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

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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. 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 (and related efforts are ongoing at both national [**?**] and state levels [**?**]). 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 such that deaths go up, reach a peak, and then go back down. This trajectory is symmetric by construction, such that the time it takes to reach the peak must equal the time it takes to go back to normal. 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 peak (the defunct Farr's Law of Epidemics notwithstanding, a law posited in 1840 before the germ theory of disease and that, like the IHME model, has no mechanistic description of the underlying basis for transmission).

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. 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 $\mathcal{R}_0 > 1$. 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, $\mathcal{R}_{eff} = 1$. But, even then, there will still be new cases which then diminish over time, until the epidemic concludes. Yet, a single peak paradigm is only robust insofar as the disease has spread sufficiently in a population to reach and exceed 'herd immunity'. In this way, the IHME (and other parametric fitting models) make a dangerous and unsupported assumption: that second peaks or other long-term trajectories are not possible. Yet, the converse is true – as long as a population remains predominantly immunologically naieve, then the risk of further infection has not passed.

The Imperial College of London model – one of the earliest and most influential epidemic models – is an example of a 'conventional' epidemic model that compares projected epidemic dynamics between 'baseline' scenarios and the outcomes expected given alternative public health interventions. Despite the many benefits of public health efforts to reduce transmission: all off their models suggest a weakness – early efforts that diminish a peak can be followed by a second peak or wave of cases precisely because the disease did not spread early, leaving the majority of the population susceptible to infection. Yet, buying time for the introduction of new therapeutics and preserving health care resources suggest that the benefits of early intervention steps exceed those of a 'herd immunity' strategy. Hence, 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.

Yet, even the ICL model has a built-in assumption: that behavior changes because of externalities, like lockdowns, school closings, and so on, that reduce infection. For a disease that is already the documented cause of more than 50,000 deaths in the United States, we posit that individuals, acting out of a sense of self-preservation, may continue to modify their behavior. 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 with a simple assumption: individuals reduce interactions when near-term death rates are high and increase interactions when near-term death rates are low. As a result, we find that epidemic dynamics can be characterized by long, danger-

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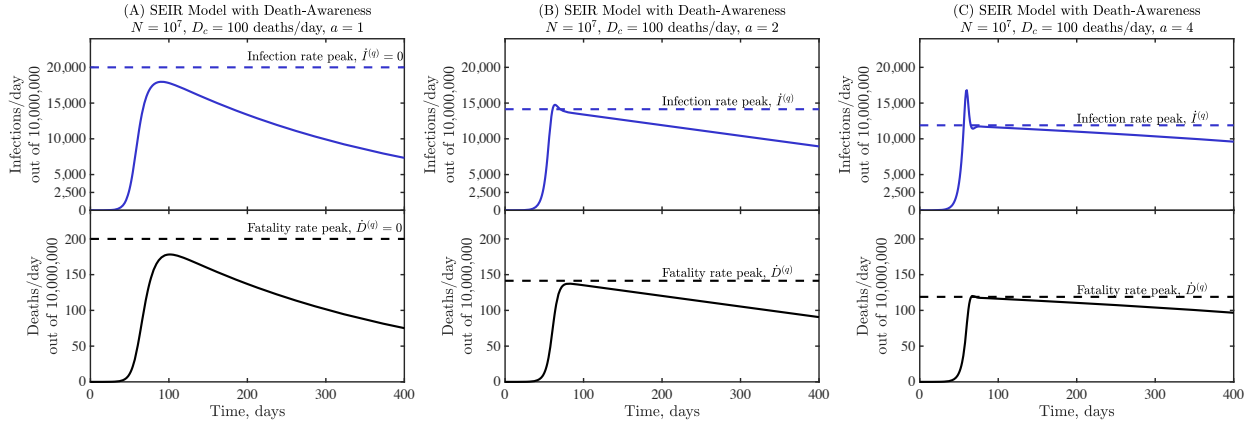

FIG. 1: Infections and deaths per day in a death-awareness based social distancing model. Awareness varies from $a = 1$, $a = 2$, to $a = 4$ in panels (A), (B), and (C).

ous plateaus – rather than peaks. We suggest that the generic nature of such plateaus should be considered in developing models that combine public health interventions that limit interactions with intrinsic, responses of individuals to the disease state – and that can combine to lead to dynamics that defy the near- and long-term predictions of parametric and even conventional epidemic models.

To begin, consider a SEIR like model:

$$\dot{S} = -\frac{\beta S I}{\left[1+\left(\dot{D}/D_c\right)^a\right]} \tag{1}$$

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

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

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

where the death rate is $\dot{D}(t) = f_D \gamma I$. Here, contacts are reduced based on current fatality rates relative to a critical level, D_c, such that when $\dot{D} \gg D_c$ then attack rates goes to 0 with a shape parameter a. Note that this model converges to the conventional SEIR model as $D_c \to \infty$. Typically, SEIR models have a single case peak, corresponding to the point where $\gamma I = \beta S I$ such that $S = 1/\mathcal{R}_0$ or when $1-\mathcal{R}_0^{-1}$ have been infected. However, when individuals decrease transmission in relationship to awareness of the aggregate severity of the disease, i.e., $\dot{D}$, then the system can 'peak' when levels of infected cases are far from herd immunity, specifically when

$$\gamma I = \frac{\beta S I}{\left[1+\left(\dot{D}/D_c\right)^a\right]}. \tag{5}$$

In the event that $D_c \ll 1$ 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 an awareness-based peak can occur early, i.e., $S(t) \approx 1$, consistent with a quasi-stationary equilibrium when

$$\dot{D}^{(q)} \approx D_c \left(\mathcal{R}_0 - 1\right)^{1/a} \tag{6}$$

and

$$\dot{I}^{(q)} \approx \frac{D_c}{f_D} \left(\mathcal{R}_0 - 1\right)^{1/a} \tag{7}$$

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

Figure 1 shows examples for $a = 1$, $a = 2$, and $a = 4$ given disease dynamics with $\beta = 0.5$, $\mu = 1/2$, $\gamma = 1/6$, $f_D = 0.01$, and $D_c = 10^{-5}$. In a population of $N = 10^7$, the critical fatality awareness level is equivalent to 100 fatalities per day, but given the duration of illness, with median generation time of 8 days, then one can consider this critical awareness level as an approximation of weekly averages. 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 $a \gg 1$, plateaus, such that deaths and constants appear constant, declining slowly because of the depletion of susceptibles. For increasing values of a, the dynamics may laso include significant overshoot in cases – given awareness of fatalities as a modulator of behavior. The shoulders and plateaus emerge because relaxation in awareness-based distancing leads to increases in deaths, and an increase in awareness.

Next, we extended this model to the case of COVID-19, including detailed information on symptomatic, asymptomatic, hospitalization, cases, age structure, and age-dependent risk. In doing so we consider a model of COVID-19 with a $\mathcal{R}_0 = 2.32$ and an incidence fatality rate of $IFR = 0.82\%$. Figure 2 shows that the insights from the SEIR model generalize to the case of COVID-19 dynamics, albeit with some differences. In particular, we find that deaths exceed D_c because of the lag between new infection and fatalities (with a mean of 20

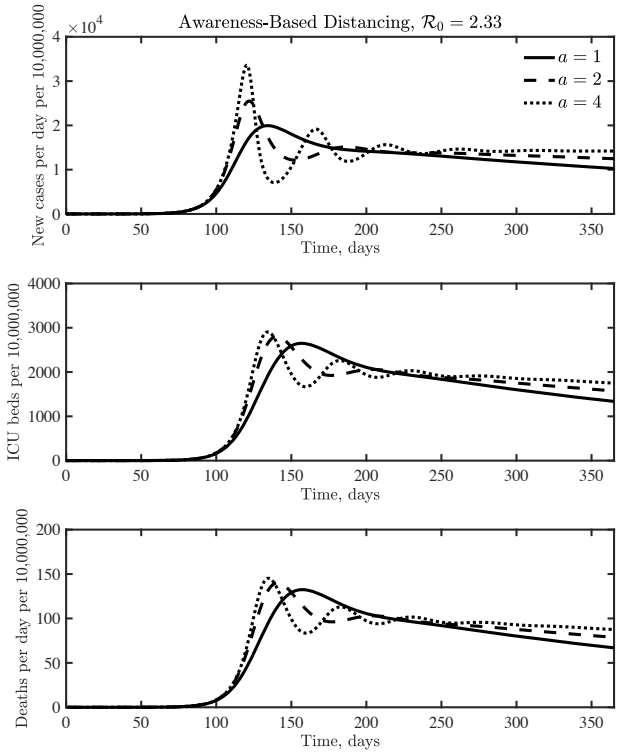

FIG. 2: COVID-19 dynamics given awareness-based social distancing model. Awareness varies from $a = 1$, $a = 2$, to $a = 4$. Epidemiological and age-specific parameters are from previous work (Weitz et al. shield immunity).

days in the current model). Further 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 of 20 days, 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 note that the scenarios studied, the bulk of the population remains susceptible throughout the dynamics. Hence, if individuals tire of social distancing policies, or begin to tolerate higher death rates, then cases can increase. We also note that long-term awareness would lead to a faster peak decline (see Eksin et al.). In reality, we expect that individual behavior is shaped by short- and long-term awareness of risks. As detailed here, if short-term awareness predominates, then COVID-19 dynamics may result in plateau- and shoulder-like behavior, including the possiblity of lag-driven oscillations. As a result, passing a 'peak' need not imply the rapid decline of risk. Instead, as cumulative data from outbreak epidemics have already shown, the asymmetric post-peak dynamics of COVID-19 may be an emergent property of awareness-driven epidemiological dynamics.