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Highlights

- We study the problem of voluntary vaccination for a transmittable disease outbreak.
- We consider vital dynamics, vaccine efficacy waning and far-sighted individuals.
- We also obtain results when part of the population has an anti-vaccination stance. 1

Canonical Modelling of Anticipatory Vaccination Behavior and Long Term Epidemic Recurrence

Julien FLAIG* Nicolas HOUY† Philippe MICHEL‡

September 27, 2017

Abstract

Vaccination is one of humanity's main tools to fight epidemics. In most countries and for most diseases, vaccination is offered on a voluntary basis. Hence, the spread of a disease can be described as two interacting opposite dynamic systems: contagion is determined by past vaccination, while individuals decide whether to vaccinate based on beliefs regarding future disease prevalence. In this study, we show how the interplay between such anticipating behavior and the otherwise biological dynamics of a disease may lead to the emergence of recurrent patterns. We provide simulation results for i) a Measles-like outbreak, ii) canonical fully rational and far-sighted individuals, iii) waning vaccine efficacy and vital dynamics, and iv) long periods of time, *i.e.* long enough to observe several vaccination peaks. For comparison, we conducted a similar analysis for individuals with adaptive behavior. As an extension, we investigated the case where part of the population has an anti-vaccination stance.

Keywords: epidemics, behavior, vaccination, game theory, forward-backward system, backward induction.

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1 Introduction

Vaccination is one of the most efficient tools humanity possesses to fight epidemics. The collective consequences of vaccination depend on its cost, its effectiveness, and disease dynamics. Many theoretical works have studied the optimal vaccination policies (from Hethcote and Waltman (1973) to Laguzet and Turinici (2015a)). When vaccination, as is often the case, is administered on a voluntary basis, a further mechanism comes into play: the interaction between disease dynamics and human behavior—a now widely aknowledged fact (see Funk et al. (2010, 2015)). On the one hand, disease dynamics has an impact on human behavior through prevalence, individual beliefs about future course of the epidemic, and spreading of information or beliefs about it. On the other hand, human actions such as vaccination, social distancing, treatment adherence, or even fleeing, influence disease dynamics. Observations of the strong impact of human behavior on disease dynamics include Philipson (1996); Jansen et al. (2003); Riley et al. (2003); Nishiura (2007); Bayham et al. (2015).

It is all the more crucial to investigate human behavior in the case of vaccination decision as vaccination contributes to herd imunity. Yet individual vaccination decisions may not be aligned with social interests: individuals make their decisions out of self-interest while their actions also bear on the whole population. Herd immunity is merely an externality of vaccination decisions, and therefore the result of voluntary vaccination is generally not socially optimal in this respect. Besides, it can be readily understood how in the long run this discrepancy between private and social interests may give rise to recurrent epidemic patterns—low prevalence may lead to low vaccination rates, which in turn may lead to high contagion, higher vaccination rates, and again low prevalence.

Theoretical modelling of vaccination behavior, however, remains a challenge. When faced with a transmittable disease, an individual may decide on a course of action based on his beliefs regarding future developments of the epidemic. That is, each individual may *anticipate* developments to come. Now, if all individuals do this, the very evolution of the epidemic is modified. Ultimately, the spread of a disease can be described as two entangled yet conflicting dynamical systems. The spread of a disease restricted to its

biological[1] features evolves *forward* in time: future developments are only determined by the current state of the epidemic. On the other hand, individuals base their vaccination decision at least in part on *backward* reasoning: they act now upon what might happen in the future. The spread of a disease is influenced by individual decisions while in turn influencing these decisions.

In order to solve this problem, several approaches have been proposed. Some authors tackled the problem with a full consideration of the forward-backward dimension described above. Yet these studies, in order to obtain tractable results, had to decrease the complexity of other dimensions. Other authors simplified, the dynamics system, at least compared to the model we will present here: Geoffard and Philipson (1996); Chen and Cottrell (2009) studied SI models, and Geoffard and Philipson (1997); Laguzet and Turinici (2015b) studied SIR models without waning vaccine efficacy. Others restricted the scope of their study. Geoffard and Philipson (1996, 1997), for instance, produced a qualitative description of some features of the solution. In the same vein, Chen and Cottrell (2009) investigated equilibrium existence, uniqueness, and potential coexistence of two equilibria in a given setting. Finally, Reluga and Galvani (2011) restricted themselves to the study of stationary states.

An alternative stream of literature somehow decreases the complexity of the coupled system by disregarding backward reasoning in human behavior. Bauch et al. (2003); Bauch and Earn (2004) sparked renewed interest in vaccination policy and individual choices with one period (*i.e.* static) models. Further instances of one period models were provided by Reeling and Horan (2015); Codeço et al. (2007); Shim et al. (2012). In order to introduce dynamic decision-making in this framework, Bauch (2005) (followed by Reluga et al. (2006); d'Onofrio et al. (2011); Fu et al. (2011); Yang et al. (2016)) proposed models with imitation behavior. Just as the spread of a transmittable disease when individual behavior is ignored, imitation only depends on past and current states of the epidemic: imitation dynamics goes forward in time. This outlook on the problem was also adopted by Fenichel et al. (2011). They assumed that individuals falsely believe that the current epidemiological state will persist (Voinson et al. (2015) added cognitive biases to this framework). Similarly,

[1]We describe epidemics as *biological* insofar as they do not depend on human behavior. As will be made clear later, this distinction depends on problem specification: some parameters may or may not be modelled as decision variables. Consider for instance the contact rate between individuals.

Buonomo et al. (2008); Epstein et al. (2008); Coelho and Codeço (2009); Funk et al. (2009); Bhattacharyya et al. (2015) all modelled information and/or beliefs with forward dynamics in time.

At this point, we must emphasize that we by no mean argue that real life vaccination decision (or for that matter any other behavior pertaining to the study of epidemics) is only driven by backward reasoning. Nor do we claim that imitation or past evolution of an epidemic are irrelevant to our case. However, we believe that there is currently a need for modelling the entangled backward and forward dynamics described above in all their complexity with canonical—though somehow unrealistic—perfectly informed, fully rational and far-sighted individuals. Simulation results are to be used as benchmarks to better evaluate the weight of the different factors that can influence decision-making in populations faced with a transmittable disease. This is to be done by measuring how real life data departs from the predictions of the canonical model proposed here.

In the present paper, we address the challenge of coupled forward-backward dynamics posed by canonical modelling of vaccination decision-making. We consider

- a SIVR (Susceptible, Infectious, Vaccinated, Removed) epidemiological dynamic model with vital dynamics and waning vaccine efficacy, and
- backward reasoning by far-sighted, fully rational, and selfish individuals.

Close to our work are Reluga (2010) and Reluga (2013) in the context of social distancing. The main difference between these studies and ours is that, since we consider waning immunity and vital dynamics with growing population, our set of equations is larger and convergence is more difficult to obtain. Indeed, the set of vaccinated individuals is not constrained to always grow in our model, which increases dramatically the array of possible vaccination strategies. We solve this complex system, and we believe that we are the first to obtain recurrent behavioral patterns (in our case, vaccination peaks) with a canonical forward-backward model and full complexity of population dynamics.

We describe our model in Section 2. Section 3 is dedicated to the results of our model for a Measles-like disease and a vaccine with waning efficacy. Our base case (Section 3.1) involves a population of identical individuals. For comparison, we provide results in the case of adaptive behavior (Section 3.2). Finally, we investigate populations in which some

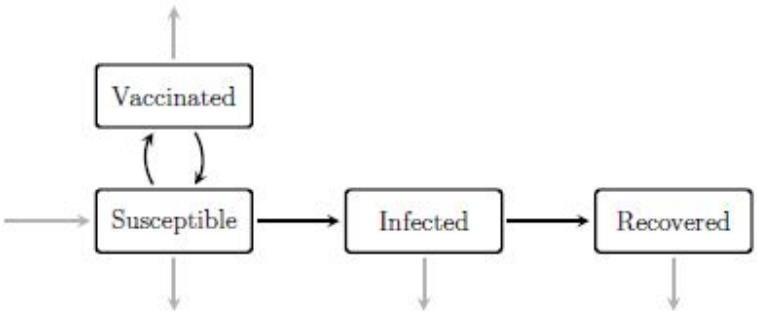


Figure 1: Illustration of the SIRV model with epidemiological transitions in black and vital dynamics transitions in gray.

individuals have an anti-vaccination stance, that is higher vaccination cost (Section 3.3). Section 4 concludes.

2 Model

We consider a SIR model with vaccination and vital dynamics (see Figure 1). Individuals can be either *susceptible* (S), *infected* (I) or *recovered* (R). In addition, susceptible individuals have the possibility to access vaccination on a voluntary basis and become *vaccinated* (V). The disease is transmitted under the assumption of homogeneous mixing of the population. Vaccination has a waning efficacy so that vaccinated individuals can become susceptible after some time. Birth and death rates can differ and hence do not necessarily imply constant population size.

A susceptible individual is assumed to base his decision to vaccinate on a rational far-sighted cost-benefit analysis. Hence, vaccination decision depends on the values the individual expects from being vaccinated and from remaining susceptible, and on the immediate cost (monetary, psychological, logistical, etc.) of vaccination. Formally, the problem of finding an individual's optimal vaccination policy over time can be solved by ways of dynamic programming via Bellman equations. Solving Bellman equations yields the *intertemporal value function* of individuals in each health status. Given his current health status, an individual's value function is the discounted future value he expects to get if he follows his optimal policy. Since we consider waning vaccine efficacy, *both* the value of remaining susceptible *and* the value of getting vaccinated at a given time depend on pre-

dictions about future epidemiological states. A vaccinated individual may lose immunity and get the value of being sucseptible with non zero probability. To our knowledge, we are the first to solve the canonical forward-backward problem with four value functions, two of them depending on contagion dynamics.

Also, we consider for the sake of realism that individuals cannot vaccinate at any time: in real life, only a fraction of them has access to vaccination simultaneously. This feature is represented by rate α (see Table 1) in our model.[2] In contrast to models where vaccine is available at once to the whole population, individuals in our model anticipate that not vaccinating when they have a chance implies waiting until the next opportunity to do so. This, however, does not remove the game theoretical dimension of our problem even though individuals do not play against each other in each instant. We have a sequential game in which Nature randomly picks the playing order in each moment, and allows a maximum αdt zero-measure set of individuals to vaccinate.

For numerical tractability, and yet certainly as a realistic assumption, we use the concept of smoothed best response (Fudenberg and Levine (1998)). When facing a choice between two alternatives leading to intertemporal values V_1 and V_2 respectively, an individual chooses V_1 with probability $\frac{e^{(V_1/\epsilon)}}{e^{(V_1/\epsilon)}+e^{(V_2/\epsilon)}}$, or introducing function $\chi_\epsilon : x \mapsto \frac{1}{1+e^{-x/\epsilon}}$ for all $\epsilon \in \mathcal{R}^+$, he chooses V_1 with probability $\chi_\epsilon(V_1 - V_2)$.[3] In Figure 2, we show function χ_ϵ for the different values of ϵ used in our simulations.[4] Notice that as ϵ tends to 0, the probability of playing any strategy that is not a best response goes to 0.

For a given ϵ, the epidemiological side—strictly speaking—of our model is governed by Equations 1a–1d. T is the final time, $s^\epsilon(t)$ (resp. $i^\epsilon(t)$, $v^\epsilon(t)$, $r^\epsilon(t)$) denotes the number of susceptible (resp. infected, vaccinated, recovered) individuals at time t in $[0, T]$. For concision, we introduced $n^\epsilon(t) = s^\epsilon(t) + i^\epsilon(t) + v^\epsilon(t) + r^\epsilon(t)$ and function $\xi_\epsilon : x \mapsto x\chi_\epsilon(x)$

[2] We performed a sensitivity analysis on α. Dividing α by two does not bear upon short term epidemiological results and vaccination decision. While the epidemic is not affected in the long run, the long term vaccination decision changes noticeably, as shown in Figure C.3.

[3] The same approach was used by Xu and Cressman (2014, 2016) with individuals making decisions based only on the present state of the epidemiology.

[4] We used two different ϵ values so as to ease equation solving for some of our simulations. This, however, is of little consequence as to our results. Consider for instance the difference in utility between being sick and being healthy for the average duration of the infectious period (see Table 1 for parameter values). This difference in utility is $5 \times (10 - 2) = 40$ on average, ignoring the discount factor for this short period. It can readily be made sure that $\chi_{1/20}(40)$ and $\chi_{1/600}(40)$ are both close enough to 1 for our purpose.χ between 0 and 1 will denote indifference in our model.

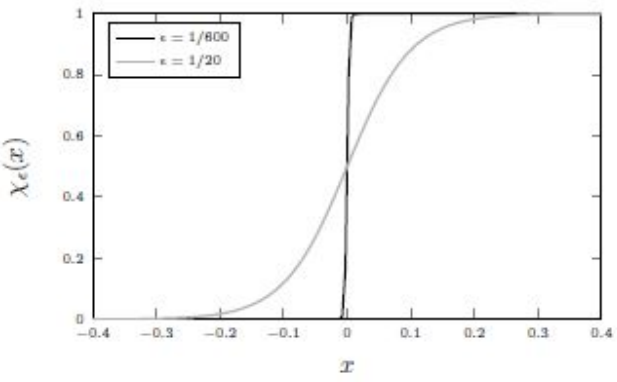


Figure 2: χ_ϵ for relevant values of ϵ.

for x in $\mathcal{R}$. The individual decision process is described by Equations 2a–2d, where $V_S^\epsilon(t)$ (resp. $V_I^\epsilon(t)$, $V_V^\epsilon(t)$, $V_R^\epsilon(t)$) is the value function of a susceptible (resp. infected, vaccinated, recovered) individual at time t in $[0, T]$.

$$\frac{d}{dt}s^\epsilon(t) = -s^\epsilon(t)\left[\alpha\chi_\epsilon(V_V^\epsilon(t) - V_S^\epsilon(t) - c) + \lambda\frac{i^\epsilon(t)}{n^\epsilon(t)} + \mu\right] + \nu n^\epsilon(t) + \gamma_V v^\epsilon(t) \tag{1a}$$

$$\frac{d}{dt}i^\epsilon(t) = -i^\epsilon(t)\left(\gamma_I - \lambda\frac{s^\epsilon(t)}{n^\epsilon(t)} + \mu\right) \tag{1b}$$

$$\frac{d}{dt}v^\epsilon(t) = -v^\epsilon(t)(\gamma_V + \mu) + \alpha s^\epsilon(t)\chi_\epsilon(V_V^\epsilon(t) - V_S^\epsilon(t) - c) \tag{1c}$$

$$\frac{d}{dt}r^\epsilon(t) = -r^\epsilon(t)\mu + \gamma_I i^\epsilon(t) \tag{1d}$$

$$-\frac{d}{dt}V_S^\epsilon(t) = u_g - (\delta+\mu)V_S^\epsilon(t) + \lambda\frac{i^\epsilon(t)}{n^\epsilon(t)}(V_I^\epsilon(t) - V_S^\epsilon(t)) + \alpha\xi_\epsilon(V_V^\epsilon(t) - V_S^\epsilon(t) - c) \tag{2a}$$

$$-\frac{d}{dt}V_I^\epsilon(t) = u_b - (\delta+\mu)V_I^\epsilon(t) + \gamma_I(V_R^\epsilon(t) - V_I^\epsilon(t)) \tag{2b}$$

$$-\frac{d}{dt}V_V^\epsilon(t) = u_g - (\delta+\mu)V_V^\epsilon(t) + \gamma_V(V_S^\epsilon(t) - V_V^\epsilon(t)) \tag{2c}$$

$$-\frac{d}{dt}V_R^\epsilon(t) = u_g - (\delta+\mu)V_R^\epsilon(t) \tag{2d}$$

A detailed description of the parameters is given in Table 1. The construction of Equations 1a–2d is made explicit by the alternative formulation in Appendix A.

Equations 1b and 1d are the same as in usual SIR models. We assume that the death rate μ is the same for healthy and infected individuals. Infected individuals are recovering at rate γ_I. Equation 1a too, is very similar to the equation describing the susceptible population in a SIR model: each day, a susceptible individual has an average λ encounters in which he could potentially get infected. A proportion $i(t)/n(t)$ of these encounters occur with an infected individual. Also, individuals are born susceptible at rate ν. Our model departs from SIR models in that susceptible individuals decide whether or not to vaccinate based on a cost-benefit analysis. At time t, the higher the net value to vaccinate $V_V^\epsilon(t) - V_S^\epsilon(t) - c$, the closer to 1 the probability of deciding to vaccinate given by function χ_ϵ.

Let us now elaborate on Equations 2a–2d satisfied by the value functions. Again, an alternative formulation of these equations is provided in Appendix A for the interested reader. u_g and u_b are the instantaneous utilities of being in good and bad health respectively. Individuals are forward-looking, hence the value functions decrease at rate u_g or u_b (depending on the considered health status) with time. δ is the time discount factor and we normalize the value of being dead to 0, so all value functions increase at rate $(\delta + \mu)$. That is the value of being say, susceptible, at time t decreases by $(\delta + \mu) \times (0 - V_S(t))$. Similarly, the value of being in a given health status decreases by the net value of each health status transition weighted by the rate of this transition.

Existence and uniqueness of a solution to Equations 1a–2d follows from Theorem 1.[5]

Theorem 1

The system of Equations 1a–2d has a unique solution for any $\epsilon > 0$.

Proof. See Appendix B.

[5]Notice that in a related study, Chen and Cottrell (2009) found possible multiple equilibria. This is due to the way they modelled imperfect vaccine efficacy, and in particular to the independance of vaccine failure at each encounter. Indeed, in their study, when vaccine efficacy is low, a high prevalence implies a high infection probability at each encounter and hence an incentive not to vaccinate balancing the incentive to vaccinate.

3 Results

3.1 Base case: population of identical individuals

Base parameter values are summarized in Table 1. We use parameter values that are characteristic of Measles. Measles is a widely studied disease whose epidemiological features allow for rich modelling in our framework. Also, Measles vaccine is offered on a voluntary basis, and has recently been in the spotlight due to alleged side effects deterring part of the population from vaccinating. We model a vaccine that is efficient for 10 years on average and costs 10.[6] Vital dynamics is characteristic of a developing country.[7]

Our base case features a perfectly mixed population of identical individuals. That is, all individuals have the same vulnerability to the disease and rate of recovery, have the same preference for being healthy over being sick, and face the same vaccination cost.

Equations 1a–2d are solved numerically using techniques close to fixed-point iterations.[8] We set initial conditions for Equations 1a–1d, and final conditions for Equations 2a–2d. Notice that from Equation 2d,

$$V_R^\epsilon = \left(V_R^\epsilon(T) - \frac{u_g}{\delta+\mu} \right) e^{-(\delta+\mu).(T-t)} + \frac{u_g}{\delta+\mu}, \tag{3}$$

and then V_I^ϵ (Equation 2b), can be solved analytically. In our model, recovered individuals stay recovered for the rest of their life, and u_g, δ and μ do not depend on time, so V_R^ϵ does not depend on time. Consequently, V_I^ϵ does not depend on time either, and we can set both V_R^ϵ and V_I^ϵ to their respective stationary values. We then use the stationary values of V_R^ϵ and V_I^ϵ to set the final value of V_S^ϵ and V_V^ϵ.[9] For all simulations, we make sure that final time T is large enough so that the influence of final conditions on the result is null.[10]

In Figures 3–4, we show the output of our model (Equations 1a–2d) for $\epsilon = 1/600$

[6] This cost represents 1/4 of the cost of being sick on average, disregarding epidemiological changes after infection (see calculation in Footnote 4).

[7] All parameter values are only illustrative and do not reflect any specific real life case. A ±10% sensisitivity analysis on all parameters is shown in Figure C.4

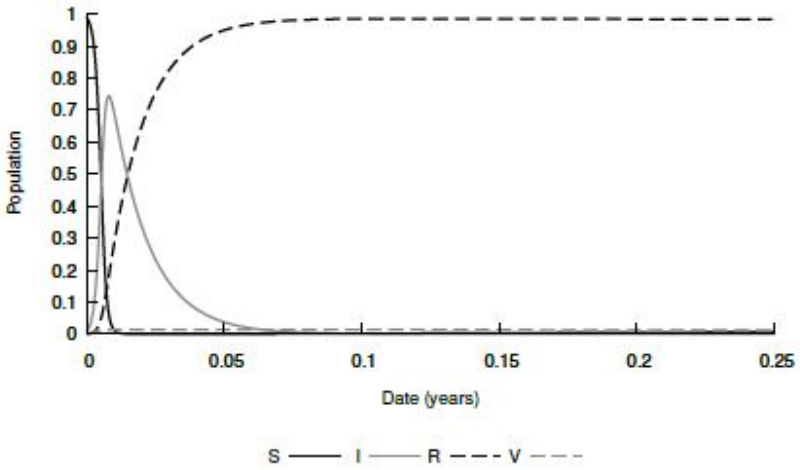
[8] Rather than shooting techniques, as is done in Reluga (2010). Source code is available on request.

[9] We know that the value of being vaccinated V_V^ϵ and the value of being susceptible V_S^ϵ are both higher than the value of being infectious V_I^ϵ, but lower than the value of having recovered V_R^ϵ. In practice, the final value of V_S^ϵ and V_V^ϵ is set to V_I^ϵ or $(V_I^\epsilon + V_R^\epsilon)/2$ depending on the simulation, with no consequence as for the results presented here.

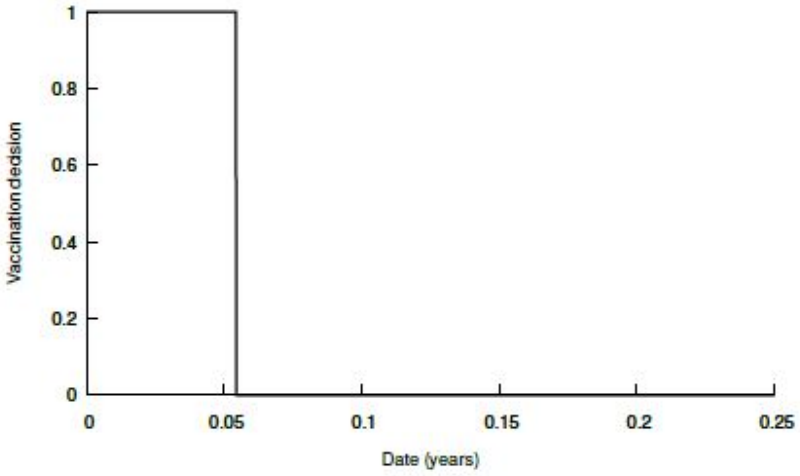
[10] Typically, T is taken larger than 350 years.

Parameter	**Base value**	**Description**
Epidemiology		
λ	2.8	Contact rate.
α	0.0068	Potential vaccination rate.
γ_V	2.74×10^{-4}	Vaccination efficacy waning rate.
γ_I	0.2	Rate of recovery.
Decision making		
u_g	2	Instantaneous utility to be in good health.
u_b	10	Instantaneous utility to be in bad health.
δ	8.1×10^{-5}	Time discount rate.
c	10	Cost of vaccination.
Vital dynamics		
μ	5.48×10^{-5}	Death rate.
ν	8.22×10^{-5}	Birth rate.
Initial State		
$s^\epsilon(0)$	$0.99 \times n^\epsilon(t)$	Initial number of susceptible individuals.
$i^\epsilon(0)$	$0.01 \times n^\epsilon(t)$	Initial number of infectious individuals.
$v^\epsilon(0)$	0	Initial number of vaccinated individuals.
$r^\epsilon(0)$	0	Initial number of recovered individuals.

Table 1: Parameter list and base values (all time dimensions in days, utility and cost are dimensionless).

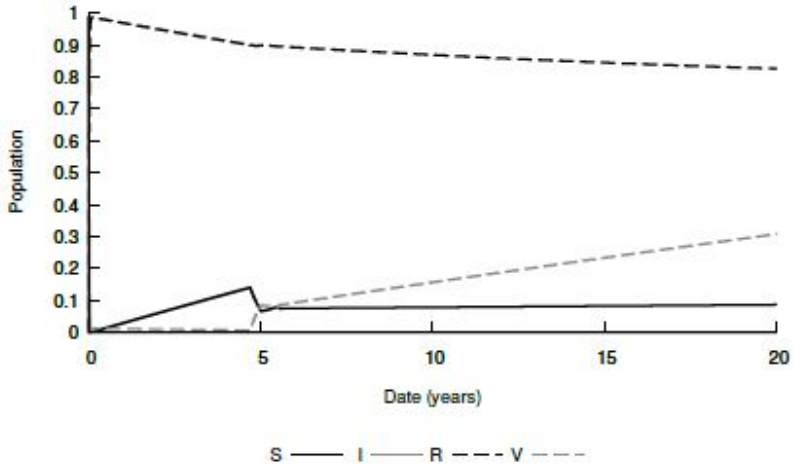


(a) Epidemiology

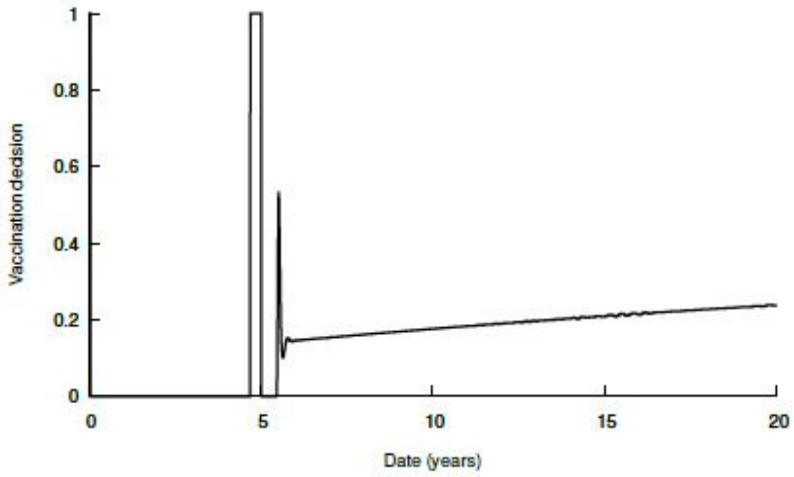


(b) Vaccination decision

Figure 3: Epidemiological results and vaccination decision ($\xi_\epsilon(V_V^\epsilon(t) - V_S^\epsilon(t) - c)$) over the first quarter of the epidemic, with $\epsilon = 1/600$ and $n^\epsilon(0) = 1$.



(a) Epidemiology



(b) Vaccination decision

Figure 4: Epidemiological results and vaccination decision ($\xi_\epsilon(V_V^\epsilon(t) - V_S^\epsilon(t) - c)$) over the first 20 years of the epidemic, with $\epsilon = 1/600$ and $n^\epsilon(0) = 1$.

over the first quarter and over the first 20 years of the epidemic.[11] In the first days of the outbreak, individuals anticipate that the prevalence of the disease will be high and hence vaccinate. At epidemic peak, more than 70% of the population is infectious and all individuals that have access to vaccination vaccinate. After that, as the pool of susceptible individuals decreases, the disease prevalence drops, and when it is low enough (and anticipated to remain so for a long time), individuals stop vaccinating. Because of vital dynamics and waning vaccination efficacy, the pool of susceptible individuals grows again and a second wave of vaccination is observed about five years after the introduction of the disease. Vaccination dynamics is then strongly damped and has an increasing frequency over time. A state is finally reached where a portion of the individuals that have access to the vaccine vaccinate at all time.

3.2 Individuals with adaptive behavior

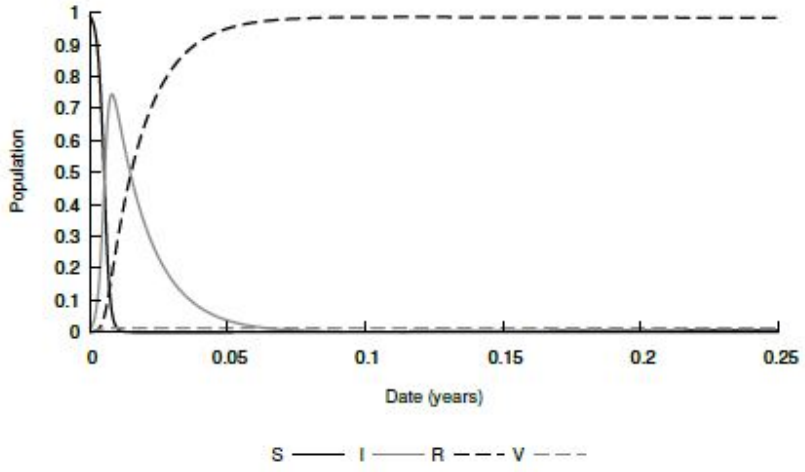
So as to draw a parallel with the existing literature, we model the same disease as in Section 3.1 in the case where individuals adopt an adaptive behavior. Individuals with adaptive behavior do not anticipate the evolution of the epidemic at an aggregate level, even though they do anticipate the evolution of their own health status. Susceptible individuals with adaptive behavior, for instance, anticipate the loss of utility corresponding to being sick for about 5 days—the average length of infection—, but mistakenly expect disease prevalence to remain unchanged in the future. Hence, in this model, value functions are stationary and solution of Equations 2a–2d under $\frac{d}{dt}V_S^\epsilon(t) = \frac{d}{dt}V_I^\epsilon(t) = \frac{d}{dt}V_V^\epsilon(t) = \frac{d}{dt}V_R^\epsilon(t) = 0$ *at any time* t.[12] The system dynamics only goes forward in time.

In Figures 5–6 we display results for a population of individuals with adaptive behavior over the first 20 years and the first quarter of the epidemic, and for $\epsilon = 1/600$.[13] The first peak of vaccination lasts more that twice longer for adpative individuals since they do not anticipate the very low prevalence to come for the following 5 years. Still, in the case of our Measles-like disease, this difference yields qualitatively almost no difference in prevalence for the first quarter of the epidemic. Indeed, the speed of contagion is so fast that when

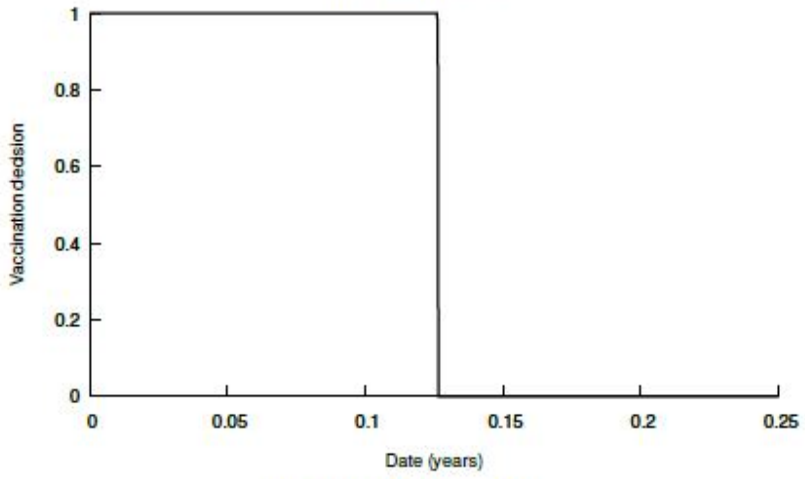
[11] Results for $\epsilon = 1/20$ are provided in Appendix C.

[12] That is, the value functions do depend on time. Numerically, we solve this system for each date, subject to the current state of the epidemic.

[13] In Figure C.5 in Appendix, we show the same results for $\epsilon = 1/20$.

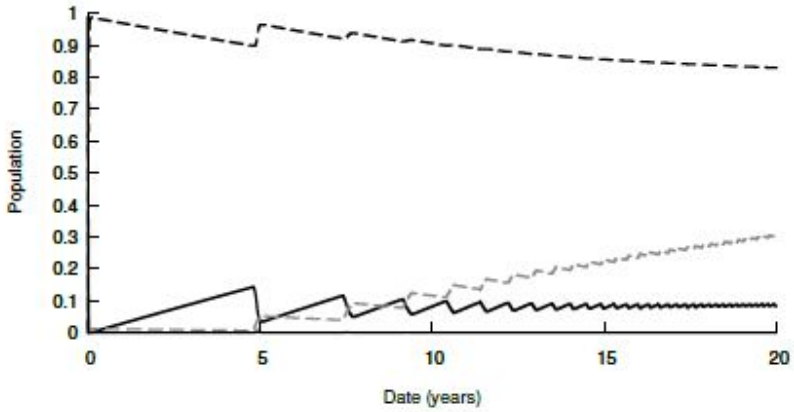


(a) Epidemiology

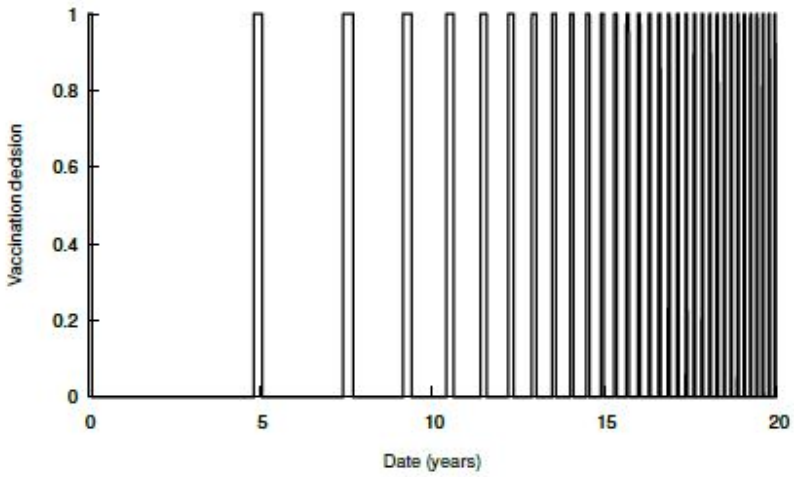


(b) Vaccination decision

Figure 5: Epidemiological results and vaccination decisions over the first quarter of the epidemic, with $\epsilon = 1/600$ and $n^{\epsilon}(0) = 1$ – Individuals with adaptive behavior.



(a) Epidemiology



(b) Vaccination decision

Figure 6: Epidemiological results and vaccination decisions over the first 20 years of the epidemic, with $\epsilon = 1/600$ and $n^{\epsilon}(0) = 1$ – Individuals with adaptive behavior.

decision differ, almost all individuals, far-sighted or with adaptive behavior have already been infected.

In the long run, the vaccination dynamics of adaptive individuals is very different from that of far-sighted individuals. While after the sixth year a portion of the latter vaccinates at all times, the former have a more polarized vaccination behavior. Either all adaptive individuals or none of them want to vaccinate. Adaptive individual vaccination peaks occur with increasing frequency. Anticipation of future epidemiological states by far-sighted individuals flattens vaccination decisions.

While we are not making policy recommendations in the present article, we can expect the selected modelling approach to have policy implications. In the case shown here, for instance, a model with adaptive agents would predict that the demand for vaccination never settles, when our model with rational expectation would predict that with time, individuals become close to indifferent to vaccination.

3.3 Populations with different costs to vaccinate

The population we have been modelling so far was made of identical individuals. Preferences, notably, were the same for all individuals. Yet we expect real life individuals to have differentiated preferences. Besides, as we are considering far-sighted individuals who need to anticipate the future of the epidemic, we need to take into account the fact that an individual's decision may be influenced by his knowledge of others' preferences and hence their influence on future epidemiological states.

In this section, we model two populations —still homogeneously mixed— only differing in their attitude toward vaccination. Different attitudes toward vaccination are modelled by different costs to vaccinate. *Population 0* has the same vaccination cost $c = 10$ as the population modelled to this point (Sections 3.1–3.2). *Population 1* has a more anti-vaccination stance and a cost to vaccinate $c = 12$.[14] Obviously, the vaccination behavior of individuals in Population 1 is different from that of individuals in Population 0. This disparity influences the course of the epidemic, which is anticipated by individuals in Population 0, in turn modifying their behavior compared to the case where they were

[14]This difference can be accounted for by ideology but it can also be interpreted more materialistically: individuals may face different insurance policies, more expensive access to medical services, etc.

the only individuals in the population. Similarly, individuals in Population 1 anticipate decisions made by individuals in Population 0.

We investigate the effects of vaccination cost heterogeneity by varying the proportion of the whole population belonging to Population 1 between 0% (all individuals are in Population 0[15]), and 100% (all individuals are in Population 1). In each simulation, 1% of Population 0 and 1% of Population 1 is initially infected.

Results are displayed in Figures 7–8.[16] Individuals in Population 0 vaccinate more that individuals in Population 1. Indeed, the former have a lower cost to vaccinate than the latter and yet face the same disease prevalence at all times.

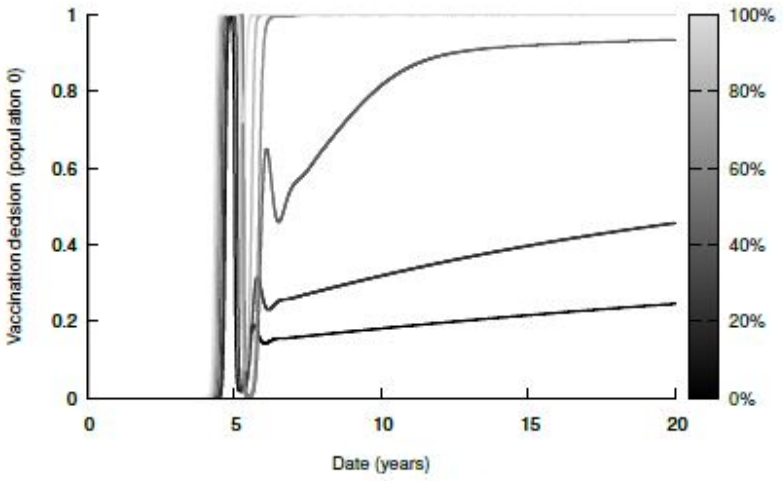
Individuals in each population tend to vaccinate more as the ratio of individuals in Population 1 increases. Indeed, more individuals in Population 1 implies a lower overall vaccination rate. Because higher prevalence is then anticipated by all individuals, more people vaccinate in each population. This reasoning fails at some points in time since the whole dynamics of the epidemic is modified by the change in vaccination policy of both populations. Indeed, a change in vaccination policy may influence the waveform of the disease dynamics and hence the lack of coherence between both cases may imply shifted local maxima and local minima of vaccination decisions.

4 Conclusion

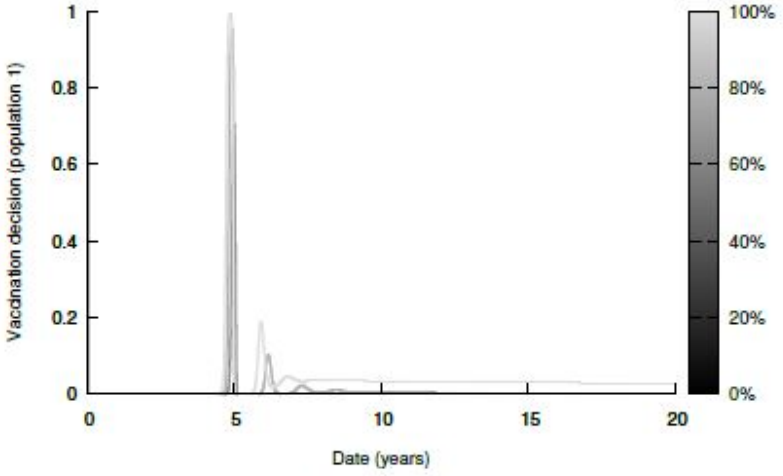
Investigating the interplay between strictly speaking *biological* dynamics of an epidemic, and individual vaccination *decision-making*, is certainly critical to the design of operational health policies. In this line, there is a need for an appropriate benchmark. We claim that this benchmark is to be provided by the behavior of canonical fully rational and far-sighted individuals. The resulting forward-backward system of equations, however, is difficult to solve: it is computationally challenging to obtain the functional fixed-point of the system. The problem is even harder to solve over long time horizon, with vital dynamics, and with waning vaccine efficacy—in this case several vaccination peaks arise, which increases dramatically the complexity of decision-making. It is even more challenging when we

[15]This corresponds in fact to the base case presented in Section 3.1.

[16]Figures D.8–D.9 in Appendix show more details. Figures D.6–D.7 and Figures D.10–D.11 in Appendix display the same results for vaccination cost in Population 1 of $c = 11$ and $c = 15$ respectively.

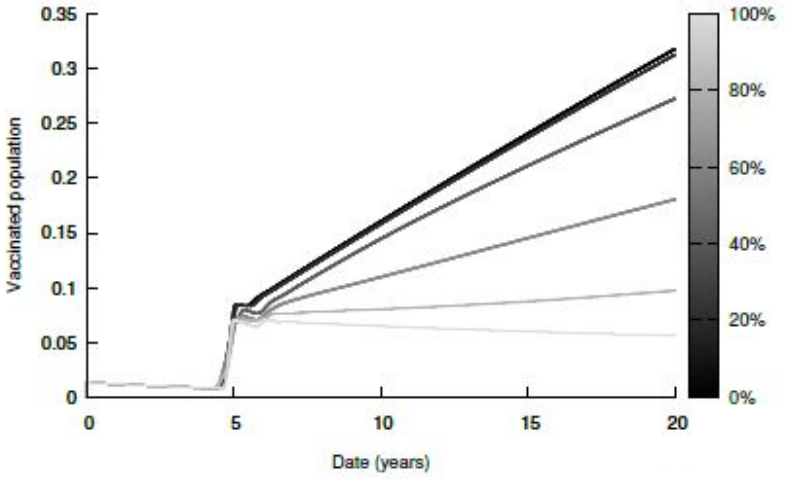


(a) Population 0

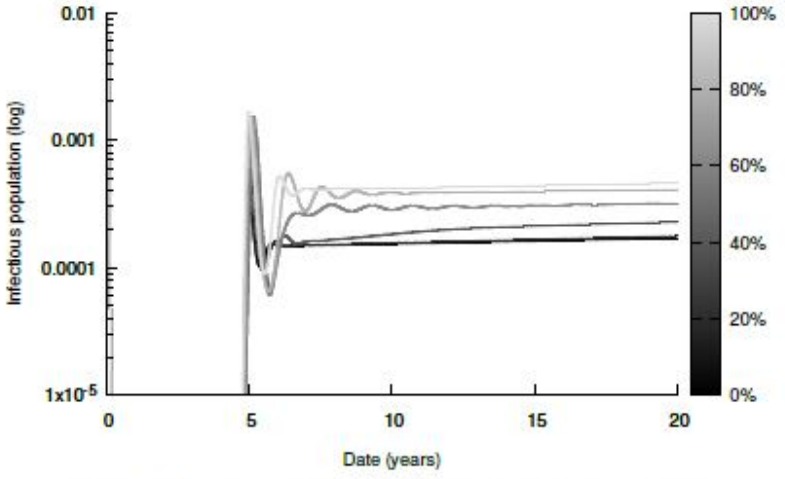


(b) Population 1

Figure 7: Vaccination decisions for two perfectly mixed populations with different costs to vaccinate. Population 0: $c = 10$, Population 1: $c = 12$. $\epsilon = 1/20$ and $n^{\epsilon}(0) = 1$. Color scale indicates the proportion of individuals in Population 1.



(a) Total number of vaccinated individuals in both populations



(b) Total Number of infectious individuals in both populations

Figure 8: Epidemiological results for two perfectly mixed populations with different costs to vaccinate. Population 0: $c = 10$, Population 1: $c = 12$. $\epsilon = 1/20$ and $n^{\epsilon}(0) = 1$. Color scale indicates the proportion of individuals in Population 1.

consider individuals with different preference types.

In this study, we exposed the evolution of such an epidemiological system taking into account 1) the forward dynamics of an epidemic, and 2) the backward individual decision-making process. We simulated a Measles-like outbreak in this setting. We obtained several vaccination peaks in the long run due to vital dynamics and waning vaccine efficacy. We compared the results of our canonical candidate benchmark with those of another possible benchmark found in the literature: adaptive vaccination decision-making. As a first extension of our model, we also modelled heterogeneous preferences in the simple case where two populations with a different stance toward vaccination coexist. Once adapted for more complex epidemic models, we believe that our approach will be able to produce benchmark results for real life epidemics in cases where vaccination is offered on a voluntary basis.

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A Alternative formulation of the problem

In Equations 4a–5d, we provide an alternative formulation of Equations 1a–2d making the construction of the model more apparent.

$$\begin{aligned} s^\epsilon(t+dt) =& s^\epsilon(t)\left[1-\alpha\chi_\epsilon(V_V^\epsilon(t+dt)-V_S^\epsilon(t+dt)-c)dt-\lambda\frac{i^\epsilon(t)}{n^\epsilon(t)}dt-\mu dt\right] \\ &+\nu n^\epsilon(t)dt+\gamma_V v^\epsilon(t)dt \end{aligned} \tag{4a}$$

$$i^\epsilon(t+dt) = i^\epsilon(t)\left(1-\gamma_I dt+\lambda\frac{s^\epsilon(t)}{n^\epsilon(t)}dt-\mu dt\right) \tag{4b}$$

$$v^\epsilon(t+dt) = v^\epsilon(t)(1-\gamma_V dt-\mu dt)+\alpha s^\epsilon(t)\chi_\epsilon(V_V^\epsilon(t+dt)-V_S^\epsilon(t+dt)-c)dt \tag{4c}$$

$$r^\epsilon(t+dt) = r^\epsilon(t)(1-\mu dt)+\gamma_I i^\epsilon(t)dt \tag{4d}$$

$$\begin{aligned} V_S^\epsilon(t) =& u_g dt+(1-\delta dt)\left\{\lambda\frac{i^\epsilon(t)}{n^\epsilon(t)}V_I^\epsilon(t+dt)dt+\alpha\xi_\epsilon(V_V^\epsilon(t+dt)-V_S^\epsilon(t+dt)-c)dt\right. \\ &\left.+\left(1-\mu dt-\lambda\frac{i^\epsilon(t)}{n^\epsilon(t)}dt\right)V_S^\epsilon(t+dt)\right\} \end{aligned} \tag{5a}$$

$$V_I^\epsilon(t) = u_b dt+(1-\delta dt)\{\gamma_I V_R^\epsilon(t+dt)dt+(1-\mu dt-\gamma_I dt)V_I(t+dt)\} \tag{5b}$$

$$V_V^\epsilon(t) = u_g dt+(1-\delta dt)\{\gamma_V V_S^\epsilon(t+dt)dt+(1-\mu dt-\gamma_V dt)V_V^\epsilon(t+dt)\} \tag{5c}$$

$$V_R^\epsilon(t) = u_g dt+(1-\delta dt)(1-\mu dt)V_R^\epsilon(t+dt) \tag{5d}$$

for t in $[0,T]$.

B Proof of Theorem 1

Without loss of generality, Equations 1a–2d can be normalized by introducing

$$\Delta_{VS}^\epsilon(t)=V_V^\epsilon(t)-V_S^\epsilon(t),\quad \Delta_{IR}^\epsilon(t)=V_I^\epsilon(t)-V_R^\epsilon(t),\quad \Delta_{IS}^\epsilon(t)=V_I^\epsilon(t)-V_S^\epsilon(t),$$
$$\overline{s^\epsilon}(t)=s^\epsilon(t)/n^\epsilon(t),\quad \overline{i^\epsilon}(t)=i^\epsilon(t)/n^\epsilon(t),\quad \overline{v^\epsilon}(t)=v^\epsilon(t)/n^\epsilon(t),\quad \overline{r^\epsilon}(t)=r^\epsilon(t)/n^\epsilon(t).$$

For all t in $[0,T]$ we get

$$\frac{d}{dt}\overline{s^\epsilon}(t) = -\overline{s^\epsilon}(t)\left(\alpha\chi_\epsilon(\Delta^\epsilon_{VS}(t)-c)+\lambda\overline{i^\epsilon}(t)+\nu\right)+\nu+\gamma_V\overline{v^\epsilon}(t) \tag{6a}$$

$$\frac{d}{dt}\overline{i^\epsilon}(t) = -\overline{i^\epsilon}(t)\left(\gamma_I-\lambda\overline{s^\epsilon}(t)+\nu\right) \tag{6b}$$

$$\frac{d}{dt}\overline{v^\epsilon}(t) = -\overline{v^\epsilon}(t)\left(\gamma_V+\nu\right)+\alpha\overline{s^\epsilon}(t)\chi_\epsilon(\Delta^\epsilon_{VS}(t)-c) \tag{6c}$$

$$\frac{d}{dt}\overline{r^\epsilon}(t) = -\overline{r^\epsilon}(t)\nu+\gamma_I\overline{i^\epsilon}(t) \tag{6d}$$

and

$$-\frac{d}{dt}\Delta^\epsilon_{VS}(t) = -(\delta+\mu+\gamma_V)\Delta^\epsilon_{VS}(t)-\alpha\xi_\epsilon(\Delta^\epsilon_{VS}(t)-c)-\lambda\overline{i^\epsilon}(t)\Delta^\epsilon_{IS}(t) \tag{7a}$$

$$-\frac{d}{dt}\Delta^\epsilon_{IS}(t) = (u_b-u_g)-(\delta+\mu+\lambda\overline{i^\epsilon}(t))\Delta^\epsilon_{IS}(t)-\alpha\xi_\epsilon(\Delta^\epsilon_{VS}(t)-c)-\gamma_I\Delta^\epsilon_{IR}(t) \tag{7b}$$

$$-\frac{d}{dt}\Delta^\epsilon_{IR}(t) = (u_b-u_g)-(\delta+\mu+\gamma_I)\Delta_{IR}(t) \tag{7c}$$

By definition, $\overline{s^\epsilon}(t)+\overline{i^\epsilon}(t)+\overline{r^\epsilon}(t)+\overline{v^\epsilon}(t)=1$. Hence we can write Equations 6a–7c as fixed-point problem

$$(\overline{s^\epsilon}(t),\overline{i^\epsilon}(t),\overline{v^\epsilon}(t),\Delta^\epsilon_{VS}(t),\Delta^\epsilon_{IS}(t),\Delta^\epsilon_{IR}(t)) = \Phi(\overline{s^\epsilon}(t),\overline{i^\epsilon}(t),\overline{v^\epsilon}(t),\Delta^\epsilon_{VS}(t),\Delta^\epsilon_{IS}(t),\Delta^\epsilon_{IR}(t)).$$

After some computation on the integral version of Equations 6a–7c, we have

$$\sup_{[0,t]}\|\Phi(u)-\Phi(v)\|(t) \le C_\epsilon(t)\sup_{[0,t]}\|u-v\|(t),$$

where

$$C_\epsilon(t) \le t\ \max\begin{bmatrix} \alpha(1+\frac{1}{4\epsilon})+2\lambda+\nu+\gamma_V, \\ \gamma_I+2\lambda+\nu, \\ \gamma_V+\nu+\alpha(1+\frac{1}{4\epsilon}), \\ \delta+\mu+\gamma_V+3\alpha+\lambda, \\ \delta+\mu+2\lambda+3\alpha+\gamma_I \end{bmatrix}.$$

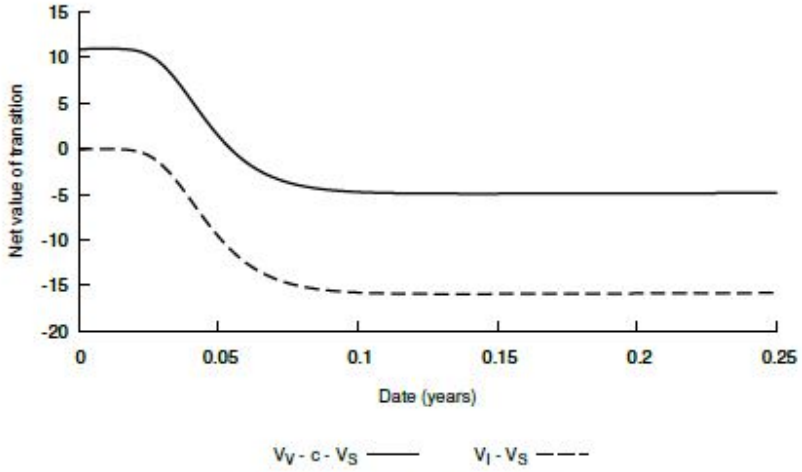
Let $M = C_\epsilon(T)+1$. Then

$$\sup_{t\in[0,T]}\|(\Phi(u)-\Phi(v))e^{-Mt}\|(t) \le \frac{C_\epsilon(T)}{M}\left(1-e^{-MT}\right)\sup_{t\in[0,T]}\|(u-v)e^{-Mt}\|(t).$$

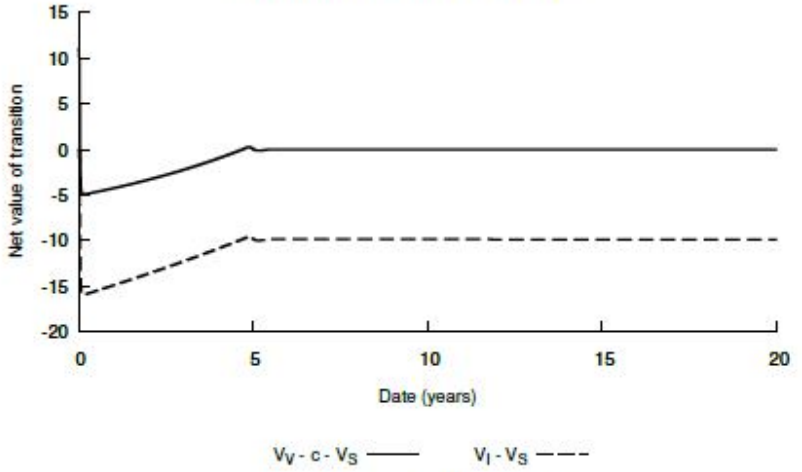
Hence, by contraction mapping theorem on $C^0([0,T])$, there exists a unique solution to Equations 6a–7c and hence to Equations 1a–2d.

C Additional figures: base case and adaptive agents

D Additional figures: heterogenous cost to vaccinate



(a) First quarter of the epidemic



(b) First 20 years of the epidemic

Figure C.1: Net value of health status transistion for vaccination cost $c = 10$, $\epsilon = 1/600$, and $n^{\epsilon}(0) = 1$.

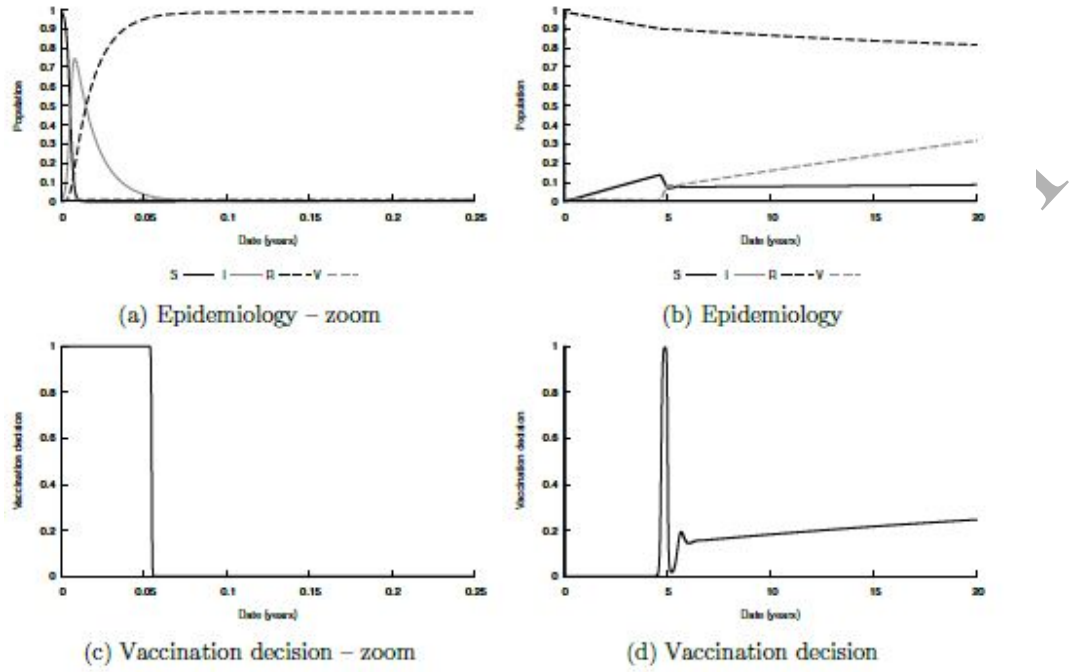


Figure C.2: Epidemiological results and vaccination decisions with $\epsilon = 1/20$ and $n^{\epsilon}(0) = 1$.

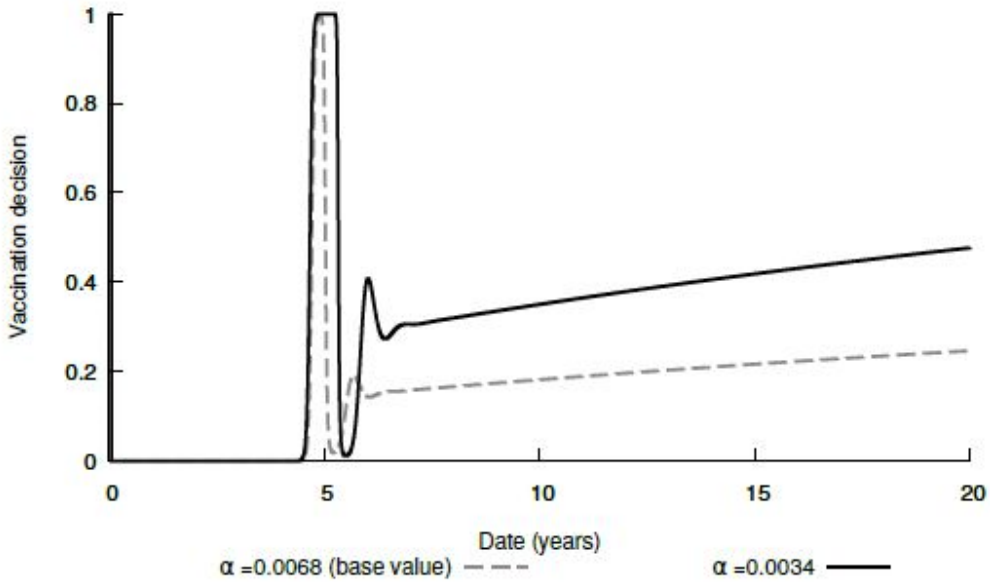
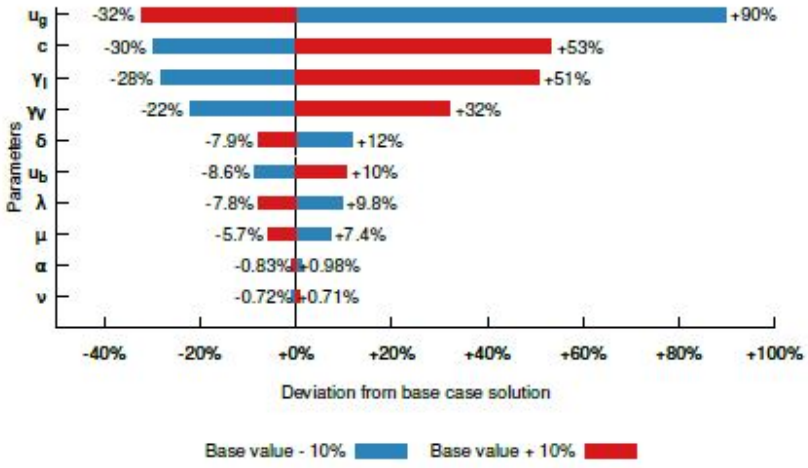
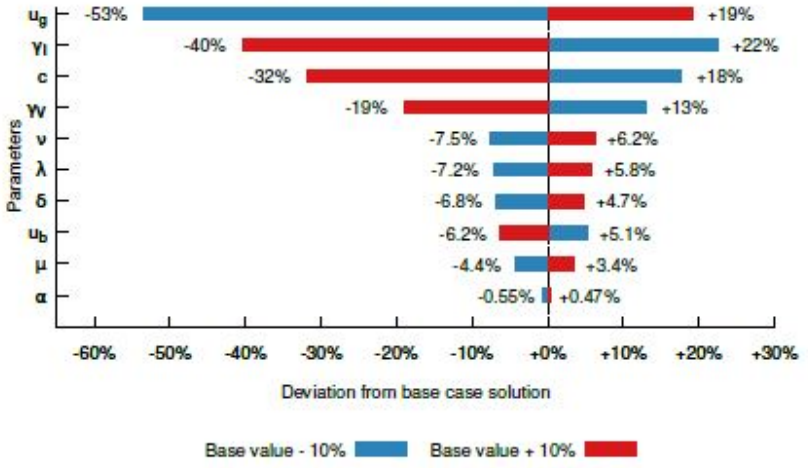


Figure C.3: Sensitivity to α of the long term vaccination decision in our base case model for $\epsilon = 1/20$.



(a) Stationary i/n sensitivity



(b) Stationary v/n sensitivity

Figure C.4: Sensitivity analysis of the stationary values of i/n and v/n in our base case model for $\epsilon = 1/20$, and a $\pm 10\%$ variation of each parameter.

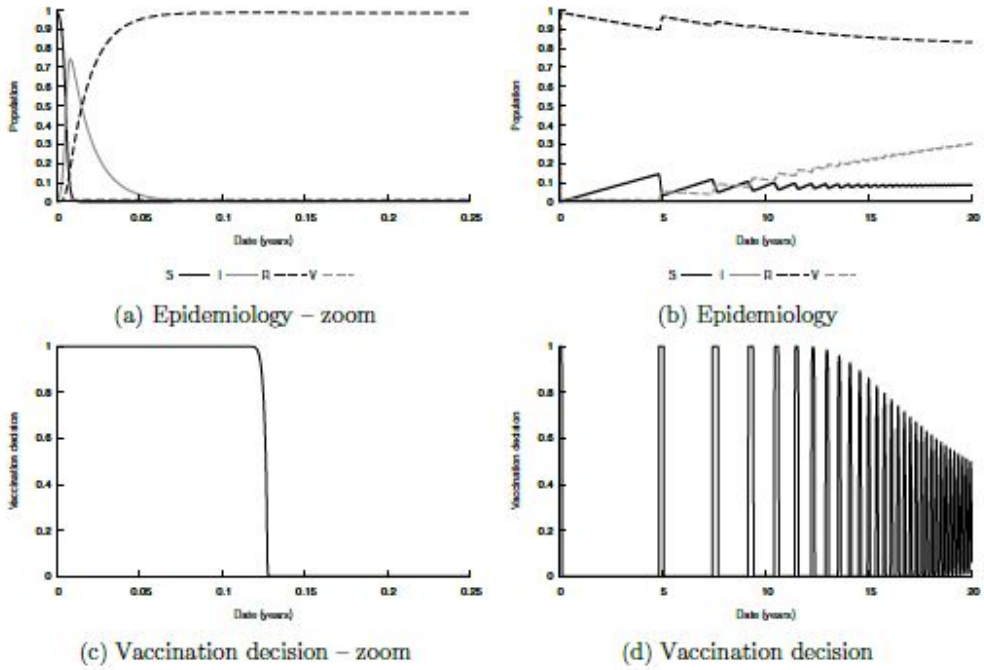
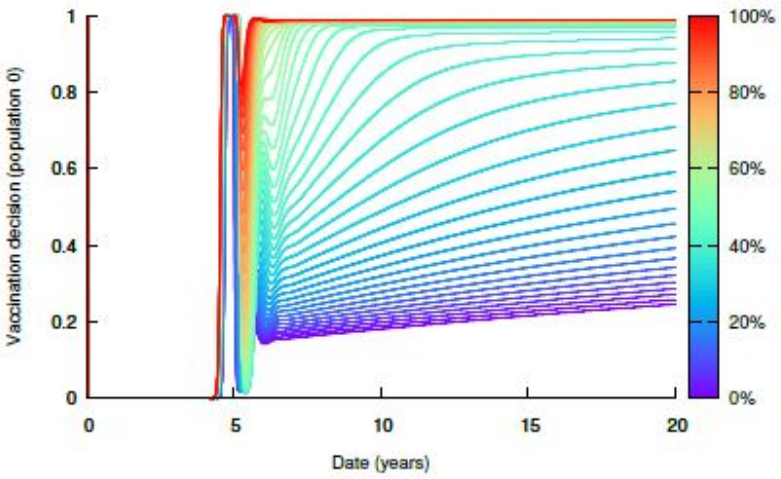
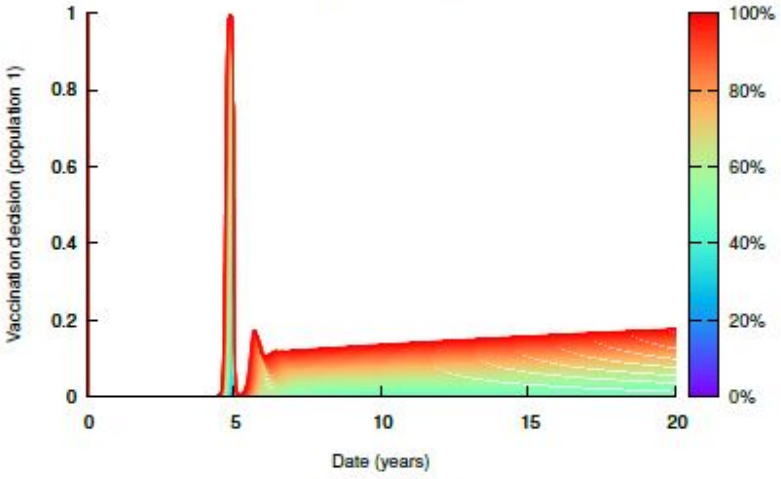


Figure C.5: Epidemiological results and vaccination decisions with $\epsilon = 1/20$ and $n^{\epsilon}(0) = 1$ – Individuals with adaptive behavior.

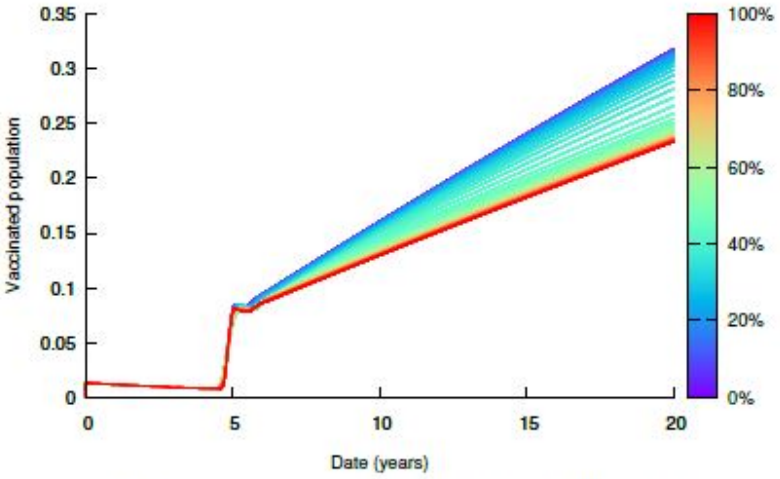


(a) Population 0

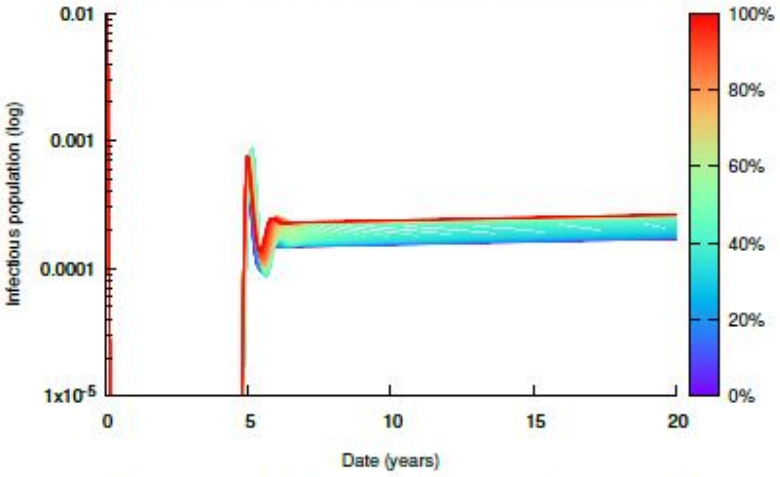


(b) Population 1

Figure D.6: Vaccination decisions for two perfectly mixed populations with different costs to vaccinate. Population 0: $c = 10$, Population 1: $c = 11$. $\epsilon = 1/20$ and $n^{\epsilon}(0) = 1$. Color scale indicates the proportion of individuals in Population 1.

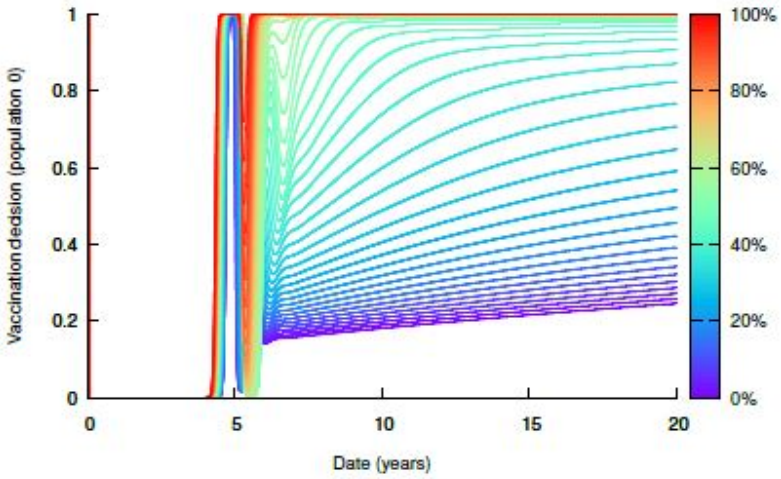


(a) Total number of vaccinated individuals in both populations

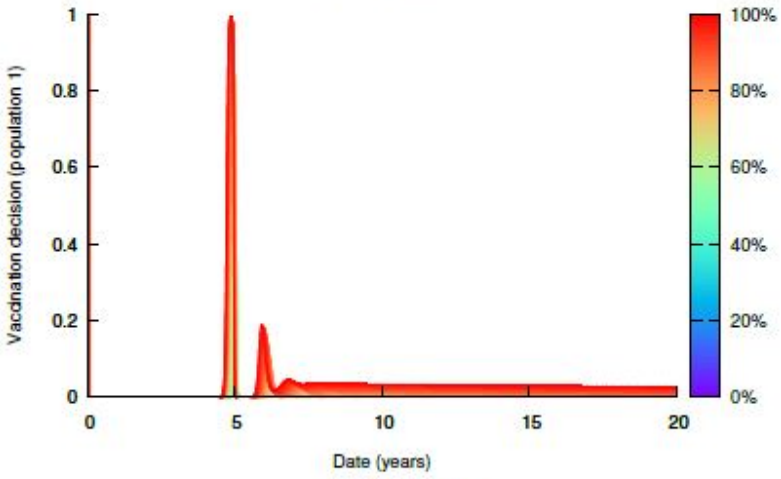


(b) Total number of infectious individuals in both populations

Figure D.7: Epidemiological results for two perfectly mixed populations with different costs to vaccinate. Population 0: $c = 10$, Population 1: $c = 11$. $\epsilon = 1/20$ and $n^{\epsilon}(0) = 1$. Color scale indicates the proportion of individuals in Population 1.

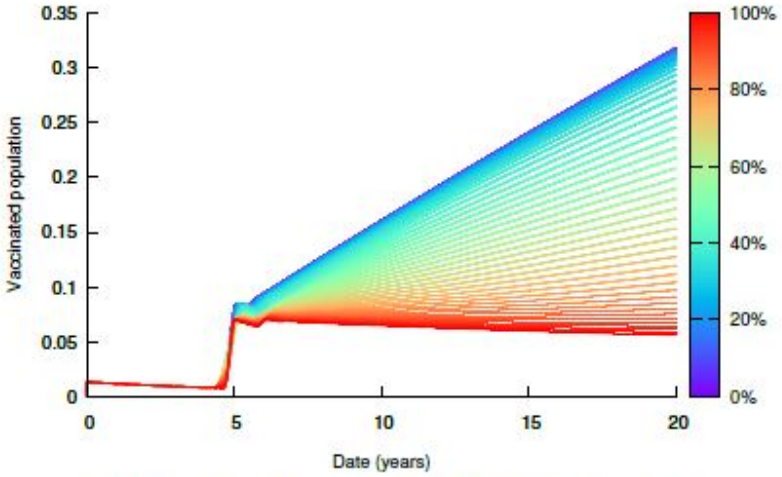


(a) Population 0

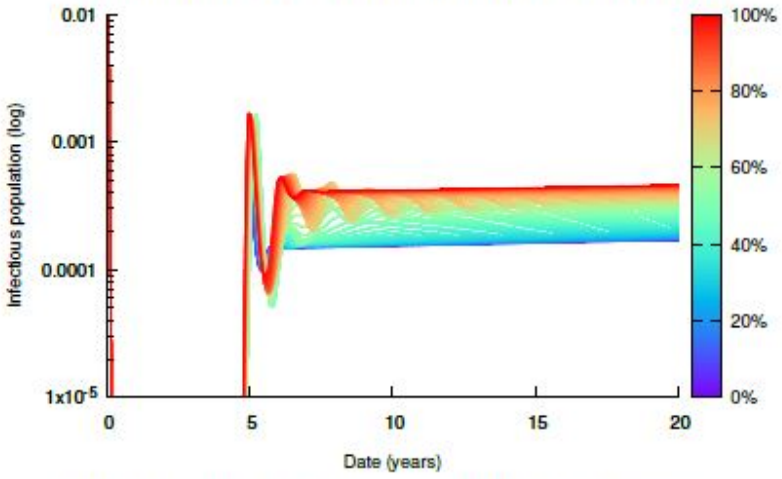


(b) Population 1

Figure D.8: Vaccination decisions for two perfectly mixed populations with different costs to vaccinate. Population 0: $c = 10$, Population 1: $c = 12$. $\epsilon = 1/20$ and $n^{\epsilon}(0) = 1$. Color scale indicates the proportion of individuals in Population 1.

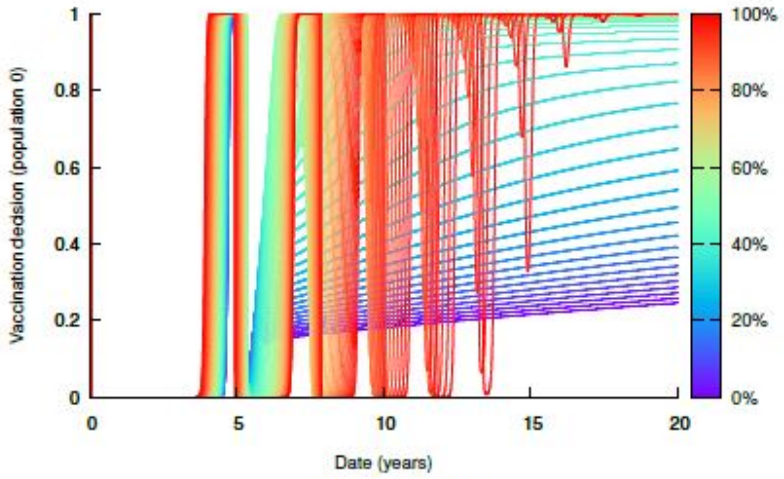


(a) Total number of vaccinated individuals in both populations

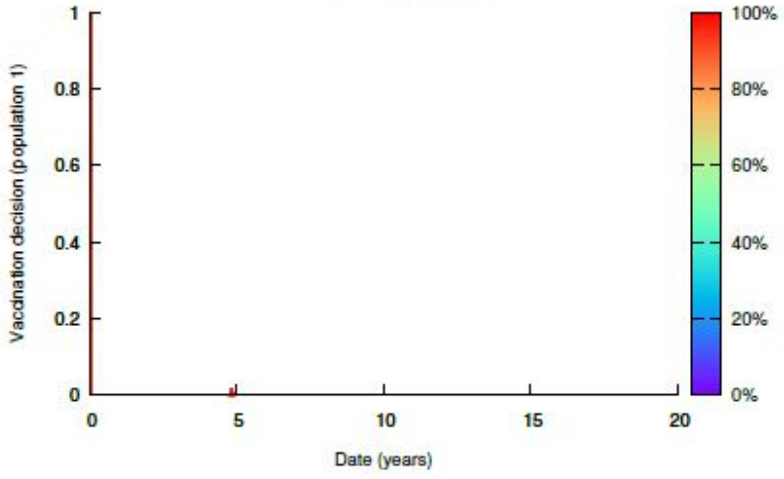


(b) Total number of infectious individuals in both populations

Figure D.9: Epidemiological results for two perfectly mixed populations with different costs to vaccinate. Population 0: $c = 10$, Population 1: $c = 12$. $\epsilon = 1/20$ and $n^{\epsilon}(0) = 1$. Color scale indicates the proportion of individuals in Population 1.

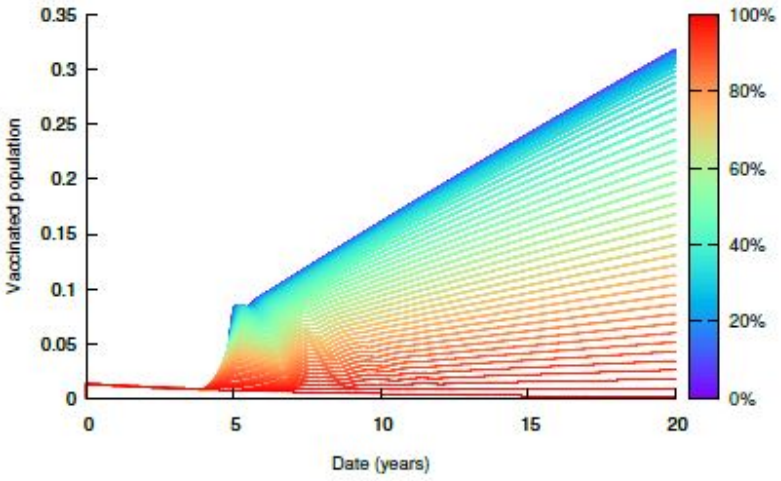


(a) Population 0

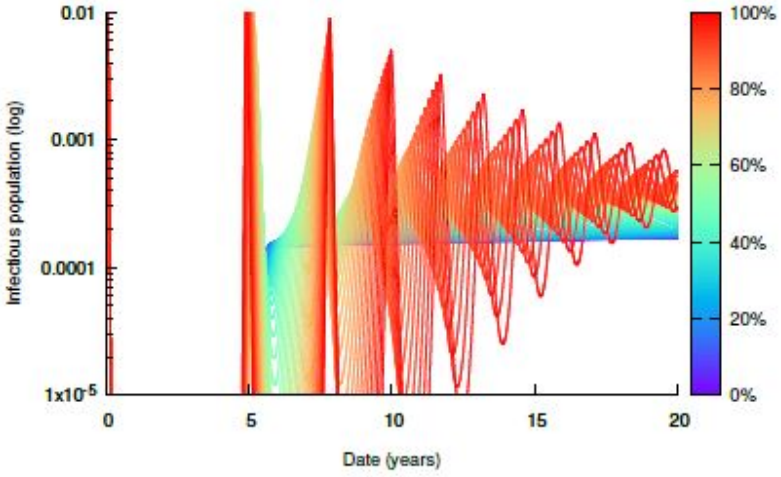


(b) Population 1

Figure D.10: Vaccination decisions for two perfectly mixed populations with different costs to vaccinate. Population 0: $c = 10$, Population 1: $c = 15$. $\epsilon = 1/20$ and $n^{\epsilon}(0) = 1$. Color scale indicates the proportion of individuals in Population 1.



(a) Total number of vaccinated individuals in both populations



(b) Total number of infectious individuals in both populations

Figure D.11: Epidemiological results for two perfectly mixed populations with different costs to vaccinate. Population 0: $c = 10$, Population 1: $c = 15$. $\epsilon = 1/20$ and $n^{\epsilon}(0) = 1$. Color scale indicates the proportion of individuals in Population 1.