# COVID-19: What If Immunity Wanes?*

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April 2, 2021

**Abstract**

*Using a simple economic model in which social-distancing reduces contagion, we study the implications of waning immunity for the epidemiological dynamics and social activity. If immunity wanes, we find that COVID-19 likely becomes endemic and that social-distancing is here to stay until the discovery of a vaccine or cure. But waning immunity does not necessarily change optimal actions on the onset of the pandemic. Decentralized equilibria are virtually independent of waning immunity until close to peak infections. For centralized equilibria, the relevance of waning immunity decreases in the probability of finding a vaccine or cure, the costs of infection (e.g., infection-fatality rate), the degree of partial immunity, and the presence of other NPIs that lower contagion (e.g., quarantining and mask use). In simulations calibrated to July 2020, our model suggests that waning immunity is virtually unimportant for centralized equilibria until at least 2021. This provides vital time for individuals and policymakers to learn about immunity against SARS-CoV-2 before it becomes critical.*

*JEL Classification*: D62; E17; I12; I18.
*Keywords*: COVID-19; SARS-CoV-2; Optimal Policy; Partial Immunity; Social-Distancing; Waning Immunity.

*We are deeply grateful for the comments and suggestions of the editor, Victor Aguirregabiria, and of the anonymous referee. They have helped us to significantly improve the paper.

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# 1 Introduction

We do not know yet the duration of immunity against severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) causing coronavirus infectious disease 2019 (COVID-19). But early evidence points to waning immunity against SARS-CoV-2 (Seow et al., 2020) and we know that immunity against other coronaviruses wanes within two years (Edridge et al., 2020; Huang et al., 2020; Kellam and Barclay, 2020).

If immunity against COVID-19 indeed wanes, then COVID-19 likely becomes endemic and herd immunity cannot be naturally reached. Therefore, ignoring waning immunity may lead to costly policies with irreversible consequences. Despite these risks, almost all the economics literature on the COVID-19 pandemic assumes permanent immunity.[1] Our paper fills this gap in the literature by assessing the implications of waning immunity for decentralized and centralized equilibria in an economic model of an epidemic.

In the model, decision makers are constrained by disease contagion and maximize the difference between the utility from social activity and the cost of infection. The utility from social activity captures, in a stylized way, all the payoffs from economic and social actions that require physical proximity. Our approach is grounded in three reasons.[2] First, the main economic impact of the pandemic has been on sectors that rely on physical proximity (Chetty et al., 2020). Second, there are also other significant

[1] In an already large and fast-growing economics literature addressing the COVID-19 pandemic, we are only aware of three papers allowing for waning immunity. We contrast our paper with these three papers below. The assumption of permanent immunity is also common outside of the economics literature: e.g., Ferguson et al. (2020) and Wang et al. (2020).

[2] Among various approaches to study epidemics in economic models, ours follows Farboodi, Jarosch and Shimer (2020), Garibaldi, Moen and Pissarides (2020), Guimarães (2020), and Toxvaerd (2020) by directly modeling the choice of social activity. Another approach is to assume contacts are a function of the level and type of i) consumption (Eichenbaum, Rebelo and Trabandt, 2020*a*,*b*; Krueger, Uhlig and Xie, 2020) and/or ii) labor (Eichenbaum, Rebelo and Trabandt, 2020*a*,*b*; Glover et al., 2020). Yet another approach is treating pandemics as exogenous shifts in state variables (e.g., human capital) (Boucekkine, Diene and Azomahou, 2008). Such an approach resembles the MIT shock assessed by Guerrieri et al. (2020) in the context of the COVID-19 pandemic. See also Philipson and Posner (1993), Kremer (1996), Chakraborty, Papageorgiou and Pérez Sebastián (2010) and Greenwood et al. (2019) for an economic perspective of HIV and malaria.

costs of constrained social activity such as anxiety, distress, fatigue, and domestic violence (Ravindran and Shah, 2020; Serafini et al., 2020). Third, contagion of virus causing respiratory diseases is mostly unrelated with consumption and work (Ferguson et al., 2006; Eichenbaum, Rebelo and Trabandt, 2020*a*) but can be influenced by behavior.

The epidemiological dynamics in the model is based on recurrence relations between three (main) health states: susceptible (S), infected (I), and recovered (R) with the flow pattern $S \to I \to R \to S$ (and hence the conventional labeling SIRS).[3] An SIRS model nests both SIR and SIS models.[4] The canonical SIR model (Kermack and McKendrick, 1927) assumes that agents are permanently immune after they recover from the infection and is widely used in the economics literature addressing the COVID-19 pandemic (e.g., Alvarez, Argente and Lippi, 2020; Atkeson, 2020; Eichenbaum, Rebelo and Trabandt, 2020*a*; Farboodi, Jarosch and Shimer, 2020). The canonical SIS model assumes that agents are never immune and, thus, is employed in studying the economics of recurrent diseases (e.g., Goenka and Liu, 2012, 2019; Goenka, Liu and Nguyen, 2014). An SIRS model is between an SIR and an SIS model by allowing agents to be immune but only temporarily. In light of the evidence on immunity against SARS-CoV-2 and other coronaviruses, an SIRS model is warranted to study the COVID-19 pandemic (Kellam and Barclay, 2020).

In the canonical SIRS model, immunity is a binary variable: agents are either immune or not. And after agents lose immunity they become as susceptible as any other susceptible agent. Waning immunity, however, does not necessarily mean that agents who lose immunity are as unprotected as those who were never infected (Punt et al., 2018; Huang et al., 2020).[5] Immunological memory (e.g., antibody count) might not be enough to avoid a reinfection but is likely enough for the body to react faster to a reinfection; put differently, agents can become partially immune after an infection. For this

[3] The flow from recovered to susceptible stems from waning immunity.
[4] For an accessible review of epidemiological models, see Hethcote (2000).
[5] In particular, Huang et al. (2020) report that individuals can be infected with the same human coronaviruses one year after first infection but with lower severity.

reason, our SIRS block allows susceptible agents to differ among themselves based on infection history. The heterogeneity in infection history and implied partial immunity can be captured by distinct i) probabilities of being infected, ii) recovery speed, iii) viral shedding, and iv) cost of infection. These possible distinctions are important as they may prevent an endemic COVID-19.

In Section 4, we analyze the simplest case in which all susceptible agents, irrespective of their infection history, are alike. We reach two main conclusions. First, if immunological memory wanes, there is no vaccine or cure, and there is no major exogenous change in the contagiousness of the virus, then COVID-19 becomes endemic because of the continuous flow of agents into the susceptible health state. In this scenario, both a social planner and decentralized individuals choose to social-distance forever. Second, the duration of immunity may not meaningfully change optimal choices in the initial months of the pandemic. We find that the decentralized equilibria is virtually independent of waning immunity for more than six months and until close to peak infections because agents abstract from how their actions affect the probability that they are reinfected later. In slight contrast, we find that the centralized equilibria may vary with waning immunity depending on the costs of infection and the probability of finding a vaccine or cure.

An endemic COVID-19, induced by waning immunity, implies a higher present value of infection costs than a non-endemic one. In response to these higher costs, the social planner mandates further social-distancing. Yet, this extra social-distancing stemming from waning immunity can be small in the short run. If a vaccine is expected in 18 months and the costs of infection reflect an infection-fatality rate of 0.64%, we find that optimal centralized policies are almost independent of waning immunity for more than 12 months. In this case, the short-term costs of infection are so high that the social planner severely constrains social activity to postpone those costs and wait for a vaccine. As social activity is already highly constrained, the marginal cost for society to further increase social-distancing is huge. Thus, the social planner finds that the

expected costs due to the endemic steady-state are of little relative importance in the short-term and almost does not react to them. In other words, when the short-term costs of the pandemic are very large, waning immunity is relatively unimportant at the early months of the pandemic.

If, on the other hand, the costs of infection are low (e.g., reflecting an infection-fatality rate close to 0.2%), the costs of the pandemic are lower and the social planner mandates less social-distancing. As the costs are lower, the marginal cost of social-distancing are not prohibitively high, giving the social planner room for maneuver to act early to the prospect of the endemic steady-state. Therefore, when immunity wanes, the social planner prefers to mandate relatively more social-distancing in the early months of the pandemic to reduce the costs of the endemic steady-state and gain time for a vaccine to arrive. Finally, a lower probability of discovering a vaccine increases the weight of future utility in the objective function in the same way as a lower discount factor does. This has an entirely different effect depending on waning immunity. When immunity is permanent, future utility is relatively high as the pandemic asymptotically disappears, which demotivates the social planner to postpone infections and mandate social-distancing. But, if immunity wanes in 10 months or two years on average, the present value of the costs of an endemic COVID-19 increase when a vaccine is expected to arrive late. Therefore, the social planner prefers to social-distance even more in the early days of the pandemic and act early to the problem of waning immunity. In sum, waning immunity only meaningfully changes centralized policies when the probability of discovering a vaccine is low or the societal marginal costs of acting early to the endemic steady-state are not unbearably high.

In Section 5, we analyze the case in which immunity wanes but susceptible agents differ based on infection history. Consistent with our previous results, if a vaccine is expected in 18 months and the costs of infections reflect an infection-fatality rate of 0.64%, knowing whether susceptible agents differ based on infection history is not critical in the initial months of the epidemic. Furthermore, if agents that lost immunity are

less likely to be infected or shed less virus, then COVID-19 does not become endemic. In this scenario, lower costs of infection and lower probability of finding a vaccine lead to markedly different choices in the short run. Thus, it is important to know whether immunity wanes and whether susceptible agents notably differ based on infection history. Finally, we find that susceptible agents that were immune can be excessively active from a social viewpoint, especially if they suffer much less from a reinfection, because they abstract from the risk of infecting others. Thus, policymakers should be aware of this extra source of risk if immunity wanes.

In Section 6, we change the starting date of the simulations. Our previous results are based on initial conditions matching the start of the COVID-19 pandemic. In this section, we account for the (epidemiological) state as of July 2020 as well as other non-pharmaceutical interventions (NPIs) in place besides social-distancing (e.g., mandatory mask use and quarantining of identified infected individuals). We find that, even if COVID-19 becomes endemic, the other NPIs in place allow for much more social activity. Furthermore, learning how the infection history affects the protection of individuals against reinfections becomes less important as contagion falls substantially. In fact, even a low probability of finding a vaccine or low costs of infection do not lead to markedly different centralized responses for many months. We conclude that individuals and policymakers have at least until 2021 to learn about the duration of immunity before it becomes truly important for decision making.

In our last set of simulations, in Section 7, we consider a further robustness check by changing our model to explicitly include vaccinated individuals. In this variant, the date and pace of vaccination are known to all individuals at the onset of epidemic, and vaccinated individuals are permanently immune. We find that the majority of our results regarding the importance of waning immunity are robust. We only find a clearer difference between our baseline model and this variant when the costs of infection are low (e.g., reflecting an infection-fatality rate close to 0.2%). In this case, if decision makers knew that vaccination would begin one year after SARS-COV-2 was identified, then

the incentives to postpone infections are extremely large. Furthermore, very few recovered individuals would be expected to lose their immunity before being vaccinated. Therefore, waning immunity becomes less important in the early days of the pandemic even when the costs of infection are relatively low.

We are aware of three papers in the economics literature allowing for waning immunity: Eichenbaum, Rebelo and Trabandt (2020*b*), Giannitsarou, Kissler and Toxvaerd (2020), and Malkov (2020). These papers, however, differ from ours in crucial aspects including the object of study, approach, and modeling choices. Eichenbaum, Rebelo and Trabandt study the role of testing and quarantines in a model with health state uncertainty and check the robustness of their findings if immunity wanes; thus, they do not fully explore how the duration of immunity affects contagion in the context of the current pandemic. Malkov focus on how waning immunity affects the epidemiological dynamics during the COVID-19 pandemic, but he does not allow individuals and the social planner to endogenously react in his simulations. Giannitsarou, Kissler and Toxvaerd assess the centralized problem during the pandemic in case immunity wanes, but they do not contrast the centralized and decentralized equilibria and their results differ from ours due to modeling and calibration choices.[6] In Section 4.2, we contrast in more detail our results with those in the three papers.

## 2 Model

We build an economic model of an epidemic in which agents face a trade-off between social activity and exposure to the virus. This trade-off results from the link between the

[6]There are three other relevant differences. As Eichenbaum, Rebelo and Trabandt and Malkov, Giannitsarou, Kissler and Toxvaerd assume that all susceptible agents are alike irrespective of infection history. And, as Eichenbaum, Rebelo and Trabandt, Giannitsarou, Kissler and Toxvaerd place their simulations at the start of the pandemic and assume that only one non-pharmaceutical intervention is in place (testing in the case of Eichenbaum, Rebelo and Trabandt and mandatory social-distancing in the case of Giannitsarou, Kissler and Toxvaerd). Section 6, in which we include the effects of other NPIs, thus, brings further insights to policy discussions. Finally, in Giannitsarou, Kissler and Toxvaerd, the terminal date of the pandemic is certain, whereas it is uncertain in our baseline model and, in Section 7, we explicitly study the roll out of vaccination and its implications for the epidemiological dynamics.

epidemiological and utility-maximization blocks of the model. The link, in turn, stems from our assumption that new infections depend on the number of susceptible and infected agents and the social activity chosen by susceptible agents. The model is set in discrete time. The population is constant and of measure one. In terms of notation, we use upper-case letters to denote aggregate variables and the respective lower-case letters to denote variables associated with one individual.

We distinguish agents that become susceptible after recovery from agents that were never infected because the former, although no longer immune, may still have some immunological memory and be partially immune. The remaining immunological memory may allow for a lower probability of infection, faster recovery, lower viral shedding, and lower costs of infection. We refer to agents that were never infected as *primary* and agents that were infected at least once as *secondary*. To further ease our exposition, we use the index $j \in \{p, q\}$, when referring primary and secondary agents, respectively.

## 2.1 Epidemiological Block

Figure 1 summarizes the epidemiological block in our model. The population in period $t$ consists of five groups of agents: primary susceptible, $S_{p,t}$, primary infected, $I_{p,t}$, recovered, $R_t$, secondary susceptible, $S_{q,t}$, and secondary infected, $I_{q,t}$.[7] The number of new infections for each type is given by

$$\beta_j A_{j,t} S_{j,t} X_t,$$

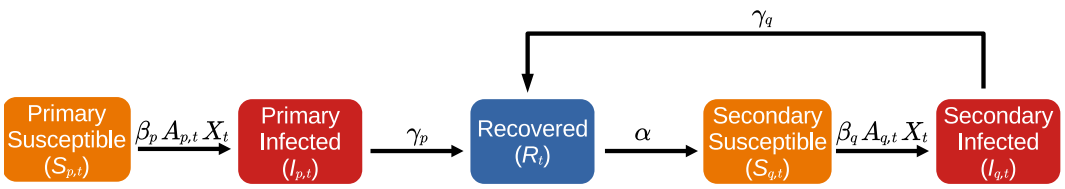
where $\beta_j$ is the measure of contagiousness for susceptible agents of type $j$ with $\beta_q \leq \beta_p$, $A_{j,t} \in [0,1]$ is the aggregate social activity of susceptible agents of type $j$, and

$$X_t = I_{p,t} + \sigma I_{q,t} \tag{1}$$

[7] Our baseline model does not include the possibility of COVID-19 related deaths. But in Appendix A we show that the results of the models with and without deaths are essentially the same in the first six years of the pandemic because the population size barely changes.

is the (effective) number of infected agents. We adjust $i_{q,t}$ with $\sigma \leq 1$ to allow secondary infected individuals shedding less virus than primary infected ones.

Figure 1: States and Flows




The laws of motion governing the transitions between health states are the following:

$$S_{p,t+1} = (1 - \beta_p A_{p,t} X_t) S_{p,t}, \tag{2}$$

$$I_{p,t+1} = \beta_p A_{p,t} S_{p,t} X_t + (1 - \gamma_p) I_{p,t}, \tag{3}$$

$$R_{t+1} = \textstyle\sum_j \gamma_j I_{j,t} + (1 - \alpha) R_t, \tag{4}$$

$$S_{q,t+1} = \alpha R_t + (1 - \beta_q A_{q,t} X_t) S_{q,t}, \tag{5}$$

$$I_{q,t+1} = \beta_q A_{q,t} S_{q,t} X_t + (1 - \gamma_q) I_{q,t}, \tag{6}$$

where $\gamma_j$ is the probability that an infected individual of type $j$ recovers and $\alpha$ is the probability that a recovered individual loses immunity. If $\alpha = 0$ and $a_{p,t} = 1$ for all $t$, the model reduces to the canonical SIR model. If $\alpha > 0$, $\sigma = 1$, $\beta_p = \beta_q$, $\gamma_p = \gamma_q$, and $a_{j,t} = 1$ for all $j$ and $t$, the model reduces to the canonical SIRS model.[8]

## 2.2 Decentralized Problem

### 2.2.1 Utility Maximization

In this section, we detail the lifetime utility maximization problem of a primary susceptible agent. Agents derive utility from their social activity, $a$. The utility function,

[8] Under permanent immunity, $\alpha = 0$, the number of secondary susceptible agents remains zero. Under waning immunity, $\alpha > 0$, with $\sigma = 1$, $\beta_p = \beta_q$, and $\gamma_p = \gamma_q$, identifying secondary agents is trivial.

denoted by $u(a)$ is single-peaked and its maximum is normalized to zero at $a = 1$. The maximization problem of a primary susceptible agent is given by

$$\max_{\{a_{p,t},a_{q,t}\}_{t=0}^{\infty}} \sum\nolimits_{t=0}^{\infty} \sum\nolimits_{j} \Lambda^t \Big( s_{j,t} u(a_{j,t}) - \kappa_j i_{j,t} \Big),$$

subject to

$$\begin{aligned}
s_{p,t+1} &= (1 - \beta_p a_{p,t} X_t) s_{p,t}, \\
i_{p,t+1} &= \beta_p a_{p,t} s_{p,t} X_t + (1 - \gamma_p) i_{p,t}, \\
r_{t+1} &= \textstyle\sum_j \gamma_j i_{j,t} + (1 - \alpha) r_t, \\
s_{q,t+1} &= \alpha r_t + (1 - \beta_q a_{q,t} X_t) s_{q,t}, \\
i_{q,t+1} &= \beta_q a_{q,t} s_{q,t} X_t + (1 - \gamma_q) i_{q,t},
\end{aligned}$$

and the initial conditions for the state variables. $s_{p,t}$, $i_{p,t}$, $r_t$, $s_{q,t}$, $i_{q,t}$ are the probabilities that the agent is in each health state in period $t$, and the constraints are the laws of motion of these probabilities. Each agent is atomistic and takes the (effective) number of infected agents, $X_t$, as given; thus, in their choice of $a_{p,t}$ and $a_{q,t}$, agents in the decentralized equilibria do not internalize the externalities of their actions. $\Lambda$ is the discount factor and $\kappa_j$ captures the costs of infection. As primary and secondary infected agents may respond differently to the infection (e.g., differ in symptoms severity), we set $\kappa_q \leq \kappa_p$. The decentralized optimum social activity is, then, governed by the transversality conditions and

$$u'(a_{j,t}) = \beta_j X_t (V_{s_j,t} - V_{i_j,t}), \tag{7}$$

$$\frac{V_{s_j,t}}{\Lambda} = u(a_{j,t+1}) + V_{s_j,t+1} - \beta_j a_{j,t+1} X_{t+1} (V_{s_j,t+1} - V_{i_j,t+1}), \tag{8}$$

$$\frac{V_{i_j,t}}{\Lambda} = -\kappa_j + V_{i_j,t+1} - \gamma_j (V_{i_j,t+1} - V_{r,t+1}), \tag{9}$$

$$\frac{V_{r,t}}{\Lambda} = V_{r,t+1} + \alpha (V_{s_q,t+1} - V_{r,t+1}), \tag{10}$$

for both $j \in \{p, q\}$ and $V_{z,t}$ denotes the (shadow) value of the agent in state $z \in \{s_p, s_q, i_p, i_q, r\}$. Eq. (7) summarizes the trade-off. Its left-hand side is the marginal util-

ity of social activity while its right-hand side is expected marginal costs resulting from the possibility of infection. Marginal costs depend on how likely they are exposed by marginally increasing activity, $\beta_j X_t$. And it also depends on the change in the value caused by exposure, which is always positive, $V_{s_j,t} - V_{i_j,t} > 0$. Thus, susceptible agents restrain their social activity, $a_{j,t} \leq 1$, to reduce exposure risk.

Eqs. (7-10), determining the behavior of primary agents, are symmetric along $j$. Given that these equations do not depend on the probability of being in any health state, the same equations also determine the behavior of secondary agents. Therefore, for brevity, we do not present the utility maximization problem of secondary agents.

### 2.2.2 Decentralized Equilibrium

Decentralized equilibria are symmetric with $a_{p,t} = A_{p,t}$ and $a_{q,t} = A_{q,t}$. Given initial values for the state variables, a decentralized equilibrium corresponds to a path of social activities, $\{A_{p,t}, A_{q,t}\}$, the number of infected agents, $X_t$, state variables, $\{S_{p,t}, S_{q,t}, I_{p,t}, I_{q,t}, R_t\}$, and shadow values, $\{V_{s_p,t}, V_{s_q,t}, V_{i_p,t}, V_{i_q,t}, V_{r,t}\}$, that satisfy the transversality conditions and Eqs. (1–10).

## 2.3 Centralized Problem

### 2.3.1 Utility Maximization

In this section, we present the maximization problem of the social planner. The social planner chooses socially optimal activity by directly influencing aggregate variables. In particular, the maximization problem of the social planner is given by

$$\max_{\{A_{p,t},A_{q,t}\}_{t=0}^{\infty}} \sum_{t=0}^{\infty} \sum_{j} \Lambda^t \Big( S_{j,t} u(A_{j,t}) - \kappa_j I_{j,t} \Big),$$

subject to Eqs. (1-6) and the initial conditions. Relative to the decentralized problem, Eq. (1) is the additional constraint because the social planner internalizes how infected individuals affect contagion; in other words, the social planner is aware of its ability to

affect $X_t$, whereas individuals are not.[9] The socially optimum social activity is, then, governed by the transversality conditions and

$$u'(A_{j,t}) = \beta_j X_t (V_{S_j,t} - V_{I_j,t}), \tag{11}$$

$$\frac{V_{S_j,t}}{\Lambda} = u(A_{j,t+1}) + V_{S_j,t+1} - \beta_j A_{j,t+1} X_{t+1} (V_{S_j,t+1} - V_{I_j,t+1}), \tag{12}$$

$$\frac{V_{I_j,t}}{\Lambda} = -\kappa_j + V_{I_j,t+1} - \gamma_j (V_{I_j,t+1} - V_{R,t+1}) - \sigma_j \textstyle\sum_j \beta_j A_{j,t+1} S_{j,t+1} (V_{S_j,t+1} - V_{I_j,t+1}) \tag{13}$$

$$\frac{V_{R,t}}{\Lambda} = V_{R,t+1} + \alpha (V_{S_q,t+1} - V_{R,t+1}), \tag{14}$$

for both $j \in \{p, q\}$, and $\sigma_j = \begin{cases} 1 & \text{if } j = p, \\ \sigma & \text{if } j = q. \end{cases}$ Comparing this set of equations governing the optimal choice of the social planner with that governing the optimal choice of agents in the decentralized problem (Eqs. 7-10), we can see that the only difference is in the shadow values of the infected states, reflecting the externality that individuals abstract from in decentralized equilibria. As a result, both $V_{I_p,t}$ and $V_{I_q,t}$ are lower than $V_{i_p,t}$ and $V_{i_q,t}$, which (*ceteris paribus*) tends to further restrain social activity relative to decentralized equilibria. Part of our objective in this paper is to analyze how the possibility of recovered agents losing immunity distances decentralized and centralized choices.

### 2.3.2 Centralized Equilibrium

Given the initial values for the state variables, a centralized equilibrium corresponds to a path of social activities, $\{A_{p,t}, A_{q,t}\}$, the number of infected agents, $X_t$, state variables, $\{S_{p,t}, S_{q,t}, I_{p,t}, I_{q,t}, R_t\}$, and shadow values, $\{V_{S_p,t}, V_{S_q,t}, V_{I_p,t}, V_{I_q,t}, V_{R,t}\}$, that satisfy the transversality conditions, Eqs. (1-6), and Eqs. (11-14).

[9] This key distinction between decentralized and centralized equilibria is well explained in Gersovitz and Hammer (2004) and Rowthorn and Toxvaerd (2020).

# 3 Calibration

We summarize our parameter choices in Table 1. Each period in the model corresponds to one day. The discount factor includes both a time discount rate, $\rho$, and the probability of finding a cure-for-all, $\delta$, which is a shock that ends the epidemic instantaneously, curing all infected individuals and/or granting permanent immunity to all.[10] In particular, we set $\Lambda = \frac{1}{1+\rho}\frac{1}{1+\delta}$, $\rho = 0.05/365$, and $\delta = 0.67/365$ reflecting a yearly discount rate of $5\%$ and the probability of finding the cure-for-all of $67\%$ within a year (see, e.g., Alvarez, Argente and Lippi, 2020; Farboodi, Jarosch and Shimer, 2020).

Table 1: Benchmark Calibration

| | |
|---|---|
| Discount factor: | $\Lambda = \frac{1}{1+0.05/365}\frac{1}{1+0.67/365}$ |
| Cost of infection: | $\kappa_p = \kappa_q = 28.44$ |
| Average number of days as infected: | $\gamma_p^{-1} = \gamma_q^{-1} = 18$ |
| Infectiousness: | $\beta_p = \beta_q = 2.4/18$ |
| Average number of days immune: | $\alpha^{-1} = 750$ |
| Relative viral shedding of secondary infected: | $\sigma = 100\%$ |

As in Farboodi, Jarosch and Shimer (2020) and Guimarães (2020), the utility of social activity is determined by:

$$u(a) = \log(a) - a + 1, \tag{15}$$

which guarantees that $u(a)$ is single-peaked with maximum at $a = 1$ and $u(1) = u'(1) = 0$. We also follow Farboodi, Jarosch and Shimer to calibrate the costs of infection, $\kappa_p$. In particular, we proxy the costs of infection using the expected utility loss per day due to the risk of dying when infected.[11] Assuming that the value of statistical life is US$10

[10] In the model, a higher probability of finding a cure-for all implies a higher discount rate, reducing the present value of the future costs of infection because the probability of infection and of requiring social-distancing are lower in the future. In Section 7, we study a variant of the model without cure-for-all but in which vaccination is known to begin at a predetermined date. Almost all our conclusions are robust to this change.

[11] Our baseline model abstracts from demographics and COVID-19 related deaths and, thus, an infection does not materialize in death. Appendix A, however, shows that the models with and without these

million and assessing how much agents would be willing to permanently reduce their consumption to permanently lower the probability of dying by $0.1\%$, we find that the value of life is $80000$ in model units. The meta-analysis of Meyerowitz-Katz and Merone (2020) suggests that about $0.64\%$ of those infected with the virus, die. Thus, we set $\kappa_p = 512\gamma_p$.

We follow Atkeson (2020) and most of the economics literature assessing the COVID-19 pandemic and assume that infected individuals remain so for 18 days, $\gamma_p^{-1} = 18$. To calibrate $\beta_p$, we follow Acemoglu et al. (2020) and assume $\beta_p = 2.4/18$, implying a basic reproduction number, $R0$, of $2.4$. This number is relatively optimistic in light of, for example, the $R0$ assumed in Alvarez, Argente and Lippi (2020) of $3.6$.

At this stage, the duration of immunity against COVID-19 and how secondary agents differ from primary agents is unknown. To calibrate the probability that recovered individuals lose immunity, we use the evidence regarding other coronaviruses surveyed in Huang et al. (2020) and also the assumption in Eichenbaum, Rebelo and Trabandt (2020*b*) and set $\alpha^{-1} = 1/750$, implying that agents have immunity for about two years. Regarding the remaining parameters, in our benchmark we simply assume that $\beta_q = \beta_p$, $\gamma_q = \gamma_p$, $\kappa_q = \kappa_p$ and $\sigma = 100\%$. Therefore, our benchmark calibration implies a SIRS model augmented with the endogenous choice of social activity.

We solve the model using a shooting algorithm as detailed in Garibaldi, Moen and Pissarides (2020). As a starting point, we assume that 1 in a million agents are primary infected, $I_p = 1/10^6$, and the remaining are primary susceptible.

# 4 Results

considerations deliver essentially the same results as long as the cost of infection and the cost of dying imply the same expected cost when an agent becomes infected. Therefore, following this calibration strategy, our model captures the effects of the risk of dying in a more stylized framework.

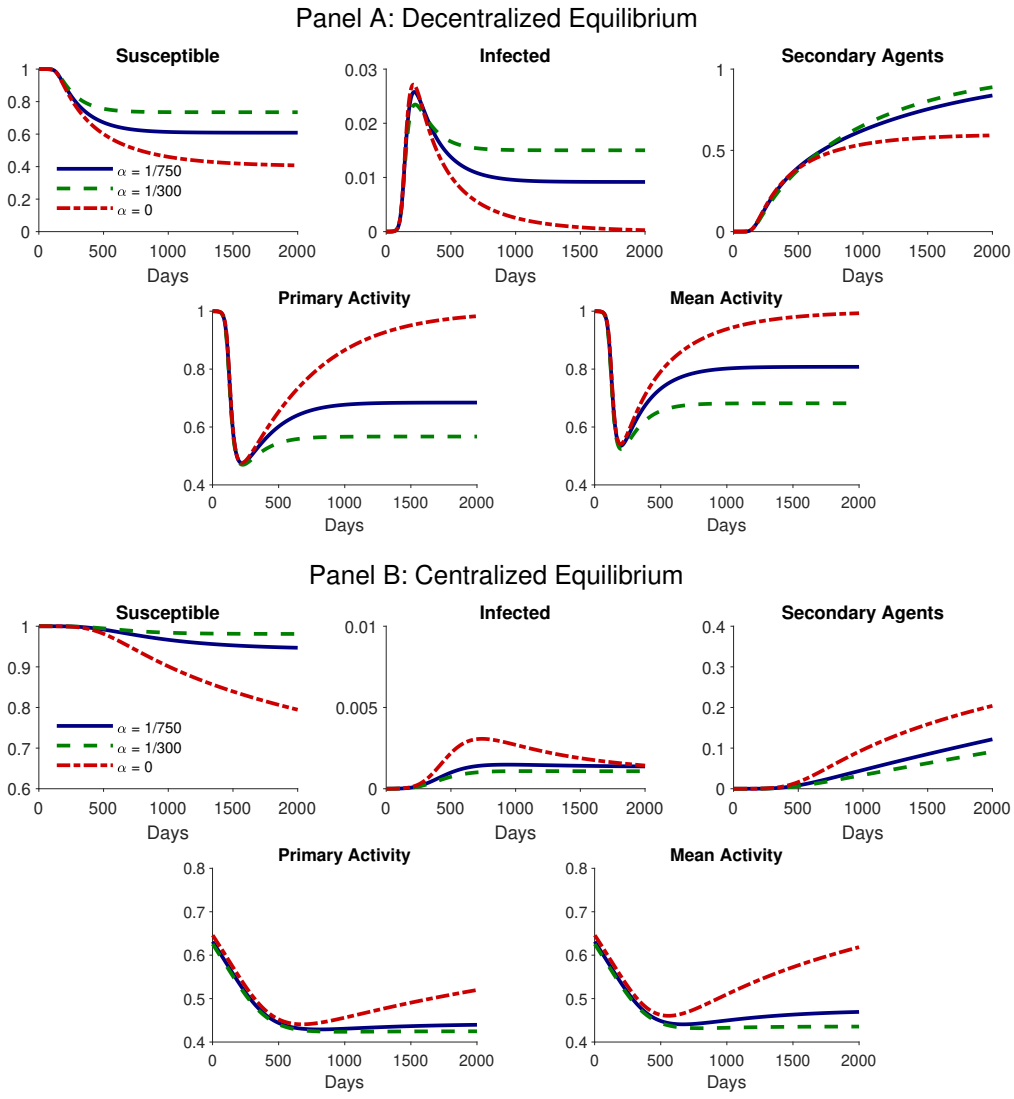
### 4.1 Main Results

Panels A and B of Figure 2 present how a waning immunological memory affects optimal decentralized and centralized dynamics, respectively. The blue (solid) lines assume our benchmark, i.e., agents are immune for two years on average. The green (dashed) lines assume, as a lower bound and consistent with Huang et al. (2020) and Kissler et al. (2020), that immunological memory lasts only 10 months. The red (dot-dashed) lines assume, as an upper bound and as in the economics literature (e.g. Alvarez, Argente and Lippi, 2020; Eichenbaum, Rebelo and Trabandt, 2020*a*; Farboodi, Jarosch and Shimer, 2020), that immunity lasts forever (implying an SIR model).

Two of our findings in Figure 2 are striking. First, if immunological memory wanes ($\alpha > 0$), then in both centralized and decentralized equilibria, social activity is severely and permanently curtailed until the discovery of a vaccine or cure. This results from the continuous flow of agents from immune to susceptible, implying a continuous flow from susceptible to infected and, therefore, a permanent exposure risk. Thus, if immunity wanes, COVID-19 reaches an endemic steady-state. In the centralized equilibrium, social activity stabilizes at about 55% lower than absent the epidemic. In the decentralized equilibrium, social activity reaches its minimum after about 200 days and then recovers slightly to its long run value, 30% lower than absent the epidemic. If agents never lose immunity, $\alpha = 0$, the results are very different. In this case, all agents return to normal activity as infections asymptotically disappear. This happens faster, albeit at a higher social cost, in the case of the decentralized equilibrium, leading to much higher peak infections. Furthermore, in the decentralized equilibrium, approximately 60% of the agents are infected at least once within three years, which differs substantially from about 5% in the centralized equilibrium.

Second, the underlying duration of immunity barely moves the initial dynamics of epidemiological variables and social activity for around 200 days in the decentralized and 400 days in the centralized equilibrium. This result is partly explained by the low accumulation of secondary agents as few agents obtain and lose immunity in the initial

Figure 2: The Role of Immunity Duration




*Note:* Susceptible agents are $S_{p,t} + S_{q,t}$; infected agents are $I_{p,t} + I_{q,t}$; secondary agents are $S_{q,t} + I_{q,t} + R_t$; primary activity is $A_{p,t}$ (which, in this case, equals secondary activity, $A_{q,t}$); and mean activity is $S_{p,t}A_{p,t} + S_{q,t}A_{q,t} + I_{p,t} + I_{q,t} + R_t$.

months of the epidemic even when immunity wanes after 10 months, $\alpha = 1/300$. But other factors play important roles, especially in centralized equilibria. In decentralized equilibria, agents do not take into account how their actions, by affecting infections, change the pace at which they might be reinfected. As a result, social activity in decentralized equilibria is mostly affected by the dynamics of infected agents. As soon

as many agents start losing immunity and become susceptible and infected again, the effects of waning immunity become visible in optimal social activities.

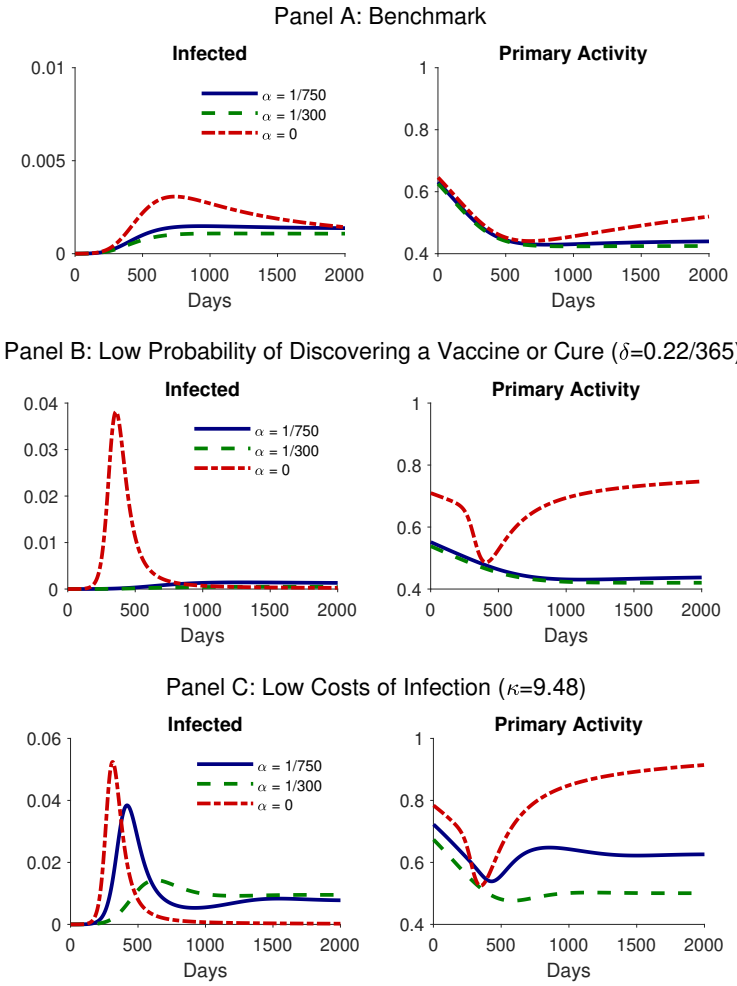
In centralized equilibria, however, the externalities of social activity are considered in decision-making. The social planner knows that by reducing social activity, it lowers and postpones infections and, thereby, decreases the number of secondary agents that lose immunity. Furthermore, the social planner is aware of the costs of the endemic steady-state. These two factors combined motivate the social planner to constrain social activity by more when waning immunity induces an endemic COVID-19. Yet, surprisingly, in our benchmark case, the optimal centralized social activity is almost unmoved by the duration of immunity for 400 days.

The social planner aims to minimize the sum of the present value of the costs of infection and of social-distancing. If immunity is permanent, Panel B of Figure 2 shows that the best option to minimize social costs is to endure high social-distancing, postpone infections, and wait for the vaccine. If, on the other hand, immunity wanes, future infection costs increase but their present value is substantially discounted because the vaccine or cure is expected in 18 months. Furthermore, as social activity is heavily constrained even if immunity is permanent, the marginal costs of social-distancing are high and very sensitive to further increases in social-distancing due to the curvature of the utility function. Put differently, the social planner lacks room to maneuver to strongly react to waning immunity in the early months of the pandemic. These two factors combined explain why waning immunity is relatively unimportant for many months in determining optimal social-distancing.

To gain further insight, in Figure 3, we show how two key parameters change the number of infected agents and social activity of primary agents in centralized equilibria. Panel A depicts again the benchmark cases to ease comparison. Panel B depicts the results when expected time to find a vaccine or cure is 4.5 years, implying $\delta$ is a third of its benchmark value. Panel C depicts the results when the infection-fatality rate is ap-

proximately 0.21%, implying $\kappa_j$ is a third of its benchmark value. This figure shows that waning immunity matters in these two deviations from benchmark in the centralized equilibria.[12] The results are particularly staggering in the case of low $\delta$: in this scenario, peak infections occur much earlier and is more than 20 times higher when immunity is persistent than when immunity wanes.

Figure 3: The Role of Immunity Duration - Centralized Equilibria




*Note:* Infected agents are $I_{p,t} + I_{q,t}$; primary activity is $A_{p,t}$ (which, in this case, equals secondary activity, $A_{q,t}$).

[12] Figure C1 in the Appendix shows that the way waning immunity affects decentralized equilibria relies much less on $\kappa_p$ and $\delta$.

A lengthier period to discover a vaccine or cure, captured by a lower $\delta$, implies that the social planner must restrict social activity for more time to avoid infections and wait for the vaccine or cure. We find that the corresponding increase in the present value of social-distancing costs greatly exceeds the increase in the present value of the costs of infection if immunity is permanent. Therefore, the social planner allows for more infections. The opposite holds when immunity wanes. Technically, a lower $\delta$ reduces the discount factor, increasing the present value of the infection costs caused by waning immunity and the endemic COVID-19. Therefore, the social planner reacts even stronger to the pandemic when it emerges if immunity wanes and the vaccine is expected later in time.

A reduction in the infection-fatality rate, captured by a lower, $\kappa_j$, implies less costs of infection and, thus, more social activity whatever is $\alpha$. But the rise in social activity increases in the duration of immunity (i.e., decreases in $\alpha$). When the costs of infection, $\kappa_j$, are lower, the implied point of the reduced social activity is under the flatter range of the curved utility.[13] Thus, the marginal cost of additional social-distancing is also relatively low, increasing the room to maneuver of the social planner. Therefore, the social planner acts stronger from the onset of the pandemic to reduce the costs of an endemic COVID-19 and gain time for the discovery of a cure or vaccine. This difference in optimal choices lead to clearly different disease dynamics: the faster immunity wanes, the more the social planner postpones and reduces peak infections.

In sum, waning immunity implies a persistent reduction in social activity either individually chosen or mandated. But because individuals lack altruism, implying a weaker link between choice and (re)infection, the early response to the pandemic in decentralized equilibria is not dependent on waning immunity. In centralized equilibria, however, waning immunity may affect the early response to the pandemic depending on the magnitude of the costs of infection and critically on how likely a vaccine or

[13] In an experiment (not reported), we varied the curvature of the utility function and find that the changes in social activity brought by waning immunity decrease in the curvature.

cure is expected to arrive. Yet, in our benchmark calibration, which we find plausible, waning immunity barely affects early optimal choices of social activity in the centralized equilibria.

### 4.2 Discussion

In this section, we contrast our findings with the three papers in the economics literature that study waning immunity. Eichenbaum, Rebelo and Trabandt (2020*b*) study the role of testing and quarantining in a model linking consumption and labor choices to contagion. They also find that decentralized individuals permanently reduce their activity (consumption and labor supply) due to the endemic steady-state caused by waning immunity. Furthermore, their Figure 9 suggests that, for over a year, waning immunity is virtually irrelevant for decentralized decisions. Yet, waning immunity affects their centralized equilibria in a way different from ours because of the different policy instruments considered. Their testing and quarantining polices rule out endemic steady states because asymptotically all individuals are continuously tested and infected ones are quarantined. Therefore, waning immunity neither restricts social planner's actions nor permanently constrains economic activity in Eichenbaum, Rebelo and Trabandt (2020*b*).

Giannitsarou, Kissler and Toxvaerd (2020) study the effects of waning immunity on social-distancing policy. Notable differences between our paper and theirs are as follows. They assume that the pandemic ends in a finite number of years (by the discovery of a vaccine), ruling out any endemic steady state. As a result, social activity returns to normal in their simulations. Moreover, the costs of infection and social-distancing are much lower in their model. They assume that the costs of infection are 10% lower output by infected and zero output by deceased individuals. The costs of social-distancing are quadratic and finite in a mandated full-lockdown, which provide a vast room to maneuver for the social planner to act. Therefore, when immunity wanes, they obtain deferment of peak infections and a negative relation between immunity duration and mandated social distancing (similar to our results in the low cost of infection case, Fig-

ure 3, Panel C).

Malkov (2020) studies how waning immunity affects the dynamics of an epidemiological model under different calibrations of the basic reproduction number. He concludes that until close to peak infections, waning immunity barely changes the disease dynamics. Although Malkov does not include endogenous decision making in his model, his findings are relatively close to our findings in the decentralized equilibria as waning immunity also only matters close to peak infections. But his findings differ substantially from our results in the centralized equilibria. In this case, the social planner takes into account the future costs of waning immunity in his early response to the pandemic, which in turn, leads to different disease dynamics.

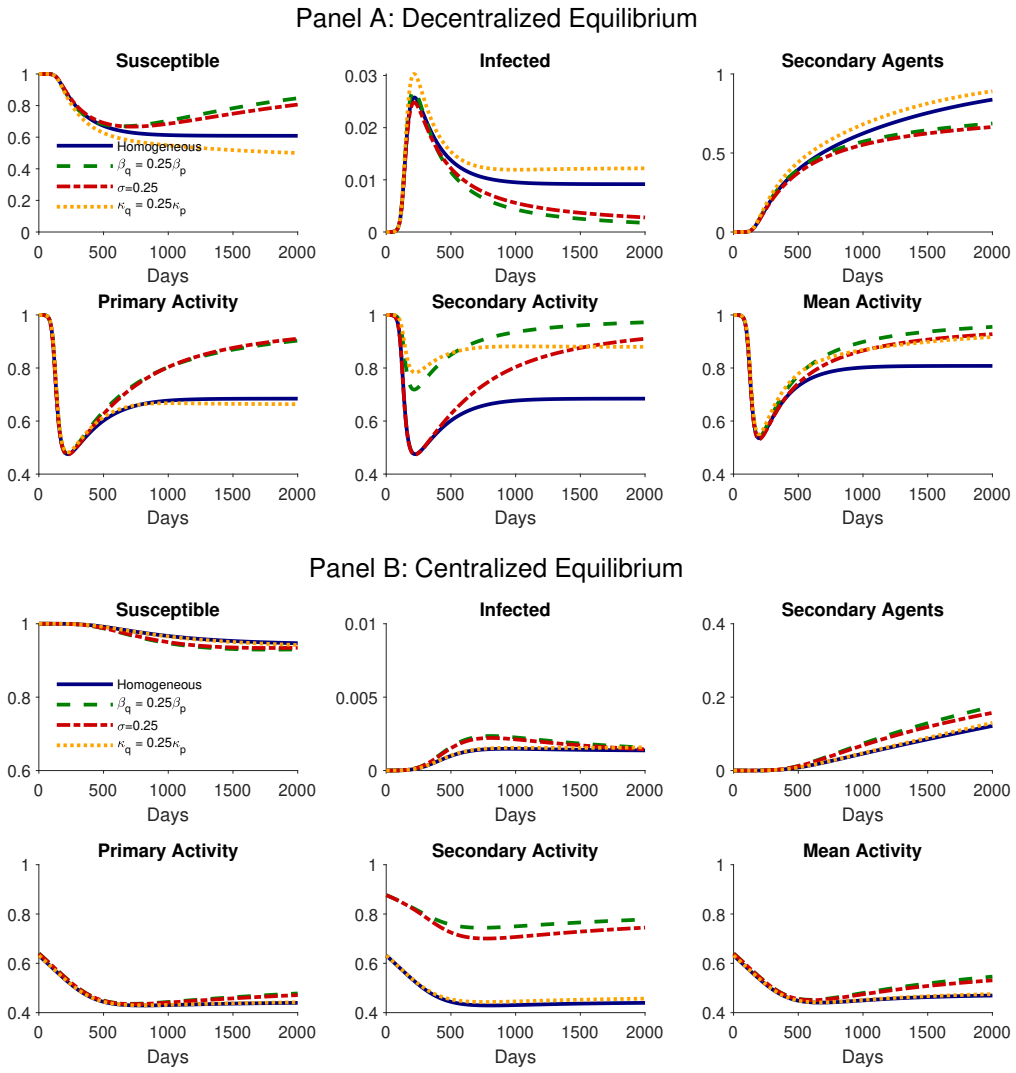
# 5 Partial Immunity

So far, we have analyzed an SIRS model augmented with endogenous social activity. Using our benchmark calibration, in Figure 4, we illustrate how our results change when secondary susceptible and infected agents differ from their primary counterparts in three aspects. Figure 5 complements our illustration in Figure 4 by showing how our results differ if $\delta$ and $\kappa_p$ are low. Green (dashed) lines show the case in which secondary susceptible agents are $75\%$ less likely to be infected than primary susceptible agents;[14] red (dot-dashed) lines show the case in which secondary infected individuals shed 75% less virus than primary infected; yellow (dotted) lines show the case in which the costs of infection are 75% lower for secondary agents; and blue (solid) lines show the benchmark. In the first two cases, even though all agents eventually lose immunity, asymptotic $R0$ is below 1 and, thus, the epidemic will asymptotically disappear as secondary agents gradually replace primary agents. In the case of $\kappa_q = 0.25\kappa_p$, the cost of a reinfection is much lower but the flows between states do not asymptotically converge to zero. That is, asymptotically, individuals are continuously infected but suffering much less than in the beginning of the epidemic. In this case, COVID-19 converges to an en-

[14] This implies a reduction of 75% in $R0$. Different combinations of changes in $\beta_j$ and $\gamma_j$ leading to the same fall in $R0$ imply similar results.

demic steady-state, which is similar to that of other coronaviruses giving rise to flu-like symptoms (Edridge et al., 2020; Huang et al., 2020; Kellam and Barclay, 2020).

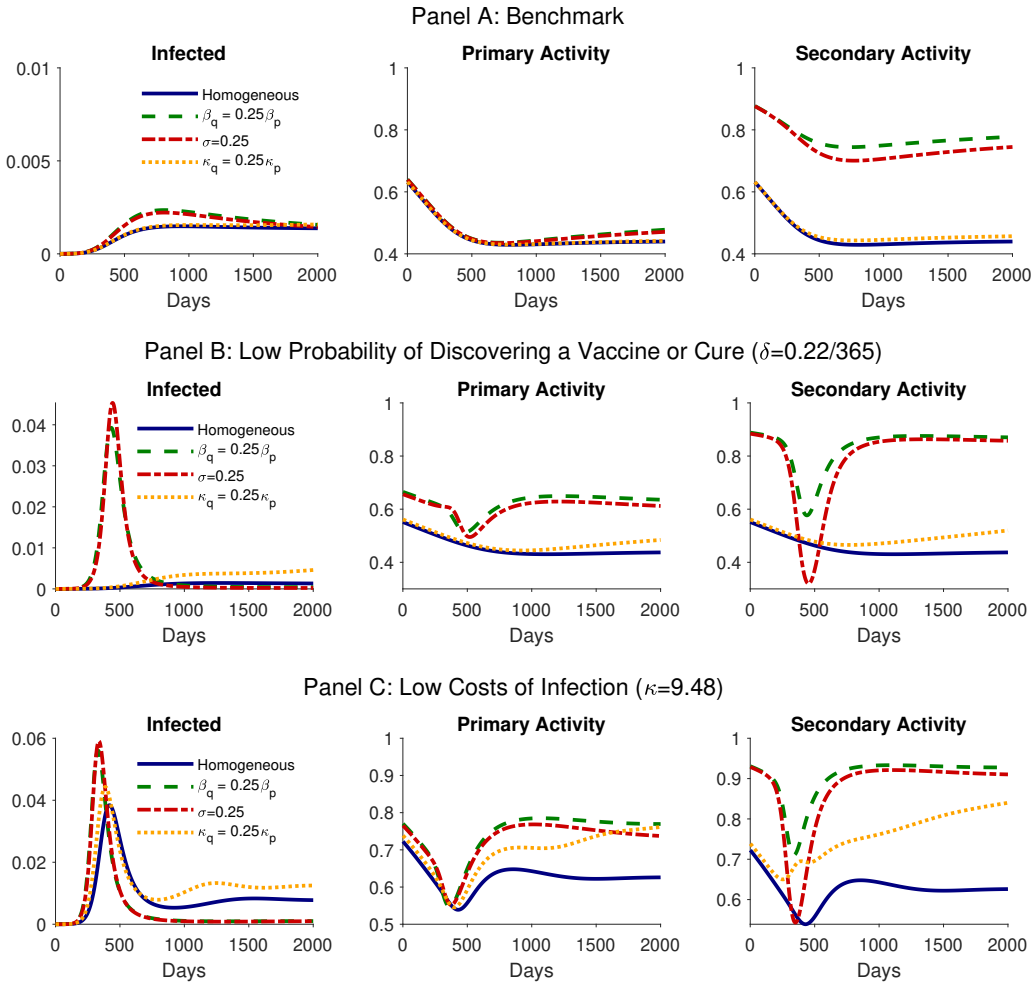
Figure 4: Partial Immunity




*Note:* Homogeneous refers to the case in which secondary and primary agents are alike. Susceptible agents are $S_{p,t} + S_{q,t}$; infected agents are $I_{p,t} + I_{q,t}$; secondary agents are $S_{q,t} + I_{q,t} + R_t$; primary activity is $A_{p,t}$; secondary activity is $A_{q,t}$; and mean activity is $S_{p,t}A_{p,t} + S_{q,t}A_{q,t} + I_{p,t} + I_{q,t} + R_t$.

Figure 4 and Panel A in Figure 5 show that if secondary and primary agents differ, there are little changes to the optimal social activity of primary susceptible agents

Figure 5: Partial Immunity - Centralized Equilibria




*Note:* Homogeneous refers to the case in which secondary and primary agents are alike. Infected agents are $I_{p,t} + I_{q,t}$; primary activity is $A_{p,t}$; secondary activity is $A_{q,t}$.

for approximately a year and a half in both centralized and decentralized equilibria. This contributes to a similar path for the number of susceptible (both primary and secondary) agents for many months. Thus, as in the previous section, our benchmark calibration implies that any uncertainty caused by waning immunity is not much relevant for several months after the start of the epidemic.

Our results in the centralized equilibria depend, again, on $\delta$ and $\kappa_p$. When it is unlikely to discover a vaccine or cure (low $\delta$), the early response to the pandemic critically

depends on whether COVID-19 becomes endemic. If it becomes endemic (benchmark and $\kappa_q = 0.25\kappa_p$), the social planner restricts social activity further as the present value of the costs of the endemic steady-state are larger. But if COVID-19 does not become endemic ($\beta_q = 0.25\beta_p$ or $\sigma = 0.25$), the social planner is more lenient. A low cost of infection of primary agents, $\kappa_p$, grants room for maneuver for the social planner to act early to endemic steady-states due to the curvature of the utility function. Therefore, mandated social-distancing visibly increases with the overall costs of the pandemic in Panel C of Figure 5.

The optimal behavior of secondary susceptible agents is much different from that of primary susceptible agents irrespective of $\delta$ and $\kappa_p$. If it is unlikely that secondary agents are reinfected ($\beta_q$ is low), they restrain social activity by much less than primary ones, which is problematic from a social perspective because they expose other agents (especially primary) significantly. Thus, even if susceptible agents are unlikely to be reinfected, policymakers should be aware that these agents are likely to be excessively active.

This problem of excessive social activity in the decentralized equilibrium is even worse if $\kappa_q = 0.25\kappa_p$. As agents are not altruistic, they only care about their own risks. A lower cost of reinfection then significantly lowers their incentives to social-distance. In contrast, the social planner would like secondary agents to substantially constrain their activity because their viral shedding and probability of infection are unchanged and many susceptible agents are still primary susceptible.[15] The scenario of $\kappa_q = 0.25\kappa_p$ also shows that agents asymptotically constrain social activity, even in the decentralized equilibrium, because COVID-19 becomes endemic and the costs of infection remain high (these costs imply a probability of dying of 0.16% in the benchmark). If these costs were lower, closer to those of endemic human coronaviruses, agents in a

[15] In this regard, secondary agents are similar to young agents in models that breakdown agents based on age (Acemoglu et al., 2020; Gollier, 2020). In those models, because young agents know that they are less likely to suffer if infected, they are too active from a social perspective as they increase exposure of older individuals.

decentralized equilibrium would behave almost as if there was no virus which is what we observed until the COVID-19 pandemic.

The results are very different if $\sigma = 0.25$. Recall that $\sigma$ measures how likely secondary infected shed virus onto susceptible. Since $\sigma$ pertains only to the externality caused by secondary agents' actions, it does not affect decisions in the decentralized equilibrium: secondary susceptible agents act as primary susceptible agents. A social planner, in contrast, would allow secondary agents to enjoy relatively more social activity. Both primary and secondary agents, however, benefit indirectly from the lower viral-shedding of secondary infected agents, which allows them to enjoy more social activity, converging asymptotically to full social activity in both equilibria.

## 6 The Role of Initial Conditions and Additional NPIs

Following the SARS-CoV2 outbreak, governments around the world have combined several NPIs to change the natural course of the pandemic. To account for this change, in this section, we base our simulations on initial conditions matching the (epidemiological) state of the COVID-19 pandemic on 1 July 2020.

In the (new) initial conditions, we accommodate a compromise between the epidemiological state in Canada, the US, and four European countries, France, Italy, Spain, and the UK. On 1 July 2020, the fraction of infected population was approximately 0.08% in Canada, 0.46% in the US, 0.09% in France and 0.02% in Italy.[16] These numbers are likely understated as authorities fail to test and identify many of infected and especially asymptomatic people (see references in Stock, 2020 for evidence on the proportion of asymptomatic). Bearing in mind the understatement and cross-country differences in the numbers, we find a compromise at $I_p = 0.2\%$. To set the initial number of recovered agents, we look at the evidence from antibody surveys. In France, Spain, and the UK, antibody surveys suggest that slightly more than 5% of the population has

[16] Statistics consulted in https://coronavirus.jhu.edu/map.html on 2 July 2020.

antibodies against SARS-CoV-2.[17] Given that the fraction of infected population ratio is two to three times higher in the US than in France, Spain, and the UK, we find a compromise at $R = 6\%$.

In all countries that we examined for this section, identified infected individuals are quarantined. This NPI naturally reduces contagion and we model it as an exogenous reduction in the social activity of some infected agents. In particular, we assume that 50% of infected agents, which is within the current estimated range of asymptomatic cases, are identified and cannot enjoy maximum social activity. In case infected individuals are identified, they enjoy 40% of normal social activity, which increases the expected costs of infection. Thus, average social activity of infected individuals falls by 30%. As of 1 July 2020, other NPIs, like mandatory mask use, differ across countries. In Canada, although some communities mandate mask use in public transport, the Public Health Agency of Canada merely recommends mask use when physical distancing is not consistently possible. In France and the UK, mask use is only mandatory in public transport, whereas in Spain, it is mandatory even in open-air spaces if it is not possible to maintain physical distance.[18] In our model, we treat mask use (mandatory or not) as an exogenous reduction in contagiousness, $\beta_p$ and $\beta_q$, by 30%. In sum, these NPIs reduce $\beta_p$ and $\beta_q$ by slightly over 50%.[19]

We depict the results in Figures 6 and 7. Blue (solid) lines assume the benchmark values for the rest of the parameters. Green (dashed) lines assume that agents are permanently immune. Red (dot-dashed) lines assume that in their contagiousness and cost of infection, secondary agents differ substantially from primary agents: $\beta_q = 0.25\beta_p$, $\sigma = 0.25$, and $\kappa_q = 0.25\kappa_p$.[20] Compared to our previous simulations, the other

[17] See the ONS COVID-19 Infection Survey for the UK; for France and Spain, see Salje et al. (2020) and Pollán et al. (2020).

[18] Since July 2020, countries have changed their mask-using rules. For example, France, UK, and some provinces in Canada have mandated mask use in indoor public places.

[19] Crucially, $R0$ is still above one as the pandemic would asymptotically disappear if $R0 < 1$. But $R0$ permanently below one seems unlikely as pointed by the second wave of infections in Australia and South Korea.

[20] In these simulations, we assume that the initial fraction of secondary susceptible and infected individuals is zero.

NPIs significantly elevate social activity because of the fall in contagiousness. Furthermore, the simulations suggest that individuals and policymakers do not need to know the duration of immunity and how secondary agents differ from primary ones until at least 2021 even if $\delta$ and $\kappa_p$ are low.[21] Thus, the combination of lower contagiousness and relatively high initial infections reduce the relevance of waning immunity even in centralized equilibria, making the social planner less responsive to future infection costs. This suggests that other NPIs provide substantial extra time to learn about the duration of immunity. Yet, given the implications of the costs of infection for social activity, and consistent with Hall, Jones and Klenow (2020), learning about the actual infection-fatality rate seems highly important.

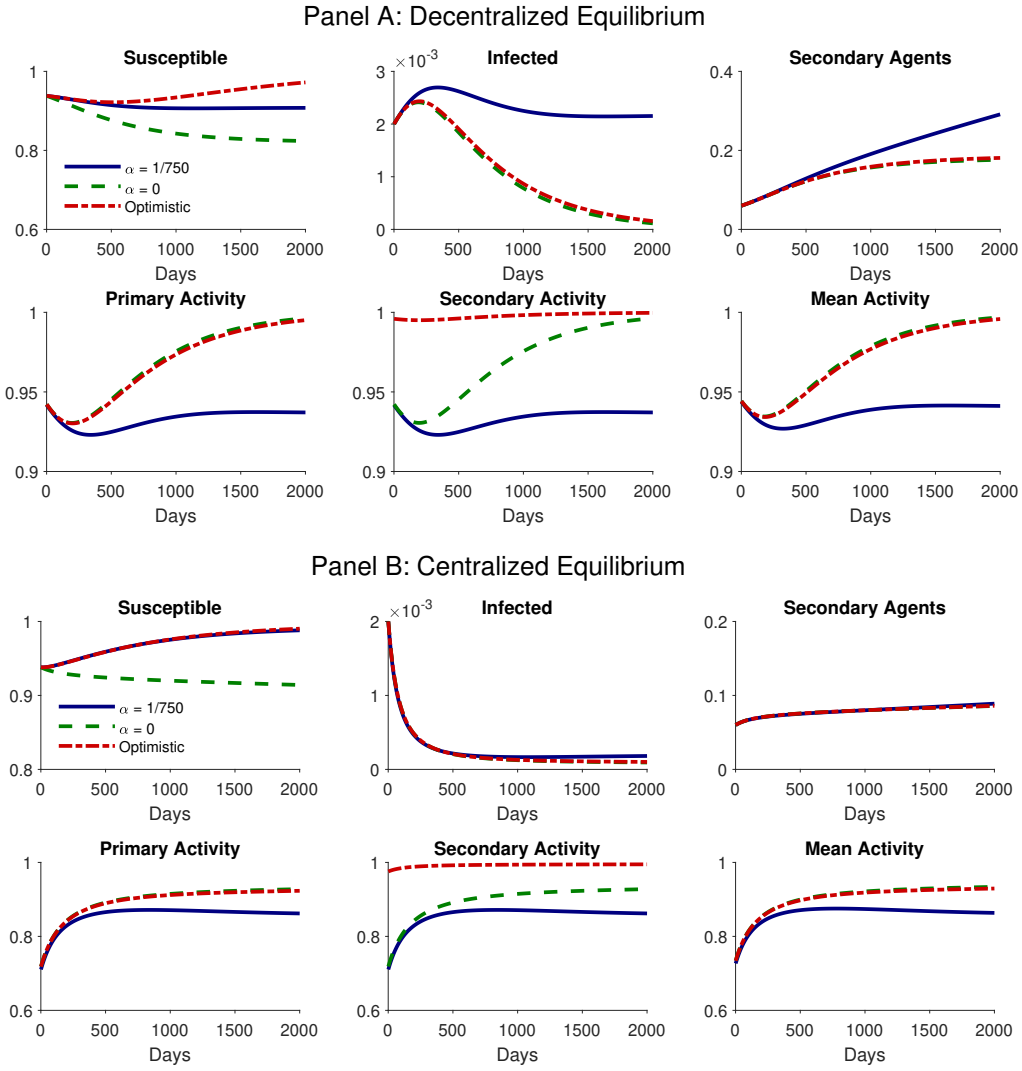
## 7 The Role of Vaccination

The calibration of the discount rate in our model assumes that a vaccine or cure might be found, instantaneously ending the pandemic. We share this assumption with Farboodi, Jarosch and Shimer (2020) and, to some extent, with, e.g., Giannitsarou, Kissler and Toxvaerd (2020), who assume that the pandemic exogenously ends at some future date. Yet, the pandemic does not end immediately when a vaccine is invented as it takes time to vaccinate the population; furthermore, as the share of the vaccinated population increases over time, the epidemiological dynamics can change.

Because of these considerations, we now assess a variant of our model in which the discount rate only reflects the time discount rate but agents might be vaccinated. We assume that the first approved vaccine is rolled out $\tau$ periods after the start of the pandemic. From period $\tau$ onwards, a proportion $\upsilon$ of recovered and (primary and secondary) susceptible individuals is vaccinated every period; thus, (primary and secondary) infected agents do not have access to the vaccine until they recover (because the vaccine does not cure). Furthermore, $\tau$ and $\upsilon$ are known to all agents from the be-

[21] Although there are slightly visible differences in terms of optimal primary activity if $\delta$ and $\kappa_p$ are low, the implied dynamics of infected individuals is almost unchanged. Optimal secondary activity depends much more on the scenario for waning immunity, but there are very few agents that are secondary susceptible.

Figure 6: Different Initial Conditions and Other NPIs

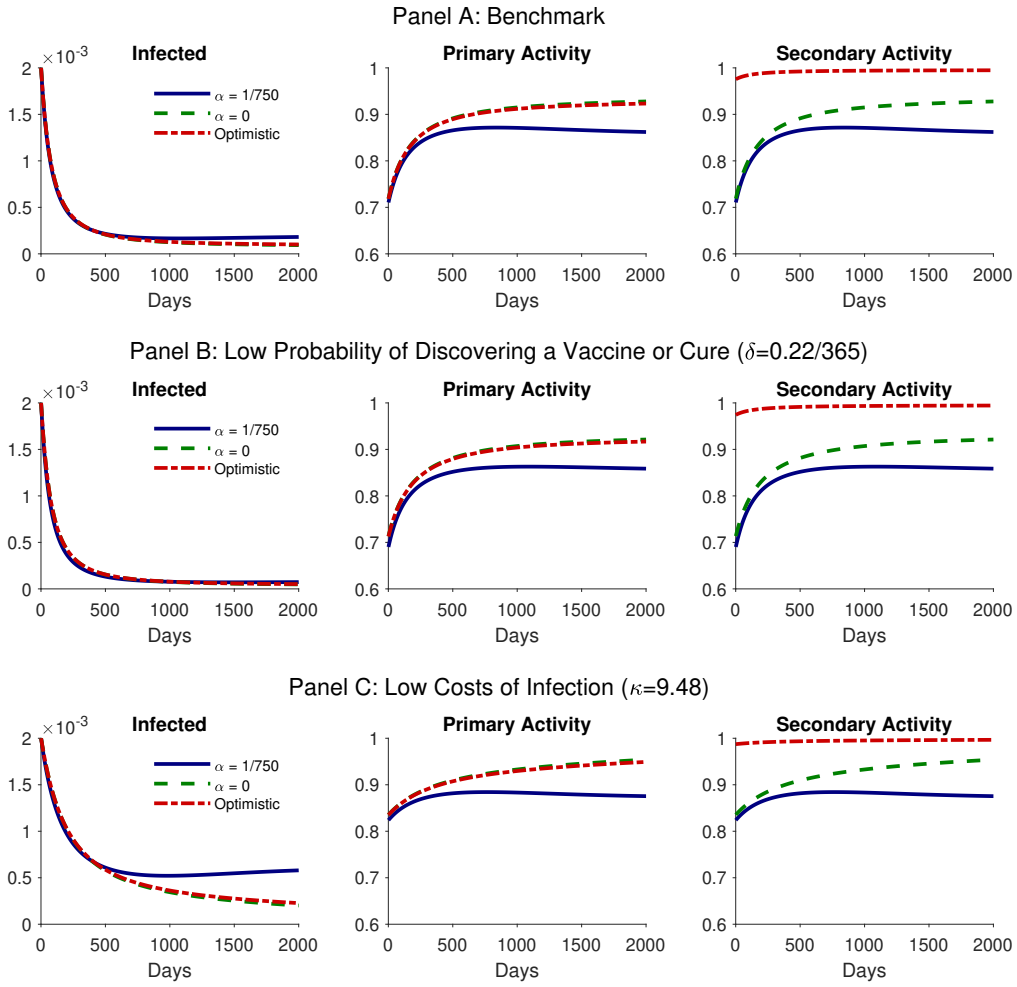



*Note:* Optimistic refers to the case in which $\beta_q = 0.25\beta_p$, $\sigma = 0.25$, and $\kappa_q = 0.25\kappa_p$. Susceptible agents are $S_{p,t} + S_{q,t}$; infected agents are $I_{p,t} + I_{q,t}$; secondary agents are $S_{q,t} + I_{q,t} + R_t$; primary activity is $A_{p,t}$; secondary activity is $A_{q,t}$; and mean activity is $S_{p,t}A_{p,t} + S_{q,t}A_{q,t} + 0.7(I_{p,t} + I_{q,t}) + R_t$.

ginning of the pandemic. Finally, we assume that the vaccine is permanently and entirely effective in protecting vaccinated individuals and in eliminating their viral shedding; in other words, vaccinated agents are permanently immune.[22]

[22]This assumption is arguably strong especially given that we do not know the extent of immunity of vaccinated agents. Yet, some approved vaccines are very effective (in excess of 90%) and, if needed, they might be readministered later, allowing for, at least approximately, permanent immunity.

Figure 7: Different Initial Conditions and Other NPIs - Centralized Equilibria

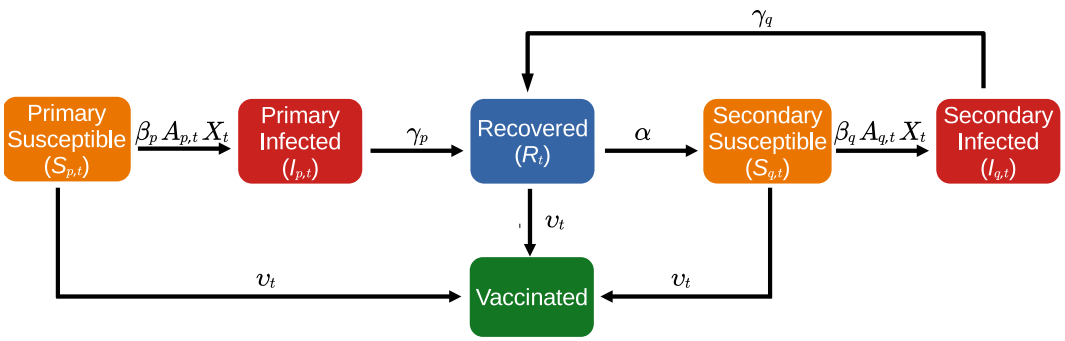



*Note:* Optimistic refers to the case in which $\beta_q = 0.25\beta_p$, $\sigma = 0.25$, and $\kappa_q = 0.25\kappa_p$. Infected agents are $I_{p,t} + I_{q,t}$; primary activity is $A_{p,t}$; secondary activity is $A_{q,t}$.

Figure 8 depicts the states and flows in this variant of our model, while Appendix B offers technical details about it. Figure 9 summarizes our results in the centralized equilibrium, in which we assume that $\tau = 365$ and $\upsilon = 1/300$ based on recent events. This implies that after one year a vaccine starts to be administered and a non-infected individual is vaccinated within an additional 10 months on average. Panel A, which reports the results using our benchmark calibration, shows that the pace of waning immunity is virtually unimportant for the optimal decision of the social planner, in line

with the results of the baseline model. Given that a vaccine is known to arrive relatively early in the pandemic, the motivation to postpone infections is very high, rendering the risk of reinfections relatively unimportant.

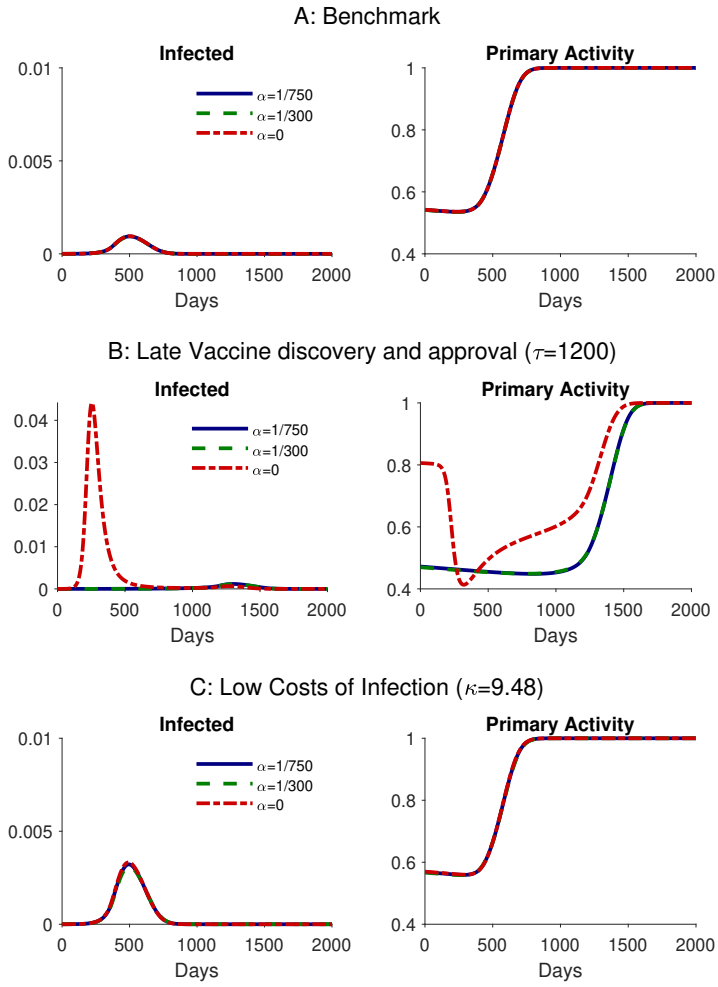
Figure 8: States and Flows with Vaccinations




Panel B, in which we report the results of this variant when a vaccine is only administered about three years after the beginning of the pandemic, is also in line with the results of the baseline model. If we compare this panel with Panel B in Fig. 3, which assumes a low probability of finding an instantaneous cure-for-all is relatively low, we can easily see the similarities in the share of infected individuals. There are, however, natural differences in terms of activity because herd immunity is known to be reached after approximately 1500 days as a result of mass vaccination.

The case in which the model with vaccinations delivers clearly different results when compared with the baseline model is when the costs of infection are low (a third of the value in benchmark). In this case, waning immunity is again unimportant for the social planner and it barely changes primary activity relative to benchmark. The reason for this result is that vaccines are known to be available within just one year from the beginning of the pandemic. Thus, virtually no recovered individual is expected to lose immunity (even when $\alpha = 1/300$) before being vaccinated and the incentives to postpone infections are extremely large. We have, however, experimented with other calibrations

Figure 9: The Role of Immunity Duration - Centralized Equilibria




*Note:* Infected agents are $I_{p,t} + I_{q,t}$; primary activity is $A_{p,t}$ (which, in this case, equals secondary activity, $A_{q,t}$).

(not reported) and we find that waning immunity is important in the early days of the pandemic in a scenario with an even lower $\kappa$ or in a scenario combining a low $\kappa$ and slightly higher $\tau$. In these scenarios, as in our baseline model, the social planner has more room to maneuver to demand higher social-distancing in the early days of the pandemic.

# 8 Concluding Remarks

It is likely that immunity against COVID-19 eventually wanes and recovered individuals face the risk of a reinfection (Edridge et al., 2020; Huang et al., 2020; Kellam and Barclay, 2020; Seow et al., 2020). This scenario is especially problematic if COVID-19 becomes endemic as other human endemic coronaviruses. We show that without the discovery of a vaccine or cure, COVID-19 reaches an endemic steady-state and social-distancing is here to stay. But, on the bright side, we also show that optimal decentralized and centralized choices do not necessarily depend on waning immunological memory for many months following the initial outbreak/contagion. This is especially the case if a vaccine is expected early in the pandemic, the costs of infection are already large in the short run, and other NPIs that lower contagiousness are in place. Before making irreversible decisions, individuals and policymakers seem to have time to learn more about immunological memory against SARS-CoV-2 and answer the call for serological studies from Kellam and Barclay (2020), Kissler et al. (2020), and Lerner et al. (2020).

Yet, in 6-12 months, without a vaccine or cure, we do need to know more about how antibodies and T-cells defend the human body against SARS-CoV-2. In particular, we must know how long immunity lasts and whether individuals that were infected (secondary agents) differ substantially from those that were never infected (primary agents). The longer immunity lasts, the less demanding should social-distancing be. And, in the limit, if immunity lasts a lifetime, then COVID-19 does not reach an endemic steady-state and social-distancing will sooner or later be unwarranted. Furthermore, if secondary agents may be reinfected but are somewhat protected against the virus, then COVID-19 may not become endemic. Yet, the way in which secondary agents differ from primary agents is crucial to design policy. For example, if most of the gains from the additional protection are private – because secondary agents are less likely to die or less likely to be reinfected – then secondary agents are excessively active from a social viewpoint. If, on the other hand, most of the gains from the additional protection are social – because secondary agents shed less virus – then the decentral-

ized and centralized equilibria are closer and less social-planning is required.

Even though most of the economics literature assumes permanent immunity, this simplification may not have dire consequences in the short run. If a vaccine or cure arrives early in the pandemic, the costs of infection are not small, and other NPIs are in place, then our model suggests that the optimal response in the initial months of the pandemic is virtually independent of waning immunity. The same is true if secondary agents, despite no longer immune, develop a strong protection against SARS-CoV-2 or shed much less virus. But, if these conditions do not hold, many of the policy prescriptions need to be revised as they rely on the possibility of herd immunity.

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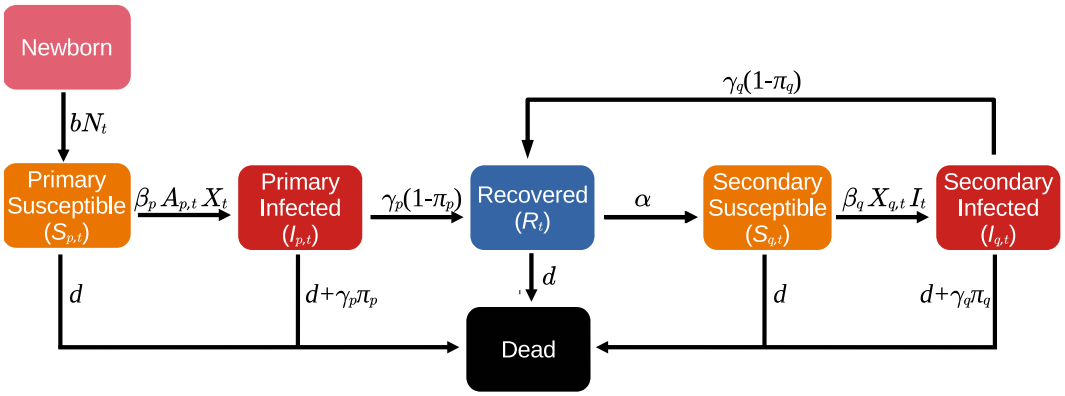
# A Birth and deaths

In this appendix, we present a variant of our model including exogenous births and deaths as well as COVID-19 related (endogenous) deaths. The results of this variant provide a robustness check of the results in the baseline model.

Figure A1: States and Flows with Vaccinations




## A.1 Epidemiological Block

The epidemiological block of the model is given by:

$$S_{p,t+1} = (1 - \beta_p A_{p,t} X_t - d) S_{p,t} + bN_t, \tag{A1}$$

$$I_{p,t+1} = \beta_p A_{p,t} S_{p,t} X_t + (1 - \gamma_p - d) I_{p,t}, \tag{A2}$$

$$R_{t+1} = \textstyle\sum_j \gamma_j (1 - \pi_j) I_{j,t} + (1 - \alpha - d) R_t, \tag{A3}$$

$$S_{q,t+1} = \alpha R_t + (1 - \beta_q A_{q,t} X_t - d) S_{q,t}, \tag{A4}$$

$$I_{q,t+1} = \beta_q A_{q,t} S_{q,t} X_t + (1 - \gamma_q - d) I_{q,t}, \tag{A5}$$

$$N_t = S_{p,t} + I_{p,t} + R_t + S_{q,t} + I_{q,t}, \tag{A6}$$

$$D_{t+1} = D_t + \textstyle\sum_j \gamma_j \pi_j I_{j,t} + dN_t.$$

There are only three differences relative to the epidemiological block in the baseline model. First, new agents are born every period and become primary susceptible; these

newborns sum to $bN_t$, where $N_t$ represents the population and $b$ is the birth rate per period. Second, a proportion $d$ of the individuals in each health state die for exogenous reasons every period; this is represented by an outflow from all health states to dead, $D_t$. Third, COVID-19 related deaths are modeled as a flow from (primary and secondary) infected agents, $I_{p,t}$ and $I_{q,t}$, to dead; in particular, we assume that a proportion $\gamma_j \pi_j$ of infected agents of type $j$ die due to the infection while $\gamma_j(1-\pi_j)$ recover.

## A.2 Decentralized Problem

### A.2.1 Utility Maximization

The lifetime utility maximization problem of a primary susceptible agent changes for two reasons. One is that we explicitly model the cost of infection as the cost associated with the risk of dying, i.e., the cost of infection is $\gamma_j \pi_j \bar{\kappa}_j$, which equals the value of life, $\bar{\kappa}_j$, multiplied by the probability that an infected agent dies, $\gamma_j \pi_j$. Yet, as explained below, we choose $\bar{\kappa}_j$ such that the results of this variant of the model are comparable to those of the baseline model (i.e., $\kappa_j$ in the baseline model equals $\gamma_j \pi_j \bar{\kappa}_j$ in this variant). The maximization problem is given by

$$\max_{\{a_{p,t}, a_{q,t}\}_{t=0}^{\infty}} \sum_{t=0}^{\infty} \sum_j \Lambda^t \Big( s_{j,t} u(a_{j,t}) - \gamma_j \pi_j \bar{\kappa}_j i_{j,t} \Big),$$

subject to

$$\begin{aligned}
s_{p,t+1} &= (1 - \beta_p a_{p,t} X_t - d) s_{p,t} + b n_t, \\
i_{p,t+1} &= \beta_p a_{p,t} s_{p,t} X_t + (1 - \gamma_p - d) i_{p,t}, \\
r_{t+1} &= \textstyle\sum_j \gamma_j (1 - \pi_j) i_{j,t} + (1 - \alpha - d) r_t, \\
s_{q,t+1} &= \alpha r_t + (1 - \beta_q a_{q,t} X_t - d) s_{q,t}, \\
i_{q,t+1} &= \beta_q a_{q,t} s_{q,t} X_t + (1 - \gamma_q - d) i_{q,t}, \\
n_t &= s_{p,t} + i_{p,t} + r_t + s_{q,t} + i_{q,t},
\end{aligned}$$

and the initial conditions. The decentralized optimum social activity is, then, governed by the transversality conditions and

$$u'(a_{j,t}) = \beta_j X_t (V_{s_j,t} - V_{i_j,t}), \tag{A7}$$

$$\frac{V_{s_j,t}}{\Lambda} = u(a_{j,t+1}) + bV_{s_p,t+1} + (1-d)V_{s_j,t+1} - \beta_j a_{j,t+1} X_{t+1}(V_{s_j,t+1} - V_{i_j,t+1}), \tag{A8}$$

$$\frac{V_{i_j,t}}{\Lambda} = (1-d)V_{i_j,t+1} + bV_{s_p,t+1} - \gamma_j(\pi_j\bar{\kappa}_j + V_{i_j,t+1} - (1-\pi_j)V_{r,t+1}), \tag{A9}$$

$$\frac{V_{r,t}}{\Lambda} = (1-d)V_{r,t+1} + bV_{s_p,t+1} + \alpha(V_{s_q,t+1} - V_{r,t+1}), \tag{A10}$$

for both $j \in \{p,q\}$. Again, these equations do not depend on the (subjective) probability of being in any health state, implying that the same equations also determine the behavior of secondary agents.

### A.2.2 Decentralized Equilibrium

Decentralized equilibria are symmetric with $a_{p,t} = A_{p,t}$ and $a_{q,t} = A_{q,t}$. Given initial values for the state variables, a decentralized equilibrium corresponds to a path of social activities, $\{A_{p,t}, A_{q,t}\}$, the number of infected agents, $X_t$, state variables, $\{S_{p,t}, S_{q,t}, I_{p,t}, I_{q,t}, R_t, N_t\}$, and shadow values, $\{V_{s_p,t}, V_{s_q,t}, V_{i_p,t}, V_{i_q,t}, V_{r,t}\}$, that satisfy the transversality conditions, Eq. (1), and Eqs. (A1–A10).

## A.3 Centralized Problem

### A.3.1 Utility Maximization

In this section, we present the maximization problem of the social planner. The social planner chooses socially optimal activity by directly influencing aggregate variables. In particular, the maximization problem of the social planner is given by

$$\max_{\{A_{p,t},A_{q,t}\}_{t=0}^{\infty}} \sum_{t=0}^{\infty} \sum_j \Lambda^t \Big( S_{j,t} u(A_{j,t}) - \gamma_j \pi_j \bar{\kappa}_j I_{j,t} \Big),$$

subject to Eqs. (1, A1–A6) and the initial conditions. The socially optimum social activity is, then, governed by the transversality conditions and

$$u'(A_{j,t}) = \beta_j X_t (V_{S_j,t} - V_{I_j,t}), \tag{A11}$$

$$\frac{V_{S_j,t}}{\Lambda} = u(A_{j,t+1}) + bV_{S_p,t+1} + (1-d)V_{S_j,t+1} - \beta_j A_{j,t+1} X_{t+1}(V_{S_j,t+1} - V_{I_j,t+1}), \tag{A12}$$

$$\begin{aligned}\frac{V_{I_j,t}}{\Lambda} = (1-d)V_{I_j,t+1} + bV_{S_p,t+1} - \gamma_j(\pi_j\bar{\kappa}_j + V_{I_j,t+1} - (1-\pi_j)V_{R,t+1}) \\ - \sigma_j \textstyle\sum_j \beta_j A_{j,t+1} S_{j,t+1}(V_{S_j,t+1} - V_{I_j,t+1})\end{aligned} \tag{A13}$$

$$\frac{V_{R,t}}{\Lambda} = (1-d)V_{R,t+1} + bV_{S_p,t+1} + \alpha(V_{S_q,t+1} - V_{R,t+1}), \tag{A14}$$

for both $j \in \{p,q\}$, and $\sigma_j = \begin{cases} 1 & \text{if } j = p, \\ \sigma & \text{if } j = q. \end{cases}$

### A.3.2 Centralized Equilibrium

Given initial values for the state variables, a centralized equilibrium corresponds to a path of social activities, $\{A_{p,t}, A_{q,t}\}$, the number of infected agents, $X_t$, state variables, $\{S_{p,t}, S_{q,t}, I_{p,t}, I_{q,t}, R_t\}$, and shadow values, $\{V_{S_p,t}, V_{S_q,t}, V_{I_p,t}, V_{I_q,t}, V_{R,t}\}$, that satisfy the transversality conditions, Eq. (1), Eqs. (A1–A6), and Eqs. (A11–A14).

## A.4 Calibration

The calibration of this variant of the model is mostly identical to that in Table 1 as we want to dissect solely the implications of birth and deaths in the model. Therefore, we set $\bar{\kappa}_j = 512/0.0064 = 80000$ and $\pi_j = 0.0064$ (based on Meyerowitz-Katz and Merone, 2020) as our benchmark.

Regarding the two new parameters, $b$ (birth rate) and $d$ (death rate), we rely on the calibration in Kissler et al. (2020), who use the US as a reference country. In the US, births sum to 3.8 million and the population is 330 million; thus, we set $b = \frac{3.8}{330}\frac{1}{365}$. And the expected lifespan in the US is 80 years, implying $d = \frac{1}{80}\frac{1}{365}$.

## A.5 Results

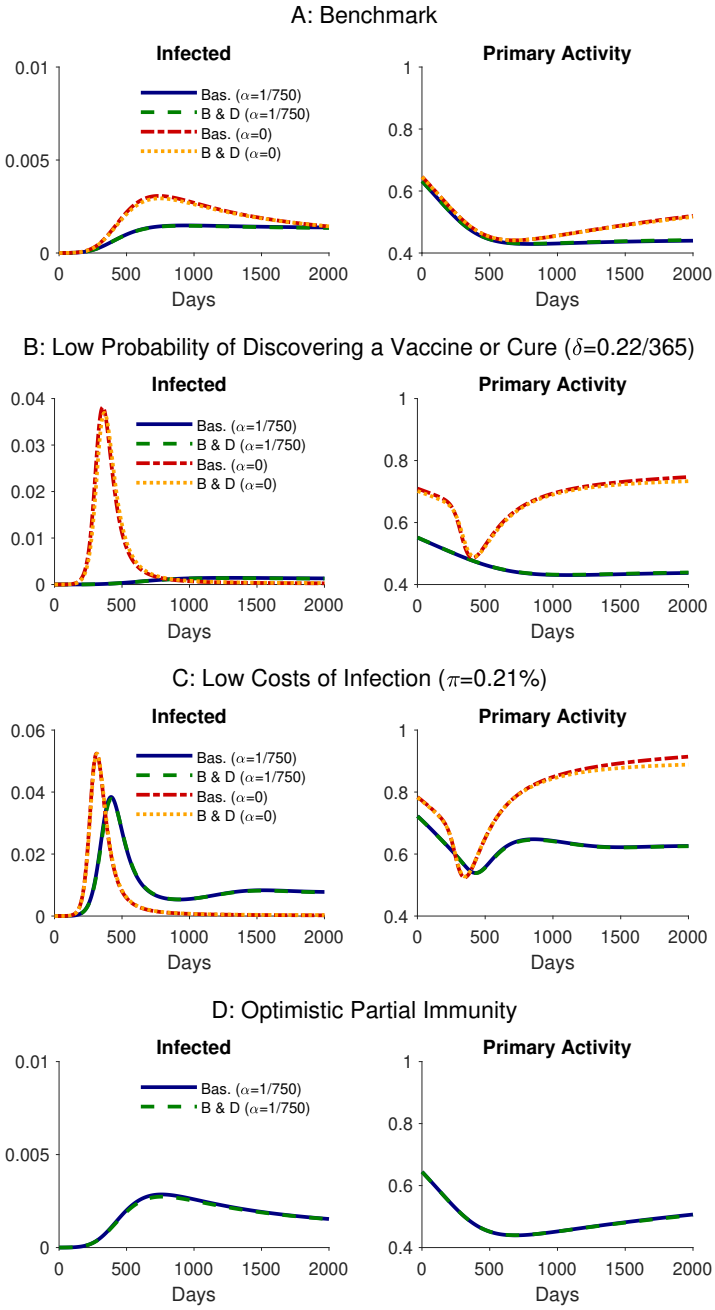
Figures A2 and A3 contrast the results of the baseline model with those of the variant with births and deaths under various calibrations of the model in the centralized and decentralized equilibrium, respectively. The results under the two variants of the model are essentially the same, particularly in the first 1000 days of the pandemic. In separate experiments (not reported), we find an even clearer overlap if $b = d = 0$.

The reason for the huge similarity in the results is that the size of the population is almost trendless and COVID-19 related deaths represent only a tiny proportion of the population. This results in part from the relatively low probability of dying (0.64%) and immunity lasting 24 months (in the experiments reported; we also experimented with $\alpha = 1/300$ and the conclusions remain unchanged). But it mostly results from social-distancing in both decentralized and centralized equilibria that greatly curb infections and COVID-19 related deaths.

If we study longer time horizons (especially beyond 2000 days), however, then births and deaths start to play a clearer role in affecting infections. Then, as the population size has a negative trend, contacts and infections tend to fall in the model because we assume mass-action dynamics. If, alternatively, we had frequency-dependent dynamics (in which, $X_t = (I_{p,t} + \sigma I_{q,t}) \frac{1}{N_t}$ instead of $X_t = I_{p,t} + \sigma I_{q,t}$), the similarity between the two variants would arguably be even larger in the early days of the pandemic.

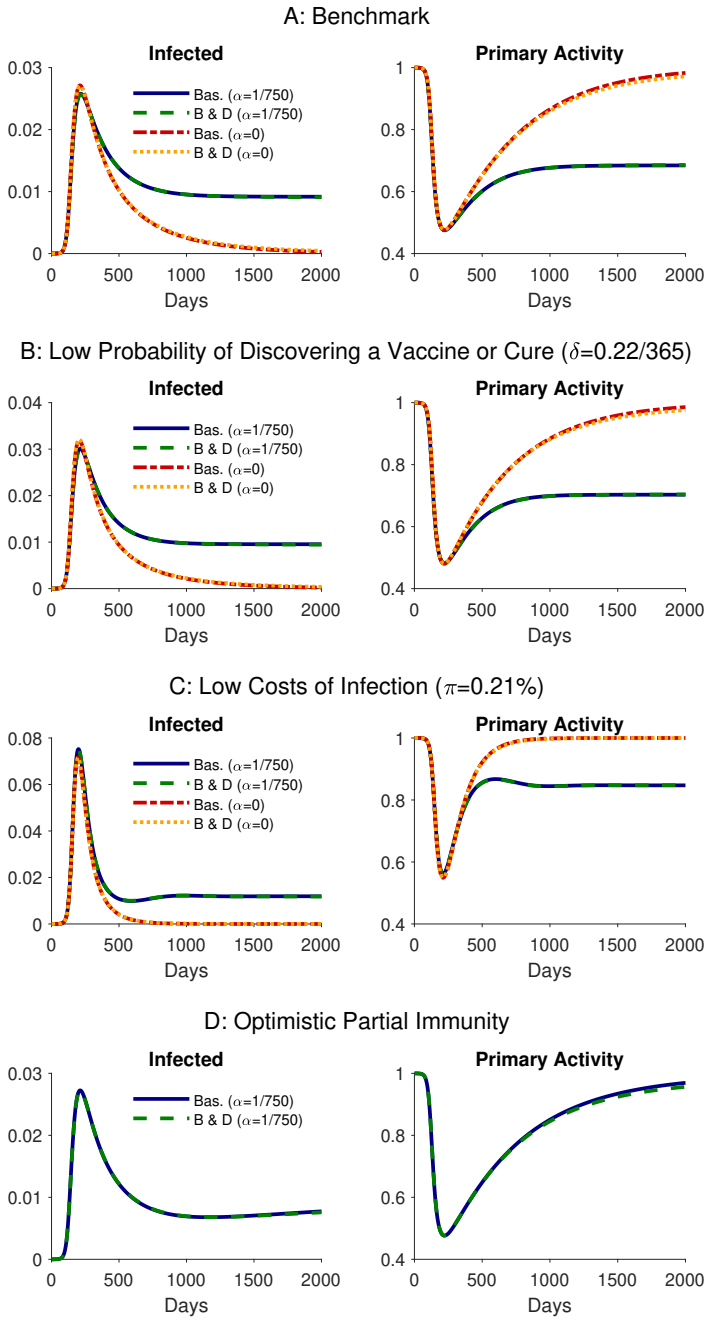
All these results, together with the fact that we mostly focus on the early response to the pandemic, justify our choice of the more stylized variant of the model (without births and deaths) as our baseline as long as $\kappa_j$ reflects the costs of dying.

Figure A2: Baseline Vs Birth Death Variant - Centralized Equilibria




*Note:* Infected agents are $I_{p,t} + I_{q,t}$; primary activity is $A_{p,t}$ (which equals secondary activity, $A_{q,t}$, in all panels except D). Optimistic partial immunity refers to the case in which $\beta_q = 0.25\beta_p$, $\sigma = 0.25$, and $\pi_q = 0.25\pi_p$. Bas. stands for the baseline model and B&D stands for the model with births and deaths presented in this appendix.

Figure A3: Baseline Vs Birth Death Variant - Decentralized Equilibria




*Note:* Infected agents are $I_{p,t} + I_{q,t}$; primary activity is $A_{p,t}$ (which equals secondary activity, $A_{q,t}$, in all panels except D). Optimistic partial immunity refers to the case in which $\beta_q = 0.25\beta_p$, $\sigma = 0.25$, and $\pi_q = 0.25\pi_p$. Bas. stands for the baseline model and B&D stands for the model with births and deaths presented in this appendix.

# B Model Featuring Vaccination

## B.1 Epidemiological Block

Most of the epidemiological block of the model is unchanged relative to the baseline model. The laws of motion governing the transitions between health states are the following:

$$S_{p,t+1} = (1 - \beta_p A_{p,t} X_t - \upsilon_t) S_{p,t}, \tag{B1}$$

$$I_{p,t+1} = \beta_p A_{p,t} S_{p,t} X_t + (1 - \gamma_p) I_{p,t}, \tag{B2}$$

$$R_{t+1} = \textstyle\sum_j \gamma_j I_{j,t} + (1 - \alpha - \upsilon_t) R_t, \tag{B3}$$

$$S_{q,t+1} = \alpha R_t + (1 - \beta_q A_{q,t} X_t - \upsilon_t) S_{q,t}, \tag{B4}$$

$$I_{q,t+1} = \beta_q A_{q,t} S_{q,t} X_t + (1 - \gamma_q) I_{q,t}, \tag{B5}$$

$$\tag{B6}$$

where $\upsilon_t = \begin{cases} 0 & \text{if } t < \tau, \\ \upsilon & \text{if } t \geq \tau \end{cases}$ is the share of non-infected agents vaccinated at time $t$.

## B.2 Decentralized Equilibrium

The objective function in the decentralized equilibrium is unchanged by explicitly considering vaccinated individuals as it is pointless to constrain activity when vaccinated and the value of being in this state is zero (i.e., not negative as for the other states). Yet, the constraints of the problem change according to the change to the epidemiological block of the model. This, in turn, implies that the solution to the optimal control

problem is given by

$$u'(a_{j,t}) = \beta_j X_t (V_{s_j,t} - V_{i_j,t}), \tag{B7}$$

$$\frac{V_{s_j,t}}{\Lambda} = u(a_{j,t+1}) + (1-\upsilon_t)V_{s_j,t+1} - \beta_j a_{j,t+1} X_{t+1}(V_{s_j,t+1} - V_{i_j,t+1}), \tag{B8}$$

$$\frac{V_{i_j,t}}{\Lambda} = -\kappa_j + V_{i_j,t+1} - \gamma_j(V_{i_j,t+1} - V_{r,t+1}), \tag{B9}$$

$$\frac{V_{r,t}}{\Lambda} = (1-\upsilon_t)V_{r,t+1} + \alpha(V_{s_q,t+1} - V_{r,t+1}), \tag{B10}$$

for both $j \in \{p,q\}$. Decentralized equilibria are symmetric with $a_{p,t} = A_{p,t}$ and $a_{q,t} = A_{q,t}$. Given initial values for the state variables, a decentralized equilibrium corresponds to a path of social activities, $\{A_{p,t}, A_{q,t}\}$, the number of infected agents, $X_t$, state variables, $\{S_{p,t}, S_{q,t}, I_{p,t}, I_{q,t}, R_t\}$, and shadow values, $\{V_{s_p,t}, V_{s_q,t}, V_{i_p,t}, V_{i_q,t}, V_{r,t}\}$, that satisfy the transversality conditions, Eq. (1), and Eqs. (B1–B10).

### B.3 Centralized Equilibrium

In the centralized equilibrium, similar to the decentralized equilibrium, the change in the constraints implies that the solution to the optimal control problem is given by

$$u'(A_{j,t}) = \beta_j X_t (V_{S_j,t} - V_{I_j,t}), \tag{B11}$$

$$\frac{V_{S_j,t}}{\Lambda} = u(A_{j,t+1}) + (1-\upsilon_t)V_{S_j,t+1} - \beta_j A_{j,t+1} X_{t+1}(V_{S_j,t+1} - V_{I_j,t+1}), \tag{B12}$$

$$\frac{V_{I_j,t}}{\Lambda} = -\kappa_j + V_{I_j,t+1} - \gamma_j(V_{I_j,t+1} - V_{R,t+1}) - \sigma_j \textstyle\sum_j \beta_j A_{j,t+1} S_{j,t+1}(V_{S_j,t+1} - V_{I_j,t+1}) \tag{B13}$$

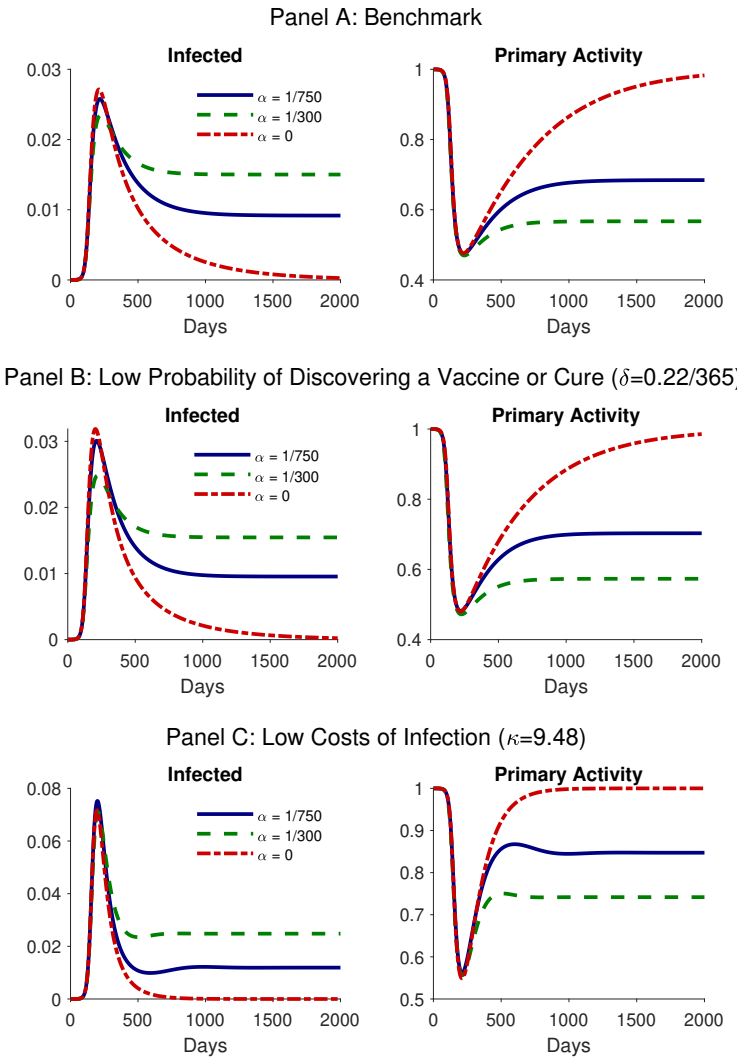
$$\frac{V_{R,t}}{\Lambda} = (1-\upsilon_t)V_{R,t+1} + \alpha(V_{S_q,t+1} - V_{R,t+1}), \tag{B14}$$

for both $j \in \{p,q\}$, and $\sigma_j = \begin{cases} 1 & \text{if } j = p, \\ \sigma & \text{if } j = q. \end{cases}$ Given initial values for the state variables, a centralized equilibrium corresponds to a path of social activities, $\{A_{p,t}, A_{q,t}\}$, the number of infected agents, $X_t$, state variables, $\{S_{p,t}, S_{q,t}, I_{p,t}, I_{q,t}, R_t\}$, and shadow values, $\{V_{S_p,t}, V_{S_q,t}, V_{I_p,t}, V_{I_q,t}, V_{R,t}\}$, that satisfy the transversality conditions, Eq. (1),

Eqs. (B1–B5), and Eqs. (B11–B14).

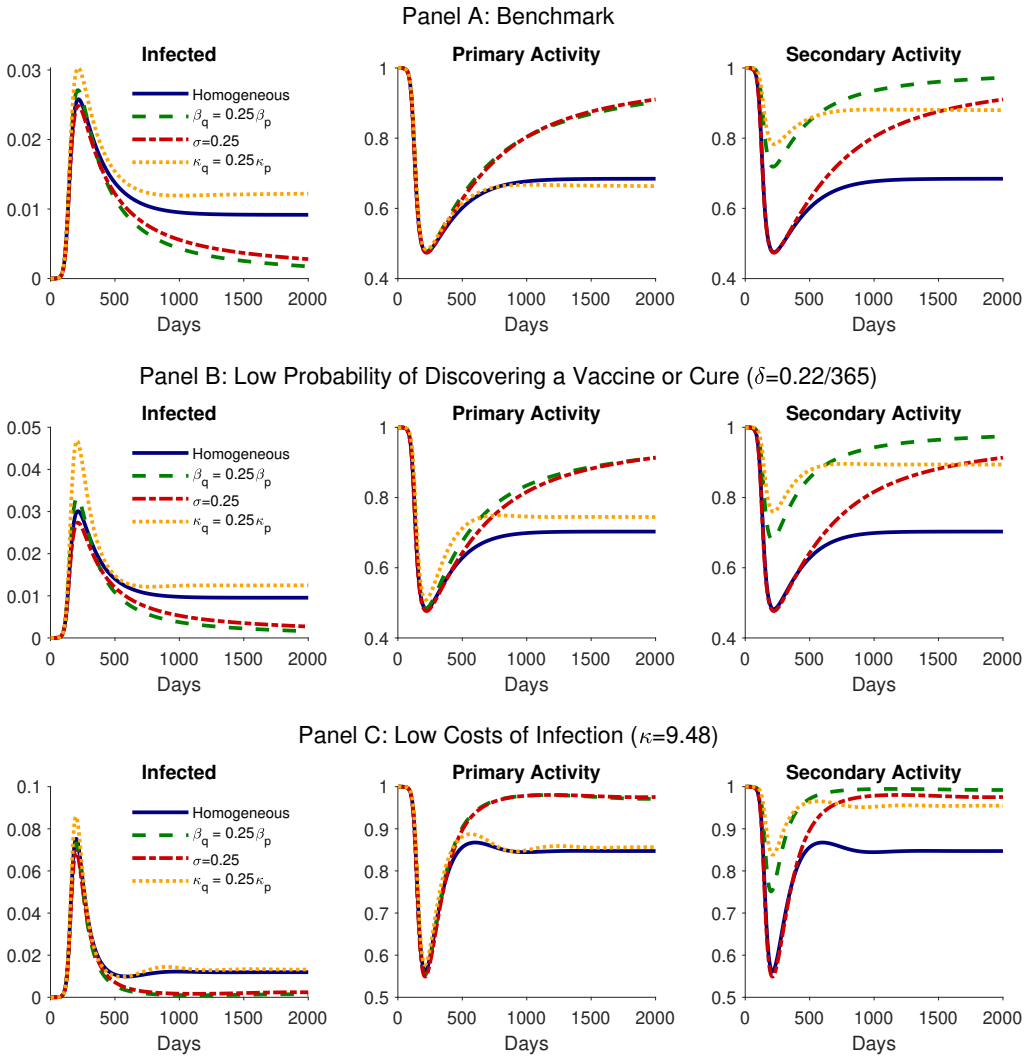
# C Robustness Checks for Decentralized Equilibria

Figure C1: The Role of Immunity Duration - Decentralized Equilibria



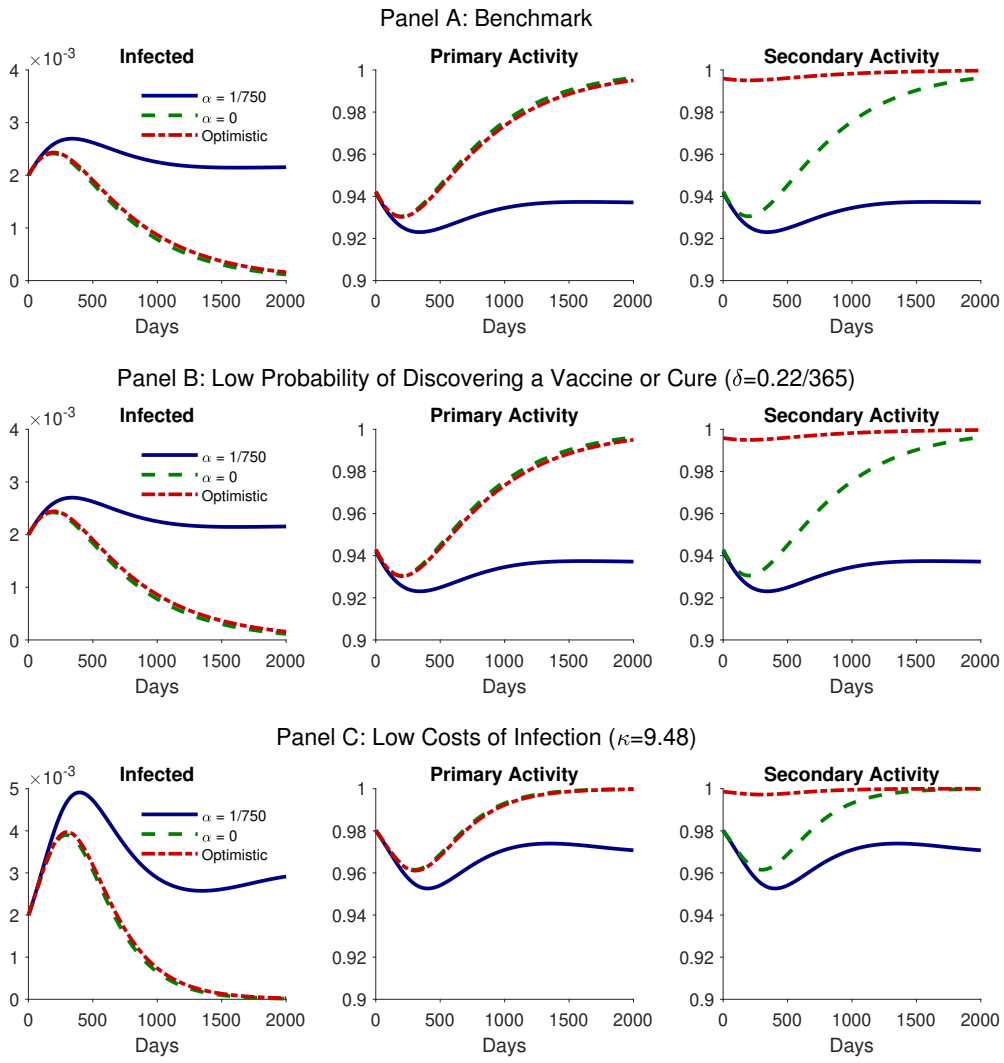

*Note:* Infected agents are $I_{p,t} + I_{q,t}$; primary activity is $A_{p,t}$ (which, in this case, equals secondary activity, $A_{q,t}$).

Figure C2: Partial Immunity - Decentralized Equilibria




*Note:* Homogeneous refers to the case in which secondary and primary agents are alike. Infected agents are $I_{p,t} + I_{q,t}$; primary activity is $A_{p,t}$; secondary activity is $A_{q,t}$.

Figure C3: Different Initial Conditions and Other NPIs - Decentralized Equilibria




*Note:* Optimistic refers to the case in which $\beta_q = 0.25\beta_p$, $\sigma = 0.25$, and $\kappa_q = 0.25\kappa_p$. Infected agents are $I_{p,t} + I_{q,t}$; primary activity is $A_{p,t}$; secondary activity is $A_{q,t}$.