ON THE NATURE OF THE GOMPERTZ IN RELATION TO THE EPIDEMIC IN 2019-2021

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ABSTRACT

Recently, a number of studies have shown that cumulative mortality followed a Gompertz curve in the initial Coronavirus epidemic period, March-April 2020. We show that the Gompertz is incompatible with the traditional communicable disease spreading hypothesis, and propose a new theory which better explain the nature of the mortality characteristics based on an environmental stressor. We show that for the Gompertz curve to emerge, the stressor has to act on everyone simultaneously, rejecting the possibility of a disease propagation stage. In the course of our presentation, we show that the population acts like a coherent organism under growth/depletion. Finally, we connect the Susceptible-Infected-Recovered (SIR) model with our new theory and show that the SIR model is compatible with the Gompertz only when all nodes in the transmission network communicate with infinite speed.

Keywords Gompertz · Coherence · Covid · Coronavirus · Network Analysis · Stochastic

Traditional communicable disease spreading theory assumes a pathogen which infects the population through a network of transmission. Following this line of reasoning it can be shown that in the early stages of an epidemic, growth follows an initial exponential growth under a constant ratio of the infection rate, β and recovery rate, α. This results in a logistic-like growth curve overall, for which analytic solutions have recently emerged [10, 13, 20, 11, 3]. However, instead of showing logistic-like growth, observed cumulative mortality exhibits a Gompertzian growth [9] where the log-transformed cumulative mortality, or log-mortality for short, is exponentially *decreasing* in time,

$$\frac{d}{dt}(\ln Y) = -b \ln Y, \tag{1}$$

with b interpreted as growth rate, whose solution is given as

$$Y(t) = Y_\infty \left(\frac{Y(t)}{Y_\infty} \right)^{\exp(-bt)} \tag{2}$$

with $Y_\infty = Y(t \to \infty)$ (see Fig. 1 for a conceptual image).

This phenomenon is also recorded by [16, 19, 5], especially clearly in Fig. 2 of Levitt et al. [14]. What is causing such a discrepancy between reality and the current theory of communicable diseases [4]?

One could approach this conundrum from parametrization of the Susceptible-Infected-Recovered (SIR) model under a time-dependent infection/recovery ratio, $\phi = \phi(t)$. How would $\phi(t)$ have to behave? Start by recalling the SIR model for a total population of N people split between the three states susceptible, $S(t)$, recovered, $R(t)$ and infected, $I(t)$,

$$\frac{dS}{dt} = -\beta \frac{IS}{N} \tag{3}$$

$$\frac{dI}{dt} = -\beta \frac{IS}{N} - \alpha I. \tag{4}$$

$$\frac{dR}{dt} = \alpha I. \tag{5}$$

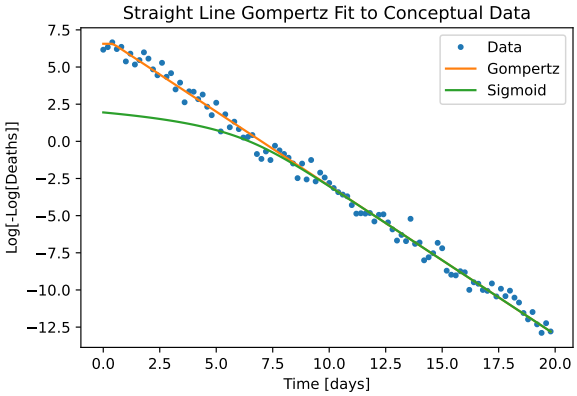

Figure 1: A conceptual display of the discrepancy seen between the logistic growth and the observations, superimposed by a Gompertz curve. Data is transformed to the straight line domain of the Gompertz curve for visual clarity, $f(Y) = \ln \ln 1/y$.

Following Rypdal and Rypdal [19], we linearize the SIR model by assuming both the number of infected, I(t), and cumulative infected, Y(t), is much less than the total population, $N \gg Y \geq I$,

$$\frac{dY}{dt} = \beta I \tag{6}$$

$$\frac{dI}{dt} = (\beta - \alpha) I. \tag{7}$$

Note that the number of recovered, $R(t)$, is under this linearization decoupled from the other variables. We also assume the number of diseased is proportional to the number of infected, offset by a time lag, which allows us to use $Y(t)$ for both without loss of generality.

Due to this linearlization, the infection/recovery ratio, ϕ, will have to change in time to accommodate for the boundary conditions. And since $I = I(Y)$, we combine (6) via an instantaneous relative growth rate, $\gamma(t) = dY(t)/(Y dt)$, parameterized by a scaling factor, ν, representing the shape of the growth,

$$\begin{aligned} \frac{dY}{dt} &= \gamma(t) Y(t) \\ \gamma(t) &= \frac{\gamma_\infty}{\nu} \left[1 - \left(\frac{Y}{Y_\infty} \right)^\nu \right], \end{aligned} \tag{8}$$

where $\gamma_\infty = \gamma(t \to \infty)$, and is the commonly used Richard's growth curve [18], also called ν-logistic growth. Note that at $\nu = 1$, the traditional logistic growth curve is obtained, where the ratio of infection and recovery rate is constant [13]. At $\nu \to \infty$ we recover the exponential (Malthusian) explosion.

The observed Gompertzian mortality curves are realized in the limit $\nu \to 0$ [17], with the relative growth rate,

$$\gamma(t) = \lim_{\nu \to 0} \frac{\gamma_\infty}{\nu} \left[1 - \left(\frac{Y}{Y_\infty} \right)^\nu \right] = \gamma_\infty \ln \frac{Y_\infty}{Y} \tag{9}$$

At this limit the growth rate approaches infinity as $Y \to 0$, which seems odd under the hypothesis that the pathogen has just started spreading. The Gompertzian limit also implies a decreasing relative growth rate from the very onset, which under the SIR model seems unlikely given the large pool of susceptible people in the beginning. One would rather expect a near-constant relative growth rate in the early times. Rypdal and Rypdal [19] suggest that the decreasing relative growth rate is caused by social and political mitigating efforts, but these hardly justify such coherent and consistent mortality characteristics across countries and even species (as shown by Gilbert and Haugen [8]). Perhaps a more likely scenario is the selective infection of central nodes in the transmission network causing relative growth to immediately decrease. On the other hand, an infection of peripheral nodes should cause immediate exponential growth. Either case seems likely, and subject to initial conditions. Herrmann and Schwartz [12] studied a networked SIR model on a variety of networks, but did not elaborate on a possible fit to a Gompertz curve. We will return to this question below.

An alternative line of reasoning is that the biosphere was perturbed by an external stressor, initiating a stress response to eventually bring mortality rates back to stability. This could explain the immediate dampening of mortality growth we see. Inspired by De Lauro et al. [6], the stressor can be modeled as a multiplicative stochastic dampening term at the microscopic level along with the countering force of the immune system. As with all multiplicative processes, it is amenable to log-transform normalized mortality with variables $F(t) = Y(t)/Y_\infty$ and $Z = \ln F$, from which a natural perturbation model emerges,

$$dZ(t) = -bZ(t)dt + \sqrt{\sigma}dW(t), \tag{10}$$

where $W(t)$ is a delta-correlated Wiener process[1]. Notice that this is simply a continuous version of an AR(1) model. The diffusion coefficient, σ, represents the strength of the perturbation, which we directly see if we recast this equation in terms of mortality,

$$dF(t) = \left\{ \frac{\sigma}{2} F(t) - bF(t) \ln \left(F(t)/K \right) \right\} dt + \sqrt{\sigma} Y(t) dW(t), \tag{11}$$

where $K = \exp\left(-\frac{\sigma}{2b}\right)$. The first term on the right hand side represents the growth due to the stressor, while the second represents the stress response. Note that mortality is treated here as dimensionless where the microscopic treatment would interpret F as cumulative probability of mortality, and the macroscopic equivalent would be the cumulative mortality.

To go from the microscopic to the macroscopic domain, we take averages of (10), and use the property that the average of log-quantities is the logarithm of the median quantity, to obtain the familiar Gompertz differential equation in (1). We now see that by comparing (8), (9) and (11) that

$$\sigma = 2\gamma_\infty \ln\left(Y_\infty\right), \tag{12}$$

showing the relationship between the diffusion coefficient and the final conditions.

Thus, a more direct interpretation of the observations is that mortality was caused by a planetary perturbation, modeled as a random process, to which organisms gradually develop resistance at a geometric rate [2, 15]. Here, a geometric rate is an exponential rate in the log-transformed domain, which is the natural transformation for many processes in nature [1].

Unifying the SIR model and the Gompertz Curve

The remarkable observation that the log-transformed domain is the natural one merits closer study. First, juxtapose the Logistic model with the Gompertz model,

$$\begin{aligned} \frac{d}{dt} F(t) &= bF(1-F) && \text{Logistic} \\ \frac{d}{dt} \ln\left(F(t)\right) &= -b \ln\left(F(t)\right) && \text{Gompertz} \end{aligned} \tag{13}$$

In the Logistic model, we recognize the right hand size as the transmission term in an SIR model, but also as a linear interaction term between the two macroscopic states. Many studies do not consider microscopic effects at all, and the solution is obtained simply by solving the macroscopic (set of ODEs) [22]. Consequently, a Gompertz curve will not emerge. Similar conclusions are obtained by modifying the macroscopic rules [3].

Because the macroscopic Logistic ODE solution never invokes the microscopic world, an imaginary microscopic solution would be modeled by splitting the system into N units, $F(t) \to f_1(t), f_2(t)$, etc, from which overall mortality would be obtained by summing the individual units,

$$Y(t) = Y_\infty \frac{1}{N} \sum_i^N f_i(t). \tag{14}$$

This procedure implies no (microscopic) correlation between the variables, and instead all dynamics are governed by independent random variables connected by a transmission rate. Intuitively, this correlation can be obtained by taking second derivatives for pairs of variables,

$$Cor(f_i, f_j) \propto \frac{\partial^2 Y}{\partial f_i \partial f_j} = 0, \tag{15}$$

[1]This perturbation model is simply an Ornstein–Uhlenbeck process

Note that one could add network interaction by letting the coefficient b be a vector instead of a scalar, necessitating a corresponding matrix version of (13), in which case we would obtain a zeroth order correlation governed by the networks transmission rate and adjacency matrix, $\{a_{i,j}\} = \mathbf{A}$, a binary matrix with ones where the i^{th} and j^{th} nodes are connected, and zeroes otherwise, viz.

$$Cor(f_i, f_j) \propto \frac{\partial^2 Y}{\partial f_i \partial f_j} = \beta a_{i,j} \tag{16}$$

Still, no Gompertz curve will emerge at the onset of the growth process. Estrada and Bartesaghi [7] provide illuminating analysis on this topic.

In contrast, as shown in (10), the Gompertz model is implied by a multiplicative stochastic perturbation and thus correlated mortality growth, viz.

$$Y(t) = Y_\infty \exp\left[\frac{1}{N}\sum_i^N Z_i(t)\right] = Y_\infty \left[\prod_i^N f_i(t)\right]^{1/N}. \tag{17}$$

Under this model, correlation is present at all orders in the original domain[2].

The remarkable observation that we see the Gompertz curve emerge at the macroscopic level implies that the system is indeed correlated, or coherent, presumably as a result of the simultaneous exposure to the same underlying stressor, but also due to the implied log-normal nature of the microscopic distributions [23]. We can now further appreciate Richard's parametrization as a transition from non-collaborative to collaborative growth as $\nu \to 0$. This feature is also obtained by Petroni et al. [17] by interpreting the $\nu-$logistic growth rate in (8) as as non-linear resource availability dependent on the overall magnitude, $Y(t)$.

But it is possible to reconcile the SIR model with the Gompertz curve from yet another argument. Since we are in a simple two state system, infected and susceptible, we can augment the interaction term to higher orders. We also vectorize the SIR model and include network effects through our previously defined adjacency matrix, $\mathbf{A}$. Let $\mathbf{F} = [f_1, f_2, ...] \in \mathbb{R}^N$ be the vector of probability of infection, and similarly $\mathbf{S} = 1_n - \mathbf{F}$ be the probability of being susceptible. Then, augment the interaction term in an SIR model with higher order terms,

$$\frac{d\mathbf{F}}{dt} = b \cdot \text{diag}(\mathbf{F})(1 + \mathbf{AS} + \mathbf{A}^2\mathbf{S}^2 + ...) - b\text{diag}(\mathbf{F}), \tag{18}$$

and the Gompertz differential equation follows immediately by using the Taylor series $1 + x + x^2 + ... = -\ln(1-x)$,

$$\frac{d\ln\mathbf{F}}{dt} = -b\ln(1 - \mathbf{AS}) - b \geq -b\ln(\mathbf{F}) - b, \tag{19}$$

where the upper bound applies for a graph in which all nodes are connected and have at least one neighbor. If all nodes are independent and identically distributed, have exactly one neighbor, and have a path to all other nodes, equality follows. Finally, for $1 \gg f_i, \forall i$, i.e. in the beginning of growth, the constant on the right hand side can be omitted.

Thus, we have shown that Gompertz growth follows from infinite interactions between the susceptible and infected states. The powers of the adjacency matrix reflect the number of nodes one can travel in one time step, dt. As dt goes to zero, the pathogen travels at infinite speed throughout the whole population. In this vein, Richard's parameter ν can be related to the number of higher order interactions in the network.

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