Logistic growth and immunity

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1 Background

It has been suggested that the classical logistic growth model be used to describe the progression of the SARS-CoV-2 virus infection. This is probably inspired by figures released by various authorities in the form of *total number infected so far*. The logistic model, however, does not take removal by immunity or death into account. Published figures, in some instances, reveal *cured*, but that information is likely to be incomplete. We present a simple model that could lead, qualitatively, to some understanding. It is based on the hypothesis that immunity occurs some time after infection.

2 The nature of logistic growth

There are two elementary linear growth models. First is *exponential growth* which states that *rate of increase (of an infection) is directly proportional to what is infected*. In mathematical terms, if $f(t)$ is the *intensity* of infection at time t,

$$f'(t) := \tfrac{d}{dt} f(t) = rf(t), \tag{1}$$

with r a positive constant, the *infection rate*.

The other is the *limited resources model* which states that *rate of increase is directly proportional to what is left to be infected*. The differential equation here is

$$f'(t) = r[A - f(t)]. \tag{2}$$

Here $A > 0$ is the *resource available* for infection. That is, the part A of the population that can be infected is finite and at time t, only $A - f(t)$ can still be infected. The quantity A may reflect the entire population but could also mean that part not immune to the infection.

If at time $t = 0$ the infection has intensity $f_0 > 0$, the solution for the exponential growth model (1) is

$$f(t) = f_0 e^{rt},$$

which represents "a total wipe-out in no time" ($e > 2.718$). When uninformed writers use the term *exponential growth* this is probably not what we should understand.

If, in the limited resources model the available resource A is constant, the solution of the equation (2) is

$$f(t) = f_0 e^{-rt} + A[1 - e^{-rt}].$$

Because of the negative exponentials in this expression, it is seen that the infection tends to wipe out all that is available as time increases. Fig.1 illustrates.

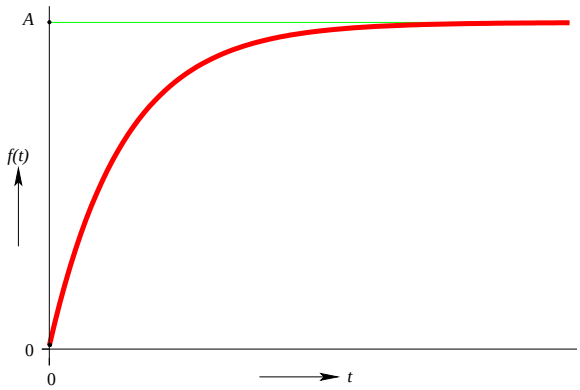

Figure 1: Limited growth

The figure also demonstrates what administrators might mean with "the flattening of the curve".

Compared to what is often observed, the curve in Fig.1 initially rises too rapidly. The *logistic growth model* compensates for that. The underlying assumption is: *rate of increase is proportional to what is infected and to what remains to be infected.* The differential equation to reflect this assumption is

$$f'(t) = rf(t)[A - f(t)]. \tag{3}$$

This equation has dimensional difficulties which, for constant A, can be overcome by considering the ratio $f(t)/A$ as a new unknown. This entails the replacements $f/A \to f$ and $Ar \to r$ so that the equation becomes

$$f'(t) = rf(t)[1 - f(t)] \tag{4}$$

in which f signifies the ratio of "infected" to "available". For $0 < f_0 < 1$ the solution of equation (4) is

$$f(t) = \frac{f_0}{f_0 + (1 - f_0)e^{-rt}}.$$

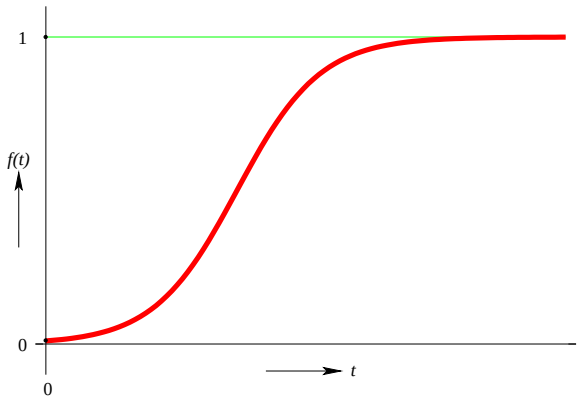

Figure 2: Logistic growth

It is seen that for large t the solution approaches 1. We illustrate in Fig.2. The graph is an example of the logistic curve first introduced by the Belgian mathematician P R Verhulst in 1838 — in the context of population growth. We note once again that the whole available population is "consumed" as time goes on. Administrators will gleefully notice the flattening of the curve everyone is waiting for.

3 Immunity

Although logistic growth shows "flattening" it offers the ironic consolation that the entire susceptible population will be wiped out. If the infection is of such a nature that immunity can be achieved after infection, one can be more optimistic. We propose a modification of the logistic model (3). In analogy to the *principle of fading memory* in continuum mechanics, we assume that the availability A is time dependent in a specific way:

$$A = A(t) = A_0 - f(t - \tau) \tag{5}$$

with $A_0 > 0$ a constant. The *immunity period* $\tau > 0$ is thought of as the time from infection to immunity, and the form of $A(t)$ suggests that availability decreases due to the onset of immunity. Thus the "raw" equation (3) may now be expressed in the form

$$f'(t) = rf(t)[A_0 - f(t - \tau) - f(t)].$$

Scaling by the constant A_0 gives rise to the following analogue of (4):

$$f'(t) = rf(t)[1 - f(t - \tau) - f(t)]. \tag{6}$$

The equation (6) is a differential equation with *delay*. This means that the initial condition $f(0) = f_0$ is insufficient. In fact one needs to know the

history of the epidemic over a time span of length τ. This we specify as follows:

$$f(t) = \phi(t) > 0 \quad \text{for } -\tau \leq t \leq 0, \tag{7}$$

with ϕ a given function.

4 The solution

To solve the equation (6) with the *initial condition* (7) we make the (standard) substitution

$$f(t) = \frac{1}{g(t)}. \tag{8}$$

This leads to the differential equation

$$g'(t) + r[1 - f(t-\tau)]g(t) = r.$$

Use of the integrating factor

$$I(t) = r\int_0^t [1 - f(s-\tau)]\,ds = rt - r\int_0^t f(s-\tau)\,ds \tag{9}$$

leads to the representation

$$e^{I(t)}g(t) = g_0 + r\int_0^t e^{I(\sigma)}\,d\sigma, \tag{10}$$

with $g_0 = 1/f_0 = 1/\phi(0)$.

The representation (10) can be sub-dived in two parts, one for $0 \leq t \leq \tau$ and the other for $t > \tau$. This is achieved by splitting the expression (9) as follows:

$$I(t) = \begin{cases} rt - r\displaystyle\int_0^t \phi(s-\tau)\,ds & \text{if } 0 \leq t \leq \tau, \\ I(\tau) + r(t-\tau) - r\displaystyle\int_\tau^t f(s-\tau)\,ds & \text{if } t > \tau. \end{cases} \tag{11}$$

For $0 \leq t \leq \tau$ the solution depends only on the *initial function* ϕ. The second part of the solution depends on the values of f in the interval $[0, t-\tau]$, already known. We see that the model assumption (5) leads to "failure of short term memory". Once these integrals are calculated, $g(t)$ can be found from (10) and $f(t)$ from (8).

5 An illustrative example

We illustrate qualitatively with a special case in which $\phi(t) = f_0$ for $-\tau \leq t \leq 0$. For this choice of ϕ the expressions in (11) become more explicit:

$$I(t) = \begin{cases} (1 - f_0)rt & \text{if } 0 \leq t \leq \tau, \\ r(t - f_0\tau) - r\displaystyle\int_\tau^t f(s - \tau)\, ds & \text{if } t > \tau. \end{cases} \tag{12}$$

The crucial integrals in (12) can be computed approximately by use of the trapezium rule. This is facilitated by the recursion

$$\begin{aligned} I(t + h) &= I(t) + rh - r\int_t^{t+h} f(s - \tau)\, ds \\ &\approx I(t) + rh - \tfrac{1}{2}rh[f(t - \tau) + f(t + h - \tau)]. \end{aligned}$$

In a similar way for the integrals in (10), $G(t) := r\int_0^t e^{I(\sigma)}\, d\sigma$:

$$G(t + h) \approx G(t) + \tfrac{1}{2}rh[e^{I(t)} + e^{I(t+h)}].$$

The result of calculations with $r = 0.25, f_0 = 0.01, \tau = 10$ and $h = 0.2$ is shown in Fig.3.

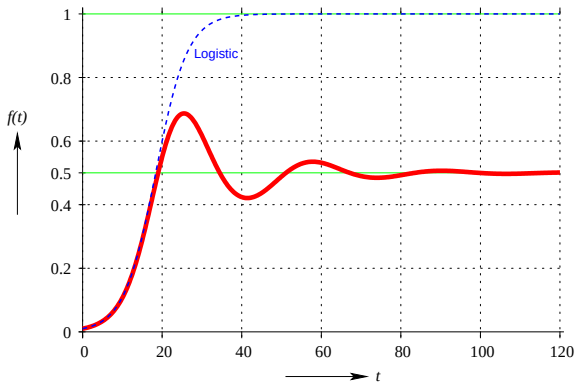

Figure 3: Logistic growth and immunity: $\tau = 10$

Also shown is the logistic curve calculated for the same values of the parameters r and f_0. Of course, in logistic growth the parameter τ has no role to play. The role of the immunity period τ in our model is illustrated in Fig.4 below. As can be expected, a longer period before immunity means a more unstable outcome.

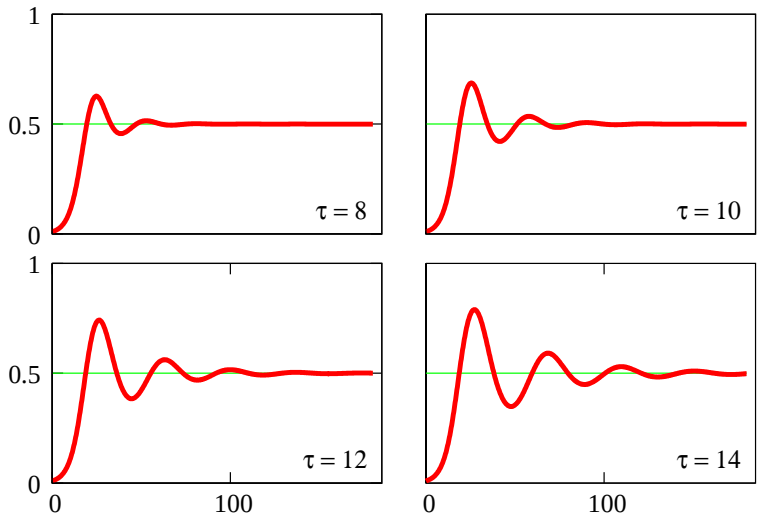

Figure 4: The role of τ

Once the levels of infection $f(t)$ have been calculated, the rate of infection can be obtained from the governing equation (6). This is shown in Fig.5. Indeed, the zeros of this graph correspond to the turning points of the growth curve.

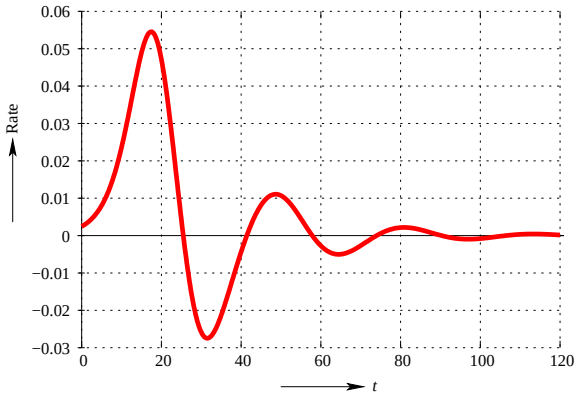

Figure 5: Growth rate

Notes

1. Figure 3 has some interesting features. One is a damped oscillation around $f = 1/2$. To explain this in terms of the model we need to consider the term $1 - f(t-\tau) - f(t)$ in the governing equation (6). At times t when this term is zero, the growth curve turns — it "flattens". This could spell danger for decision makers. The favourable flattening

is the asymptote $f_\infty = \lim_{t\to\infty} f(t)$. In this case $1 - 2f_\infty = 0$ or $f_\infty = 1/2$.

2. The model (5) for availability is perhaps too simple and could be modified to

$$A(t) = A_0 - \ell f(t-\tau) - f(t)$$

with ℓ denoting the fraction of infections where immunity is achieved. This would have the effect that $f_\infty = 1/(1+\ell)$.

3. Published data often present growth rate as *number of new cases*. This can be visualized in a meaningful way as the ratio: *new cases to new tests*. If suitable data on number of recoveries were available, this could be brought in line with the model discussed here.

4. The model presented here does not take infection by diffusion — from higher to lower concentration — into account. For this a formidable amount of additional information, including population density, would be needed. Without diffusion we are simply looking at a "well-stirred" environment.

5. A physical analogue of the limited resources model is the build-up of charge in a capacitor though a resistor with a battery of fixed potential (voltage) supplying charge. This is a little too simple though. The potential of the battery is depleted in the process of charging the capacitor, and that brings us closer to the model discussed above.

6. My sincere thanks to Jean Lubuma who brought to my attention a paper that reviews the rich and diverse literature on differential equations with delay as applied to population dynamics. The reference is:

   Ruan, S. Delay differential equations in single species dynamics. In: O. Arino et al. (eds), *Delay Differential Equations and Applications*, Springer, Berlin, 2006, pp.477–517.