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

Joshua S. Weitz,[1,2,3,*] Sang Woo Park,[4] Ceyhun Eksin,[5] and Jonathan Dushoff[6,7]

[1] *School of Biological Sciences, Georgia Institute of Technology, Atlanta, GA, USA*
[2] *School of Physics, Georgia Institute of Technology, Atlanta, GA, USA*
[3] *Center for Microbial Dynamics and Infection, Georgia Institute of Technology, Atlanta, GA, USA*
[4] *Department of Ecology and Evolutionary Biology, Princeton University, Princeton, NJ, USA*
[5] *Department of Industrial and Systems Engineering, Texas A&M, College Station, Texas, USA*
[6] *Department of Biology, McMaster University, Hamilton, ON, Canada*
[7] *DeGroote Institute for Infectious Disease Research, McMaster University, Hamilton, ON, Canada*

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The COVID-19 pandemic has caused more than 200,000 reported deaths globally, of which more than 50,000 have been reported in the United States. 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 the severity of the short- and long-term epidemic is likely to play a critical role in disease dynamics, beyond that imposed by intervention-driven policies.

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 represent populations in terms of their 'status' vis a vis the infectious agent, in this case SARS-CoV-2 (e.g., [3–9]), e.g., susceptible, exposed, infectious, hospitalized, and recovered. 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, $\mathcal{R}_{\rm eff} = 1$. 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 as the disease has spread sufficiently in a population to reach and exceed 'herd immunity'. The converse is also true in the case of COVID-19 – 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 50,000 deaths in the United States, we posit that individuals, are likely to continue to modify their behavior even after lockdowns are lifted. Hence, here, we use a simple model to ask the question: what is

*Electronic address: jsweitz@gatech.edu; URL: http://ecotheory.biology.gatech.edu

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 a simple, initial assumption: individuals reduce interactions when death rates are high and increase interactions when death rates are low.

To begin, consider a SEIR like model

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

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

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

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

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

where S, E, I, R, and D denote the proportions of susceptible, exposed, infectious, recovered, and deaths, respectively. 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 ($k \geq 1$). Since δ is proportional to I, this model is closely related to a recently proposed awareness-based distancing model [14]. Note that the present model converges to the conventional SEIR model as $\delta_c \to \infty$.

Typically, epidemics arising in SEIR models have a single case peak, corresponding to the point where $\gamma I = \beta SI$ such that $S = 1/\mathcal{R}_0$, equivalent to when the herd immunity level proportion of individuals $1 - 1/\mathcal{R}_0$ have been infected. However, when individuals decrease transmission in relationship to awareness of the current severity of the disease, $\delta(t)$, then the system can 'peak' when levels of infected cases are far from herd immunity, specifically when

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

When δ_c is small compared to the 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

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

and the infection rate is

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

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

We evaluate this hypothesis in Figure 1 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, increasing nonlinearity of awareness a leads to shoulders where incidence decreases very slowly after a peak. We interpret this finding to mean that as the awareness exponent k increases, individuals become less sensitive to fatality rates where $\delta < \delta_c$ and more sensitive to fatality rates where $\delta > \delta_c$. The 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.

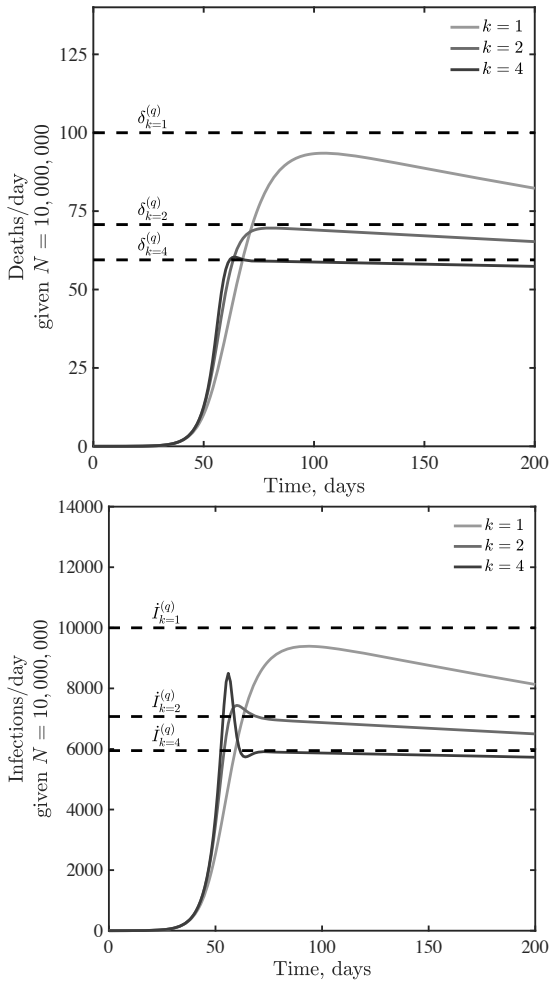

FIG. 1: Infections and deaths per day in a death-awareness based social distancing model. Simulations have the epidemiological parameters $\beta = 0.5$ /day, $\mu = 1/2$ /day, $\gamma = 1/6$/day, and $f_D = 0.01$, with variation in $k = 1$, 2 and 4.

These results suggest a generic outcome: first fatalities will grow exponential before plateauing near to the fatality awareness level δ_c. In the event that $\delta_c/(\gamma f_D)$ is sufficiently high then susceptible depletion will lead to the decline of cases and fatalities. Figure 2 shows the results of dynamics given δ_c values over a range equiv-

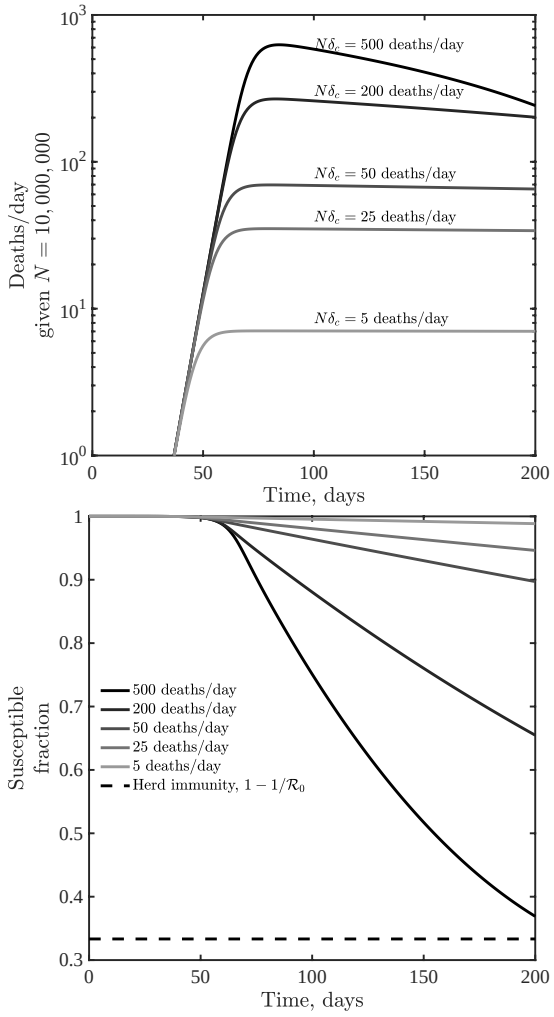

FIG. 2: Dynamics given variation in the critical fatality awareness level, D_c for awareness $k = 2$. Panels show deaths/day (top) and the susceptible fraction as a function of time (bottom), the latter compared to a herd immunity level when only $S = 1/\mathcal{R}_0$ remain. These simulations share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

alent 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, and are included in the `github` repository). We find that fatalities are sustained at near-constant levels (top) even as the population remains susceptible at levels far above herd immunity (bottom). We observed that as k increases, then fatalities may overshoot the plateau. This arises because individuals initiate protective measures closer to when a critical fatality rate has been reached. These overshoots may lead to oscillatory dynamics when there are larger lags between new 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 explicit detailed information on symptomatic transmission, asymptomatic transmission, hospitalization outcome, age structure, and age-dependent risk (as in [3]). Instead, consider the extended SEIR model:

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

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

$$\dot{I} = \mu E - \gamma I \tag{11}$$

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

$$\dot{H} = f_D \gamma I - \gamma_H H \tag{13}$$

$$\dot{D} = \gamma_H H \tag{14}$$

where $T_H = 1/\gamma_H$ defines the average time in a hospital stay before a fatality. The earlier analysis of the quasi-stationary equilibrium in fatalities holds; 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 3 for $k = 2$ with qualitatively similar results for $k = 1$ and $k = 4$ on the `github`). In essence, delays between infection and fatalities can lead to larger oscillations, as individual behavior changes due to local awareness of deaths. Reduction in contacts when fatality rates exceed the critical awareness level do not translate into reductions for a period similar to that of T_H , such that deaths can then be driven below this critical value, then contacts increase, and deaths increase (again with a lag), and so on. We note that these oscillations could be amplified in stochastic models.

Finally, we recognize that awareness can vary in duration. 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, here we consider an extension of the SEIR model with lags between infection and fatalities that incorporates both short-term and long-term awareness:

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

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

$$\dot{I} = \mu E - \gamma I \tag{17}$$

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

$$\dot{H} = f_D \gamma I - \gamma_H H \tag{19}$$

$$\dot{D} = \gamma_H H \tag{20}$$

where D_c denotes a critical cumulative fatality level. Note that the relative importance of short- and long-term

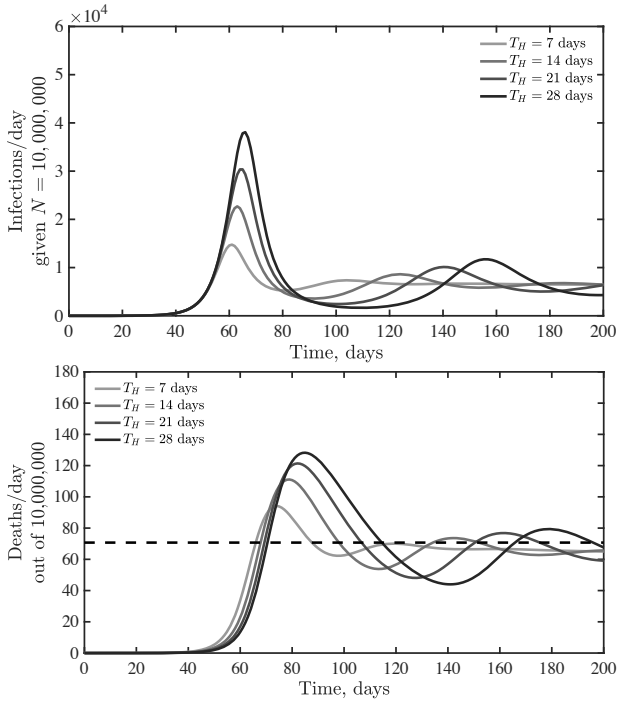

FIG. 3: Emergence of oscillatory dynamics in a death-driven awareness model of social distancing given lags between infection and fatality. Awareness is $k = 2$ and all other parameters as in Figure 2. The dashed lines for fatalities expected quasi-stationary value $\delta^{(q)}$.

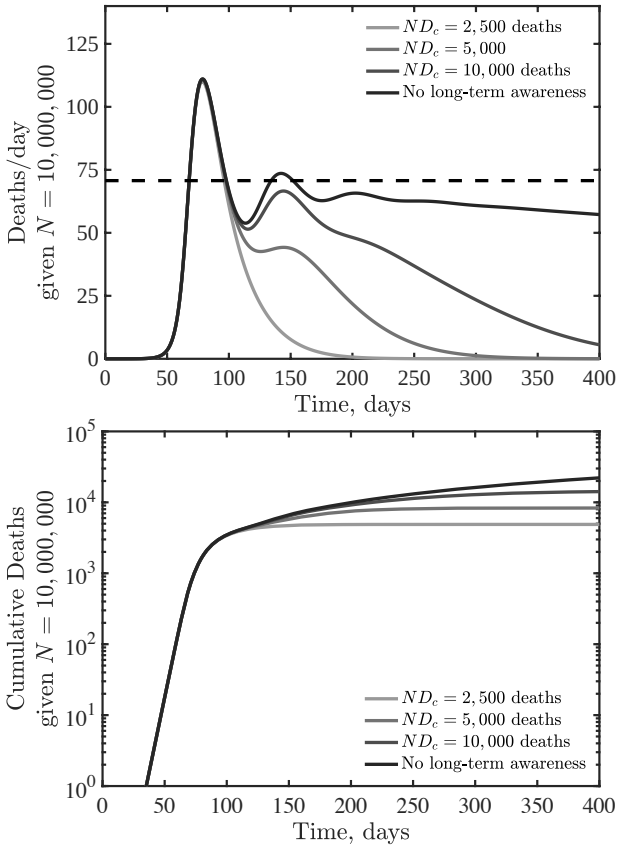

FIG. 4: SEIR dynamics with short- and long-term awareness. Model parameters are $\beta = 0.5$ /day, $\mu = 1/2$ /day, $\gamma = 1/6$ /day, $T_H = 14$ days, $f_D = 0.01$, $N = 10^7$, $k = 2$, $N\delta_c = 50$ /day (short-term awareness), with varying ND_c (long-term awareness) as shown in the legend. The dashed line (top) denotes $\delta^{(q)}$ due to short-term distancing alone.

awareness can be modulated by δ_c and D_c respectively. Figure 4 shows cumulative fatalities (left) and daily fatalities (right) for a 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].

In summary, we have shown how awareness-driven avoidance of transmission when fatality rates exceed critical levels can lead to sustained fatalities even as the majority of the population remains susceptible. Hence, if individuals are unable to sustain social distancing policies, or begin to tolerate higher death rates, then cases could increase. As a result, 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. Moving forward, we hope that our findings highlight the 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, we contend that as cumulative data from COVID-19 outbreaks already indicate, the asymmetric post-peak dynamics of COVID-19, including slow declines and plateau-like behavior, may be an emergent property of awareness-driven epidemiological dynamics.

Data availability: All simulation and codes used in the development of this manuscript are available at `https://github.com/jsweitz/covid19-git-plateaus`.

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