Moving Beyond a Peak Mentality: Plateaus, Shoulders, Oscillations and Other 'Anomalous' Behavior-Driven Shapes in COVID-19 Outbreaks

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The COVID-19 pandemic has caused more than 300,000 reported deaths globally, of which more than 83,000 have been reported in the United States as of May 16, 2020. Public health interventions have had significant impacts in reducing transmission and in averting even more deaths. Nonetheless, in many jurisdictions (both at national and local levels) the decline of cases and fatalities after apparent epidemic peaks has not been rapid. Instead, the asymmetric decline in cases appears, in some cases, to be consistent with plateau- or shoulder-like phenomena. Here we explore a model of fatality-driven awareness in which individual protective measures increase with death rates. In this model, epidemic dynamics can be characterized by plateaus, shoulders, and lag-driven oscillations after exponential rises at the outset of disease dynamics. We also show that incorporating long-term awareness can avoid peak resurgence and accelerate epidemic decline. We suggest that awareness of epidemic severiy is likely to play a critical role in disease dynamics, beyond that imposed by intervention-driven policies.

Significance statement: In contrast to predictions of conventional epidemic models, COVID-19 cases and fatalities have asymmetric shapes at both national and sub-national scales, with cases and fatalities declining much more slowly than they rose. This manuscript evaluates how awareness-driven behavior modulates the shape of epidemics. We find that short-term awareness of fatalities leads to emergent plateaus, persistent shoulder-like dynamics, and lag-driven oscillations in a SEIR-like model. Hence, new cases and fatalities persist at a near-constant rate given an almost entirely susceptible population. We also find that incorporating long-term awareness accelerates epidemic decline, leading to a switch from plateaus back to peak-like dynamics. These findings suggest the need to incorporate feedback between outbreak and behavior in forecasting models and in evaluating public health campaigns to control epidemic spread.

I. INTRODUCTION

The spread of COVID-19 has elevated the importance of epidemiological models as a means to forecast both near- and long-term spread. In the United States, the Institute for Health Metrics and Evaluation (IHME) model has emerged as a key influencer of state- and national-level policy [1]. The IHME model includes a detailed characterization of the variation in hospital bed capacity, ICU beds, and ventilators between and within states. Predicting the projected strains on underlying health resources is critical to supporting planning efforts. However such projections require an epidemic 'forecast'. The IHME's epidemic forecast differs from conventional epidemic models in a significant way – IHME assumes that the cumulative deaths in the COVID-19 epidemic follow a symmetric, Gaussian-like trajectory. For example, the IHME model predicts that if the peak is 2 weeks away then in 4 weeks cases will return to the level of the present, and continue to diminish rapidly. But, epidemics need not have one symmetric peak – the archaic Farr's Law of Epidemics notwithstanding (see [2] for a cautionary tale of using Farr's law as applied to the HIV epidemic).

Conventional epidemic models of COVID-19 represent populations in terms of their 'status' vis a vis the infectious agent, i.e., susceptible, exposed, infectious, hospitalized, and recovered [3–9]. New transmission can lead to an exponential increases in cases when the basic reproduction number $\mathcal{R}_0 > 1$ (the basic reproduction number denotes the average number of new infections caused by a single, typical individual in an otherwise susceptible population [10]). Subsequent spread, if left unchecked, would yield a single peak – in theory. That peak corresponds to when 'herd immunity' is reached, such that the effective reproduction number, $\mathcal{R}_{\text{eff}} = 1$. The effective reproduction number denotes the number of new infectious cases caused by a single infectious individual in a population with pre-existing circulation. But, even when herd immunity is reached, there will still be new cases which then diminish over time, until the epidemic concludes. A single-peak paradigm is only robust insofar

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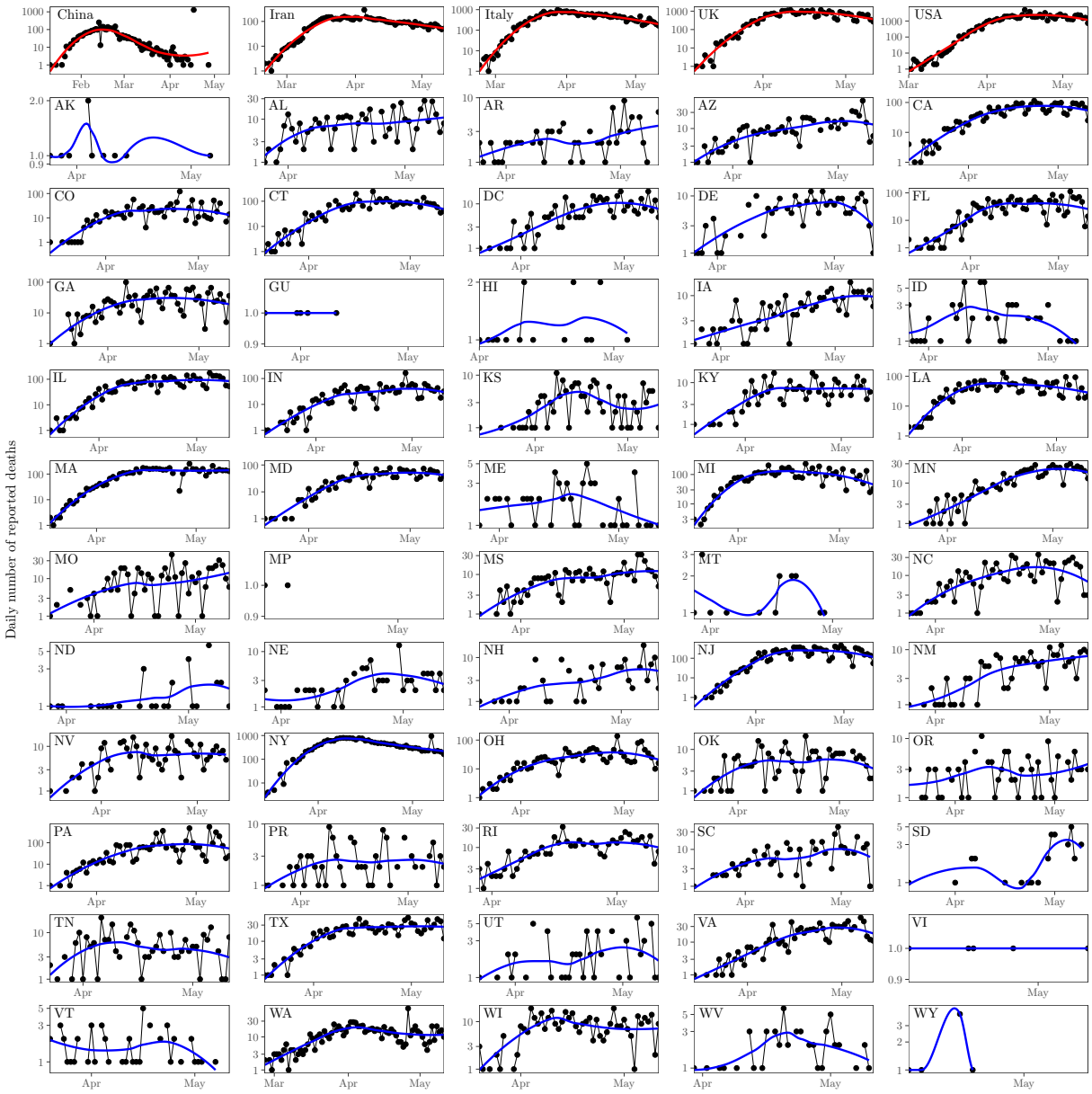

FIG. 1: Locally estimated scatterplot smoothing (LOESS) of daily number of deaths for COVID-19. Daily number of deaths is averaged in log space, only including days with one or more reported deaths. (Top; points and red lines) Selected national level averages; (Bottom; points and blue lines) US state level averages.

as the disease has spread sufficiently in a population to reach and exceed 'herd immunity'. The converse is also true – as long as a population remains predominantly immunologically naive, then the risk of further infection has not passed.

The Imperial College of London (ICL) model [3] is one of the most influential of epidemiogical models shaping public health responses to COVID-19. The ICL model is an example of a 'conventional' epidemic model that shows the benefits of early intervention steps in reducing transmission and preserving health system resources vs. a 'herd immunity' strategy. The ICL model assumes that transmission is reduced because of externalities, like lockdowns, school closings, and so on. As a result, the ICL model suggests that lifting of large-scale public health interventions could be followed by a second wave of cases. Yet, for a disease that is already the documented cause of more than 83,000 deaths in the United States alone, we posit that individuals are likely to continue to modify their behavior even after lockdowns are lifted. Indeed, the peak death rates in the United States and globally are not as high as potential maximums in the event that COVID-19 had spread unhindered in the population [3]. Moreover, rather than a peak and decline, there

is evidence at both national and within-US state scales of plateaus and shoulder like behavior for daily fatality rates (Figure 1).

In this manuscript we use a nonlinear model of epidemiological dynamics to ask the question: what is the anticipated shape of an epidemic if individuals modify their behavior in direct response to the impact of a disease at the population level? In doing so, we build upon earlier work on awareness based models (e.g. [11–14]) with an initial assumption: individuals reduce interactions when death rates are high and increase interactions when death rates are low. As we show, short-term awareness can lead to dramatic reductions in death rates compared to models without accounting for behavior, leading to plateaus, shoulders, and lag-driven oscillations in death rates. We also show that dynamics can be driven from persistent dynamics to elimination when awareness shifts from short- to long-term.

II. RESULTS AND DISCUSSION

A. SEIR Model with Short-Term Awareness of Risk

Consider an SEIR like model

$$\dot{S} = -\frac{\beta S I}{\left[1+(\delta/\delta_c)^k\right]} \quad (1)$$

$$\dot{E} = \frac{\beta S I}{\left[1+(\delta/\delta_c)^k\right]} - \mu E \quad (2)$$

$$\dot{I} = \mu E - \gamma I \quad (3)$$

$$\dot{R} = (1-f_D)\gamma I \quad (4)$$

$$\dot{D} = f_D \gamma I \quad (5)$$

where S, E, I, R, and D denote the proportions of susceptible, exposed, infectious, recovered, and deaths, respectively, given transmission rate β /day, transition to infectious rate μ /day, recovery rate γ /day, where f_D is the infection fatality probability. The awareness-based distancing is controlled by the death rate $\delta \equiv \dot{D}$, the half-saturation constant ($\delta_c > 0$), and the sharpness of change in the force of infection ($k \geq 1$) (see Figure 2 for a schematic). Since δ is proportional to I, this model is closely related to a recently proposed awareness-based distancing model [14] and to an independently derived feedback SIR model [15]. Note that the present model converges to the conventional SEIR model as $\delta_c \to \infty$.

Uncontrolled epidemics in SEIR models have a single case peak, corresponding to the point where $\gamma I = \beta S I$ such that the population obtains herd immunity when only a proportion $S = 1/\mathcal{R}_0$ have yet to be infected. However, when individuals decrease transmission in relationship to awareness of the population impacts of the disease, $\delta(t)$, then the system can 'peak' when levels of infected cases are far from herd immunity, specifically

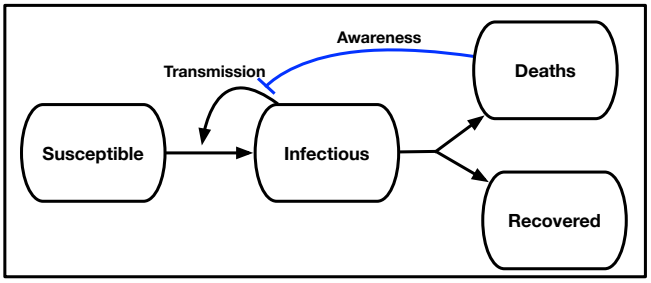

FIG. 2: Schematic of an SEIR model with awareness-driven social distancing. Transmission is reduced based on short- and/or long-term awareness of population-level disease severity (i.e., fatalities).

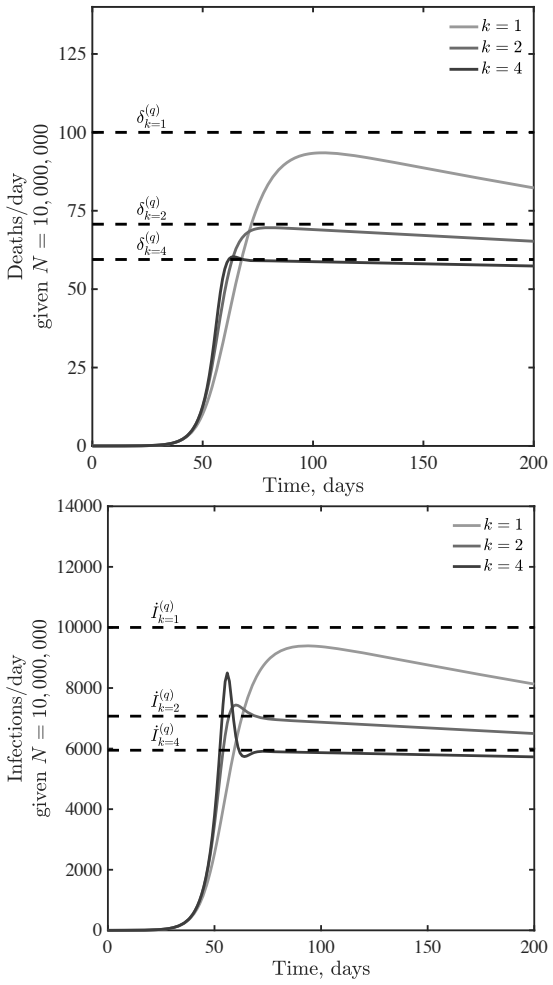

FIG. 3: Infections and deaths per day in a death-awareness based social distancing model. Simulations have the epidemiological parameters $\beta = 0.5$ /day, $\mu = 1/2$ /day, $\gamma = 1/6$/day, and $f_D = 0.01$, with variation in $k = 1$, 2 and 4. We assume $N\delta_c = 50$ /day in all cases.

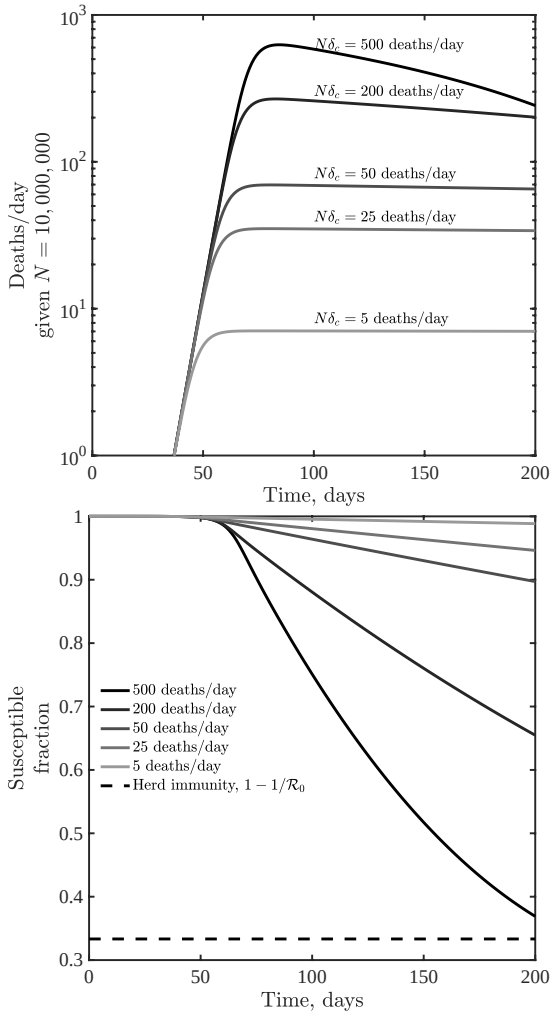

FIG. 4: Dynamics given variation in the critical fatality awareness level, δ_c for awareness $k = 2$. Panels show deaths/day (top) and the susceptible fraction as a function of time (bottom), the latter compared to a herd immunity level when only a fraction $1/\mathcal{R}_0$ remain susceptible. These simulations share the epidemiological parameters $\beta = 0.5$ /day, $\mu = 1/2$ /day, $\gamma = 1/6$ /day, and $f_D = 0.01$.

when

$$\gamma I = \frac{\beta SI}{\left[1 + (\delta/\delta_c)^k\right]}. \tag{6}$$

When δ_c is small compared to the per-capita death rate of infectious individuals (γf_D) we anticipate that individual behavior will respond quickly to the disease outbreak. Hence, we hypothesize that the emergence of an awareness-based peak can occur early, i.e., $S(t) \approx 1$, consistent with a quasi-stationary equilibrium when the death rate is

$$\delta^{(q)} \approx \delta_c \left(\mathcal{R}_0 - 1\right)^{1/k} \tag{7}$$

and the infection rate is

$$\dot{I}^{(q)} \approx \frac{\delta_c}{f_D} \left(\mathcal{R}_0 - 1\right)^{1/k}. \tag{8}$$

These early onset peak rates should arise not because of herd immunity but because of changes in behavior.

We evaluate this hypothesis in Figure 3 for $k = 1$, $k = 2$, and $k = 4$ given disease dynamics with $\beta = 0.5$ /day, $\mu = 1/2$ /day, $\gamma = 1/6$ /day, $f_D = 0.01$, $N = 10^7$, and $N\delta_c = 50$ /day. As is evident, the rise and decline from peaks are not symmetric. Instead, incorporating awareness leads to dynamics where incidence decreases very slowly after a peak. The peaks occur at levels of infection far from that associated with herd immunity. Post-peak, shoulders and plateaus emerge because of the balance between relaxation of awareness-based distancing (which leads to increases in cases and deaths) and an increase in awareness in response to increases in cases and deaths.

We also observe some differences in dynamics given increases in the awareness exponent k. As k increases, individuals become less sensitive to fatality rates where $\delta < \delta_c$ and more sensitive to fatality rates where $\delta > \delta_c$. This leads to sharper dynamics. In addition, infections can over-shoot the expected plateau given that awareness is driven by fatalities which are offset with respect to new infections.

B. Short-term awareness and long-term plateaus

Initial analysis of an SEIR model with short-term awareness of population-level severity suggests a generic outcome: fatalities will increases exponentially before plateauing near to a level δ_c. Figure 4 shows the results of dynamics given δ_c values over a range equivalent to 5 to 500 deaths/day given a population of 10^7 for $k = 2$ (we note that results for $k = 1$ and $k = 4$ lead to similar findings, see Figure S1). When δ_c is small (compared to (γf_D), fatalities can be sustained at near-constant levels for a long time. When δ_c is higher then the decline of cases and fatalities due to susceptible depletion is relatively fast. Hence, even given significant variation in the critical daily fatality rates, the population remains largely susceptible even as sustained fatalities continue for a period far greater than the time it took to reach the plateau.

C. Emergent oscillations given lags between cases and fatalities

To explore the impacts of lags on dynamics, we incorporated an additional class H, assuming that fatalities follow potentially prolonged hospital stays. We do not include detailed information on symptomatic transmission, asymptomatic transmission, hospitalization outcome, age structure, and age-dependent risk (as in [3]).

Instead, we consider the extended SEIR model:

$$\dot{S} = -\frac{\beta SI}{\left[1+(\delta/\delta_c)^k\right]} \quad (9)$$

$$\dot{E} = \frac{\beta SI}{\left[1+(\delta/\delta_c)^k\right]} - \mu E \quad (10)$$

$$\dot{I} = \mu E - \gamma I \quad (11)$$

$$\dot{R} = (1-f_D)\gamma I \quad (12)$$

$$\dot{H} = f_D\gamma I - \gamma_H H \quad (13)$$

$$\dot{D} = \gamma_H H \quad (14)$$

where $T_H = 1/\gamma_H$ defines the average time in a hospital stay before a fatality. Note, we recognize that many individuals recover from COVID-19 after hospitalization; this model's hospital compartment functions as a prefilter.

The earlier analysis of the quasi-stationary equilibrium in fatalities holds in the case of a SEIR model with additional classes before fatalities. Hence, we anticipate that dynamics should converge to $\delta = \delta^{(q)}$ at early times. However, increased delays between cases and fatalities could lead to oscillations in both. Indeed, this is what we find via examination of models in which T_H ranges from 7 to 35 days, with increasing magnitude of oscillations as T_H increases (see Figure 5 for $k = 2$ with qualitatively similar results for $k = 1$ and $k = 4$ shown in Figure S2).

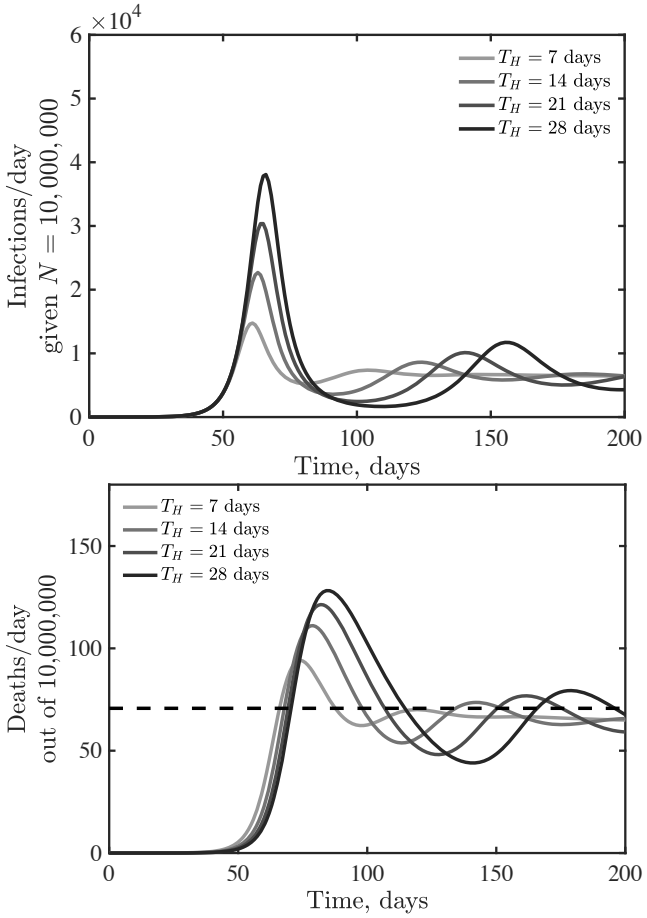

FIG. 5: Emergence of oscillatory dynamics in a death-driven awareness model of social distancing given lags between infection and fatality. Awareness is $k = 2$ and all other parameters as in Figure 3. The dashed lines for fatalities expected quasi-stationary value $\delta^{(q)}$.

D. Dynamical consequences of short-term and long-term awareness

Awareness can vary in duration, e.g., awareness of SARS-1 may prepare individuals to more readily adopt and retain social distancing measures [16, 17]. In previous work, long-term awareness of cumulative incidence was shown to lead to substantial decreases in final size of epidemics compared to baseline expectations from inferred strength [14]. Hence, we consider an extension of the SEIR model with lags between infection and fatalities that incorporates both short-term and long-term awareness:

$$\dot{S} = -\frac{\beta SI}{\left[1+(\delta/\delta_c)^k+(D/D_c)^k\right]} \quad (15)$$

$$\dot{E} = \frac{\beta SI}{\left[1+(\delta/\delta_c)^k+(D/D_c)^k\right]} - \mu E \quad (16)$$

$$\dot{I} = \mu E - \gamma I \quad (17)$$

$$\dot{R} = (1-f_D)\gamma I \quad (18)$$

$$\dot{H} = f_D\gamma I - \gamma_H H \quad (19)$$

$$\dot{D} = \gamma_H H \quad (20)$$

where D_c denotes a critical cumulative fatality level (and formally a half-saturation constant for the impact of long-term awareness on distancing). Note that the relative importance of short- and long-term awareness can be modulated by δ_c and D_c respectively. Figure 6 shows daily fatalities (top) and cumulative fatalities (bottom) for an SEIR model with $\mathcal{R}_0 = 2.5$, $T_H = 14$ days, and $N\delta_c = 50$ fatalities per day and critical cumulative fatalities of $ND_c = 2,500$, 5,000, 10,000 as well as a comparison case with vanishing long-term awareness. As is evident, long-term awareness drives dynamics towards rapid declines after reaching a peak. This decline arises because D monotonically increases; increasing fatalities beyond D_c leads to rapid suppression of transmission. However, when δ_c rather than D_c drives dynamics, then shoulders and plateaus can re-emerge. In reality, we expect that individual behavior is shaped by short- and long-term awareness of risks, including the potential for 'decay' of long-term awareness [11, 12].

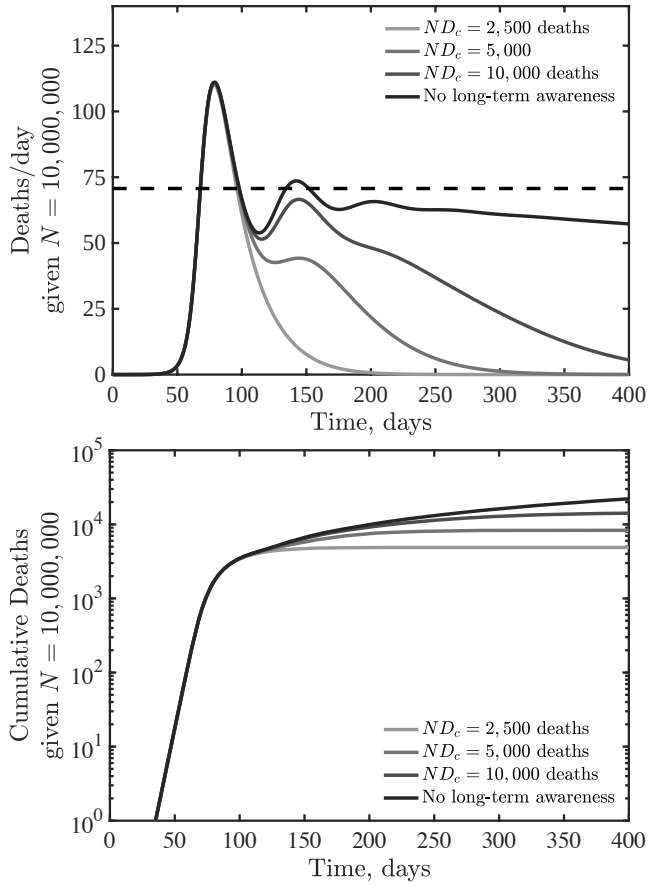

FIG. 6: SEIR dynamics with short- and long-term awareness. Model parameters are $\beta = 0.5$ /day, $\mu = 1/2$ /day, $\gamma = 1/6$ /day, $T_H = 14$ days, $f_D = 0.01$, $N = 10^7$, $k = 2$, $N\delta_c = 50$ /day (short-term awareness), with varying ND_c (long-term awareness) as shown in the legend. The dashed line (top) denotes $\delta^{(q)}$ due to short-term distancing alone.

III. CONCLUSIONS

We have developed and analyzed a model that assumes awareness of disease-induced death can reduce transmission and shown that such awareness-driven feedback can lead to highly asymmetric epidemic curves. Asymmetric curves exhibit extended periods of near-constant cases even as the majority of the population remains susceptible. In these conditions, if individuals are unable to sustain social distancing policies, or begin to tolerate higher death rates, then cases could increase (similar results have also been proposed in a recent, independently derived feedback SIR model [15]). Hence: passing a 'peak' need not imply the rapid decline of risk. These types of impacts of awareness-driven endogenous changes in $\mathcal{R}_{\text{eff}}$ are typically absent in models that form the basis for public policy and strategic planning. Our findings highlight the potential impacts of short-term and long-term awareness in efforts to shape information campaigns to reduce transmission after early onset 'peaks', particularly when populations remain predominantly immunologically naive.

Although the models here are intentionally simple, it seems likely that observed asymmetric dynamics of COVID-19, including slow declines and plateau-like behavior, may be an emergent property of awareness-driven epidemiological dynamics. We recognize that qualititatively similar effects could arise if responses are due to case reports or to illnesses with a personal connection. They may also be driven by official and media responses, as well as purely individual ones. In addition, ongoing ascertainment biases may also influence the shape of reported, rather than actual, case curves.

Moving forward, it is essential to fill in significant gaps in understanding how awareness of disease risk and severity shape behavior [18]. Mobility data is a proxy but not equivalent to a direct indicator of transmission risk. Nonetheless, measurements of community mobility have been used as a leading indicator for epidemic outcomes. Prior work has shown significant impacts of changes in mobility and behavior on the COVID-19 outbreak [7]. Here we have shown the importance of looking at a complementary feedback mechanism, i.e., from outbreak to behavior. Understanding the drivers behind emergent plateaus observed at national and sub-national levels could help decision makers structure intervention efforts appropriately to effectively communicate awareness campaigns that may aid in collective efforts to control the ongoing COVID-19 pandemic.

Data availability: All simulation codes, figures, and data used in the development of this manuscript are available at `https://github.com/jsweitz/covid19-git-plateaus`. Daily number of reported deaths as of May 11, 2020, is obtained from The COVID Tracking Project (covidtracking.com; for US states and territories) and the European Centre for Disease Prevention and Control (https://www.ecdc.europa.eu/en; for 5 countries).

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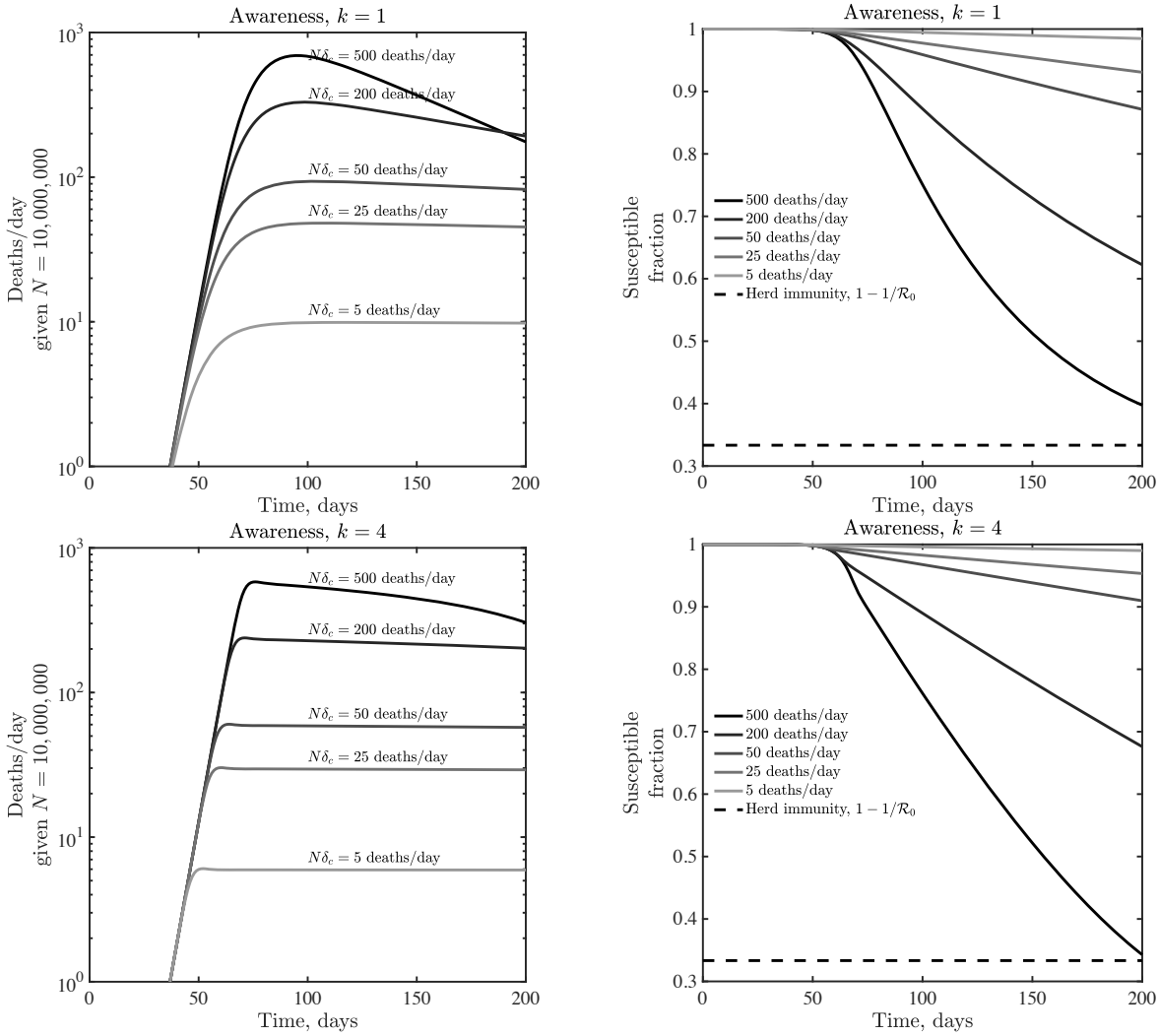

FIG. S1: Dynamics given variation in the critical fatality awareness level, δ_c for awareness $k = 1$ (top) and $k = 4$ (bottom). Panels show deaths/day (top) and the susceptible fraction as a function of time (bottom), the latter compared to a herd immunity level when only a fraction $1/\mathcal{R}_0$ remain susecptible. These simulations share the epidemiological parameters $\beta = 0.5$ /day, $\mu = 1/2$ /day, $\gamma = 1/6$ /day, and $f_D = 0.01$.

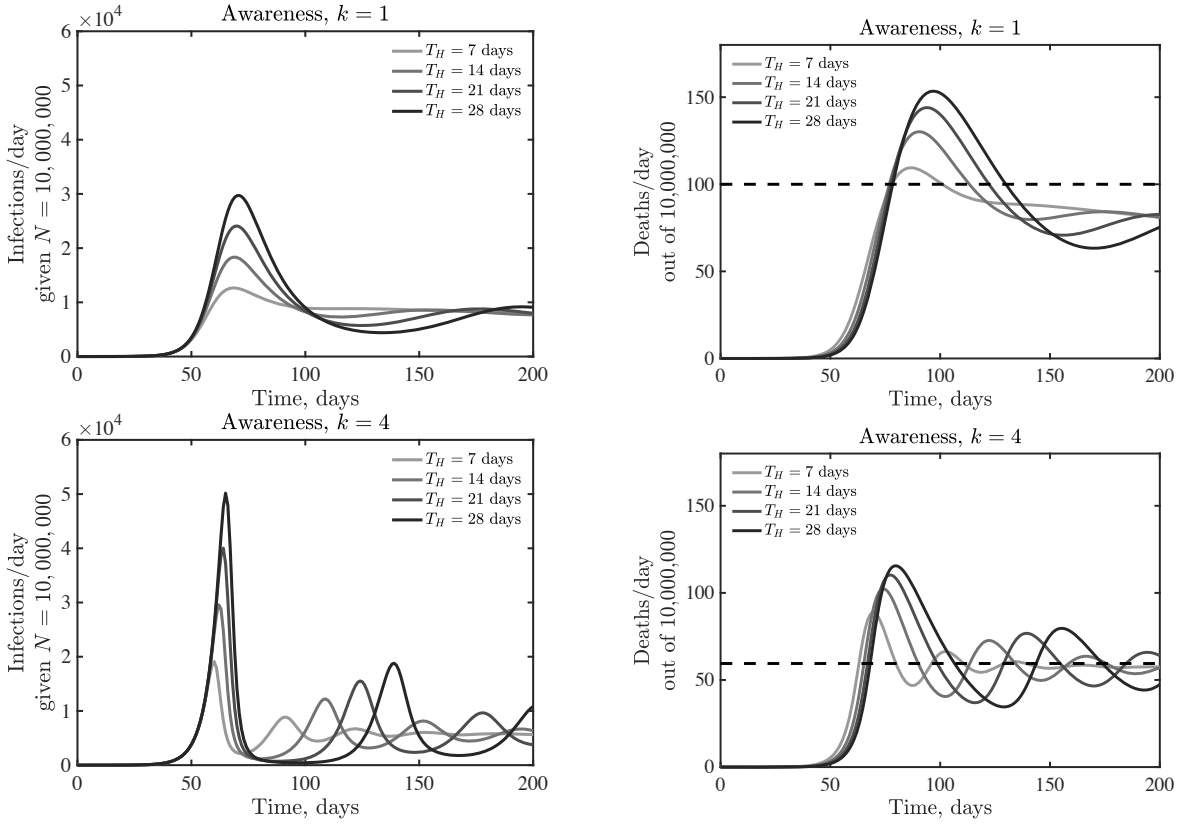

FIG. S2: Emergence of oscillatory dynamics in a death-driven awareness model of social distancing given lags between infection and fatality. Awareness is $k = 1$ (top) and $k = 4$ (bottom), all other parameters as in Figure 3. The dashed lines for fatalities expected quasi-stationary value $\delta^{(q)}$.