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 200,000 reported death2 globally of which more than 50,000 deaths have been reported in the United States alone. 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 propose a model of fatality-driven awareness in which individual protective measures increase as death rates exceed critical awareness levels. 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 near- and long-term epidemic is likely to play a critical role in disease dynamics, beyond that imposed by intervention-driven policies.

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 predefined, 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, there is no epidemiological rule that says epidemics must have one symmetric peak, the defunct Farr's Law of Epidemics notwithstanding (see [2] for a cautionary tale of the misapplication of Farr's law to the HIV epidemic).

Instead, conventional COVID-19 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]), That is, individuals in a population are represented in different compartments, e.g., susceptible, exposed, infectious, hospitalized, and recovered. In many cases, epidemic models might further categorize individuals by age and disease status, or age, occupation, and disease status, and so on. As a result, increases in cases are a result of transmission events between infectious and susceptible individuals. The compounding effects of 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, $\mathcal{R}_{eff} = 1$. The effective reproduction number denotes the number of new infectious cases caused by a single infectious individual in a population with pre-existing circulation; this number varies with time depending on population state, disease characteristics, and social behavior. 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'. Yet, the converse is 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 earliest and most influential epidemic models that has shaped 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

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in reducing transmission and preserving health system resources vs. a 'herd immunity' strategy. Unlike the IHME model, the ICL model suggests that more than one peak is possible, precisely because the model integrates latent states of the population as part of a representation of epidemiological mechanisms. The ICL model assumes that transmission is reduced because of externalities, like lockdowns, school closings, and so on. 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 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

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

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

$$\dot{I} = \mu E - \gamma I \tag{3}$$

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

$$\dot{D} = f_D \gamma I \tag{5}$$

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 ($k \geq 1$). As a result of the proportionality between δ and I, this model is a variant of a recently proposed local 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 $\gamma I = \beta S I$ 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

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

In the event that $\delta_c/(\gamma f_D) \ll 1$ we anticipate that most of the population remains susceptible when individual behavior changes markedly due to awareness of disease severity. Hence, we hypothesize that the emergence of

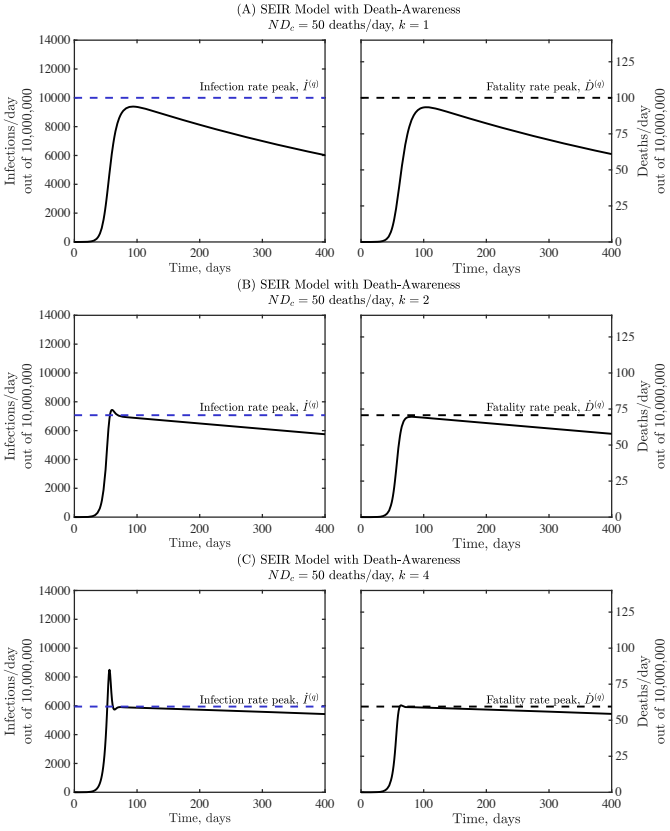

FIG. 1: Infections and deaths per day in a death-awareness based social distancing model. Awareness varies from $k = 1$, $k = 2$, to $k = 4$ in panels (A), (B), and (C).

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 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 lead to shoulders and, in the limit of $k \gg 1$, plateaus, such that deaths and cases appear nearly constant and close to the quasi-stationary equilibrium, declining slowly because of the depletion of susceptibles. 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

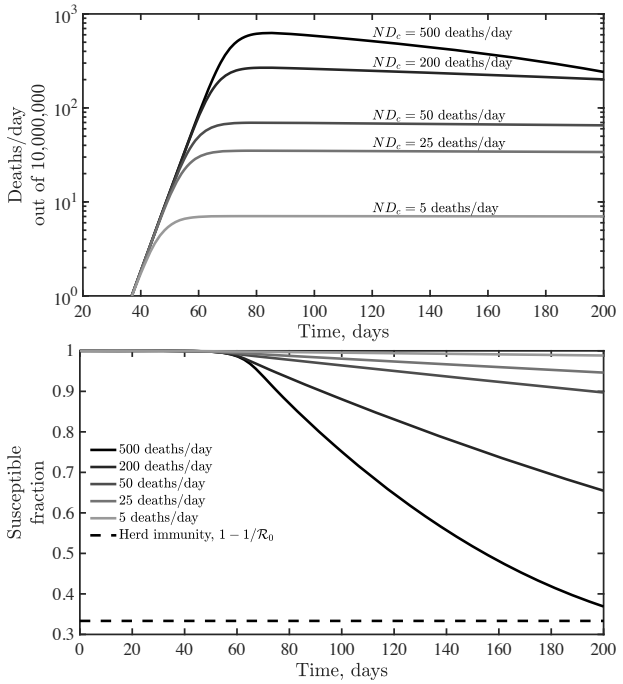

FIG. 2: Dynamics given variation in the critical fatality awareness level, D_c for awareness $k = 2$. Panels show deaths/day (top) and the susceptible fraction as a function of time (bottom), the latter compared to a herd immunity level when only $S = 1/\mathcal{R}_0$ remain. These simulations share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

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.

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 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, and are included in the `github` repository). We find that fatalities are sustained at near-constant levels (left) even as the population remains susceptible at levels far above herd immunity (right). We observed that as k increases, then fatalities may overshoot the plateau. This arises because individuals wait to initiate distancing 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 (whether due to reporting or due to delays arising because of disease etiology and treatment).

To explore the impacts of oscillations 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. Instead, consider the extended SEIR model:

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

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

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

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

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

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

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 also incorporates both short-term and long-term awareness:

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

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

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

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

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

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

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

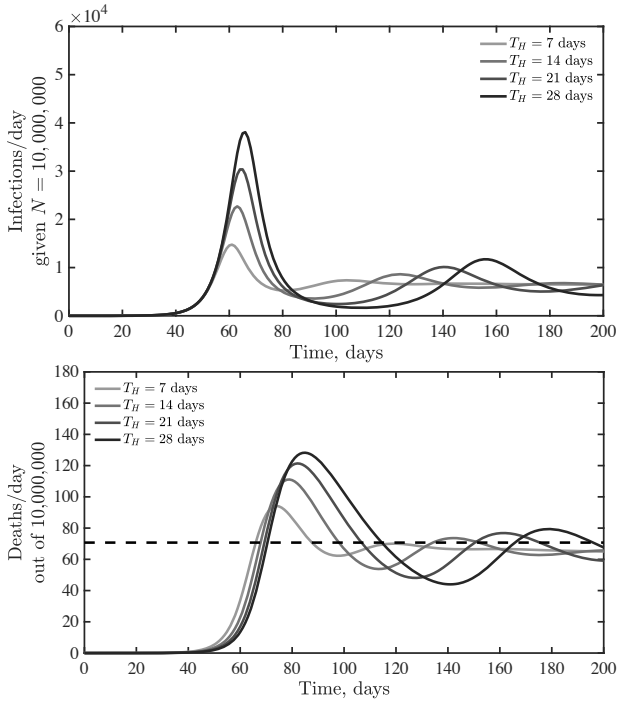

FIG. 3: Emergence of oscillatory dynamics in a death-driven awareness model of social distancing given lags between infection and fatality. Awareness is $k = 2$ and all other parameters as in Figure 2. The dashed lines for fatalities expected quasi-stationary value $\delta^{(q)}$.

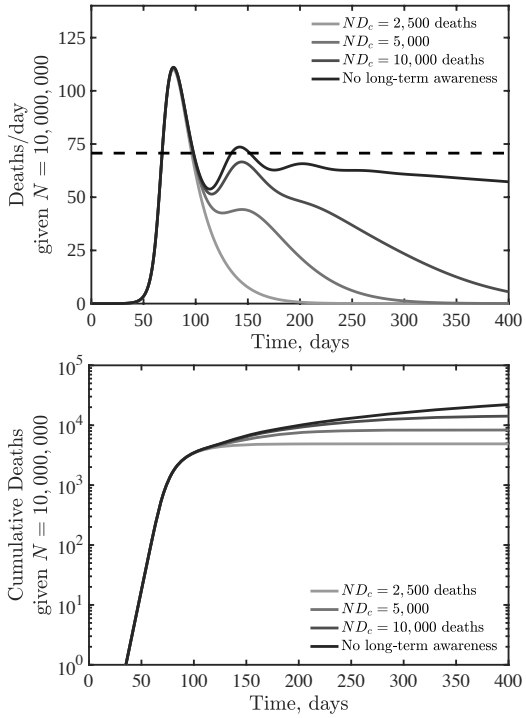

FIG. 4: SEIR dynamics with short- and long-term awareness. Model parameters are $\beta = 0.5$/day, $\mu = 1/2$/day, $\gamma = 1/6$/day, $T_H = 14$ days, $f_D = 0.01$, $N = 10^7$, $k = 2$, $N\delta_c = 50$ /day (short-term awareness), with varying ND_c (long-term awareness) as shown in the legend. The 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 plateaus, shoulders, and lag-driven oscillatory dynamics in epidemiological dynamics. Sustained levels of fatalities occur even as the majority of the population remains susceptible. Hence, if individuals tire of or 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. The impacts of awareness-driven endogenous changes in $\mathcal{R}_{eff}$ is often absent in models that form the basis for public policy and strategic planning. More optimistically, we hope that our findings highlight the value of 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 COVID19 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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