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 symmetric peak, the defunct Farr's Law of Epidemics notwithstanding.

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 $\mathcal{R}_0 > 1$ (the basic reproduction number denotes the average number of new infections caused by a single, typical individual in an otherwise susceptible population). 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 reprooduction 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. 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 naive, 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:

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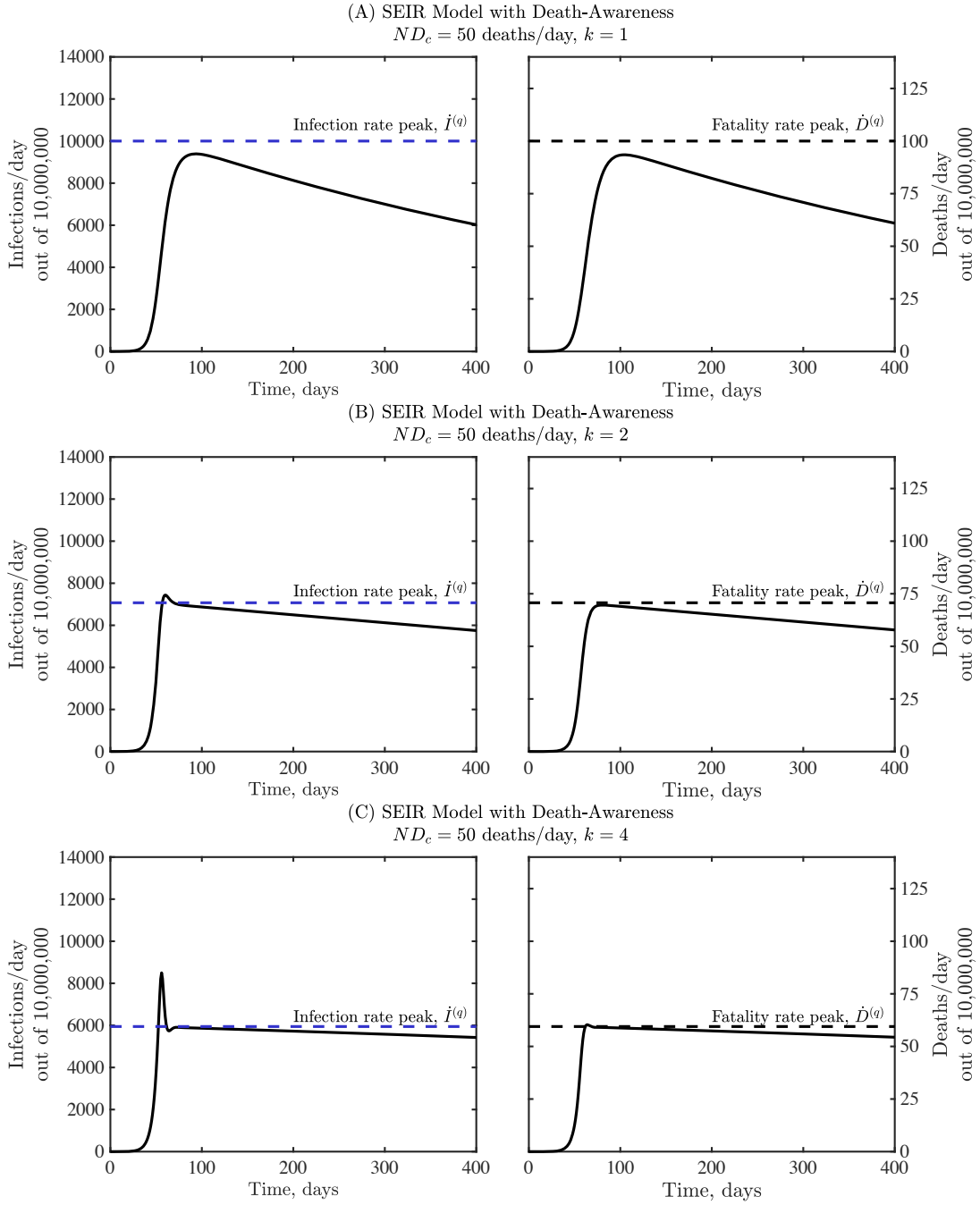

FIG. 1: Infections and deaths per day in a death-awareness based social distancing model. Awareness varies from $k = 1$, $k = 2$, to $k = 4$ in panels (A), (B), and (C).

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, dangerous plateaus – rather than peaks. We suggest that the generic nature of such plateaus should be considered in developing models that combine public health interven-

tions 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

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

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

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

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

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

where S, E, I, R, and D denote the proportions of susecptible, exposed, infectious, recovered, and deaths, respectively. The awareness-based distancing is controlled by the half-saturation constant ($D_c > 0$) and the sharpness of change in the force of infection ($k \geq 1$). As a result of the proportionalaity betweeen $\dot{D}$ and I, the present model is a variant of a recently proposed local awareness based distancing model [**?**]. Note that the present model converges to the conventional SEIR model as $D_c \to \infty$.

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 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

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

In the event that $D_c/\gamma \ll 1$ 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

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

and

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

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, $f_D = 0.01$, and $D_c = 10^{-5}$ /day. In a population of $N = 10^7$, the critical fatality awareness level is equivalent to a rate 100 fatalities per day, but given the duration of illness, with mean generation interval of 8 days, then one can consider this critical awareness level as an approximation of daily fatality rates over week-long periods. 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 $\dot{D} < D_c$ and more sensitive to fatality rates where $\dot{D} > D_c$. The shoulders and plateaus emerge because relaxation in awareness-based distancing leads to increases in deaths, and an increase in awareness.

These results suggest a generic outcome: first fatalities will grow exponential before plataeuing near to the fatality awareness level D_c. In the event that γD_c is sufficiently high then susceptible depletion will lead to the decline of cases and fatalities. Figure 2 shows $f_D = 0.01$, and D_c values over a range equivalent to 5 to 500 deaths/day given a population of 10^7. 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 plateu. 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:

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

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

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

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

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

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

where $T_H = 1/\gamma_H$ defines the average time in a hospital stay before a fatality. The earlier analysis of the

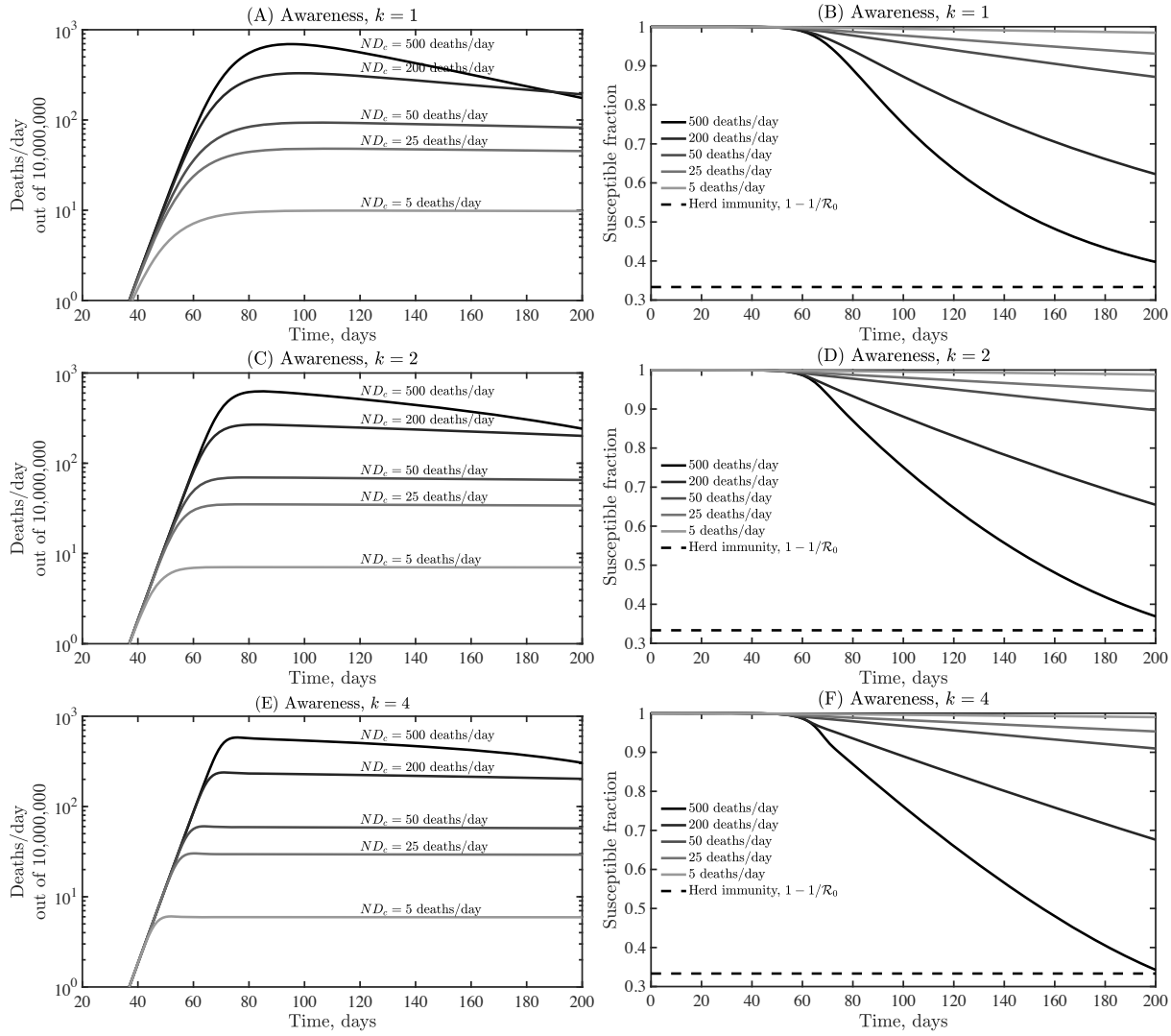

FIG. 2: Dynamics given variation in the critical fatality awareness level, D_c and k. Panels (A), (C), (E) show deaths/day as a function of time. Panels (B), (D), (F) show the fraction of susceptibles as a function of time, with respect to a herd immunity level when only $S = 1/\mathcal{R}_0$ remain. These simulations share the epidemiological parameters $\beta = 0.5$ /day, $\mu = 1/2$ /day, $\gamma = 1/6$/day, and $f_D = 0.01$.

quasi-stationary equilibrium in fatalities holds; hence we anticipate that dynamics shold converge to $\dot{D} = \dot{D}^{(q)}$ 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). 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.

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. We note that these oscillations could be amplified in stochastic models. As a result, passing a 'peak' need not imply the rapid decline of risk. This simple message has gone unheeded in models that now form the basis for public policy and strategic planning, e.g., like the IHME model. The IHME model predicts peaks and rapid declines,

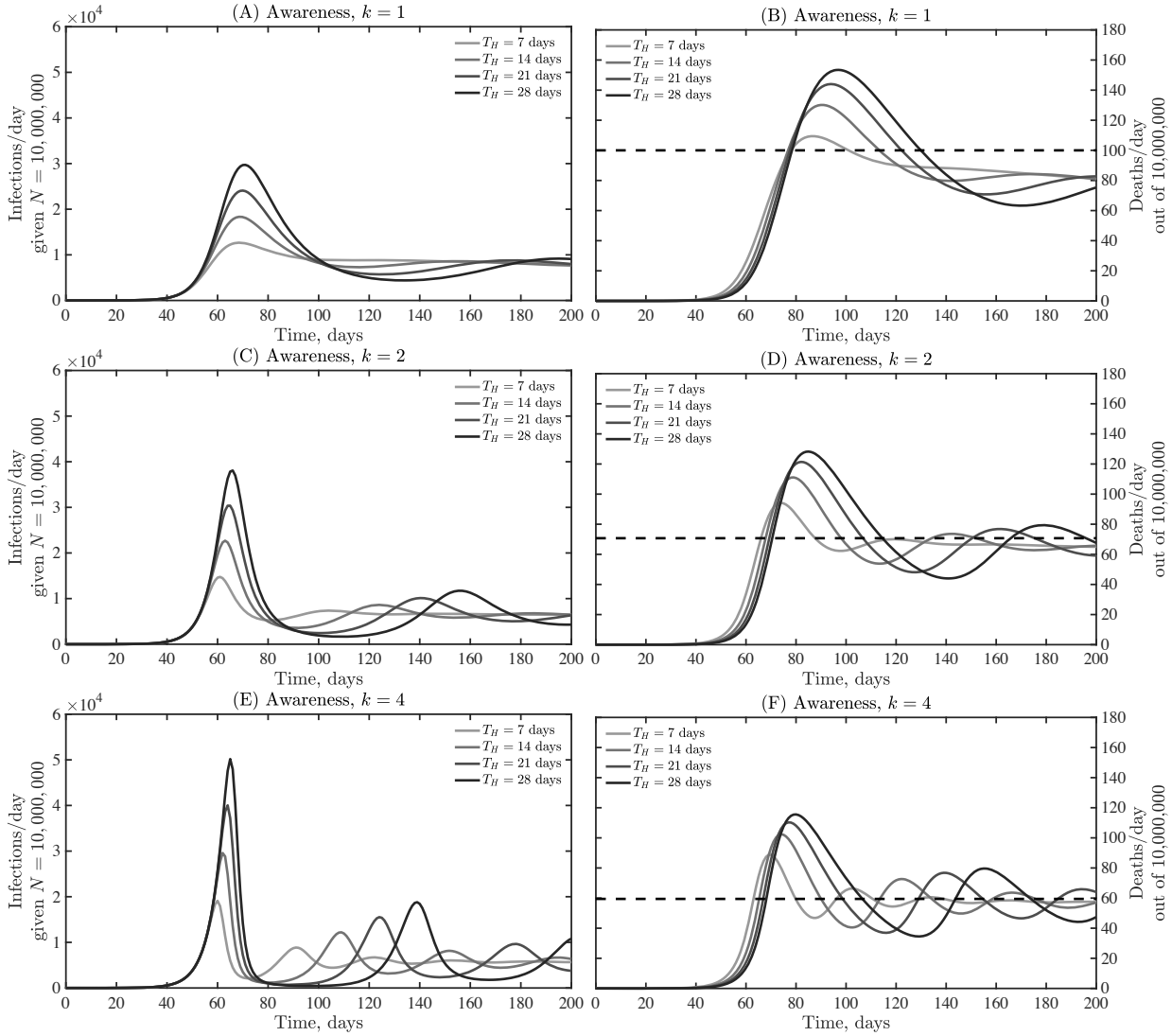

FIG. 3: Emergence of oscillatory dynamics in a death-driven awareness model of social distancing given lags between infection and fatality. Awareness varies from $k = 1$, $k = 2$, to $k = 4$. The dashed lines in panels (B), (D), and (F), denote the expected quasi-stationary value $\dot{D}^{(q)}$.

building upon the curve-fitting approach of Farr's law posited in 1840 before the germ theory of disease. Thirty years ago reserachers revived Farr's Law, in part, to predict the scope of the HIV epidemic, e.g., in 1990 the law was used to extrapolate to a peak HIV epidemic size of 200,000 cases by the mid-1990s[**?**]; in contrast there were nearly 2 million new AIDS cases reported last year alone. Relying on curve fits to early cases to forecast early declines in the absence of population-wide immunity can be exponentially wrong. Instead 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.