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# Some fractal thoughts about the COVID-19 infection outbreak

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ABSTRACT

Some ideas are presented about a geometric motivation of the apparent capacity of generalized logistic equations to describe the outbreak of quite many epidemics, possibly including that of the COVID-19 infection. This interpretation pivots on the complex, possibly fractal, structure of the locus describing the "contagion event set", and on what can be learnt from the models of trophic webs with "herd behaviour".

Under the hypothesis that the total number of cases, as a function of time, is fitted by a solution of the Generalized Richards Model, it is argued that the exponents appearing in that differential equation, usually determined empirically, represent the geometric signature of the non-space filling, network-like locus on which contagious contacts take place.



## 1. Introduction

In these days of worldwide mourning for the human tragedy due to the COVID-19 pandemic, while experiencing a heavy lockdown, and the fear of a possible forthcoming economical and social crisis, I think every scientist is reasoning about how to be of help, in terms of 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. I wrote down these thoughts when I went through the paper [1], in which the authors perform a deep investigation of the behaviour with time of the "total number $C(t)$ of people who have been 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. It is important to stress that [1] appeared on March 12, 2020, so the data those authors could use were just the ones available at that stage of the epidemics: all the reasonings about their finding should be understood to describe the contagion dynamics up to that time.

What has attracted my attention in [1], among the various results reported, is the apparently good performance, in fitting the contagion data, of the law $C(t)$ solving the Generalized Richards Model (GRM) [2]; the latter is a modification of the well known logistic equation

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

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

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

(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, the question here is which "first principle meaning" should be given to those powers $p$ and $\alpha$ in (2). In the literature, those parameters are determined empirically, so to fit the experimental curve $C(t)$ a posteriori, once the epidemic is completely developed. Some study noticeably put the $(p, \alpha)$ 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 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" appear to be what gives the *contagion network* 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 $C(t)$, as in classical dynam-

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ics of systems with complex structures in phase space. I am myself involved, together with Giuseppe Consolini, in a study searching for an ODE possibly solved by the best fit of the COVID-19 curve in Italy [5]: indeed, the effects of dynamics with finite waiting time (possibly encoding a complex phase space topology) appear to be relevant.

All in all, it is clear that deviations of the equation for $C(t)$ 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, in order for $C$ to satisfy (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]: it is assumed 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 the population. This, roughly, gives rise to integer powers of population variables in the ODEs. The argument here is that Eq. (2) may 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 a violation of the mass hypothesis take place? The answer suggested here in inspired by the *Trophic Web Theory* (TWT), where the dynamics of interacting populations in ecosystems are represented via coupled ODEs. In TWT 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 equations with non-integer powers, exactly as in the GRM, so popular in epidemiology, and that the authors of [1] claim to fit the curves of the COVID-19 epidemic. Here the 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] (it may be of some use to stress that the term "herd behaviour" here has nothing to do with the expression "herd immunity" of epidemiology. Moreover, no obvious relationship between the two "herd things" appear).

The paper is organized as follows.

In Section 2 the logistic ODE is recalled, and its non-integer power generalization is presented. In Section 3 the concept of herd behaviour in TWT is treated, with some examples from ecology and ecoepidemic models. Then, its interpretation of the parameters $p$ and $\alpha$ in (2) is presented.

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

## 2. Logistic and generalized logistic equations

As stated in the Section 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 $C_{\rm eq}$ so that $\dot{C}(C_{\rm eq})=0$, that is realized for $C_{\rm eq}=K$: 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

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

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): we have a population dynamics in which all the $C$ individuals take part to both the exponetial growth and the self-limitation. In a chemio-kinetic framework, the term $rC$ would mean that, for each of the $C$ units, one more unit will be "created", in a "reaction" of the form $\mathrm{C}\longrightarrow\mathrm{C}+\mathrm{C}$, every $\Delta t_{+1}=\frac{1}{r}$ units of time; meanwhile, the term $-\frac{r}{K}C^2$ would mean that, whenever each of the $C$ individuals meets another one of the $C$ individuals, one individual is destroyed in a process $\mathrm{C}+\mathrm{C}\longrightarrow\mathrm{C}$, that takes place every $\Delta t_{-1}=\frac{K}{rC}$ units of time (this $\Delta t_{-1}$ becomes smaller and smaller as the amount $C$ increases, making the frequency of destructive matches grow). Using (3) to describe the total-cases-up-to-now in an epidemic, the sensible interpretation of $-\frac{r}{K}C^2$ is that the $rCdt$ new cases produced by the $C$ individuals in the $dt$ should be diminished by a term $\Delta C^2 dt$ proportional to the number of matches of two infected people, in which no new infected one is produced, being $\Delta=\frac{r}{K}$. $r$ is the effectiveness of the $+1$ production process, while $\frac{r}{K}$ measures the effectiveness of the $-1$ destruction process (here, better understood as "failure of $+1$ process"). Expression (3) is also referred to as *Verhulst Equation* (VE) [8].

It is important to underline the relationship between the mass hypothesis and the mathematical nature of (3). Indeed, the fact that the creation rate reads $rC$ means that *all of the C individuals of the population do take part to the production process*; in the same way, the expression $-\frac{r}{K}C^2$ for the limitation rate means that *there is a possible "failure to spread" 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 limits the contagion activity of *all* its fellows.

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

$$\dot{C}=rC^p-\frac{r}{K^\alpha}C^{p+\alpha}\ : \tag{4}$$

we now have a production term $rC^p$, with $p<1$, and a self-competition term $-\frac{r}{K^\alpha}C^{p+\alpha}$, with $p+\alpha<2$. This expression (4) is the one suggesting the interpretation presented here, in terms of herd behaviour.

## 3. Herd behaviour and fractals

To introduce the concept of herd behaviour in TWT, let us consider two populations $X$ and $Y$, respectively of prey and predators, living on a surface, e.g. the savannah, 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

$$\dot{X}_Y=-kXY \tag{5}$$

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

$$\dot{X}_Y=-\frac{h}{b+X}XY, \tag{6}$$

if the model is more sophisticated and a Holling Type II response function is adopted to describe predation, as in [9] ($k$, $h$ and $b$ are constants). In $\dot{X}_Y(X,Y)$ the number of prey and predators appears to the first power: in (5), each of the $X$ prey units may "couple" with each of the $Y$ predators with the same destruction rate $k$; in (6), this happens, but with a rate $\frac{h}{b+X}$ decreasing with the total amount of prey. Under the idea that *every prey can be reached by every predator*, there is clearly the mass hypothesis discussed in Section 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 us suppose that the $X$ prey units move in compact groups of finite size, that cannot be penetrated by predators: each of the $Y$ predators can only pick their prey from the border of those groups. The right hypothesis is, then, not that each prey is attacked by each predator, but that just the $X_\partial$ prey units 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 prey units really involved in this predation, the $X_\partial$ ones sitting on the group border. Because the

scene is 2-dimensional, under the hypothesis of homogeneos surface density (a necessary one, in order 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" $\ell$, clearly the surface has a measure $A=\mathbb{O}\left(\ell^2\right)$, so that $\ell=\mathbb{O}\left(X^{\frac{1}{2}}\right)$, while the perimeter scales as $P=\mathbb{O}(\ell)$: one may conclude

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

As the prey 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 outside these groups, the predation terms in (5) and (6) will be re-written as

$$\dot{X}_Y=-k'\sqrt{X}Y \text{ and } \dot{X}_Y=-\frac{h'\sqrt{X}}{b'+\sqrt{X}}Y \tag{7}$$

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 prey and predators move in $\mathbb{R}^3$, as it happens to nekton animals in the sea, then one may state $X=\mathbb{O}\left(\ell^3\right)$ and $X_\partial=\mathbb{O}\left(\ell^2\right)$, so that the predation terms (7) will read:

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

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

$$\dot{X}_Y=-\hat{k}X^\eta Y,\quad \dot{X}_Y=-\frac{\hat{h}X^\eta}{\hat{b}+X^\eta}Y,\quad \eta=\frac{m}{n}\leq 1 \tag{8}$$

will appear in the prey population ODE. Note that, in the expressions from (5) to (8), a mass hypothesis is still active on predators, that are supposed to be "very mobile" and "enough mixed" outside the groups of prey. If, instead, also the predators are scarcely mobile or slow, possibly packs of predators interact with herds of prey just via their borders, so that, for instance, one should write

$$\dot{X}_Y=-\tilde{k}\sqrt{XY} \text{ and } \dot{X}_Y=-\frac{\tilde{h}\sqrt{XY}}{\tilde{b}+\sqrt{X}} \tag{9}$$

instead of (7), and so on. The introduction of terms due to herd behaviours in population ODEs is studied in a wide literature, ranging from more properly ecological system theory, as in [11–18], where applications to epidemiology are however suggested; to more strictly epidemiological cases, as in [19–21]. Very recently [22], the use of terms as in (9) has been introduced to describe competition between populations of algae, that live on the 2-dimensional seabed and show border competition and border grazing by their "predators".

Applying these concepts to the epidemic growth given by Eq. (4) 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 those described in Section 2: if all the individuals living in $\mathbb{E}$ take part to both the creation and the limitation processes, clearly the Verhulst Eq. (3) will rule the evolution of $C$. 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 $\mathbb{E}_{\text{act}}\subset\mathbb{E}$, so that $\dim\mathbb{E}_{\text{act}}=\mu\leq\nu$. It is straightforward to show that the active portion of population is

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

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

$$\dot{C}=rC^p-\frac{r}{K^p}C^{2p}, \tag{10}$$

that is precisely the same as (4), or (2), with $\alpha=p$.

Now, what if $\alpha\neq p$ in (4)? If one has $\alpha>p$ as in [1], it is sensible to put $\alpha=p+\delta$, with $\delta>0$, and then to re-write (4) as:

$$\dot{C}=rC^p-\frac{rC^\delta}{K^{p+\delta}}C^{2p}. \tag{11}$$

The only true difference between this case and the ODE (10) is 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 limiting process depends on the population itself* as

$$\Delta_{\text{eff}}(C)=\frac{r}{K^{p+\delta}}C^\delta, \tag{12}$$

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

$$K_{\text{eff}}(C)=\frac{K^{p+\delta}}{C^\delta}, \tag{13}$$

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 (10) is that in (4), and hence in (11), the rush towards the limit $C=K$ gets slower and slower, with respect to the logistic ODE tempo, while the total population $C$ is increasing).

Another possibile interpretation of the self-competition term in (4) could be that, next to the infected people able to infect the others, i.e. $C_{\text{act}}=\mathbb{O}(C^p)$, there is a class of infected people with whom those $C_{\text{act}}$ come in contact uneffectively, that is some $C_{\text{lim}}=\mathbb{O}(C^\alpha)$*limiting* the contagion, as $-\frac{r}{K^\alpha}C^pC^\alpha$. In this vision, one should define some geometric locus $\mathbb{E}_{\text{lim}}$, with Hausdorff dimension $\alpha\nu$, to which the uneffective contacts are restricted (it is very likely that $\mathbb{E}_{\text{act}}\cap\mathbb{E}_{\text{lim}}\neq\emptyset$ holds).

Now, the crucial point is *to understand why* the COVID-19 contagion growth, together with that of 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 Section 4.

## 4. Conclusions and possible applications

About the interpretation of the powers appearing in (2), for sure one may state that the portion of the infected people $C_{\text{act}}$, truely active in spreading the contagion further, is a very particular function of the whole number of infected ones, as:

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

Similarly, the self-competition term limiting the growth of $C$ according to (2) is a power law of the total of infected persons, i.e.

$$\dot{C}_{\text{lim}}\propto C^{p+\alpha}.$$

The possible interpretation of $p$ and $\alpha$ suggested here is that those non-integer powers should represent *the geometric locus where "contagion reactions" take place*.

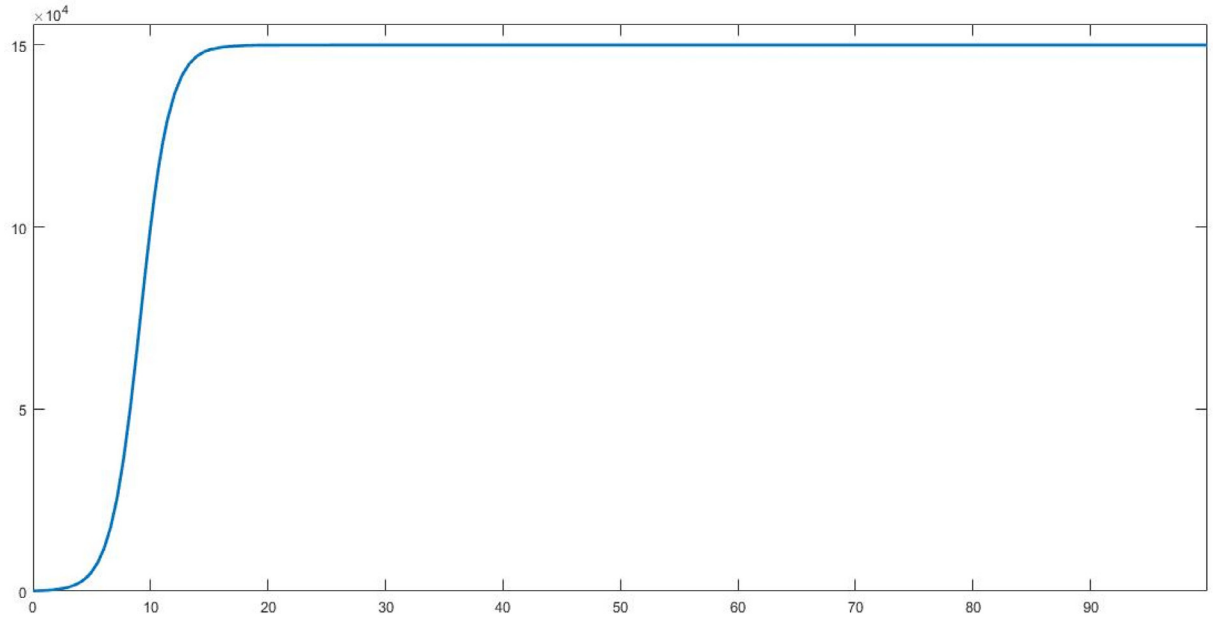


**Fig. 1.** A curve solving the ODE (1), i.e. Eq. (2) with $p = \alpha = 1$, and with $r = 0.8\text{day}^{-1}$. The initial value of infected individuals is $C(0) = 100$, with a carrying capacity of $K = 15 \times 10^4$.

In the herd behaviour of TWT, real powers of population variables represent a measure of the physical places where predators and prey 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 (7)), so that one could state $\dim \mathbb{E} = 2$, and $\dim \mathbb{E}_{\text{act}} = 1$, 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 $\dim \mathbb{E} = \nu$ (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 $\mathbb{E}_{\text{act}}$: attributing a value $\dim \mathbb{E}_{\text{act}} = \mu \leq \nu$ to the "dimension" of $\mathbb{E}_{\text{act}}$ 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 $\mathbb{E}_{\text{act}}$ 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 $C_{\text{act}}(C)$: provided things work as in the herd behaviour of TWT, i.e. provided $\mathbb{E}_{\text{act}}$ is a *non-space-filling subset* of $\mathbb{E}$, hence with a Hausdorff dimension smaller than $\dim \mathbb{E}$, one may state

$$C_{\text{act}} \propto C^{\frac{\dim \mathbb{E}_{\text{act}}}{\dim \mathbb{E}}} = C^{\frac{\mu}{\nu}} = C^p. \tag{14}$$

The explanation for the case of $\alpha = p$ is just given by the foregoing assumption (14). In order to understand the case $p < \alpha = p + \delta$ one may either think that the "active" contagious people $C_{\text{act}}$ will interact with "slightly more" individuals 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 is 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 $\dim \mathbb{E}$, $\dim \mathbb{E}_{\text{act}}$ and $\dim \mathbb{E}_{\text{lim}}$, 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 $\nu \leq \rho \leq 2$. An indication about $\dim \mathcal{H}$, in agreement with the arguments here, may be found in [23], for example. When one goes from $\dim \mathbb{E} = \nu$ to the value of $\dim \mathbb{E}_{\text{act}}$, 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 $\mathbb{E}_{\text{act}}$ 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 $\dim \mathbb{E}_{\text{act}} = \mu \leq \nu$, and hence $p$. Similar considerations will lead to figuring out what $\alpha$ could be.

As it is understandable from the aforementioned arguments, "predicting" the numbers $\nu$, $\mu$, $p$ and $\alpha$ 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 $\mathbb{E}_{\text{act}}$ 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 Fig. 1: looking at that plot, one sees that the maximum value of infected people is reached in practice between the $20^{\text{th}}$ and the $30^{\text{th}}$ day. If one puts, instead, $p = 0.7$ and $\alpha = 1$, the result is that of Fig. 2: in this case, we see that the value $C \simeq K$ is reached not before $t = 140\text{days}$, 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 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 $\alpha$ may modify the shape of the curve $C(t)$.

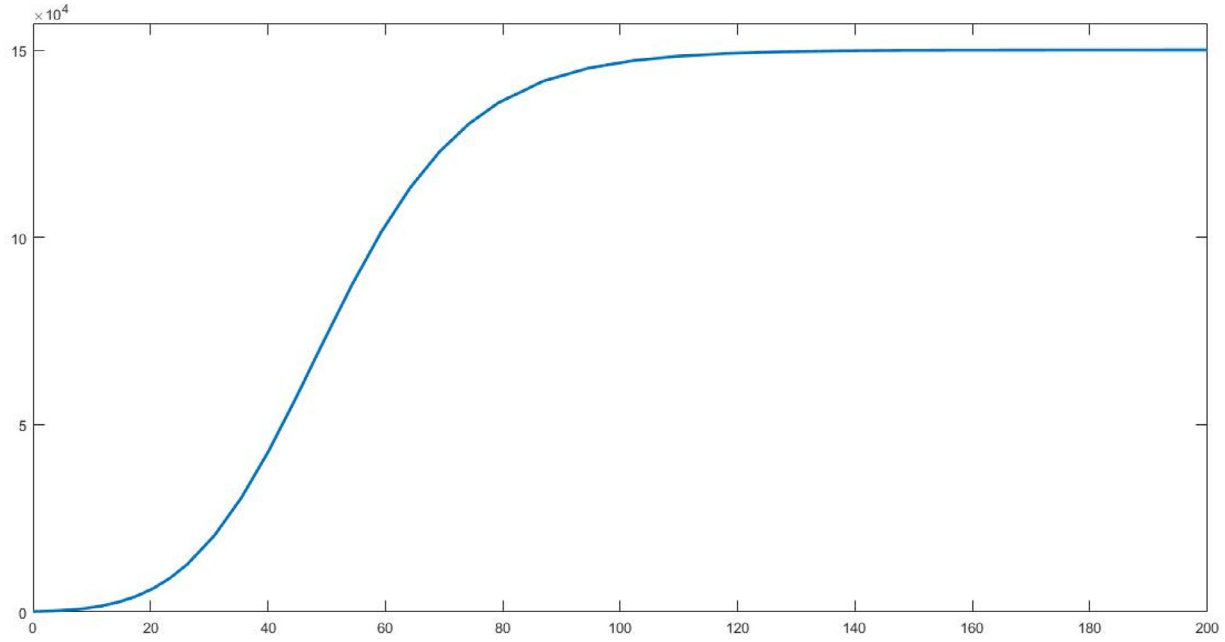


**Fig. 2.** A curve solving the ODE (2) with $p = 0.8$, $\alpha = 1$ and $r = 0.8\text{people}^{0.2} \cdot \text{day}^{-1}$. The initial value of infected individuals is $C(0) = 100$, with a carrying capacity of $K = 15 \times 10^4$.

Provided Eq. (2) describes an epidemic 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 $\alpha$ 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 $\delta$ in (12), so to render less effective the "spreading" term $rC^p$ and more and more important the "liming" term $-\Delta_{\text{eff}}(C)C^{2p}$.

## Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## CRediT authorship contribution statement

**Massimo Materassi:** Conceptualization, Methodology, Software, Writing - original draft, Investigation, Writing - review & editing.

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