GOMPERTZ GROWTH AS INFINITELY CORRELATED NON-LOCAL GROWTH

A PREPRINT

Matz A. Haugen[*]
Independent Reseacher, Oslo
matzhaugen@gmail.com

Dorothea Gilbert
Independent Reseacher, Oslo

March 23, 2024

ABSTRACT

Logistic and Gompertz growth has traditionally been connected by augmenting the logistic model with an extra parameter to obtain what is known as θ-logistic growth or the Richards model for growth. In spite of this bridge, the biological foundation of Gompertz growth is only vaguely understood. To add to this biological foundation, we show that there is another way to connect the two growth modes by adding higher order terms in the growth equation, and present both a macroscopic and microscopic representation of growth where the latter involves a network of entities. The microscopic representation elucidates the source of Gompertz growth as the potential abundance and not the realized abundance, and an example of such a network is presented. For finite higher order terms we show that the growth equation can be represented by Gauss' Hypergeometric function, and only for infinitely many terms does the abundance lend itself conveniently to a log-transformation wherein the log-abundance growth rate decreases linearly, the trademark of Gompertz growth. We thus equate Gompertz growth with correlated microscopic entities that exhibit long-range or non-local interaction. This conclusion adds to the interpretation of biological systems that undergo Gompertz growth where long-range interaction could be interpreted as the source of growth being external to the individual entities, like a field that stimulates growth simultaneously across all entities. This field could be interpreted as an environmental stressor like air-pollution, radiation, etc, or an environmental stimulant like an electromagnetic field. Our new discovery allows for a unification of well-known growth models as follows: time independent and uncorrelated growth yields the exponential growth function, time dependent and uncorrelated growth yields Gaussian growth, time independent and pairwise correlated growth yields the logistic function and time independent and infinitely correlated growth yields the Gompertz function.

Keywords Gompertz · Coherence · Network Analysis · Self-organization

Highlights

- Logistic and Gompertz growth is connected with a novel framework based on microscopic principles.
- Gompertz growth is connected with ubiquitous growth-activating field justified by physical and mathematical principles.
- Growth processes is put in a larger contextual framework based on varying dependence in spacetime, including the Exponential, Gaussian and Poisson distributions.

1 Introduction

In 1825, Gompertz published a paper on the nature of mortality where he discovered that from adulthood and onward "the power to avoid destruction" decreases exponentially with age. Equivalently, mortality rates increase in geometric progression for arithmetic increases in age, an observation now commonly referred to as Gompertz' law of mortality [1].

Makeham (1889) later provided an interpretation to Gompertz' law where the recuperative force of a human, or "vital force", becomes less efficient with time [2]. Gompertz' law has later been known as the Gompertz model or Gompertz growth.

1.1 Gompertz' law in existing work

This simple law has been shown to generalize to situations where mortality rates are substituted by many types of abundance variables, like the growth of animals, plants, bacteria, and cancer tumors [3–13], exposing Gompertz growth as nearly ubiquitous in biological growth processes. The idea that the Gompertz' law can be applied to other biological systems was first proposed by Weymouth (1931) [3, 4] and Winsor (1932) [14]. Gompertz' law has also been shown to apply to societal phenomena like growth in railway traffic, and the demand for goods and services, and sales of tobacco [15–17].

Alongside the many contexts wherein Gompertz' law has been observed, an almost equal number of efforts have been made to interpret and derive this law from various phenomenological principles [18]. This effort has often been accompanied by a phenomenological comparison of another popular growth model called logistic growth, or Verhulst growth, where "the power to avoid destruction" decreases linearly with time, as opposed to decreasing exponentially with time as seen with the Gompertz model. In a study comparing the Gompertz and the logistic growth models, Petroni et al. (2020) [19] show that Gompertz growth can be interpreted as *maximally coherent* growth based on long-range interaction or correlation, while logistic growth is minimally coherent with non-collaborating elementary entities. With this interpretation, one is lead to postulate that the Gompertz model is a result of an external stimulus with perceived long-range interactions while the logistic model is a result of an internal stimulus with local interaction. As such, the logistic model has been popular both for communicable disease models [20–23] and and for predator-prey theory [24], where disease or injury spreads through pairwise interactions.

A number of studies in the biological context have argued that Gompertz growth could be activated from an external factor which is ubiquitously and instantaneously introduced to the system, e.g. as a toxic agent or an environmental stressor to which the system gradually adapts [25–27]. In the original case of mortality rates as a function of age, this interpretation would imply that time itself is the environmental ubiquitous stressor. Other environmental stressors like chronic radiation exposure have been observed to shift the Gompertz curve, an observation also theoretically developed by Sacher (1956) ([28]). Sacher modeled the toxic stressor as a stochastic perturbation to the physiologic state of a population wherein death of an entity would occur beyond some perturbation threshold. The interpretation of Gompertz growth as the result of a ubiquitous stochastic perturbation was recently revived by De Lauro et al. (2014) [29] who arrived at the same result without alluding to a threshold value beyond which death was inevitable, but rather by letting the stochastic perturbation be the source of growth.

Another framework used to show how the Gompertz model can result from an external or ubiquitous stressor is chemical reaction theory. In reaction theory, a set of reactants combine to produce products, possibly in the presence of a catalyst. Morkov (2019) [30] showed that the Gompertz model can be interpreted as growth under a catalyst whose effect diminishes at a rate independent of the growth rate of the reactant. In other words, the catalyst can be considered as an external and ubiquitous stimulant or stressor to the reactants, perfectly in line with the ubiquitous stochastic perturbation model from Sacher and De Lauro et al. On the other hand, if the logistic growth equation is recast into the reaction network equivalent, the catalyst is diminished by the growth of the reactant, which suggests that the source of growth is internal or at least dependent on the internal state. So also in reaction theory the Gompertz model could be seen as a result of a ubiquitous field while the logistic model could be seen as a result of internal interactions of the reactants.

It is worth noting that the notion that Gompertz growth is maximally coherent and non-local has also been derived from quantum mechanical principles [31]. Molski and Konarski (2003) showed that the Gompertz equation is the solution of a form of the time-independent Schrodinger equation. From this result, they derive the quantum coherent states which are non-local in space while moving along a classical time trajectory. This adds to our exhibits that the Gompertz model can be interpreted as non-local in origin. Coherent quantum states was originally formulated in the context of electromagnetic fields by Glauber [32] (for which he received the Nobel Prize). Some biological macroscopic growth phenomena are also due to such fields. For example, the role of internal electrical fields in regenerative growth is well established and is based on negative electrical potentials that act on sensitive cells [33].

In fact, it has been shown that cancer malignancies are mostly *negative* in electric polarity with respect to the rest of the body, while nonmalignant (or benign) tumor pathology is mostly *positive* in polarity [34]. Gompertz growth has also been widely observed in cancerous cells [35, 5]. One is then led to the interpretation that certain biological growth processes that exhibit Gompertz growth are coupled with an electromagnetic field of specific character. Consequently and unsurprisingly, the artificial disruption of such a field has been found to disrupt the mitotic cell cycle [36–39].

On the topic of disease modelling, the Gompertz model has been shown by Wang et al. [40] to be excluded from a subset of models in the communicable disease paradigm. They used the common Susceptible-Infected-Recovered (SIR) model by Kermack and McKendrick [41] augmented by the Richards parameter and showed that the allowed parameter range with the communicable disease assumption does not cover the parameter value taken in the Gompertz scenario. If one incorporates a social proximity network on which the disease spreads, Zonta and Levitt [42] observed through computer simulations that a near-Gompertz growth can occur but only for scale-free networks, a type of network that has been shown to be rare in human social networks [43–45]. We will show that one can obtain a true Gompertz curve from a ubiquitous stressor scenario based on microscopic interactions. While many studies show the SIR model (and its extensions) as the fitted model in epidemics [46–51], some recent studies have successfully fitted a Gompertz model [52–56].

1.2 Main Results and Roadmap

In this paper, our primary aim is to investigate the source of growth in self-organized processes. In particular, we posit that the source of growth can be elucidated by considering interactions in the microscopic domain which can be aggregated to recover the traditional macroscopic growth equations. Second, we will elaborate on the idea that the logistic model results from local interactions while the Gompertz model results from a field or equivalently from an infinite set of higher order interactions between the microscopic entities involved in the growth. We will devise a slightly different parametrization than the Richards model, the generalized Gompertz-Logistic equation which is commonly used to compare the two models (e.g. by Petroni et al. [19], Tjørve and Tjørve [57], and Wang et al. [40]). In particular, instead of considering a decimal number as the unifying exponent we will appeal to integer exponents with a number of terms that tend towards infinity as we approach the Gompertz model. As we shall see, this is rooted in the microscopic interpretation of the growth system, where each subgroup of N elementary entities interacts with all other equally sized subgroups each composed of mutually exclusive elementary entities. If these subgroups are present at all orders of cardinality, the Gompertz model emerges, while only pairwise interactions yield the logistic model. As seen in the aforementioned references, this is in line both with experimental and theoretical results. In presenting this argument, we present a novel bridge between the logistic and the Gompertz model which offers both a mathematical and biological foundation to the source of growth. Lastly, we will present a unifying framework in the growth context for the common functions Gaussian, Exponential, Logistic, Gompertz and Poisson.

Our paper is structured as follows: we will first present a new macroscopic foundation of the Gompertz-Logistic spectrum which offers an alternative to the traditional Richards model. Then, we present a microscopic foundation which acts as the building blocks of the macroscopic phenomena. This is where we show that the Gompertz model results from an infinite set of higher order interactions between the elementary entities involved in the growth, which could alternatively be seen as a ubiquitous field or in the biological context: an environmental stressor. We proceed with a discussion that summarizes our findings in the context of the other well-known growth modalities involving the Gaussian and the Laplacian model and then conclude.

2 Macroscopic growth

First consider a system that grows according to logistic growth. If we let the variable $X(t)$ denote the abundance of a quantity in a normalized system where its maximum size is 1, the equation that determines its size as a function of time is

$$\frac{1}{X}\frac{d}{dt}X(t) = \beta(1 - X(t)), \tag{1}$$

where $\beta > 0$ is a growth parameter. Here, the right hand side is commonly interpreted as representing "resources available for growth" which decrease linearly with respect to the relative abundance's growth rate on the left hand side. One way to generalize logistic growth is to introduce a parameter that exponentiates the resource availability in the following way,

$$\frac{1}{X}\frac{d}{dt}X(t) = \frac{\beta}{\theta}(1 - X^{\theta}(t)), \tag{2}$$

also called θ-logistic growth or Richards model [58]. Petroni et al. (2020) argued that this exponentiation can be interpreted as non-linear interaction effects where there is a level of cooperation in the microscopic domain. They showed that in the limit where the system becomes *maximally cooperative* or *coherent*, $\theta \to 0$ and the θ-logistic equation reduces in this limit to the Gompertz growth model,

$$\frac{1}{X}\frac{d}{dt}X(t) = -\beta \ln X(t). \tag{3}$$

In this limit, the relative growth goes to infinity as $\lim_{t\to 0}$, which could be seen as a verification of the maximally coherent characteristic of the system. One is reminded of a system that is perturbed and left to reach a new equilibrium.

Petroni's definition of maximal coherence in the context of the Gompertz equation was shown to rigorously correspond to the definition of coherence used in quantum mechanical systems [31] attributed to Glauber (1963) [32].

Another way to generalize the logistic equation is to augment the resource terms to higher orders as follows,

$$\frac{1}{X}\frac{d}{dt}X(t) = \beta\left[(1-X(t)) + \frac{1}{2}(1-X(t))^2 + ...\frac{1}{K}(1-X(t))^K\right], \tag{4}$$

where fractional prefactors to each term will be justified in the sequel. After some algebra (Appendix A) and letting $\beta = 1$ for simplicity, this augmented logistic equation reduces to a closed form expression involving Gauss' Hypergeometric function, ${}_1F_2$, viz.

$$\frac{d}{dt}\ln X(t) = -\frac{{}_1F_2(1, K+1, K+2; 1-X(t))}{K+1}(1-X(t))^{K+1} - \ln X(t). \tag{5}$$

In the context of higher order terms, the equivalent of $\theta \to 0$ in θ-logistic growth is that the higher order terms go to infinity, i.e. $K \to \infty$, which again reduces to the Gompertz equation by virtue of the Hypergeometric term going to zero.

The emergence of Gompertz growth from higher order logistic growth can be seen as another way to encode non-linear effects that were seen as coherence in accordance with Petroni and Molski. In practice, the augmented logistic model has a slower growth rate decay than the familiar θ-logistic growth (Figure 1). If we interpret deceleration of relative growth as degree of coherence, the augmented logistic growth would be more coherent than the θ-logistic model for a given starting value.

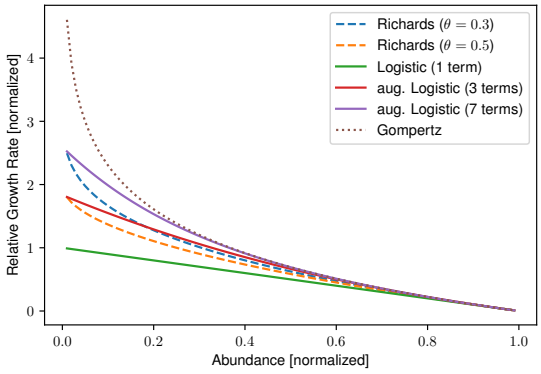

Figure 1: Comparison relative growth rate in the Richards θ-logistic model (Eq. 2), Gompertz model (Eq. 3), and the augmented logistic model (Eq. 5), all as a function of the abundance variable. Both dependent and independent variables are normalized so that final abundance (or size) is set to unity. The Richards θ-parameter is annotated in brackets in the legend, in addition to the number of terms used in the augmented logistic model.

In spite of the connection of the Gompertz model with higher order terms in an augmented logistic model, it is not clear whether the source of growth is external or internal to the system. The right hand side "resource" term is not specific enough so as to specify the location of the source. One could argue that the since the Gompertz model has asymptotically infinite initial growth rate, the system could be seen as exposed to a ubiquitous signal (or catalyst in Markov's language) that initiates the growth and subsequently decays exponentially like a memoryless process. To further explore the source of growth we turn to the microscopic domain.

3 Microscopic Growth

Consider N entities represented by a set of random abundance variables, $x_i(t), \quad i = 1, ..., N$, which are unitless and take values between 0 and 1, where 0 represents no realized growth while 1 represents the maximum potential growth having been realized. In other words, these variables represent the *realized potential*, either in the form of individual volume, mass, likelihood of infection or mortality by being preyed on, etc. Let these variables have a shared expected value as a function of time, $\mu(t)$, and corresponding higher moments, i.e. each variable follows the same statistical

distribution. With these variables it will also be convenient to also define an *abundance potential* variable, $s_i = 1 - x_i$, where we omit the temporal variable for ease of exposition. An abundance potential could represent the future capacity to grow, e.g. encoded in an organism's DNA combined with its environmental factors. Our goal now is to investigate under which conditions we can achieve logistic or Gompertz growth.

Preliminarily, consider a network between these N entities along a graph with a set of edges and nodes which delineates the interaction of the growth variables, where we define interaction in this context as the transfer of one entity's potential growth to another entity which converts potential to realized growth. Examples of such networks could be cells that communicate either through electron or protein transfer, predator-prey systems or even human interactions through mutual trade, spreading of knowledge, or disease.

3.1 Microscopic Logistic Growth

Now consider the growth equation with only pairwise interactions, or *first order* interactions, governed by an adjacency matrix a_{ij} as a binary matrix with ones where the i^{th} and j^{th} nodes are connected, and zeroes otherwise, viz.

$$\frac{dx_i}{dt} = x_i \sum_{j \in \mathcal{N}(i)} a_{ij} s_j \quad \forall i, \tag{6}$$

or

$$\frac{d}{dt} \ln x_i = \sum_{j \in \mathcal{N}(i)} a_{ij} s_j \quad \forall i, \tag{7}$$

where $\mathcal{N}(i)$ is the neighborhood of nodes connected to node i, excluding node i itself. For example, if the adjacency matrix has only 1s and no 0s, this means that every node is connected to every other node. The diagonal of the adjacency matrix is zero as a node is not connected to itself by definition.

Let us look at the simplest case of two connected entities only, viz.

$$\frac{dx_1}{dt} = x_1 s_2, \tag{8}$$

$$\frac{dx_2}{dt} = x_2 s_1. \tag{9}$$

Rearranging and summing the two equations, we can express the variables in terms of their averages,

$$\frac{1}{2}\left\{ \frac{1}{x_1}\frac{dx_1}{dt} + \frac{1}{x_2}\frac{dx_2}{dt} \right\} = \frac{1}{2}(s_1 + s_2) = 1 - \frac{1}{2}(x_1 + x_2), \tag{10}$$

where the left hand side represents the average of the relative growth, while the right hand side represents the average abundance potential. Thus, even for a simple two-variable system we see that the system can be described in terms of summary statistics, which we shall see that we will be able to generalize.

We choose to work in a unitless domain without parameters for the interaction strength and carrying capacity for simplicity of exposition. These can be added without changing the conclusions of these arguments.

Now as we let N grow, we impose a simple rule: *Each entity can only receive growth potential from a single entity and create growth potential in a single possibly different entity*. This condition translates into an adjacency matrix with each column containing a single non-zero entry. Using this rule and rearranging and summing across all entities we obtain the following relationship,

$$\frac{d}{dt} \ln \left[\prod_i^N x_i \right] = \sum_j^N s_j. \tag{11}$$

Dividing by the total number of entities, we obtain the simplest form of logistic growth of the geometric mean of the ensemble driven by the arithmetic mean of the inverse abundance, or the *potential abundance*,

$$\frac{d}{dt} \ln \left[\prod_i^N x_i \right]^{1/N} = \frac{1}{N} \sum_j^N s_j. \tag{12}$$

Now note that the time derivative of the geometric mean translates into the *average relative growth rate*, where if we let $v_i = \frac{1}{x_i}\frac{d}{dt}x_i$, we see that

$$\frac{1}{N}\sum_{j}^{N} v_i = \frac{1}{N}\sum_{j}^{N} s_j. \tag{13}$$

At this point we have shown how macroscopic variables can be obtained from their microscopic counterparts. We have not needed to evoke the randomness of our variables, but have obtained a macroscopic understanding only using sample quantities starting from pairwise microscopic principles.

However, if we assume our set of random variables, $\{x_i\}$, converge to a distributional mean as N grows, both of the relative growth rate average and the abundance average will converge to their distributional counterparts. Specifically and with a slight abuse of notation in the arguments, letting $\bar{x}(N)$ represent the sample abundance mean for N nodes which converges to its distributional counterpart $\mu(t)$, it follows from the continuous mapping theorem that $\bar{v}(N)$ will converge to $\frac{1}{\mu(t)}\frac{1}{dt}\mu(t)$ so long as the time derivative is smooth. We thus have

$$\begin{aligned}
\lim_{N\to\infty} \frac{1}{N}\sum_{j}^{N} v_i &= \lim_{N\to\infty} \frac{1}{N}\sum_{i}^{N} s_i \\
\lim_{N\to\infty} \bar{v}(N) &= \lim_{N\to\infty} (1-\bar{x}(N)) \\
\frac{1}{\mu(t)}\frac{d}{dt}\mu(t) &= 1-\mu(t),
\end{aligned}$$

where we make the time dependency explicit for clarity of exposition. Now we see that we have obtained the logistic model in terms of a macroscopic ensemble average.

We make special note that logistic growth emerges from pairwise interactions only and without any independence assumption across the variables. This does not mean that the logistic growth cannot emerge from other microscopic settings but it nevertheless provides one possible scenario under which the logistic curve can emerge. Now, as we shall allow higher order interactions we will see the Gompertz curve emerge.

3.2 Microscopic Gompertz Growth

Instead of limiting the growth equation to pairwise interactions, we allow higher order interactions in the form of triplets, quadruplets, and so on. Analogous to the pairwise exclusivity rule, each entity can again only participate in *one* interaction group at any given order and any given direction. For example, any triplet can only interact with one other triplet and have exclusive possession of its entities at the triplet level (Figure 2).

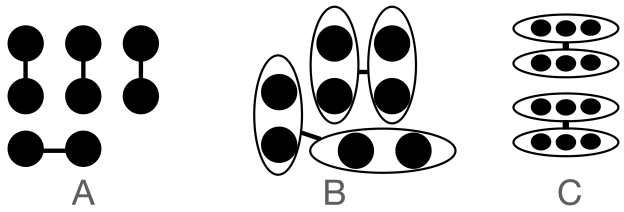

Figure 2: Exclusive interaction groups at each order: A) First order interaction, each entity is paired with a single other entity. B) Second order interaction, mutually exclusive groups of two entities interact with each other. B) Third order interaction, same as B) but for groups of three entities.

Thus, let S_k be the set of all mutually exclusive k-sized groups where $k = 1, ..., N/2$, where the highest order are capped at $N/2$ since that would be the largest number of entities in a single group that could interact with an identically sized group. Thus, the aggregated growth is represented by the sum of each interaction order,

$$\frac{d}{dt}\ln\left[\prod_{i}^{N} x_i\right] = \sum_{j\in S_1}^{N} s_j + \sum_{j_1,j_2\in S_2} s_{j_1}s_{j_2} + ... + \sum_{j_1,j_2,...,j_{N/2}\in S_{N/2}} s_{j_1}s_{j_2}\dots s_{j_{N/2}}, \tag{14}$$

where S_k is the set of all mutually exclusive $k-$sized groups. In each of the sums, the total number of terms is equal to the total number of groups which at each order is N/k where k is the interaction order.

Now, we can use the identical distribution assumption and divide the right hand side by N to identify convergence properties as both k and N grow:

1. As the number of entities grow each term will converge in expectation to higher powers of the mean of s_i, multiplied by the prefactor $1, 1/2, 1/3$, etc.,

$$\lim_{N\to\infty} \frac{1}{N} \sum_{j_1,j_2,\ldots,j_k \in S_k} s_{j_1} s_{j_2} \ldots s_{j_k} = \frac{1}{N}\frac{N}{k}\mu^k = \frac{1}{k}\mu^k. \tag{15}$$

2. As the higher order interaction terms grow

$$\lim_{k\to\infty} \frac{1}{N} \sum_{j_1,j_2,\ldots,j_k \in S_k} s_{j_1} s_{j_2} \ldots s_{j_k} = 0 \tag{16}$$

Thus, as each higher order term is composed of a decreasing number of summations and an increasing number of multiplications, the product of s_i goes to zero since each random variable is bounded between 0 and 1. Now, using the Taylor series identity $\mu + \mu^2/2 + ... = -\ln(1-\mu)$, we obtain Gompertz growth in terms of the arithmetic means of the microscopic entities

$$\lim_{N\to\infty} \frac{d}{dt} \ln\left[\prod_i^N x_i\right]^{1/N} = \lim_{N\to\infty}\left[\sum_j^N s_j + \sum_{j_1,j_2\in S_2} s_{j_1}s_{j_2} + ...\right] \tag{17}$$

$$\lim_{N\to\infty} \bar{v}(N) = \mu + \frac{1}{2}\mu^2 + ... \tag{18}$$

$$\frac{1}{\mu(t)}\frac{d}{dt}\mu(t) = -\ln(1-\mu). \tag{19}$$

Note that for the right hand side to exhibit asymptotically infinite growth rate at the onset of growth, it is not only sufficient but also necessary to have higher order terms. This is seen by virtue of the infinite polynomial representation of the logarithm and implies that *Gompertz growth cannot result from pairwise interactions under the assumptions presented in this model*, but rather requires all higher order interactions to be present.

Since all higher order interaction would necessarily imply non-local interaction, the natural interpretation would be that the growth is activated by a ubiquitous field accessible by all entities simultaneously. In this way one could think of the activating field as an *effectively* external field, even though it is possible that the field emerges as a collective emergent phenomenon purely from within the system or organism. Examples of this field could be sound waves or the electromagnetic field.

Another way to see the source of growth as effectively external is to recognize that the source of growth in the higher order terms is coming from the abundance potential and not the realized potential, while in the logistic growth model this is not necessarily the case. To see this, first swap the role of x_i and s_i on the right hand side of Equation 6 to see that the same logistic dependency can be obtained under the exclusivity condition. This could be interpreted as a causality reversal of the growth process. Then note that when augmenting the growth equation with higher order terms on the right-hand side of the same equation, the same swapping will not lead to the Gompertz model (see Appendices B and C).

Causality reversal is indeed what is done when modelling communicable diseases in a microscopic network context, where x_i represents the infected nodes and s_i represents the susceptible nodes (see e.g. Estrada and Bartesaghi [59]). But because higher order growth does not allow this causality reversal, where the infected nodes are the source of spread, the Gompertz model cannot be used since it would imply that the susceptible nodes are the source of the disease spreading, which would turn communicable disease theory on its head. In this context, the Gompertz model would rather suggest a system disturbed by a ubiquitous, simultaneous, and non-local stressor eliciting a corresponding stress response through which a new stress tolerance baseline is gradually established. Thus, with the necessary requirement of higher order interactions between nodes, *the Gompertz model cannot be used as a communicable disease model*.

4 Unifying the Gaussian and Exponential curves with the Logistic and the Gompertz curves

Up until now we have seen how logistic growth emerges from pairwise correlated growth while Gompertz growth emerges from infinitely correlated growth. These are both growth modes where there is some degree of connectivity between the entities. But what if there is no connectivity between the entities but each entity is independent? In such a

case we can formulate a growth equation where the relative growth is only linearly dependent on time, where we make a slight modification and model the *potential abundance*, $s(t)$, instead of the realized abundance,

$$\frac{d}{dt}s(t) = -\beta t s(t), \tag{20}$$

or

$$\frac{d}{dt}\ln s(t) = -\beta t, \tag{21}$$

for some constant $\beta > 0$.

Solving this equation yields the familiar Gaussian function,

$$s(t) = Ae^{-\beta t^2/2}, \tag{22}$$

where β can be interpreted as the inverse variance or dispersion of the growth phenomena and where $A = 1$ when $s(0) = 1$. Thus, not surprisingly Gaussian growth emerges for mutually independent events that have a linear time dependence. The same result was originally formulated by Gauss with space as the independent variable instead of time and with spatial estimation error as the dependent variable instead of abundance, all in the context of the prediction of the heavenly object called Ceres [60] (see [61] for details). In the context of measurement error, one would equate the above equation to the statement that the relative measurement error decreases linearly with size.

If we remove the dependency on time altogether, we observe the exponential growth equation

$$\frac{d}{dt}\ln s(t) = -\beta, \tag{23}$$

which yields

$$s(t) = Ae^{-\beta t}. \tag{24}$$

This exponential equation implies a complete independence on both time and of the mutual entities involved in the growth. In the context of measurement error this function would be the Laplacian error which states that absolute measurement error decreases linearly with the error itself [2].

In general, our choice of time as the dependent variable can be substituted for any suitable metric of choice. Another example of space as the dependent variable in the Gompertz setting is given by Molski and Konarski [31].

4.1 Unification with the Poisson distribution

As a side note, the Poisson distribution can be included in this discussion as the distribution which governs the infinitesimal growth rate of a coherent or infinitely correlated group of entities. By letting the growth rate be the rate at which a zero-count is observed with the Poisson mean parameter equal to $-\beta t$, one recovers the Gompertz model in Equation 3. This argument was put forth by Shklovskii [62], and Glauber [32] was the first to point out the connection where the Poisson distribution reflects a coherent system. Glauber also showed that incoherent systems, or systems with independent entities, follow the Gaussian distribution which is in accordance with our results and discussion here. The mapping between coherence and incoherence has also been postulated as a medical diagnostic technique by Zhang and Popp [63], where incoherence is a sign of disease while coherence is a sign of well-being.

5 Discussion and Conclusion

To summarize, we have seen that both time independent and uncorrelated growth yields the exponential growth function, time dependent and uncorrelated growth yields Gaussian growth, time independent and pairwise correlated growth yields the logistic function and time independent and infinitely correlated growth yields the Gompertz function.

We have further shown how Gompertz growth emerges both at the microscopic and the macroscopic level as a non-local effect. A novel bridge between logistic and Gompertz growth is established involving Gauss' Hypergeometric function that serves as a replacement for the Richards model and is derived from first principles. This bridge presupposes the source of growth to be the potential abundance as opposed to the realized abundance. On the microscopic level, the Gompertz model emerges through an exclusivity rule at every order of interaction. As a corollary, this thesis shows how the logistic function is the result of local growth processes with pairwise-interactions only, in line with its observation in the fields of predator-prey models and in communicable diseases [64].

[2] Or as Laplace originally stated: "[...] we have no reason to suppose a different law for the ordinates than for their differences" [61].

An infinitely interacting system where entities are classical in nature with well-defined position and momentum, breaks the law of causality unless we allow the source of growth to be a ubiquitous field, externally or internally created, which affects all microscopic entities instantaneously [3]. With a such a source, entities need not be interacting with each other but rather respond to the field-induced stimuli. Examples of such models are already given by De Lauro (2014), Sacher (1956) and Markov (2019). De Lauro et al. [29] and Sacher [28] consider the source of growth to be stochastic in nature, and while Lauro et al. do not discuss the external nature of such a source, their model does not preclude it. More generally, with growth as stochastically rooted it is also ubiquitously present uniformly across all entities from the very onset of growth. Markov (2019)[30] discusses growth within the context of chemical reactions, where the source of growth is a catalytic agent that is implicitly available ubiquitously to all reactants to the same degree, exactly in line with De Lauro and Sacher's models.

For many growth processes within biological organisms, it has been well established that the mediator is the electromagnetic field [33], which suggests that the accompanying growth pattern in these processes follows the Gompertz model as the result of a ubiquitous growth-inducing field. For growth in larger ecological or societal systems other catalysts can be air pollution, oxygen deprivation, or even instant psychologically induced catalysts like collective fear.

We finally note that the original formulation of the Gompertz model made by Gompertz himself was concerning mortality rates as a function of age. He noticed that mortality rates increased at a geometric rate with age. In this context the ubiquitous stressor postulated above would be the living conditions on our planet, for which time acts as a proxy. One could further postulate this stressor as a field surrounding the earth, e.g. in its electromagnetic footprint often related with the Schumann Resonance [65, 66]. It is indeed curious that we do not see mortality rates behave as linearly dependent on time, for which a Gaussian curve would be appropriate. Nor do we see any finite-ordered polynomial time dependence on mortality rates. Instead we see growth of mortality rates as sourced from other entities at all interaction orders, which in light of our discussion above is equivalent to mortality coming from an environmental stressor.

Competing interests

The authors declare no competing interests.

Appendices

A A closed-form finite-interaction model

Here we arrive at a closed-form expression for a finite number of interaction terms in this modified logistic model, which represents the infection term in the SIR model. Working with a single unitless growth variable and omitting the multivariate network without loss of generality, we start with the logistic growth equation augmented by K terms and using the nomenclature defined in Section 3, viz.

$$\frac{d\ln x}{dt} = \sum_{i=1}^{K} s^i/i. \tag{25}$$

Using standard integrals we see that this sum amounts to a Gompertz growth term adjusted by a Hypergeometric function,

$$\begin{aligned}\sum_{i=1}^{K} s^i/i &= \int_s \sum_{i=0}^{K-1} s^i ds \\ &= \int_s \frac{1-s^K}{1-s} ds \\ &= -\frac{{}_1F_2(1, K+1, K+2; s)}{K+1} s^{K+1} - \ln(1-s),\end{aligned}$$

where ${}_1F_2$ is Gauss' Hypergeometric function, and integration limits are indefinite. In summary, the modified logistic function emerges as

$$\frac{d}{dt}\ln x(t) = -\frac{{}_1F_2(1, K+1, K+2; 1-x(t))}{K+1}(1-x(t))^{K+1} - \ln x(t), \tag{26}$$

[3] Or at speeds much greater than the differential times in the growth equations

which represents the novel bridge between logistic and Gompertz growth. Note that for $K = 1$, this relative growth rate becomes the standard logistic function, while increasing the number of higher order terms will move the equation closer to the Gompertz curve as the Hypergeometric term approaches zero. This modification to the logistic function does not have a 1-to-1 correspondence with the Richards model, but serves instead as an alternative when encoding non-linear or collaborative growth effects (see Fig. 1).

B The invariance of logistic growth under causality reversal

Consider a causality reversal of logistic growth such that the realized growth drives potential growth. Then Equation 6 must be modified by swapping x and s on the RHS as follows,

$$\frac{dx_i}{dt} = s_i \sum_{j \in \mathcal{N}(i)} a_{ij} x_j \quad \forall i. \tag{27}$$

Using the identity,

$$\sum_i \frac{1}{s_i}\frac{dx_i}{dt} = -\sum_i \frac{1}{s_i}\frac{ds_i}{dt} = -\frac{d}{dt}\ln\left[\prod_i^N s_i\right], \tag{28}$$

and using the equivalent connectivity rule as outlined in Section 3, we have,

$$-\frac{d}{dt}\ln\left[\prod_i^N s_i\right] = \sum_j^N x_j. \tag{29}$$

And using the same convergence argument as before we get $\bar{s}(N) \to \gamma(t) = 1 - \mu(t)$

$$-\frac{1}{\gamma(t)}\frac{d}{dt}\gamma(t) = (1 - \gamma(t)), \tag{30}$$

which solves to

$$\gamma(t) = \frac{1}{1 + A\exp(t)}, \tag{31}$$

which implies that

$$\mu(t) = \frac{A\exp(t)}{1 + A\exp(t)}. \tag{32}$$

So even when realized growth is responsible for growth thus reversing causality in the pairwise growth setting, the logistic emerges. Thus, we can say that the *microscopic logistic model is invariant under causality reversal*.

C The lack of invariance of Gompertz growth under causality reversal

Similar to our procedure with the logistic model we now swap potential growth with actual growth on the RHS of the microscopic Gompertz model to simulate a causality reversal,

$$-\frac{d}{dt}\ln\left[\prod_i^N s_i\right] = \sum_{j \in S_1}^N x_j + \sum_{j_1, j_2 \in S_2} x_{j_1}x_{j_2} + ... + \sum_{j_1, j_2, ..., j_{N/2} \in S_{N/2}} x_{j_1}x_{j_2}\ldots x_{j_{N/2}}. \tag{33}$$

Make a special note of the new minus sign on the LHS due to a change of variable as done with the logistic treatment (Eq. 29).

Now, with our continuity argument as outlined in the main text we obtain the macroscopic version of this equation as

$$\frac{d}{dt}\ln\gamma(t) = \ln\gamma(t), \tag{34}$$

with $\gamma(t) = 1 - \mu(t)$ and where we are now rid of the minus sign on the RHS of the regular Gompertz model (Eq. 3 due to causality reversal.

This equation solves to

$$\mu(t) = 1 - \exp\left[A\exp(t)\right], \tag{35}$$

which is not of the Gompertz form and furthermore does not exhibit the initially fast growth which is characteristic of the Gompertz model (see Figure 3). Thus we have shown that the *microscopic Gompertz model is not invariant under causality reversal* and is thus not suitable for a communicable disease setting where infected nodes infect uninfected nodes.

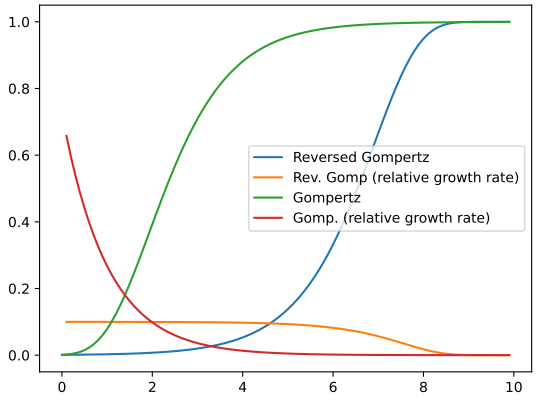

Figure 3: A comparison of the growth profiles of the Gompertz and the causality-reversed Gompertz models. Their relative growth rates defined as the time derivative of the logarithm of the abundance ($\mu(t)$) are also shown. To solve for the constants involved we apply the initial condition $\mu(0) = 0.001$ (a very small positive number).

References

[1] Gompertz, B. XXIV. On the nature of the function expressive of the law of human mortality, and on a new mode of determining the value of life contingencies. in a letter to francis baily, esq. f. r. s. &c. *Philosophical Transactions of the Royal Society of London*, 115:513–583, dec 1825. doi:10.1098/rstl.1825.0026.

[2] Makeham, W. M. On the further development of gompertz's law. *Journal of the Institute of Actuaries*, 28(2): 152–159, 1889.

[3] Weymouth, F. W. and McMillin, H. C. *Relative growth and mortality of the Pacific razor clam (Siliqua patula, Dixon) and their bearing on the commercial fishery*. Number 1099. US Government Printing Office, 1931.

[4] Weymouth, F. W. and Thompson, S. H. *The age and growth of the pacific cockle (Cardium corbis, Martyn)*. Number 1101. US Government Printing Office, 1931.

[5] Laird, A. K. Dynamics of tumour growth. *British journal of cancer*, 18(3):490, 1964.

[6] Zwietering, M. H., Jongenburger, I., Rombouts, F. M., and Van't Riet, K. Modeling of the bacterial growth curve. *Applied and environmental microbiology*, 56(6):1875–1881, 1990.

[7] Skinner, G. E., Larkin, J. W., and Rhodehamel, E. J. Mathematical modeling of microbial growth: a review. *Journal of food safety*, 14(3):175–217, 1994.

[8] Starck, J. M. and Ricklefs, R. E. *Avian growth and development: evolution within the altricial-precocial spectrum*. Number 8. Oxford University Press on Demand, 1998.

[9] Aggrey, S. Comparison of three nonlinear and spline regression models for describing chicken growth curves. *Poultry science*, 81(12):1782–1788, 2002.

[10] Paine, C. T., Marthews, T. R., Vogt, D. R., Purves, D., Rees, M., Hector, A., and Turnbull, L. A. How to fit nonlinear plant growth models and calculate growth rates: an update for ecologists. *Methods in Ecology and Evolution*, 3(2):245–256, 2012.

[11] Benzekry, S., Lamont, C., Beheshti, A., Tracz, A., Ebos, J. M., Hlatky, L., and Hahnfeldt, P. Classical mathematical models for description and prediction of experimental tumor growth. *PLoS computational biology*, 10(8):e1003800, 2014.

[12] Halmi, M., Shukor, M., Johari, W., and Shukor, M. Evaluation of several mathematical models for fitting the growth of the algae dunaliella tertiolecta. *Asian Journal of Plant Biology*, 2(1):1–6, 2014.

[13] Tjørve, K. M. and Tjørve, E. Shapes and functions of bird-growth models: how to characterise chick postnatal growth. *Zoology*, 113(6):326–333, 2010.

[14] Winsor, C. P. The gompertz curve as a growth curve. *Proceedings of the National Academy of Sciences*, 18(1): 1–8, jan 1932. doi:10.1073/pnas.18.1.1.

[15] Olshansky, S. J. and Carnes, B. A. Ever since gompertz. *Demography*, 34:1–15, 1997.

[16] Prescott, R. B. Law of growth in forecasting demand. *Journal of the American Statistical Association*, 18(140): 471–479, 1922.

[17] Peabody, L. E. Growth curves and railway traffic. *Journal of the American Statistical Association*, 19(148): 476–483, 1924.

[18] Bajzer, Ž., Vuk-Pavlović, S., and Huzak, M. Mathematical modeling of tumor growth kinetics. In *A survey of models for tumor-immune system dynamics*, pages 89–133. Springer, 1997.

[19] Petroni, N. C., De Martino, S., and De Siena, S. Logistic and θ-logistic models in population dynamics: General analysis and exact results. *Journal of Physics A: Mathematical and Theoretical*, 53(44):445005, 2020.

[20] Harko, T., Lobo, F. S., and Mak, M. Exact analytical solutions of the susceptible-infected-recovered (sir) epidemic model and of the sir model with equal death and birth rates. *Applied Mathematics and Computation*, 236:184–194, 2014.

[21] Kröger, M. and Schlickeiser, R. Analytical solution of the SIR-model for the temporal evolution of epidemics. part a: time-independent reproduction factor. *Journal of Physics A: Mathematical and Theoretical*, 53(50):505601, 2020.

[22] Schlickeiser, R. and Kröger, M. Analytical solution of the SIR-model for the temporal evolution of epidemics: part b. semi-time case. *Journal of Physics A: Mathematical and Theoretical*, 54(17):175601, 2021.

[23] Heng, K. and Althaus, C. L. The approximately universal shapes of epidemic curves in the susceptible-exposed-infectious-recovered (SEIR) model. *Scientific Reports*, 10(1):1–6, 2020.

[24] Berryman, A. A. The orgins and evolution of predator-prey theory. *Ecology*, 73(5):1530–1535, 1992.

[25] Neafsey, P. J., Boxenbaum, H., Ciraulo, D. A., and Fournier, D. J. A gompertz age-specific mortality rate model of aging, hormesis, and toxicity: Fixed-dose studies. *Drug metabolism reviews*, 19(3-4):369–401, 1988.

[26] Neafsey, P. J., Ciraulo, D. A., Boxenbaum, H., and Fournier, D. J. A gompertz age-specific mortality rate model of aging, hormesis, and toxicity: dose-response studies. *Drug Metabolism Reviews*, 20(1):111–150, 1989.

[27] Thompson, G. A., Smithers, J., and Boxenbaum, H. Biphasic mortality response of chipmunks in the wild to single doses of ionizing radiation: toxicity and longevity hormesis. *Drug metabolism reviews*, 22(2-3):269–289, 1990.

[28] Sacher, G. A. On the statistical nature of mortality, with especial reference to chronic radiation mortality. *Radiology*, 67(2):250–258, 1956.

[29] De Lauro, E., De Martino, S., De Siena, S., and Giorno, V. Stochastic roots of growth phenomena. *Physica A: Statistical Mechanics and its Applications*, 401:207–213, 2014.

[30] Markov, S. M. Reaction networks reveal new links between gompertz and verhulst growth functions. *Biomath*, 8 (1):ID–1904167, 2019.

[31] Molski, M. and Konarski, J. Coherent states of gompertzian growth. *Physical review E*, 68(2):021916, 2003.

[32] Glauber, R. J. Coherent and incoherent states of the radiation field. *Physical Review*, 131(6):2766, 1963.

[33] Becker, R. O. Electromagnetic controls over biological growth processes. *Journal of Bioelectricity*, 3(1-2): 105–118, 1984.

[34] Langman, L. and Burr, H. A technique to aid in the detection of malignancy of the female genital tract. *American Journal of Obstetrics & Gynecology*, 57(2):274–281, 1949.

[35] Kozusko, F. and Bajzer, Ž. Combining gompertzian growth and cell population dynamics. *Mathematical biosciences*, 185(2):153–167, 2003.

[36] Goodman, R., Bassett, C. A. L., and Henderson, A. S. Pulsing electromagnetic fields induce cellular transcription. *Science*, 220(4603):1283–1285, 1983.

[37] Liboff, A., Williams Jr, T., Strong, D., and Wistar Jr, R. Alternating magnetic fields enhance dna synthesis in fibroblastic cells. *Bioelectrical Repair and Growth Society (BRAGS)*, 1981.

[38] Norton, L., Hanley, K., and Turkewicz, J. Bioelectric perturbations of bone: Research directions and clinical applications. *The Angle Orthodontist*, 54(1):73–87, 1984.

[39] Liboff, A., Williams Jr, T., Strong, D., and Wistar Jr, R. Time-varying magnetic fields: effect on dna synthesis. *Science*, 223(4638):818–820, 1984.

[40] Wang, X.-S., Wu, J., and Yang, Y. Richards model revisited: Validation by and application to infection dynamics. *Journal of theoretical biology*, 313:12–19, 2012.

[41] Kermack, W. O. and McKendrick, A. G. A contribution to the mathematical theory of epidemics. *Proceedings of the royal society of london. Series A, Containing papers of a mathematical and physical character*, 115(772): 700–721, 1927.

[42] Zonta, F. and Levitt, M. Virus spread on a scale-free network reproduces the gompertz growth observed in isolated covid-19 outbreaks. *Advances in Biological Regulation*, 86:100915, 2022.

[43] Clauset, A., Shalizi, C. R., and Newman, M. E. Power-law distributions in empirical data. *SIAM review*, 51(4): 661–703, 2009.

[44] Broido, A. D. and Clauset, A. Scale-free networks are rare. *Nature communications*, 10(1):1–10, 2019.

[45] Holme, P. Rare and everywhere: Perspectives on scale-free networks. *Nature communications*, 10(1):1016, 2019.

[46] Carletti, T., Fanelli, D., and Piazza, F. COVID-19: The unreasonable effectiveness of simple models. *Chaos, Solitons & Fractals: X*, 5:100034, 2020.

[47] Cooper, I., Mondal, A., and Antonopoulos, C. G. A SIR model assumption for the spread of COVID-19 in different communities. *Chaos, Solitons & Fractals*, 139:110057, 2020.

[48] Postnikov, E. B. Estimation of COVID-19 dynamics "on a back-of-envelope": Does the simplest SIR model provide quantitative parameters and predictions? *Chaos, Solitons & Fractals*, 135:109841, 2020.

[49] Muñoz-Fernández, G. A., Seoane, J. M., and Seoane-Sepúlveda, J. B. A SIR-type model describing the successive waves of COVID-19. *Chaos, Solitons & Fractals*, 144:110682, 2021.

[50] Cooper, I., Mondal, A., Antonopoulos, C. G., and Mishra, A. Dynamical analysis of the infection status in diverse communities due to COVID-19 using a modified SIR model. *Nonlinear Dynamics*, pages 1–14, 2022.

[51] Saikia, D., Bora, K., and Bora, M. P. COVID-19 outbreak in india: an SEIR model-based analysis. *Nonlinear Dynamics*, 104(4):4727–4751, 2021.

[52] Ohnishi, A., Namekawa, Y., and Fukui, T. Universality in COVID-19 spread in view of the gompertz function. *Progress of Theoretical and Experimental Physics*, 2020(12), oct 2020. doi:10.1093/ptep/ptaa148.

[53] Rypdal, K. and Rypdal, M. A parsimonious description and cross-country analysis of COVID-19 epidemic curves. *International Journal of Environmental Research and Public Health*, 17(18):6487, sep 2020. doi:10.3390/ijerph17186487.

[54] Català, M., Alonso, S., Alvarez-Lacalle, E., López, D., Cardona, P.-J., and Prats, C. Empirical model for short-time prediction of COVID-19 spreading. *PLOS Computational Biology*, 16(12):e1008431, dec 2020. doi:10.1371/journal.pcbi.1008431.

[55] Rodrigues, T. and Helene, O. Monte carlo approach to model COVID-19 deaths and infections using gompertz functions. *Physical Review Research*, 2(4):043381, 2020.

[56] Levitt, M., Scaiewicz, A., and Zonta, F. Predicting the trajectory of any COVID19 epidemic from the best straight line. *medRxiv*, jun 2020. doi:10.1101/2020.06.26.20140814.

[57] Tjørve, K. M. and Tjørve, E. The use of gompertz models in growth analyses, and new gompertz-model approach: An addition to the unified-richards family. *PloS one*, 12(6):e0178691, 2017.

[58] Richards, F. A flexible growth function for empirical use. *Journal of experimental Botany*, 10(2):290–301, 1959.

[59] Estrada, E. and Bartesaghi, P. From networked SIS model to the gompertz function. *Applied Mathematics and Computation*, 419:126882, 2022.

[60] Gauss, C. F. and Davis, C. H. *Theory of the motion of the heavenly bodies moving about the sun in conic sections: a translation of Gauss's" Theoria Motus." with an appendix*. Little, Brown, 1857.

[61] Stahl, S. The evolution of the normal distribution. *Mathematics magazine*, 79(2):96–113, 2006.

[62] Shklovskii, B. A simple derivation of the gompertz law for human mortality. *Theory in Biosciences*, 123(4): 431–433, 2005.

[63] Zhang, C.-L. and Popp, F.-A. Log-normal distribution of physiological parameters and the coherence of biological systems. *Medical Hypotheses*, 43(1):11–16, 1994.

[64] May, R. and McLean, A. R. *Theoretical ecology: principles and applications*. Oxford University Press, 2007.

[65] Cherry, N. Schumann resonances, a plausible biophysical mechanism for the human health effects of solar. *Natural hazards*, 26(3):279–331, 2002.

[66] Schumann, W. O. Über die strahlungslosen eigenschwingungen einer leitenden kugel, die von einer luftschicht und einer ionosphärenhülle umgeben ist. *Zeitschrift für Naturforschung A*, 7(2):149–154, 1952.