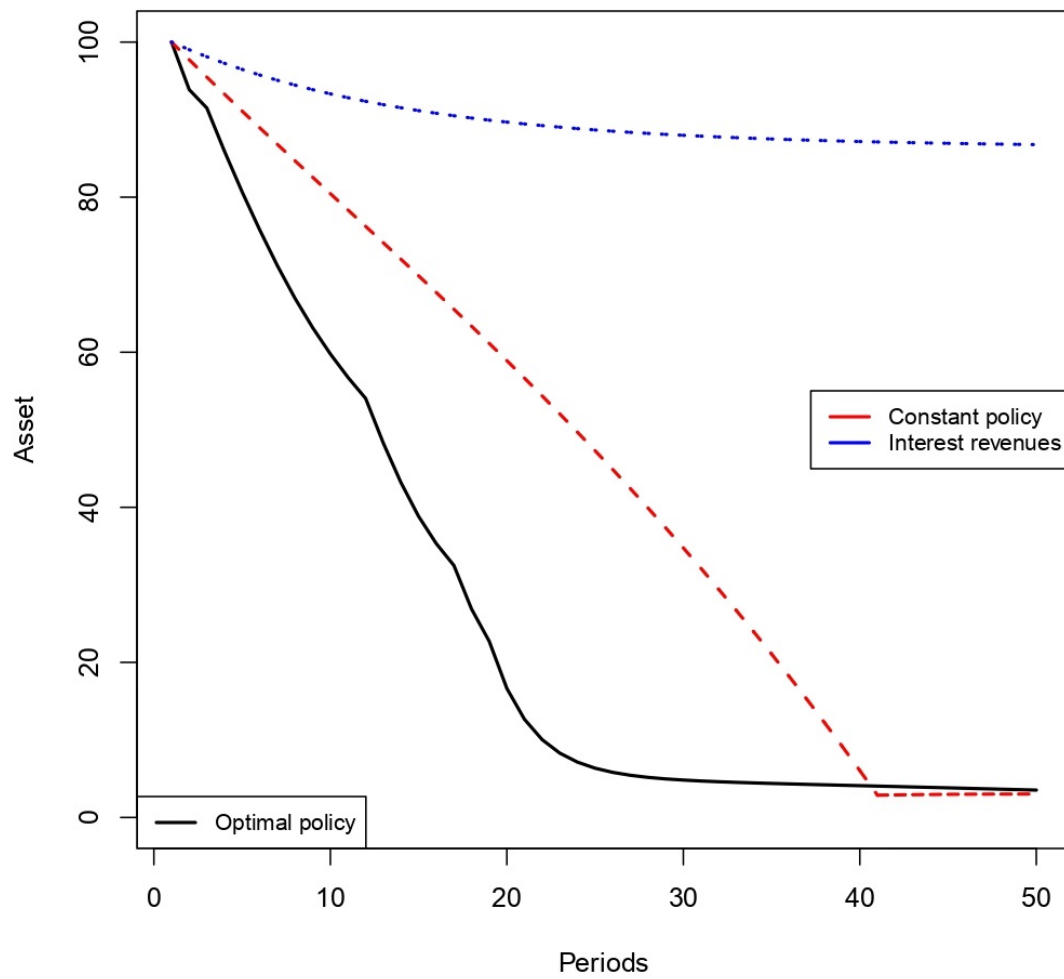
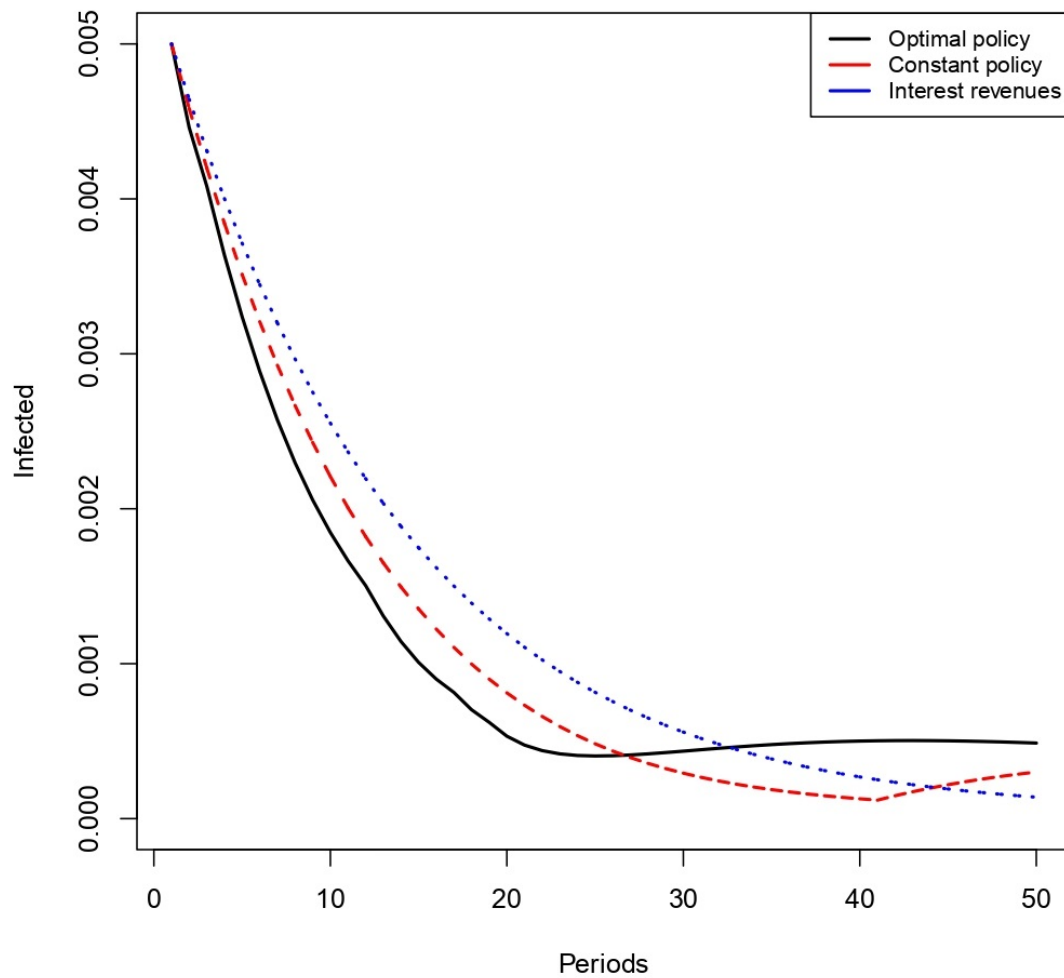Figure 3: Variational budget impact on state variables

Figure 4: 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.

Note: Assets are normalized to 100 initially. "Constant policy" is the fixed budget policy in which the level of assets at period 40 is the same as the optimal policy and interest revenues is the one using interests only as defined in the text

Figure 5: Profile of infected over time
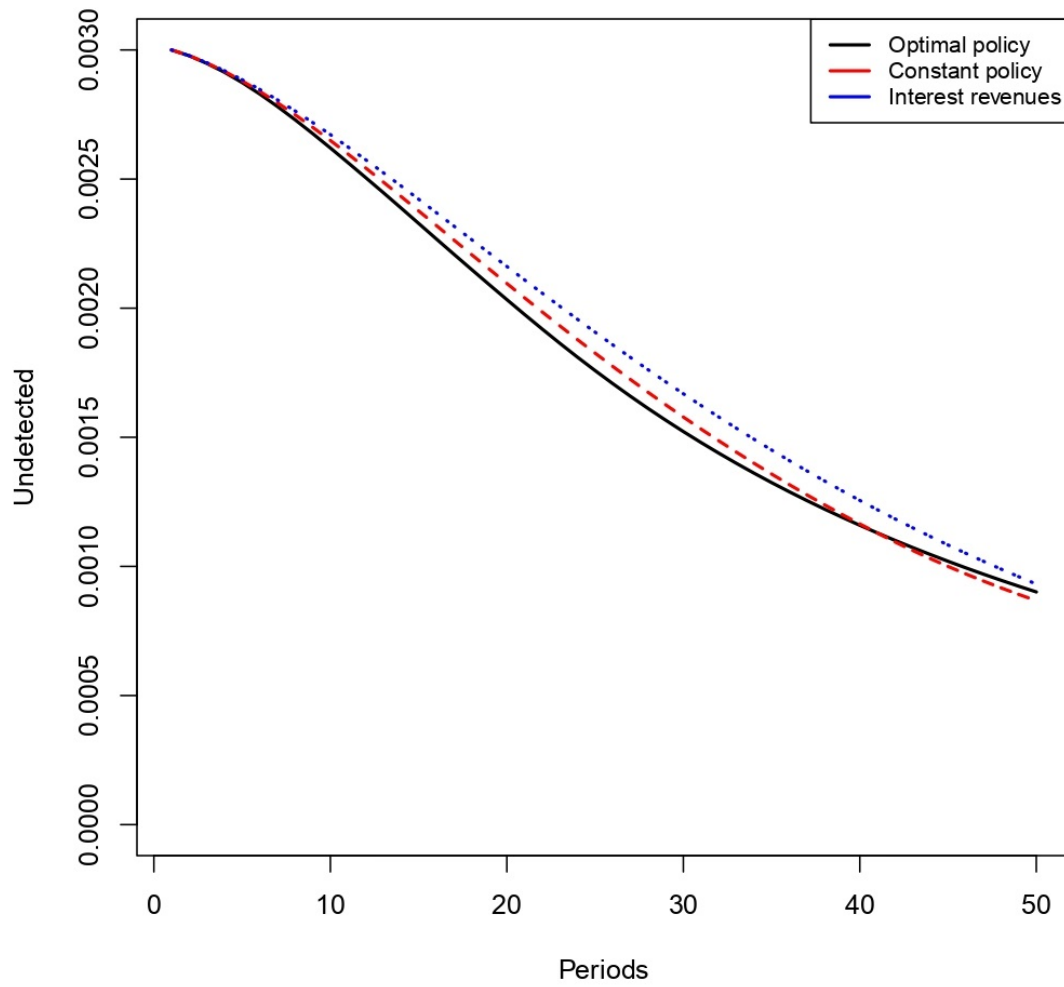
Note: Undetected infected is the rate of infection undetected in the population initialized at its calibrated value. Constant policy is the fixed budget policy and interest revenues is the one using interests only as defined in the text.

Figure 6: Profile of undetected infected over time