# Behavior and the Dynamics of Epidemics

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During the first half of the Twentieth Century, Americans enjoyed tremendous gains in health and life-expectancy as infectious diseases were drastically curtailed thanks to major medical advances and massive investments in sanitation and public health. Annual mortality rates from infectious disease in the United States fell by an order of magnitude: from nearly 800 per 100,000 in 1900 to under 50 per 100,000 by 1960, in a steady downward trend, interrupted, dramatically, by the 1918-1919 outbreak of the Spanish Flu.[i] But as the HIV/AIDS pandemic made evident, and the COVID-19 pandemic reinforced, infectious diseases are far from vanquished.[ii] In fact, the risk that we experience another pandemic in the not too distant future is considerable. For example, according to a September of 2019 estimate by the President's Council of Economic Advisors, there is a four percent probability at an annual rate of a pandemic influenza that, at the high end, would cause in the United States nearly $4 trillion in economic damage and over half a million deaths.[iii]

Given that we are likely to see significant outbreaks of infectious disease in the future, this seems an opportune time to re-examine our models of disease dynamics. What dynamics of infections and deaths should we expect to see from a pandemic? What are our options for mitigating the impact of a pandemic on public health? How might this mitigation be done in a manner to reduce the negative impact of a pandemic on the economy? These are questions that will provoke new research in the light of worldwide data from this COVID pandemic for years to come.

But, after one year of data on COVID, one conclusion seems clear: the endogenous response of both public and private behavior to the prevalence of COVID-19 has transformed this epidemic from what might have been predicted to be a short, but exceedingly intense, episode into a milder but chronic pandemic that will impact public health and the economy over several years, until, with luck, the technological solution of vaccination brings this disease under much greater control.

In this paper, I use a simple model of our experience with COVID-19 in the United States over the past year to explore how the interaction of disease and behavior changes the dynamics of an epidemic and constrains our options for mitigating the impact of a pandemic on public health absent a technological solution such as vaccines. I then re-examine the goals of public health measures to contain a pandemic when there is a decent chance that a technological solution such as a vaccine might be found. I conclude with some discussion of examples of policies that might allow for better public health with a smaller economic impact over the long term.

## I.    Epidemic dynamics with and without behavior

The public health policies enacted around the world to combat COVID-19 have been guided by standard epidemiological models built on the SIR framework developed by Kermack and McKendrick[iv]. These models simulate disease transmission as arising when infected individuals (I) interact with others. Through this interaction, a virus or other disease succeeds in infecting those who have no immunity and are thus susceptible (S), turning such agents into newly infectious individuals (I). Individuals who gain immunity from prior infections or vaccinations are said to be removed (R) as they no longer contribute to the transmission of the disease.

When applied to COVID-19, three quantitative implications of this standard model stand out.[v] First, the model gives dire forecasts for the peak of the disease's first wave --- 10 to 20% of Americans were predicted to be sick with COVID-19 simultaneously at the first peak of infections absent drastic efforts (such as lockdowns and quarantines) to slow transmission. At current estimates of the infection fatality rate for COVID-19, this rate of infection would have corresponded to peak death rates on the order of 30,000 to 60,000 per day. Second, this model forecast that if efforts to slow transmission were applied early but were only temporary, this dramatic first peak would be delayed but not prevented: cases and deaths would explode again once efforts to slow transmission were relaxed. Third, this standard model offered dramatic long-run predictions made famous by Angela Merkel in March of 2020[vi] —more than 2/3 of the population were forecast to experience infections (if not vaccinated) before the pandemic would end through herd immunity. Again, applying current estimates of the average infection fatality rate for COVID-19 in the US, this implies a long-run death toll on the order of 1.25 million or more.

These implication of a standard epidemiological model for the magnitude of the first peak and the long-run impact of COVID-19 in terms of infections are driven by a single parameter known as the basic reproduction number of the virus (the $R_0$)[vii]. The implications of these

infections for deaths from COVID-19 are determined by the average infection fatality rate across the infected population. While we now know that the infection fatality rate from COVID-19 varies widely with age and other factors, estimates of the disease burden from COVID-19 from the CDC are consistent with an average infection fatality rate of 0.45% across the entire infected population in the United States for 2020.[viii] The emergence of new, more transmissible, virus variants with higher basic reproduction numbers make the predictions of standard epidemiological models for peak infections and long run impact even more dire.

It is now clear that the first prediction of standard epidemiological models for the first peak of infections and deaths were off by at least an order of magnitude --- it is unlikely that more than 2 percent of Americans have ever been infected simultaneously, and the peak of daily deaths in America from COVID-19 has fortunately stayed in four digits. Looking at data worldwide, it appears that the second prediction of standard epidemiological models is also off perhaps by an order of magnitude. While many locations within the United States and abroad have suffered severe second or third waves of COVID-19 deaths after relaxing costly public measures to control disease transmission, these waves have been much smaller than predicted by a standard SIR model.

In contrast, the standard SIR model's third prediction, regarding long-run impact, looks to be closer to the mark. While the precise threshold of herd immunity—the fraction of the population that has to gain immunity through infection or vaccination before the pandemic can end—is not yet empirically resolved,  available data from locations such as Manaus, in Brazil, that have experienced high rates of infection, and from Israel, which has high vaccination rates, indicate that the predictions of a standard epidemiological model for the long-run impact of COVID-19 are likely correct: this pandemic will not resolve until high proportions of the population have acquired immunity either through infection or vaccination.[ix]

### I.A. Behavior regulates disease dynamics

How does consideration of the impact of behavior on the progression of a pandemic help us understand this relationship between the standard SIR model predictions and observed outcomes?

Within economics, Tomas Philipson pioneered the study of the interaction of behavior and the spread of disease in his work on the HIV/AIDS pandemic. In a 1999 handbook chapter[x] summarizing work on that pandemic, Philipson argued that epidemiological models should

incorporate prevalence-elastic private demand for costly measures to prevent of the spread of infectious disease. Such models, he maintained, offered two fundamental economic insights.

The first insight is that costly private efforts to prevent disease transmission are self-limiting—as disease incidence falls, these costly efforts to control disease spread are relaxed and the disease re-emerges. Within the United States, it appears that this observation holds for public policies aimed at COVID-19 as well --- state and local disease control measures are often conditioned on measures of disease prevalence such as infections or hospitalizations, and these public measures aimed at the control of COVID-19 are relaxed as disease prevalence falls.

The second insight is that the private response to changing disease prevalence partially offsets public interventions aimed at disease control. In short, the effect of public interventions is limited by their success as private efforts aimed at disease control are relaxed in response.

That both public and private prevalence-elastic demand for costly measures to control disease is self-limiting is a particularly powerful insight for understanding where the standard epidemiological model fails as a description of disease dynamics and where it succeeds. In joint work with Karen Kopecky and Tao Zha, I find[xi] that the data on the progression of the COVID-19 pandemic across many countries and U.S. states throughout 2020 conform strikingly well with a core prediction of the standard epidemiological model modified to include prevalence-elastic demand for disease prevention—that after the first phase of the pandemic in which disease grows rapidly, the growth rates of infections and deaths should remain in a relatively narrow band around zero until the pandemic is over.[xii]

The intuition for this prediction regarding disease dynamics in the context of a model with prevalence-elastic demand for disease prevention is simple. If new infections and daily deaths from the disease grow too high, people and governments take costly efforts to avoid interaction and thus slow disease spread.  Likewise, if the prevalence of the disease falls, people and government relax those costly efforts at disease prevalence and the prevalence of the disease rises again. The reaction of behavior, both public and private, to the prevalence of the disease regulates the equilibrium prevalence of the disease in the same way that a cruise control regulates the velocity of a car on the highway that winds up and down hills. The equilibrium level of daily deaths, corresponding in this analogy to the velocity of the car, remains within a relatively narrow band (relative to that predicted by a standard SIR model) in response to shocks impacting disease transmission because of the stabilizing role of endogenous prevalence-elastic public and private disease avoidance

behavior. The impact of this behavior then is to transform what would otherwise be a short and sharp disease episode into a much more slowly evolving and drawn-out phenomenon.

What are the implications of a model with prevalence-elastic demand for disease prevention for the long run impact of an epidemic? Here the insight that the demand for disease prevention is self-limiting is particularly relevant. For an epidemic to end, the prevalence of the disease must fall towards zero. As disease prevalence falls towards zero, the demand for costly disease prevention efforts also falls towards zero, and hence the disease will come back unless the population has already achieved herd immunity measured at pre-pandemic levels of behavior. That is, the predictions for the long-run impact of COVID-19 using a standard epidemiological model should continue to hold. [xiii] Given estimates of the basic reproduction number in the range of 2.5 (or now higher with new variants), this herd immunity threshold should kick in when significantly less than 40% of the population remains susceptible.

This logic implies that, absent a vaccine, the implications of a model that includes a prevalence-elastic demand for disease prevention for the long-run impact of a pandemic in terms of cumulative infections and deaths should be similar to that of a standard epidemiological model. In the case of COVID-19 in the United States, this would be a cumulative death toll on the order of 1.25 million.

### I.B. A Quantitative Illustration

To illustrate these points regarding the predictions of a standard epidemiological model and one with a prevalence elastic demand for disease prevention for the dynamics of an epidemic, I turn to a simple model of the dynamics of deaths from the COVID-19 epidemic in the United States that I presented in a recent working paper and which is included as an online appendix to this paper[xiv]. This model accounts for the dynamics of deaths from COVID-19 in the United States over the past year with natural shocks to transmission rates due to seasonality, due to the emergence of a new, more transmissible, variant of the novel coronavirus, and due to potential changes in the prevalence-elasticity of demand for costly measures to mitigate disease transmission. (I refer to this third shock as "pandemic fatigue" as a short-hand description of a decline in the responsiveness of private and public demand for costly disease prevention measures to changes in disease prevalence.)

This model accounts remarkably well for the pandemic's evolution in the United States over the past year. A seasonal decline in transmission rates, the model suggests, explains why the

prevalence of COVID-19 dropped to relatively low levels in the summer of 2020. It also suggests that a decline in the strength of the behavioral response to disease prevalence in late fall—"pandemic fatigue"—helps explain the large waves of infections and deaths seen in the late fall and winter. I use this model to generate forecasts for the pandemic over the next two years for both countries, with a new, more contagious variant of COVID-19, arriving in the United States from the United Kingdom and/or other sources in December 2020.

In Figure 1, I show (the prediction (in blue) for daily deaths from COVID-19 in the United States from mid-February 2020 to mid-February 2022, and data (red) on the seven-day moving average of daily deaths in the United States over the past year downloaded from the CDC's new COVID data tracker website[xv]. The behavioral model matches the data on deaths over the past year quite well, and it forecasts, absent vaccines, a continuation of the pandemic well into 2022. The predicted peak of deaths in late Spring of this year shown in this figure is driven by the spread of the new, more contagious, virus variant in the model. This new variant becomes the dominant variety by summer of 2021 in this forecast.

The long-run cumulative death toll in this forecast run of the model in Figure 1 is 1.27 million. The forecast shown in this figure does not include any consideration of the impact of vaccines, both to permit comparison with projections from a standard epidemiological model, and to serve as a benchmark for the impact of vaccination efforts.
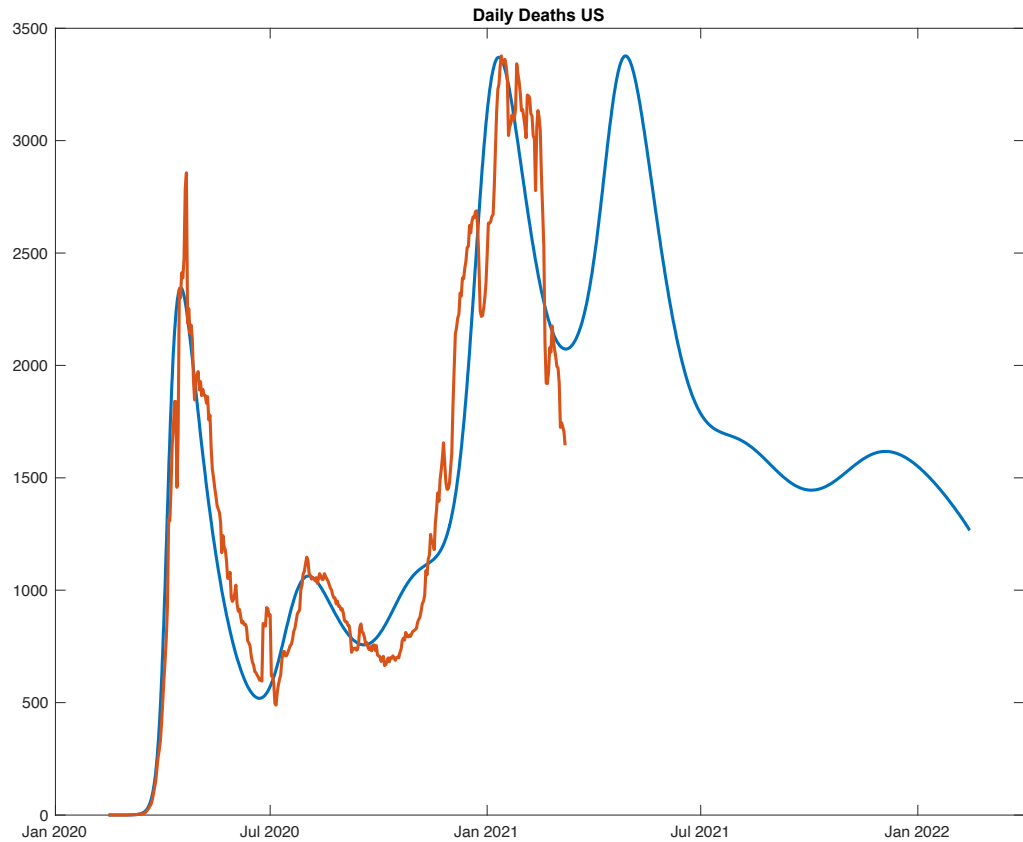
**Figure 1:** Behavioral model implications for daily deaths in the United States from mid-February, 2020 through mid-February 2022 are shown in blue. Transmission rates in the model are impacted by seasonal variation, the introduction of a more contagious variant in December of 2020, and prevalence elastic demand for costly measures to slow disease transmission. The wave of deaths forecast to occur later this spring is driven in the model by the introduction of the new, more contagious variant of the virus. Data on the seven-day moving average of daily deaths in the United States over the past year are shown in red. The forecast for cumulative deaths over the long-run implied by this model is 1.27 million.

To clarify the importance of the behavioral response in shaping disease dynamics, in Figure 2, I show the prediction for daily deaths of the same model with the behavioral response to disease prevalence turned off (in blue), relative to data on the seven-day moving average of daily deaths (in red). As we see in this figure, this standard epidemiological model without a behavioral response overstates the first peak of daily deaths by at least an order of magnitude (these peak at over 30,000/day), but then the pandemic comes quickly to an end in the fall of 2020. The cumulative death toll in this model forecast is 1.5 million. This prediction for the cumulative death toll is certainly larger than in the model with a behavioral response, but the gap between the two models in this dimension is much smaller than in their predictions for the initial peak and the time scale of the pandemic[xvi].

What is evident from these figures is that incorporating a response of public and private behavior to disease prevalence gives a dramatically different forecast for the severity of disease peaks, and the speed with which this epidemic passes through the population. This is true even with a relaxation of mitigation behavior. In this, behavioral model, the pandemic takes two-and-a-half years to play out rather than six-to-nine months as forecast by the basic model. The model's implications, however, for the long-run impact of the disease are not much altered by the consideration of behavior. In both basic and behavior variations, the model forecasts that a substantial majority of the population must become immune through infection or vaccination for the pandemic to end.
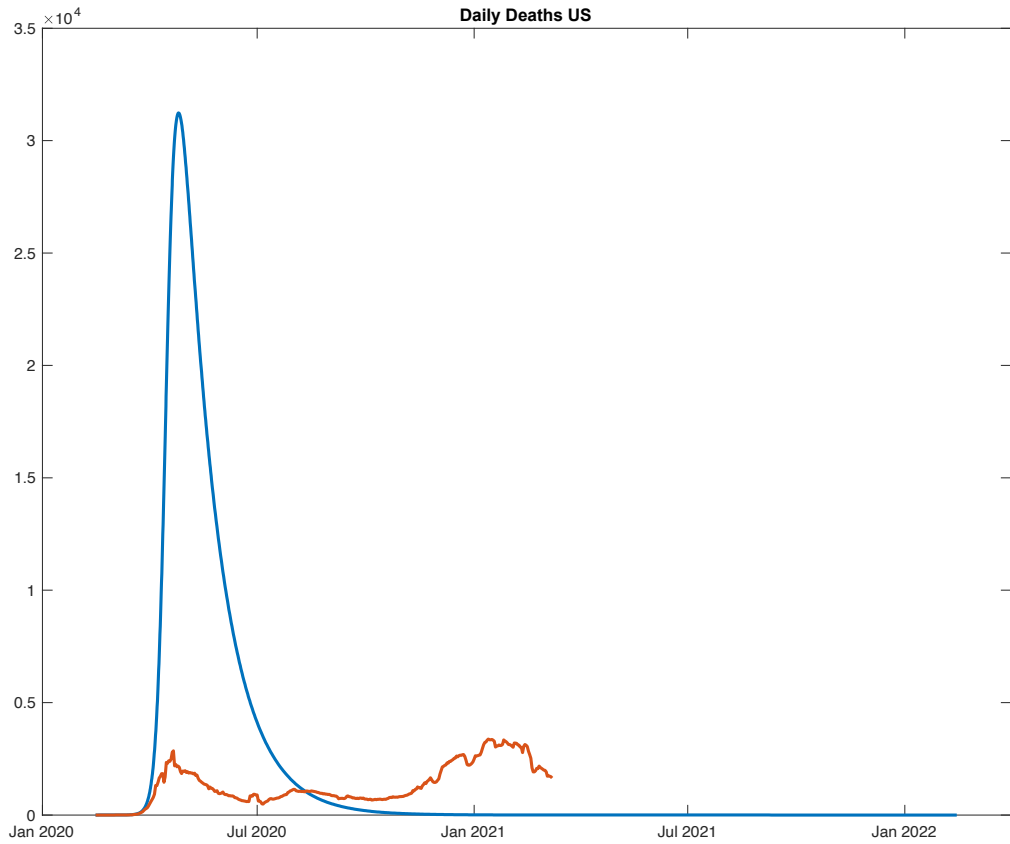
**Figure 2:** Standard model implications for daily deaths in the United States from mid-February, 2020 through mid-February 2023 are shown in blue. Transmission rates in the model are impacted by seasonal variation, the introduction of a more contagious variant in December of 2020, but the model has no prevalence elastic demand for costly measures to slow disease transmission. Data on the seven-day moving average of daily deaths in the United States over the past year are shown in red. The forecast for cumulative deaths over this three-year period is 1.5 million.

## II.    Private Behavior and Constraints on Policy

Given these insights on the impact of prevalence elastic demand for disease prevention on the dynamics of an epidemic, what are our options for using public policy to mitigate the impact of a pandemic on public health? One insight, that we have already mentioned, is that there is likely to be an offsetting private behavioral response to public measures that limit the spread of disease --- that is, that public measures to control an epidemic may well be partially undone by private responses to declining disease prevalence. The other insight is that public measures at disease prevention have to be essentially permanent to result in a meaningful reduction of the long-run impact of an epidemic absent a technological solution such as a vaccine or a cure.

We can use our simple behavioral model to illustrate the quantitative implications of these two insights. Imagine that through public policies facilitating a wide range of disease control measures such as masking and social distancing protocols, testing and contact tracing with isolation of the infectious, it was possible to reduce the transmission rate of COVID-19 in half, holding fixed seasonality and the level of costly disease control measures undertaken by both private agents and state and local authorities.

In Figure 3, I show a simulation of the model with such measures put in place for a two-year period from May 1, 2020 through May 1, 2022. I show the model implications for daily deaths over a five-year period in blue and the data on the seven-day moving average of daily deaths in red. As we see in this figure, these disease control measures, when imposed on top of those arising in equilibrium from the prevalence-elastic demand of both private agents and public authorities for costly measures to control disease, have a significant impact in reducing deaths from the disease in the first year. Then, in this simulation, in early 2021, the arrival of the new variant and, in mid 2022 the abandonment of these disease control measures, leads to significant spikes in forecast deaths. A significant level of daily deaths then continues into the first half of 2023 and, over the long run, the cumulative death tool is 1.27 --- almost exactly what we found in the simulation in Figure 1 that had no such disease control measures imposed.
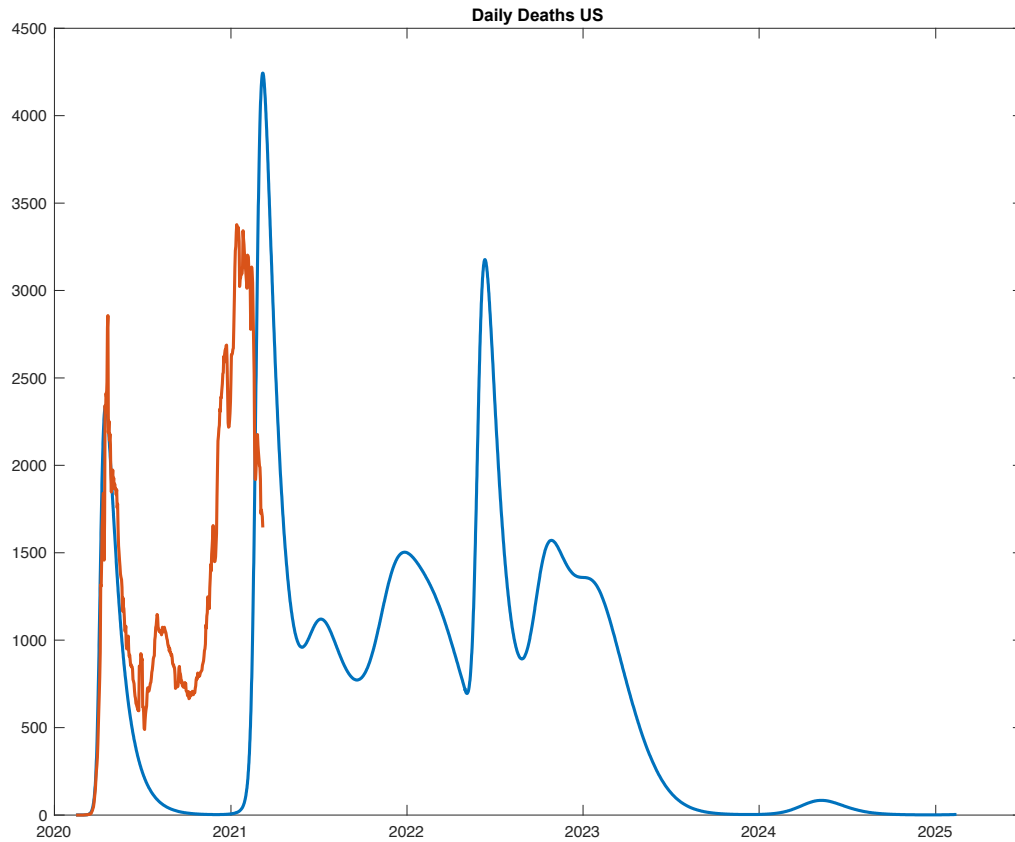
**Figure 3:** Predictions of the model for the evolution of daily deaths from COVID-19 in a version of the model in which disease control measures such as masks, social distancing, testing with contact tracing and isolation of the infected cut the transmission rate of the disease in half holding fixed the level of private and state and local disease control efforts undertaken in response to the prevalence of the disease. These measures are assumed to be in place for two years from May 1, 2020 to May 1, 2022. While these disease control measures are effective in reducing deaths in the first year, they do not succeed in later years. The data on the seven-day moving average of daily deaths are shown in red. The model-implied cumulative death toll is 1.27 million --- the same as we found in the simulation in Figure 1.

### II.A. Waiting for a vaccine

We saw in Figure 3 that temporary disease control measures do not significantly reduce the long run public health impact of the epidemic in the absence of a technological solution such

as a vaccine or a cure. How does the analysis of the impact of such measures change when there is a good prospect that a vaccine or cure might arrive? Here I use the model to show that such measures can have a significant long-run public health benefit in reducing deaths from disease while waiting for the arrival of that technological solution.

In figure 4, I show the implications of the model for the evolution of daily deaths (in blue) when a program of vaccination starts on January 1, 2021 at a pace sufficiently fast to succeed in protecting half of the United States population by July 1, 2021. This vaccine is assumed to prevent both illness and disease transmission by the vaccinated. The data on the seven-day moving average of daily deaths is again shown in red. To see the model-implied impact of this vaccination program on the epidemic, one can compare the blue lines in Figures 1 and 4. Here we see that in the model, this vaccination program significantly reduces the forecast impact of the new variant later this Spring and brings the epidemic to an end late this summer or fall. Note that here the vaccination program succeeds despite the model-implied relaxation of public and private efforts at disease prevention.

We also see that the model predictions for the long-run death toll with this vaccination program is 672 thousand, a bit over half of what is forecast in the absence of a vaccine (in the simulations in Figures 1 and 3). In this sense, the vaccination program succeeds in reducing cumulative deaths in a manner that a two-year program of disease mitigation absent a vaccine does not.

But now consider the model-implied scenario for cumulative deaths if the temporary disease mitigation measures used in the simulation in Figure 3 had been imposed starting May 1, 2020 and the same vaccination program applied in the simulation in Figure 4 had started on January 1, 2021. With this combination of temporary disease mitigation measures and a successful vaccination program, the cumulative death toll implied by the model would have been only 292 thousand. Clearly, the combination of temporary disease control measures applied while waiting for a technological solution can save many lives.
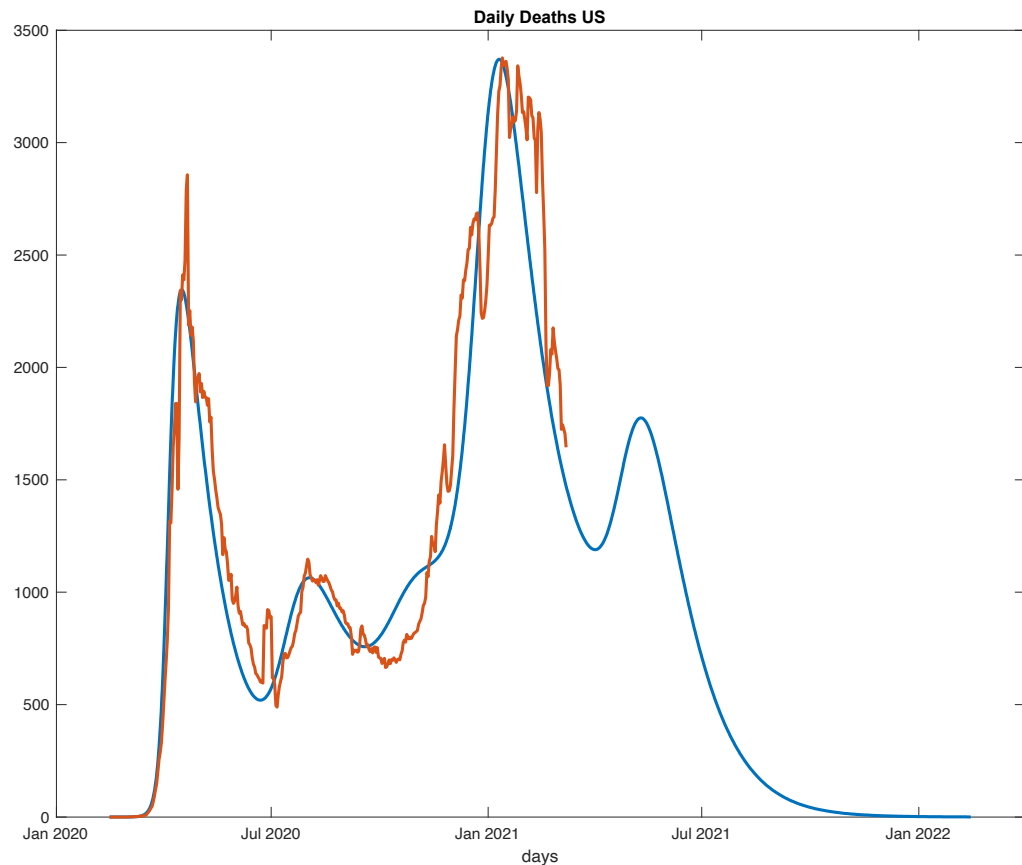
**Figure 4** Predictions of the model for daily deaths from COVID-19 (in blue) in a simulation with a vaccination program starting on Janaury 1, 2021 that proceeds at a rate fast enough to protect half of the population by July 1, 2021. In this simulation, the vaccine is assumed to protect against illness and to prevent disease transmission by the vaccinated. The data on the seven-day moving average of daily deaths are shown in red. To see the predicted impact of the vaccine on the dynamics of the epidemic, compare the blue line for model-implied daily deaths 2021 in Figure 1 to the blue line here. The vaccination program is forecast to significantly mitigate the spread of the new variant of the virus and to bring the pandemic to an end in late summer or early fall of 2021. The long-run cumulative death toll in this simulation is 672 thousand.

## III.    Conclusion

The global COVID pandemic has clearly demonstrated that the risks from the emergence of new infectious diseases that epidemiologists have been speaking about for years is terribly real. This pandemic has also posed a severe test of public health strategies and capabilities worldwide. In many countries, the associated economic impact has been as severe as any downturn seen since the Great Depression. How might we do better next time?

Based on the lessons about the interaction of behavior and disease dynamics discussed here, I suggest the following three-part strategy to improve our public health and economic response to emerging infectious disease.

First, we need to invest in our disease surveillance capabilities worldwide, perhaps using the infrastructure developed for worldwide influenza surveillance as a model.[xvii] It is certainly worth a lot of money to have the capacity to contain and eliminate a new infectious disease anywhere in the world before it gets going.

Second, we need to invest in new models for accelerating the development, financing, and distribution of vaccines and cures for emergent disease. In the end, it is these technological solutions that will allow us to contain the long run impact of new pandemics once they become global.

Third, we must consolidate all that has been learned about the implementation of public health measures for disease control over the past year so that we might be able to quickly implement those measures that effectively slow disease spread with the least cost to the economy. As we have seen from these model simulations, the strategy underlying such measures should be to allow us to wait for the development of a technological solution to a global pandemic with minimal loss of life and economic damage.

To illustrate the urgency of addressing these public health priorities, consider one final model scenario. There is increasing evidence that, through mutation, COVID-19 might evolve to evade the immunity conferred by prior infection and vaccines. In such a scenario, COVID-19 would be an endemic, seasonal, disease that might require essentially permanent efforts at disease control.[xviii] To illustrate how such a scenario might play out, I simulate the model with vaccines shown in Figure 4 but in which immunity from infection and/or vaccination lasts on average for only 18 months. I show the resulting forecast path of daily deaths from COVID over a five year period in Figure 5. In this simulation, I assume that the vaccination program continues at a constant

rate of roughly 1.3 million vaccinations per day throughout the forecast period. As one can see in this figure, the epidemic is forecast in this scenario to settle into a regular seasonal pattern killing over 100,000 Americans per year even with new vaccines and a response of public and private behavior to the changing prevalence of the disease. Clearly, in such a scenario, we would benefit greatly from finding ways to mitigate this disease on an ongoing basis at a lower economic cost.
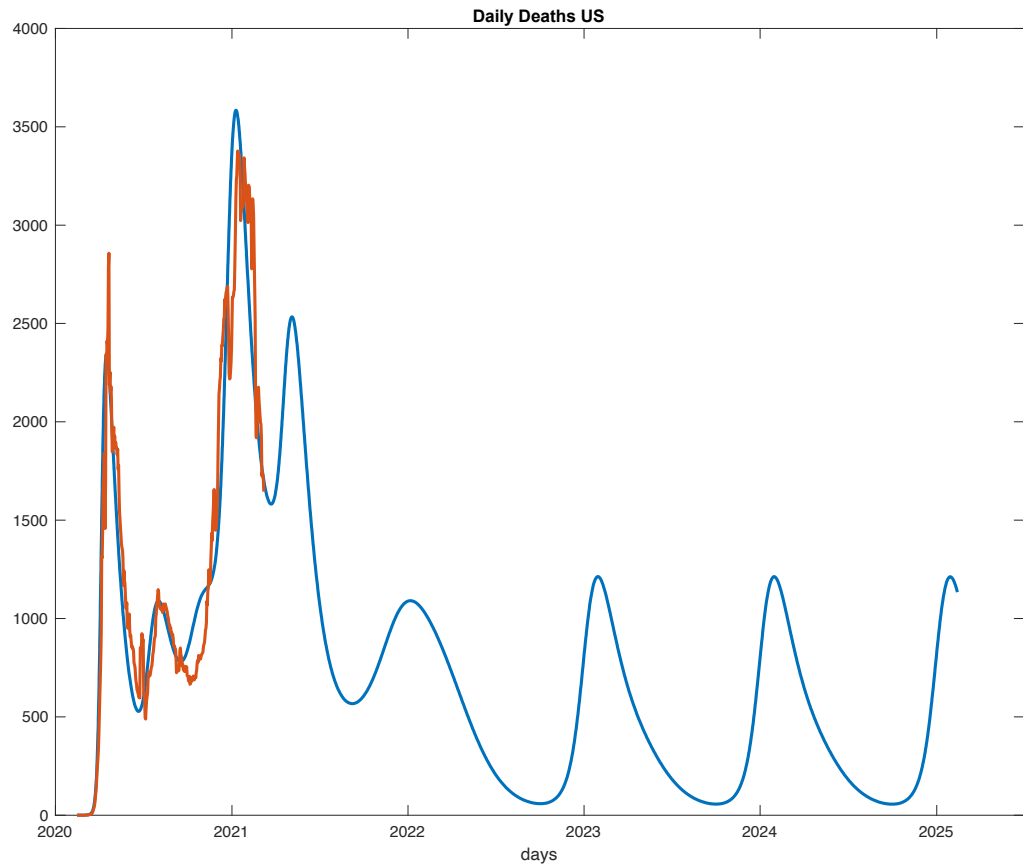


**Figure 5:** Predictions of the model for daily deaths from COVID-19 (in blue) in a simulation with a vaccination program starting on Janaury 1, 2021 that proceeds at a rate fast enough to protect half of the population by July 1, 2021. The data on the seven-day moving average of daily deaths are shown in red. In this simulation, immunity acquired from prior infection or vaccination is assumed to last 18 months on average. The vaccination program is assumed to continue at a constant rate throughout the entire period with presumably new booster shots conferring immunity against new variants as they occur. Even with this program of booster

vaccines and continued prevalence elastic behavior, in this simulation, more than 100,000 Americans die each year from COVID on a persistent basis.

ACKNOWLEDGMENTS Contents follow here on this line.

[i] See Armstrong, Gregory L., Conn, Laura A., Pinner, Robert W. "Trends in Infectious Disease Mortality in the United States During the 20th Century" *JAMA.* 1999;281(1):61-66. doi:10.1001/jama.281.1.61 https://jamanetwork.com/journals/jama/fullarticle/768249 . To place the mortality from COVID-19 in historical perspective, note that COVID mortality in the United States was roughly 100 in 100,000 in 2020 and may very well reach this level again in 2021. So while mortality from COVID will not reach the levels reached during the Spanish Flu, it will clearly be the most significant short term increase in mortality from infectious disease in the United States in at least 60 years.

[ii] See Morens, David M. and Fauci, Anthony S. "Emerging Pandemic Diseases: How we got to COVID-19" Cell *182*, September 3, 2020 https://doi.org/10.1016/j.cell.2020.08.021

[iii] See  this September 2019 report from the President's Council of Economic Advisors on the potential public health and economic impact of pandemic influenza.
[iv] Kermack, W. O. and McKendrick, A. G. "A Contribution to the Mathematical Theory of Epidemics." *Proc. Roy. Soc. Lond. A* **115**, 700-721, 1927.

[v] See https://www.nber.org/papers/w26867 and https://www.nber.org/system/files/working_papers/w26902/w26902.pdf for expositions of these predictions of standard SIR models from one year ago.
[vi] See https://www.bundesregierung.de/breg-de/themen/coronavirus/statement-chancellor-1732296 and https://edition.cnn.com/world/live-news/coronavirus-outbreak-03-11-20-intl-hnk/h_ab9bb8236fa91a9bf63cdbc7a69e0f10
[vii] See https://www.cell.com/immunity/fulltext/S1074-7613(20)30170-9 for a description of the calculations and considerations involved.
[viii] See https://www.cdc.gov/coronavirus/2019-ncov/cases-updates/burden.html. Here the CDC estimates that 83 million Americans experienced COVID infection over the course of 2020 with cumulative deaths from the disease reaching 375,000 by mid-January 2021.
[ix] See https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(21)00183-5 and https://www.nature.com/articles/d41586-021-00316-4 regarding data from Manaus and Israel on the empirical herd immunity threshold.
[x] NBER working paper 7037 published here https://www.sciencedirect.com/science/article/abs/pii/S1574006400800463

[xi] https://www.minneapolisfed.org/research/staff-reports/behavior-and-the-transmission-of-covid19

[xii] Joshua Gans reviews the implications of epidemiological models with a prevalence-elastic demand for costly measures to prevent disease transmission and much of the work by NBER affiliates on this topic in https://www.nber.org/papers/w27632.

[xiii] More complex models that emphasize heterogeneity and the network structure of human interaction potentially offer more optimistic implications for the long-run impact of COVID. See, for example https://www.nber.org/papers/w27373, https://www.nber.org/papers/w27374, https://www.nber.org/papers/w27741, and https://www.nber.org/papers/w28282. Recent research https://science.sciencemag.org/content/371/6530/741 forecasts that COVID-19 may become endemic if immunity is only temporary, as is the case for other coronaviruses.

[xiv] https://www.nber.org/papers/w28434
[xv] https://covid.cdc.gov/covid-data-tracker/#datatracker-home

xvi This difference between the cumulative death toll forecast in the model run in Figure 2 and that in Figure 1 is due to what is known as "overshooting" of herd immunity in the model without behavior in Figure 2. See https://www.nytimes.com/2020/05/01/opinion/sunday/coronavirus-herd-immunity.html for an explanation of this concept.
xvii https://www.who.int/influenza/gisrs_laboratory/en/

xviii See for example Christopher Murray and Peter Piot "The Potential Future of the COVID-19 Pandemic: Will SARS-CoV-2 Become a Recurrent Seasonal Infection?" Journal of the American Medical Association (JAMA) , published online March 3, 2021  doi:10.1001/jama.2021.2828

# Online Appendix for "Behavior and the Dynamics of Epidemics" for the Spring 2021 BPEA [*]

Andrew G. Atkeson[†]

March 8, 2021

# 1   Introduction

This appendix presents the model and parameters used in "Behavior and the Dynamics of Epidemics" by Andrew Atkeson for the Brookings Panel on Economic Activity Spring 2021. This model is based closely on that presented in "A Parsimonious Behavioral SEIR Model of the 2020 COVID Epidemic in the United States and United Kingdom" which is available as NBER working paper 28434 and as Federal Reserve Bank of Minneapolis Staff Report 619. This appendix discusses the model extended to include vaccines and the potential for waning immunity. It is applied to the United States.

This model is a an SEIR model (with compartments for agents who are susceptible, S, exposed, E, infectious, I, and recovered and hence removed R) modified to include a compartment for those infected agents who end up with serious disease. I refer to this compartment as H, for hospitalized. Agents who die from COVID are assumed to transition from infection I to death, D, through this compartment H. The expected time that agents spend in this compartment is set to 30 days to capture the delay between serious illness, death, and the reporting of that death. Behavior in this model is assumed to respond to daily death rates. It is assumed that behavior does not respond immediately to new infections as these are not directly observed. As discussed by John Cochrane[1] and Weitz et. al. 2020[2] the delay between infection and death introduced by this compartment H implies that this simple behavioral model has oscillatory endogenous dynamics that are helpful in allowing the model to reproduce the data with only a few shocks.

The three shocks considered in this paper are as follows. First, I add a standard seasonal variation in the baseline transmission rate of the virus from a winter peak to a low in midsummer. Second, I introduce a one-time change in behavior modeled as

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[1]See https://johnhcochrane.blogspot.com/2020/05/an-sir-model-with-behavior.html

[2]Joshua Weitz, Sang Woo Park, Ceyhun Eksin, and Jonathan Dushoff, "Awareness-driven behavior changes can shift the shape of epidemics away from peaks and toward plateaus, shoulders, and oscillations" , *Proceedings of the National Academy of Science*, vol. 117, no. 51, December 22, 2020

a reduction in the semi-elasticity of the transmission rate with respect to the daily death rate from an initial level to a new, permanently lower level. I refer to this second shock as the onset of pandemic fatigue. In the United States, pandemic fatigue sets in late in 2020. Third, I introduce a more contagious variant of COVID to the United States on December 1, 2020. The transmissibility of this variant is calibrated off of the experience in the United Kingdom. The model implies that this new variant becomes the dominant variant circulating in the United States by summer of 2021. I discuss the role that these shocks play in allowing the model to match the data on daily deaths from COVID in the paper "A Parsimonious Behavioral SEIR Model of the 2020 COVID Epidemic in the United States and United Kingdom" .

I model the impact of vaccines as moving agents from the susceptible compartment $S$ directly to the removed compartment $R$ at a rate $\lambda$ per day. With this assumption, I impose that the vaccine blocks both transmission by the vaccinated and disease in the vaccinated. I model waning immunity as a movement of agents from the removed compartment $R$ back to the susceptible compartment at a rate $\xi$ per day. I do not consider population growth in the model.

For all model forecasts, I leave the behavioral parameter $\kappa$ which determined the semi-elasticity of the transmission rate with respect to daily deaths fixed at its final value at the end of the model estimation period of one year. Thus, I assume that there are no further changes in behavior going forward.

## 2  Model and Parameters

The model is as follows.

The SEIHR model extends the SIR model by adding both the exposed state $E$ and the hospitalized state $H$. In this version of the model the total population $N$ is given by the sum of susceptible agents in state $S$, exposed in state $E$, infected in $I$, hospitalized in $H$, recovered in $R$, and dead in $D$.

To model the introduction of a new variant, I add separate compartments $E_v$ and $I_v$ for those exposed to and infectious with the new variant. The transmission rate of the original variant is denoted by $\beta(t)$. That for the new variant is denoted by $\beta_v(t)$.

The dynamics of the model are given by

$$\frac{dS(t)}{dt} = -(\beta(t)I(t) + \beta_v(t)I_v(t))S(t) - \lambda(t)S(t) + \xi R(t)$$

$$\frac{dE(t)}{dt} = \beta(t)I(t)S(t) - \sigma E(t)$$

$$\frac{dE_v(t)}{dt} = \beta_v(t)I_v(t)S(t) - \sigma E_v(t) + \bar{E}_v(t)$$

$$\frac{dI(t)}{dt} = \sigma E(t) - \gamma I(t),$$

$$\frac{dI_v(t)}{dt} = \sigma E_v(t) - \gamma I_v(t)$$

$$\frac{dH(t)}{dt} = \eta\gamma(I(t) + I_v(t)) - \zeta H(t)$$

$$\frac{dR(t)}{dt} = (1-\nu)\zeta H(t) + (1-\eta)\gamma(I(t) + I_v(t)) - \bar{E}_v(t) + \lambda(t)S(t) - \xi R(t)$$

$$\frac{dD(t)}{dt} = \nu\zeta H(t),$$

The reduced-form for the behavioral response of the transmission rate to the level of daily deaths is given by

$$\beta(t) = \bar{\beta}\exp(-\kappa(t)\frac{dD(t)}{dt} + \psi(t))$$

$$\beta_v(t) = \bar{\beta}_v\exp(-\kappa(t)\frac{dD(t)}{dt} + \psi(t))$$

where the parameters $\bar{\beta}$ and $\bar{\beta}_v$ control the baseline transmissibility of the normal and variant of COVID, the parameter $\psi(t)$ is used to introduce seasonality in trans-

mission, and $\kappa(t)$ is the semi-elasticity of transmission with respect to the level of daily deaths.

This reduced form response of transmission to daily deaths can be obtained as a result of a two-equation system in which the transmission rate is given as a function of activity $Y(t)$ with

$$\beta(t) = \bar{\beta} Y(t)^{\alpha} \exp(\psi(t))$$

and activity is given as a declining function of daily deaths

$$Y(t) = \exp(-\frac{\kappa(t)}{\alpha} \frac{dD(t)}{dt})$$

This decline in activity with daily deaths can be interpreted as arising either from a change in private behavior or public mandates that are conditioned on the prevalence of the disease. Seasonality as captured by $\psi(t)$ is modeled as a shift in the relationship between activity and transmission.

The new variant is introduced by setting $\bar{E}_v(t) = 1$ for one day on a specified date $t_v$ and equal to zero otherwise. Note that this quantity is subtracted off of the change in the $R$ compartment simply to keep the population constant. Since this shift is only one person for one day, it does not impact the quantitative implications of the model for large populations.

To model seasonality in the transmission of the virus, we set

$$\psi(t) = seasonalsize * (\cos((t + seasonalposition) * 2\pi/365) - 1)/2$$

where *seasonalsize* controls the magnitude of the seasonal fluctuations in transmissibility holding behavior fixed and *seasonalposition* controls the location of the seasonal peak in transmission. Note that $t$ is indexed to $t = 0$ on February 15, 2020.

To model pandemic fatigue, we set

$$\kappa(t) = \bar{\kappa} * (1 - normcdf(t, fatiguemean, fatiguesig)) +$$

4

$$fatiguesize * \bar{\kappa} * normcdf(t, fatiguemean, fatiguesig)$$

where $\bar{\kappa}$ sets the initial semi-elasticity of transmission with respect to daily deaths, $fatiguesize$ sets the percentage reduction in this semi-elasticity in the long run, $normcdf$ is the normal CDF, $fatiguemean$ sets the date at which the transition in $\kappa(t)$ from its initial to new long run level is halfway complete, and $fatiguesig$ sets the speed with which that transition occurs.

Initial conditions are $E(0) > 0$, $E_v(0) = I(0) = I_v(0) = R(0) = H(0) = D(0) = 0$, $S(0) = 1 - E(0)$. For the United States, $E(0) = 33$ on February 15 out of a population of 330 million.

I set the epidemiological parameters as follows: $\gamma = 0.4$, $\sigma = 0.425$, $\eta = 0.025$, $\nu = 0.2$, $\zeta = 1/30$. The parameter $\sigma$ corresponds to an expected time before and exposed agent becomes infectious of 2.35 days and the parameter $\gamma$ corresponds to an expected time for which an infected individual is infectious of 2.5 days. These two parameters together imply a generation time of 4.85 days. [3]

The parameter $\zeta$ corresponds to the rate at which those hospitalized flow either to death or recovery. This rate is chosen to have an average stay in compartment $H$ of 30 days, which corresponds to an average stay in the hospital of two weeks for those with serious illness and a reporting delay of deaths of two weeks. The infection fatality rate is given by $\eta\nu = 0.005$ which is the product of a rate of serious illness of 2.5% of total infections and a fatality rate of 20% for those with serious illness.[4]

The basic reproduction number of the virus at peak transmissibility is $\mathcal{R}_0(t) = \bar{\beta}/\gamma$ for the original virus and $\mathcal{R}_0(t) = \bar{\beta}_v/\gamma$ for the new variant. For the United States, I set $\bar{\beta} = 3\gamma$ giving a peak basic reproduction in Winter of 3. This number is well within the range of estimates of this parameter from the early phase of the pandemic.

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[3]See https://www.cdc.gov/coronavirus/2019-ncov/hcp/planning-scenarios.html. On that web-page, the CDC notes a mean time of approximately six days between symptom onset in one person to symptom onset in another person infected by that individual.

[4]See https://www.cdc.gov/coronavirus/2019-ncov/hcp/planning-scenarios.html. On that web-page, the CDC notes a median time from symptom onset to death of approximately two weeks and a median time from death to reporting just under three weeks.

For the new variant of the virus, I set $\bar{\beta}_v = 5\gamma$ giving a basic reproduction in Winter of 5. This implies that the new variant is 67% more transmissible than the original variant in the US. Note that the other epidemiological parameters associated with this new variant are assumed to stay the same, including the infection fatality rate.

To model seasonality of transmission in the United States, I set $seasonalsize = 0.35$ and $seasonalposition = 20$. Figure 1 shows the basic reproduction number corresponding to no reduction in transmission due to a behavioral response (the transmisibility of the virus with behavior at pre-pandemic patterns) for the United States. We see that the assumed pattern for seasonality in the US introduces a 35% reduction in transmissibility of the virus holding behavior fixed from the winter peak and the summer low.
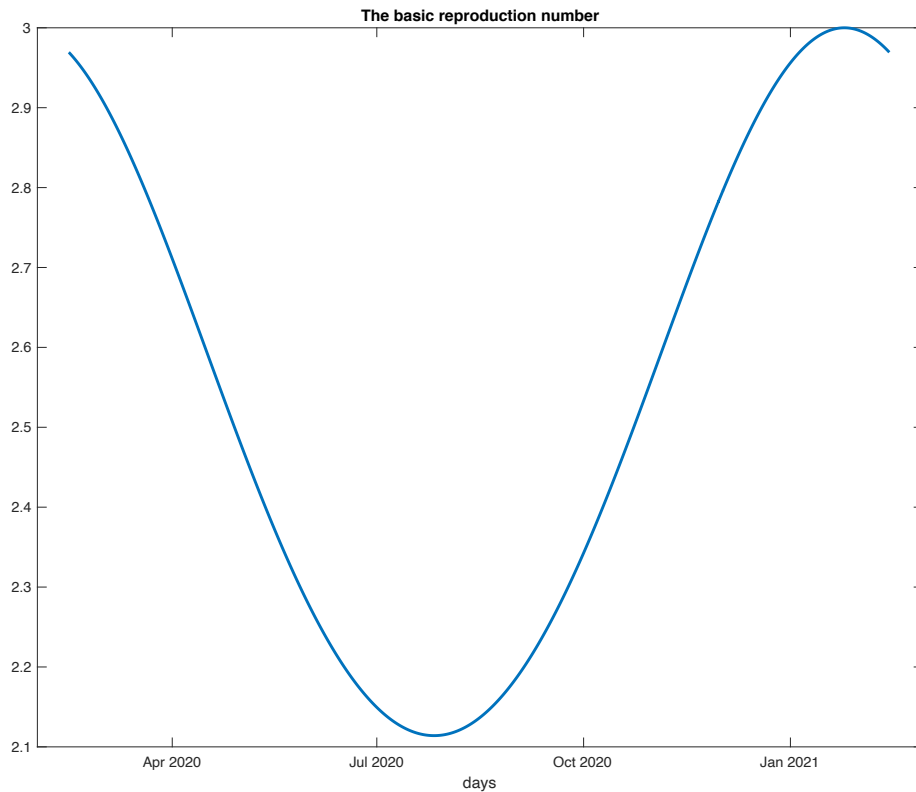
**Figure 1:** Assumed seasonality in the basic reproduction number.

The initial semi-elasticity of transmission with respect to daily deaths (measured as a fraction of the population) for the United States is $\bar{\kappa} = 250000$. To model the onset of pandemic fatigue in the United States, I set $fatiguesize = 0.375$, $fatiguemean = 285$ and $fatiguesig = 15$. Figure 2 shows the ratio of the semi-elasticity of the transmission rate with respect to the level of daily deaths relative to its initial level for the United States. We see in that figure that this semi-elasticity is assumed to fall to 37.5% of its original level late in the year.
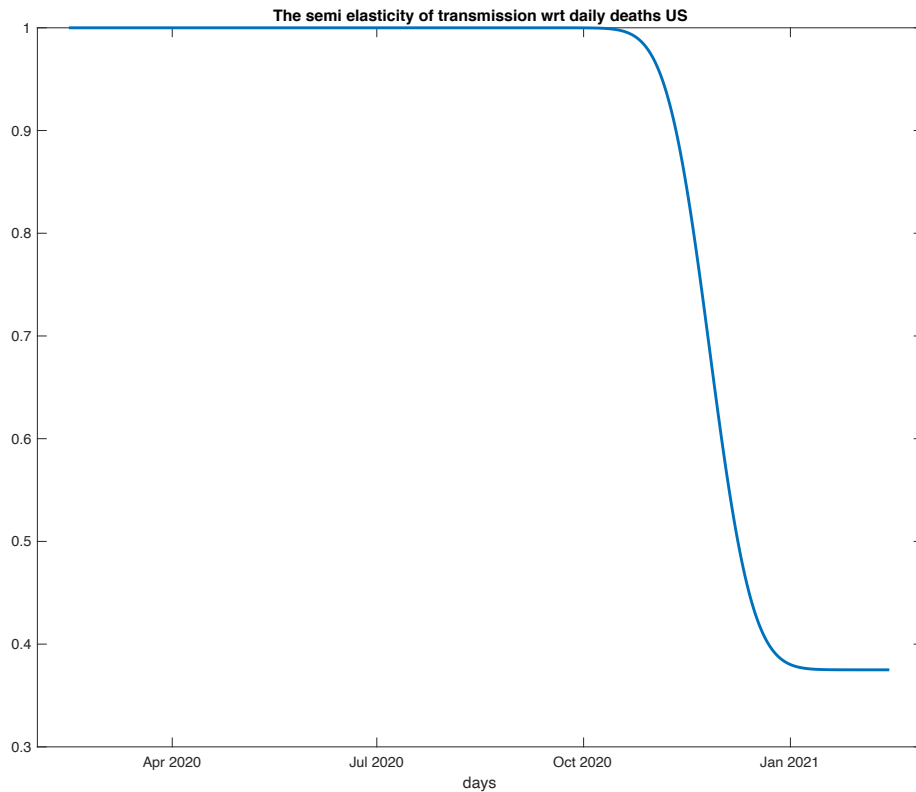
7

**Figure 2:** Assumed pandemic fatigue. The blue line shows the evolution of the semi-elasticity of transmission with respect to daily deaths relative to its initial level.

In simulations in which I include a vaccine, I set $\lambda(t) = 0.004$ starting on January 1, 2021. I assume that vaccinations are offered to the general population. This implies that in a population of 330 million, the daily number of vaccines administered is close to 1.3 million. In comparing this number to data on vaccinations, one must take into account that most of the vaccines administered require two doses for full effect. This assumption implies that roughly 50% of the population is fully vaccinated by July 1, 2021.

In simulations in which I assume waning immunity, I set $\xi = 1/547.5$ which corresponds to an expected time to loss of immunity of 18 months.