Some fractal thoughts about the COVID-19 infection outbreak

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Abstract

Some ideas are presented about the physical motivation of the apparent capacity of generalized logistic equations to describe the outbreak of the COVID-19 infection, and in general of quite many other epidemics. The main focuses here are: the complex, possibly fractal, structure of the locus describing the "contagion event set"; what can be learnt from the models of trophic webs with "herd behaviour".

1 Introduction

In these days of worldwide mourning for the human tragedy due to the COVID-19 pandemic, while experiencing the heavy lockdown, in the fear of a possible forthcoming economical and social crisis, I think every scientist is thinking about how to be of help, in terms of her/his ideas and technical culture. Besides humbly admitting this to be, first of all, the work of doctors, nurses, virologists, biologists and epidemiologists, not to mention the engineers who design medical and life-saving devices, each of us wonders what technical tools among ours might either help the fight, or at least teach something about how the pandemic appears to work. Speaking for myself, I am doing some job on the biomathematics of epidemics partly for scientific curiosity and intellectual challenge, and partly (I'm sure) just to do something in contact with what is happening out there, be it the epical tragedy or the difficult clash against the virus. The antidote my research is looking for is, at least, the one against the feeling of personal powerlessness.

I felt the need to publish these notes when I went through the paper [1], in which the authors perform a deep investigation about the behaviour with time of the "total number C(t) of people infected so far", for the Chinese Province of Hubei, the other Chinese Provinces, and few other countries undergoing the outbreak of COVID-19, i.e. South Korea, Japan, Iran and Italy. The fact

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attracting my attention, among the various results reported, was the apparent good performance, in fitting the contagion data, of the law C(t) solving the Generalized Richards Model (GRM) [2]; this is a modification of the logistic equation

$$\dot{C} = rC\left(1 - \frac{C}{K}\right),\tag{1}$$

in which the population variable appears with powers different from 1, typically between zero and 1:

$$\dot{C} = rC^p \left[1 - \left(\frac{C}{K}\right)^{\alpha} \right], \ p, \alpha \in [0, 1], \ \alpha \ge p$$
(2)

(despite they are omonimous, the coefficients r in (1) and (2) are not the same quantity: in particular, in (1) one has $[r] = t^{-1}$, while in (2) one has $[r] = C^{1-p}t^{-1}$).

The Ordinary Differential Equation (ODE) (2) has been largely and succesfully employed in epidemiology, so there is no real surprise in its applicability to the COVID-19 outbreak. However, as theorists usually do, I wonder which "first principle meaning" should be given to those powers p and α in (2). In the literature, those parameters appear to be mostly guessed/adapted empirically, in order to fit the experimental curve C(t) a posteriori, once the epidemic is completely developed. Some study noticeably put the (p, α) values in relationship with the "microscopic contagion dynamics" [3]: it is shown that these constants depend on the geography of contagion (whether the epidemic develops in regions with or without clusters of population and communications, like towns of various size, or not) and on the sociology of contagion (in the case of HIV, for instance, whether this takes place via sexual intercourse, or via needle sharing); these "microscopic conditions" appears to be what gives the *contagion networks* a different topology. Recently, the specific study [4] on the COVID-19 outbreak argued how the scale-free complex clusterization of contagion events could motivate a fractional kinetics for C(t); I am myself involved, together with Giuseppe Consolini, in a study about fractional ODEs possibly solved by the COVID-19 C(t) best fit in Italy [5].

All in all, it is clear that deviations of C(t) from the pure logistic ODE (1) points towards the departure of the real laws governing the infection spread from the hypotheses one has to assume to think a population C satisfies (1). One of the most important assumptions of the logistic equation, and of all ODEs regulating the kinetics of population growth, is the so called mass hypothesis [6, 7]: one assumes that the mixing of individuals is such, that all the microscopic actions represented in the ODE are taken (on the average) by all the elements of that population. This, roughly, gives rise to integer powers of population variables in the ODEs. In a certain sense, one might expect equation (2) to be a consequence of mass hypothesis violation by the statistics of the microscopic actions (contagion events), of which it represents a mean field description.

Why should that take place?

The answer I suggest here in inspired by the *Trophic Web Theory* (TWT), where the dynamics of interacting populations in ecosystems are represented

via coupled ODEs, in which a form of segregation may take place, namely *herd* behaviour: this assumption, that represents a topological correction to the mass hypothesis, leads the population variables to appear in their ODEs with real, possibly non-integer, powers, exactly as in the GRM that the authors of [1] claim to fit well the curves of the COVID-19 epidemic.

Here I present a personal vision on how to interpret the powers p and α in (2), according to the idea of herd behaviour in TWT: my suggestion is that the "abstract geometrical locus of contagion events" may have a *fractal dimension*, under the assumption that the network of human contacts giving contagion may be a scale-free network, with clusters of links on many scales, just reflecting those geographical and sociological aspects evoked by the authors of [3].

The paper is organized as follows.

In § 2 the logistic ODE is recalled, and its non-integer power generalizations are presented. In § 3 the concept of herd behaviour in TWT is introduced, with some examples from ecology. Then, its interpretation of the parameters p and α is presented.

Conclusions, and possible developments of these ideas, are given in § 4.

Before starting, I would like to stress how this work *is not* about fitting the contagion curve, or predicting anything; rather, it is an exercise about the possible theoretical motivations for (2) to describe well various cases of epidemic; this does not mean these reasonings to be completely useless in applicative terms.

2 Logistic, and generalized logistic, equations

As stated in the § 1, the ODE (2) is a form of generalized logistic ODE, because it extends the original law (1), to which it reduces for $p = \alpha = 1$. The dynamics (1) describes a population C that may grow from arbitrarily small positive amounts up to an equilibrium value for C_{eq} so that $\dot{C}(C_{eq}) = 0$, that is realized for $C_{eq} = K$: this parameter K is referred to as carrying capacity.

In order to understand a little bit more the roles of the various terms in (1), it is better to re-write it as

$$\dot{C} = rC - \frac{r}{K}C^2:$$
(3)

in this expression, one may distinguish the competition between an exponential growth term rC and a self-limitation term $-\frac{r}{K}C^2$. The expression (3) is of help in understading the rationale of (1): as made clear in the expression (3), we have a population dynamics in which all the *C* individuals take part to both the exponential growth and the the self-limitation. The term rC means that, for each of the *C* units, one more unit will be "created", in a "reaction" of the form

$$C \longrightarrow C + C,$$
 (4)

every $\Delta t_{+1} = \frac{1}{r}$ units of time; meanwhile, the term $-\frac{r}{K}C^2$ means that, whenever each of the *C* individuals meets another one of the *C* individuals, one individual

is destroyed in a process

$$C + C \longrightarrow C,$$
 (5)

that takes place every $\Delta t_{-1} = \frac{K}{rC}$ units of time (this Δt_{-1} becomes smaller and smaller as the amount *C* increases). Before closing the simple reasoning about (3), let me stress that *r* is the effectiveness of the +1 production process, while $\frac{r}{K}$ measures the effectiveness of the -1 destruction process. Expression (3) is often referred to as *Verhulst Equation* (VE) [8].

It is important to underline the relationship between the mass hypothesis, and the mathematical way how the two "reactions" (4) and (5) are implemented in the kinetics (3) of the population C. Indeed, the fact that the creation rate reads rC means that all of the C individuals of the population do take part to (4); in the same way, the expression $-\frac{r}{K}C^2$ for the destruction rate means that there is a possible "annihilation" for each and every couple of the C individuals, being those couples as many as $\mathbb{O}(C^2)$, because each of the C units competes with all its fellows.

Having these considerations about (1) in mind, we may re-write the *generalized logistic equation* (2) as follows

$$\dot{C} = rC^p - \frac{r}{K^{\alpha}}C^{p+\alpha}:$$
(6)

we now have a production term rC^p , with p < 1, and a destruction (self-competition) term $-\frac{r}{K^{\alpha}}C^{p+\alpha}$, with $p + \alpha < 2$. The interpretation of (2) under the point of view of (fractal) herd behaviour, that is described in § 3, starts from here.

3 Herd behaviour and fractals

Let us consider, for instance, two populations X and Y, respectively of preys and predators, living on a surface, e.g. the savnnah, or a regular portion of the seabed, i.e., 2-dimensional environments. The predator-prey interaction, consisting of simple predation, gives rise to a term

$$\dot{X}_Y = -kXY \tag{7}$$

in a simple Lotka-Volterra model, or to tomething like

$$\dot{X}_Y = -\frac{h}{b+X}XY,\tag{8}$$

if the model is more sophisticated and a Holling Type II response function is adopted to describe predation, as in [9] (k and h are constants). In $\dot{X}_Y(X,Y)$ the number of preys and predators appears to the first power: in (7), each of the X preys may "couple" with each of the Y predators with the same destruction rate k; in (8), this happens, but with a rate $\frac{h}{b+X}$ decreasing with the total amount of preys. Under the idea that every prey can be reached by every predator, there is clearly the mass hypothesis discussed in § 1. In TWT a condition has been introduced [10], that changes this hypothesis and, accordingly, modifies the response terms, the so called *herd behaviour*. Let's suppose that the X preys move in compact groups of finite size, that cannot be penetrated by predators: each of the Y predators can only pick preys from the border of those groups. The right hypothesis is, then, not that each prey is attacked by each predator, but that just the X_{∂} preys along the group border will be. So, in the place of X in $\dot{X}_Y(X,Y)$, one has to put the number of preys really involved in this predation, i.e. those X_{∂} ones sitting on the group border. Because the scene is 2-dimensional, under the hypothesis of homogeneos surface density (that must be done if we want to discuss *space-implicit models*, describing everything via ODEs), comparing the number of individuals along the border of a geometrical figure with that of all the ones all over the figure is just as comparing the length of the perimeter with the measure of the surface. If the figure at hand has a "size" ℓ , clearly the surface has a measure $A = \mathbb{O}(\ell^2)$, so that $\ell = \mathbb{O}(X^{\frac{1}{2}})$, while the perimeter scales as $P = \mathbb{O}(\ell)$: one may conclude

$$X_{\partial} = \mathbb{O}\left(X^{\frac{1}{2}}\right).$$

As the preys move just in compact, predator-impenetrable groups, i.e. as they show herd behaviour, while predators are free to move all over the 2d space aoutside these groups, the predation terms in (7) and (8) will be re-written as

$$\dot{X}_Y = -k'\sqrt{X}Y$$
 and $\dot{X}_Y = -\frac{h'\sqrt{X}}{b'+\sqrt{X}}Y$ (9)

respectively.

That of the savannah is an \mathbb{R}^2 example, but the herd behaviour can be generalized to other geometrical enrivonments: for instance, if preys and predators move in \mathbb{R}^3 , as it happens to nekton animals in the sea, then one may state $X = \mathbb{O}(\ell^3)$ and $X_{\partial} = \mathbb{O}(\ell^2)$, so that the predation terms (9) will read:

$$\dot{X}_Y = -k'' X^{\frac{2}{3}} Y, \ \dot{X}_Y = -\frac{h'' X^{\frac{2}{3}}}{b'' + X^{\frac{2}{3}}} Y$$

More in general, if those species live in some \mathbb{R}^n , but the preys that can be preyed on are segregated in a sub-ambient \mathbb{E}_{act} of dimension dim $\mathbb{E}_{act} = m \leq n$, clearly some terms as

$$\dot{X}_Y = -\hat{k}X^{\eta}Y, \ \dot{X}_Y = -\frac{\dot{h}X^{\eta}}{\hat{b} + X^{\eta}}Y, \ /\eta = \frac{m}{n} \le 1$$
 (10)

will appear in the prey population ODE. Note that, in the expressions from (7) to (10), a mass hypothesis is still active on predators, that are supposed to be "very mobile" and "enough mixed" outside the groups of preys. If, instead, also the predators are scarcely mobile or slow, possibly packs of predators interact

with herds of preys just via their borders [11], so that, for instance, one should write $\tilde{}$

$$\dot{X}_Y = -\tilde{k}\sqrt{XY}$$
 and $\dot{X}_Y = -\frac{h\sqrt{XY}}{\tilde{b}+\sqrt{X}}$

instead of (9), and so on.

Applying these concepts to the epidemic growth given by equation (6) requires some generalization of the ecological examples just described. Suppose to deal with a population of infected people C occupying an environment \mathbb{E} of dimension dim $\mathbb{E} = \nu$: this number can be, in principle, any real, positive number, as we are imaging populations living in any fractal subset of \mathbb{R}^n . Suppose that these individuals undergo processes as (4) and (5): if all the individuals living in \mathbb{E} take part to both these processes, clearly the Verhulst equation (3) will be solved by C. Instead, suppose that, in order to be "active" pruducing new individuals (when an infected unit meets a susceptible one), or limiting each other (because when two infected units meet, no new one appears), those units have to be segregated in a sub-environment of \mathbb{E} , namely some $\mathbb{E}_{act} \subset \mathbb{E}$, so that dim $\mathbb{E}_{act} = \mu \leq \nu$. It is straightforward to convince ourselves that the active portion of population is

$$C_{\text{act}} = \mathbb{O}(C^p), \ p = \frac{\mu}{\nu} \le 1.$$

It is then obvious to write the generalization of (3) to which such a species would undergo:

$$\dot{C} = rC^p - \frac{r}{K^p}C^{2p},\tag{11}$$

that is precisely the same as (6), or (2), with $\alpha = p$.

Now, what if $\alpha \neq p$ in (6)?

Typically, one has $\alpha > p$ as in [1], so it is sensible to put $\alpha = p + \delta$, with $\delta > 0$, and then re-write (6) as:

$$\dot{C} = rC^p - \frac{rC^\delta}{K^{p+\delta}}C^{2p}.$$
(12)

The only true difference between this case and the ODE (11) is the fact that the coefficient of C^{2p} depends on C itself. This can be interpreted in two equivalent ways: on the one hand, one may say that the effectiveness of the -1 limiting process depends on the population itself as

$$\Delta_{\text{eff}} (C) \stackrel{\text{def}}{=} \frac{r}{K^{p+\delta}} C^{\delta}, \qquad (13)$$

so that the larger the poulation is, the more destructive the self-limitation turns out to be among the individuals in \mathbb{E}_{act} ; on the other hand, one might as well state that there is an *C*-local effective carrying capacity $K_{eff}(C)$ decreasing with C

$$K_{\text{eff}}(C) \stackrel{\text{def}}{=} \frac{K^{p+\delta}}{C^{\delta}},\tag{14}$$

so that, as the population increases, its dynamics "sees" a smaller and smaller carrying capacity (even if the asymptotic value is still C = K: the only difference with respect to the cases (1) and (11) is that in (6), and hence in (12), the rush towards the limit C = K gets slower and slower, with respect to the logistic ODE tempo, while the total population C increases). Another possibile interpretation of the self-competition term in (6) could be that, next to the infected people able to infect the others, i.e. $C_{act} = \mathbb{O}(C^p)$, there is a class of infected people with whom those C_{act} come in contact uneffectively, that is some $C_{lim} = \mathbb{O}(C^{\alpha})$ limiting the contagion, as $-\frac{r}{K^{\alpha}}C^{p}C^{\alpha}$. In this vision, one should define some geometric locus \mathbb{E}_{lim} , with Hausdorff dimension α , to which the uneffective contacts are restricted (it is very likely that we well have $\mathbb{E}_{act} \cap \mathbb{E}_{lim} \neq \emptyset$).

Now, the crucial point is to understand why the COVID-19 contagion growth, together with other epidemics well studied in the past, should behave in this way, in terms of the segregation of the various classes of individuals: a point of view on this, is given in the following \S 4.

4 Conclusions and possible applications

About the interpretation of the powers appearing in (2), for sure one may state that the truly active portion of the infected people C_{act} si a very particular function of the whole number of infected ones, as:

$$C_{\rm act} \propto C^p$$
.

Similarly, the self-competition term limiting the growth of C according to (2) is a power law in terms of the total of infected persons, i.e. $\dot{C}_{\text{lim}} \propto C^{p+\alpha}$. The possible physical interpretation of p and α , the one I am suggesting in this note, is that those non-integer powers should represent the geometric locus where "contagion reactions" take place.

In the herd behaviour of TWT, real powers of population variables represent a measure of the physical places where predators and preys meet, or where competition takes place, but here such powers must be attributed a more subtle meaning. While animals in the savannah move in a 2-dimensional space, see (9), so that one could state dim $\mathbb{E} = 2$, and dim $\mathbb{E}_{act} = 1$ (being \mathbb{E}_{act} the locus where preys can be caught by predators, the border of prey groups), things are different for humans infecting each other. The locus \mathbb{E} "where infected humans live" must be understood as a subset of the place where people live, work and move, i.e., of the network of inhabited centers and the links connecting them. Let us put dim $\mathbb{E} = \nu$ (consider this is far from being easily defined). Moreover, a sub-locus of this \mathbb{E} , i.e. where contagion events really take place, is indicated as \mathbb{E}_{act} : attributing a value dim $\mathbb{E}_{act} = \mu \leq \nu$ to the "dimension" of \mathbb{E}_{act} means understanding which part of the total infected people is really in contact with susceptible ones, being able to "produce new infectious people". Once this \mathbb{E}_{act} is identified, its "size" should be expressed as a function of the "size" of the whole \mathbb{E} , so to be able to write the expression $C_{act}(C)$: provided things work as in the herd behaviour case, i.e. provided \mathbb{E}_{act} is a *non-space-filling subset* of \mathbb{E} , hence of Hausdorff dimension smaller than dim \mathbb{E} , one may state

$$C_{\rm act} \propto C^{\frac{\dim \mathbb{E}_{\rm act}}{\dim \mathbb{E}}} = C^{\frac{\mu}{\nu}} \stackrel{\text{def}}{=} C^p.$$
(15)

The explanation for the case of $\alpha = p$ is just given by the foregoing assumption (15), while, in order to understand the case $p < \alpha = p + \delta$ one may either think that the "active" contagious people C_{act} will interact with "slightly" more infected people than themselves alone, so that the limiting locus will be $\mathbb{E}_{\text{lim}} \supset \mathbb{E}_{\text{act}} / \dim \mathbb{E}_{\text{lim}} = \alpha \nu$: this gives rise to the competition term $-\frac{r}{K^{\alpha}}C^{p} \cdot C^{\alpha}$ in the ODE; or think that the coefficient of C^{2p} term in a "regular" competition term with a coefficient depending on C explicitly, as $\dot{C}_{\text{lim}} = -\frac{rC^{\alpha-p}}{K^{\alpha}}C^{2p}$, be this a competition strength growing as $\mathbb{O}(C^{\alpha-p})$, or a carrying capacity decreasing as $\mathbb{O}(C^{p-\alpha})$.

The great question is, then, how to compute $\dim \mathbb{E}$, $\dim \mathbb{E}_{act}$ and $\dim \mathbb{E}_{lim}$, provided it makes sense at all to represent the behaviour of infected humans and of the contagion via fractal geometrical loci. In this vision, the locus $\mathbb E$ should depend on the human behaviour and society: in particular, it must retrace the locus where people are concentrated, i.e. the web of the inhabited centers and communications \mathcal{H} . As a fantasy, we could say dim $\mathcal{H} = \rho \leq 2$, since we can at most occupy the 2-dimensional surface of a country: so $\mathbb{E} \subseteq \mathcal{H}$ will mean $\nu < \rho < 2$. An indication about dim \mathcal{H} , in agreement with the arguments here, may be found in [12], for example. When one goes from dim $\mathbb{E} = \nu$ to the value of dim \mathbb{E}_{act} , things become more complicated, because now we have to consider not only the distribution and behaviour of humans, but also the "contagion dynamics", a contribution given by the nature of the virus. For COVID-19, the contagion seems to take place via rather close contact, so that particles of the breath of an infected person are received by the susceptible individual: one may imagine to select \mathbb{E}_{act} considering the sub-locus of \mathbb{E} of the close contacts of the single individual, i.e. possibly the "network of personal relationships" and "of casual encounters". Possibly, this will give dim $\mathbb{E}_{act} = \mu \leq \nu$, and hence p. Similar considerations will lead to figuring out what α could be.

As it is understandable from the aforementioned arguments, "predicing" the numbers ν , μ , p and α from what we may study about the distribution and communications of humans, their relationship networks, and from what we know about the behaviour of COVID-19, will be a very tough interdisciplinary task. What one can say by intuition is that, as the locus \mathbb{E}_{act} is more sparse, the behaviour of the outbreak C(t) will be slower and slower. For instance: considering $K = 15 \times 10^4$ and $r = 0.8 \text{ people}^{1-p} \cdot \text{day}^{-1}$, the curve C(t) solving the ODE with $p = \alpha = 1$ is the one illustrated in Figure 1: looking at that plot, one sees that the maximum value of infected people is reached in practice between the 20th and the 30th day. If one puts, instead, p = 0.7 and $\alpha = 1$, the result is that of Figure 2: in this case, we see that the value $C \simeq K$ is reached not before t = 140 days, i.e. the growth is much slower as p decreases.

The slowness of C(t) towards K, increasing with decreasing p, teaches that, with smaller p, the outbreak of contagion gives much more time to the public



Figure 1: A curve solving the ODE (1), i.e. with $p = \alpha = 1$ and $r = 0.8 \text{ day}^{-1}$. The initial value of infected individuals is C(0) = 100.



Figure 2: A curve solving the ODE (1), i.e. with p = 0.8 and $\alpha = 1$, and $r = 0.8 \text{ people}^{0.2} \cdot \text{day}^{-1}$. The initial value of infected individuals is C(0) = 100.

healthcare administration to take anti-contagion measures. The faster the reach of K is, the more crowded the hospitals will be, the more difficult will be to assist ill people, and the larger the number of dead can be, and this can be mitigated acting precisely on p. Clearly, also acting on α may modify the shape of the curve C(t).

Provided equation (2) describes COVID-19 outbreak with time, acting on the exponents in (2) may regulate the time given to a national healthcare administration to confront it. As argued before, the exponents p and α depend on the physical distribution of people, on their behaviour and on their relationship network: under this point of view, one has to hope that the lockdown meaures taken by many Governments are acting in the direction of diminishing p, and increasing δ in (13), so to render less and effective the "creation" term rC^p and more and more rapid the "cancellation" term $-\Delta_{\text{eff}}(C) C^{2p}$.

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