MOMENTUM MANAGING EPIDEMIC SPREAD
AND BESSEL FUNCTIONS

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Abstract. Starting with the power law for the total number of infections during the middle stages of epidemics, we propose differential equations describing the process of momentum epidemic management, which is a set of measures aimed at reducing the epidemic spread via timely response to the dynamic of the number of infections. In the most aggressive mode, the saturation of the number of infections can be achieved sufficiently quickly, though it can be not the end of the epidemic. The square root of the intensity of hard measures is qualitatively inversely proportional to the epidemic duration. We use the theory of Bessel functions. For Covid-19, they appeared surprisingly efficient for modeling the whole period of intensive spread, presumably including the late stages; we discuss the USA, UK, Austria, Israel and Sweden.

Key words: epidemic spread, epidemic psychology, Bessel functions
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1. Our approach and findings. In this work, we propose a system of differential equations describing the effects of momentum management of epidemics, which can be defined as a system of measures of any nature aimed at reducing the epidemic spread by regulating the intensity of these measures on the basis of the latest absolute or average numbers of infections. The usage of the absolute numbers here is combined with "hard measures"; detection and isolation of infected people and closing the places where the spread is the most likely are the key. We demonstrate mathematically that the epidemic will reach some saturation under such management, which may be followed by a period of linear growth and even result in the recurrence. Our approach seems the first one where the cornerstone is momentum epidemic management. Bessel functions play the key role here; they model very well almost all periods of the intensive epidemic spread, presumably including the late stages. As an application we suggest a forecasting tool
for the spread Covid-19; the USA, UK, Austria, Israel and Sweden are considered. Figures 1 and 7 presents this tool for the USA data.

A somewhat different approach is required for the preventive measures like wearing the protective masks and social distancing. The impact of such and similar "soft" measures can be significant for reducing the transmission rate, but they alone are insufficient for ending epidemics. These are mathematical findings, but they match well real-life observations and expectations. For instance, we demonstrate that the square root of the intensity of hard measures is essentially inversely proportional to the duration of the epidemic. A more exact estimate is based on Bessel functions.

**Classical theory.** The simplest equation for the spread of communicable diseases results in exponential growth of the number of infections, which is mostly applicable to the initial stages of epidemics. We focus on the middle stages, where the growth is no greater than some power functions in time, which requires different equations.

The equally classical logistic equations for the spread, as well as the SIR and SID models, assume that the number of infections is comparable with the whole population, which do not impose. The spread of this kind was not really the case with the epidemics we faced during the last 100 years, even when the numbers of infections were big. This is obviously due to better disease control worldwide.

Anyway, the reality now is the power-type growth of epidemics, at least during their middle stages. This must be the starting point of any analysis if we want our mathematical models to be up to date, the challenges with the latest Covid-19 included. Our approach is entirely based on this assumption: power law of epidemics of some sort.

**Beyond epidemics.** There is a strong connection of our approach with behavioral science, including behavioral finance. We actually closely follow paper [Ch] devoted to momentum risk-taking with momentum investing as the main application. For instance, the aggressive management of type (A) from Section 2 is almost a direct counterpart of profit taking from Section 2.6 of this paper. The measures of type (B) are parallel to the investing regimes discussed in Section 2.4. The corresponding differential equations are essentially the same. The key link to financial mathematics is that the price function from [Ch] is a counterpart of the protection function in the present paper.

Paper [Ch] can be considered as part of a program toward general purpose artificial intelligence, the most difficult and ambitious among all AI–related research directions. Momentum risk-taking is a very general concept. We even argue in [Ch] that the mathematical mechanisms we propose can be present in neural processes in our brain, which would explain their universality. See Section 1.4, including an instructional example of self-driving cars. Generally, this is about the ways
our brain processes information and manages risks real-time, which is certainly applicable to behavioral aspects of epidemics.

2. Two kinds of management. There is a long history and many aspects of mathematical modeling epidemic spread; see e.g. [He] for a review. We restrict ourselves only with the dynamic of momentum managing epidemics, naturally mostly focusing on the middle stages, when our actions must be as precise as possible. The two basic approaches we consider are essentially as follows:

(A) aggressive enforcement of the measures of immediate impact reacting to the current absolute numbers of infections and equally aggressive reduction of these measures when these numbers decrease;

(B) a more balanced and more defensive approach when mathematically we react to the average numbers of infections to date and the employed measures are mostly of indirect and palliative nature.

Hard and soft measures. To clarify, an action of type (A) can be a prompt detection and isolation of infected people and those of high risk to be infected, closing factories and other places where the spread is the most likely and so on. The measures like wearing protective masks, recommended self-isolation, restrictions on the size of events and social distancing are typical for (B). This distinction heavily impact the differential equations we propose. However the main difference between the modes, (A) vs. (B), is the way the number of infections is treated: the absolute number of infections is the trigger for (A), whereas the average number of infections to date is what we monitor under (B).

Generally, the (A)—type approach provides the fastest possible and "hard" response to the changes with the number of infections, whereas we somewhat postpone with our actions until the averages reach proper levels under (B), and the measures we implement are "softer". Mathematically, the latter way is slower but better protected against stochastic fluctuations, but (B) alone cannot lead to the termination of the epidemic, which we justify mathematically within our approach. So these modes significantly influence the results of the management.

There is almost a direct analogy with investing, especially with profit taking. Practically, a trader either directly uses the price targets or prefers to rely on the so-called technical analysis, based on some chart averages. Let us emphasize that by "actions", we means not only those by the authorities in charge of the epidemic. Our own individual ways of reacting to epidemic figures are essentially no different. Epidemiology has very strong roots in behavioral science, psychology, sociology, mass and collective behavior; see e.g. [St].

Focus on the "periodicity". Both modes, (A) and (B), are momentum, since they are entirely based on the current trend, but they are quite
different qualitatively and quantitatively. As we will see, both provide essentially the same kind of power growth of the spread, approximately \( t^{c/2} \) in terms of time \( t \) for the initial transmission coefficient \( c \). The main difference is that \((A)\), significantly more aggressive than \((B)\), results in *quasi-periodicity* of the corresponding solutions. By quasi-periodic, we mean an asymptotically periodic function in \( t \) multiplied by its *envelope*, which will be a power function in this paper. This type of periodicity is not related to periodicity models based on seasonal factors, various delays and other mechanisms of this nature; see e.g. [HL]. Our one is entirely due to active *momentum* management.

The main objective of any managing epidemics is to end them. However the termination based on such (any) aggressive ”interference” can lead to the recurrence of the epidemic too: the ”quasi-periodicity” may begin working ”against us”. This can be avoided only if we continue to stick to the prevention measures as much as possible even when the number of new infections goes down significantly. Reducing them too much on the first signs of improvement is a way to the recurrence of the epidemic, which we see mathematically within our approach.

3. Power law of epidemics. Here and below we will assume that the number of people perceptive to the virus is unlimited, i.e. we do not take into consideration in this paper any kind of saturation when the number of infected people is comparable with the whole population. Accordingly, *herd immunity* and similar factors are not considered. Also, we disregard the average duration of the disease and that for the quarantine periods imposed; the total number of infections regardless of the output is what we are going to model, which is commonly used.

For any choice of units, days are the most common, let \( U_n \) be the number of infected individuals at the moments \( n = 0, 1, 2, \ldots \). The simplest equation of the epidemic spread and its solution are:

\[
U_n - U_{n-1} = \sigma U_{n-1} \quad \text{for} \quad n = 1, 2, \ldots, \quad \text{and} \quad U_n = C(1+\sigma)^n
\]

for some constant \( C \) and the intensity \( \sigma \). Here \( \sigma \) is the *transmission rate*, which is the number of infections transmitted by an average infected individual during 1 day (or other time-unit), assuming that the ”pool” of non-infected perceptive people is unlimited. So this model results in exponential growth of the number of infections, which can be practically present only at relatively early stages of epidemics, especially with the epidemics we faced during the last 100 years.

**Some biological aspects.** The *viral fitness* is an obvious component of \( \sigma \). Its diminishing over time can be expected, but this is involved. This can happen because of the virus replication errors, especially typical for *RNA* viruses, which are of highly variable and adaptable nature. The *RNA* viruses, *Covid-19* included, replicate with fidelity that is...
close to error catastrophe. See e.g. [CJLP, Co] for some review, perspectives and interesting predictions. Such matters are well beyond this paper, but one biological aspect must be mentioned, concerning the asymptomatic cases.

The viruses mutate at very high rates. They can ”soften” over time to better coexist with the hosts, though fast and efficient spread is of course the ”prime objective” of any virus. Such softening can result in an increase of asymptomatic cases, difficult to detect. So this can contribute to diminishing \( \sigma \) we observe, though this is not because of the decrease of the spread of the disease. We model the available (posted) numbers \( U_n \), which mostly reflect the symptomatic cases.

To summarize, it is not impossible that the replication errors and ”softening the virus” may result in diminishing \( \sigma \) at later stages of the epidemic, but we think that the reduction of the contacts of infected people with the others dominates here, which is directly linked to behavioral science, sociology and psychology. This is different for animal epidemics, though there are some behavioral aspects there too.

Transmission rate psychologically. When reducing our contacts during an epidemic, we rely on the latest posted number of infections, which is \( U_{n-1} \); the response of those infected (or suspecting to be infected) is the key here. Our claim is that \( U_{n-1}/(n-1) \) is more likely to be the actual trigger for us than \( U_{n-1} \). Indeed, knowing the trend is what we need to decide. Generally, \( (U_{n-1} - U_{n-2}) \) gives it, but it may be not what we really ”calculate” in this and similar decision-making situations.

First, this difference (or the corresponding derivative in continuous setting) is poorly protected against the random fluctuations of \( U_n \), which can be extreme. Though the pieces of news like ”the biggest daily increase ever” are of course indicative for the trend.

Second, momentum decision-making rely very much on our instincts. Storing and processing the prior \( U \)-numbers in our brain is more involved than ”keeping” \( U_{n-1}/(n-1) \), an almost perfect substitute for the corresponding differences or derivatives in such and similar ”cases”.

Last but not least, the actions of infected people or those who think they can be infected certainly become less chaotic over time; also, they can receive better treatment at later stages. This undoubtedly reduces the transmission of the virus too.

Within our brain, we actually do not need to ”divide by time” with this information of that of similar sort. Our brain can deal directly with the input like ”yesterday number is \( U_{n-1} \) and this took \( (n-1) \) days”, and with much more complex data. The mechanisms here are complicated, but processing such information (involving time or other ”denominators”) consciously or subconsciously is not a problem at all, and not only for humans. This is built-in at the level of instincts.
Master equation revisited. We conclude that a reasonable equation describing the middle stages of epidemics and the corresponding asymptotic growth of its solutions are as follows:

\begin{equation}
U_n - U_{n-1} = \frac{\sigma}{n-1} U_{n-1}, \ n = 1, 2, \ldots, \ U_n \approx Cn^\sigma
\end{equation}

for some constant \( C \). When \( \sigma = 1 \), \( U_n \) is exactly \( Cn \). As we outlined, the main mechanism for this power law is likely to be of behavioral nature, but it can include biological factors too. Needless to say that power laws are fundamental everywhere in natural sciences.

Power-law models for epidemics and the spread of infectious are not really something new; see e.g. [MH]. However, our approach is very different; it describes our response to the epidemic information available. Actually it is not that important in this paper what are the exact reasons for dividing \( \sigma \) by the time, though we suggest some underlying principles. What is the key for us is that the switch from (1) to (2) is a mathematical necessity; the exponential growth is unsustainable.

The power growth can be of course unsustainable long-term too, but the quadratic and then linear growth can last during epidemics, including Covid-19, where both types are clearly present. Let us mention that there are some indications that the ratio between all infected people and those "seriously or and critically ill" during Covid-19 has a tendency to be proportional to \( t \) during the middle periods, but this is far from solid. Thus the \( \sigma/(n-1) \) can be due to some microbiological processes: the virus is probably getting "softer" inversely proportionally to time, but this is very preliminary and qualitative.

Anyway, our focus is on the middle periods, when the precise management is of key importance. The management depends mostly on projections and considerations of global nature in the beginning; and it is relatively straightforward: "restrict what can be restricted".

4. Hard measures. In the realm of differential equations, our starting equation (2) becomes as follows:

\begin{equation}
\frac{dU(t)}{dt} = c \frac{U(t)}{t}, \ \text{where} \ t \ \text{is time and} \ c \ \text{replaces} \ \sigma.
\end{equation}

We apply Taylor formula to \( U_n - U_{n-1} \), so \( c \) is "essentially" \( \sigma \); we will use the most relevant one below depending on the context, mostly \( c \).

The aim is to reduce the "free" spread of the epidemic modeled in (3) by aggressive imposing protection measures. Accordingly, the protection function \( P(t) \) is the output of a measure or measures. It is a counterpart of the price function in [Ch]. Essentially we have two types of measures: (a) isolating infected people, and (b) general diminishing the number of contacts. We will call them "hard" and
"soft". They will correspond to the management modes (A) and (B); see below and Section 2. Let us begin with the "hard" ones.

Protection function. The main "hard" measure is testing and then detection of infected individuals, followed by their isolation. This is hard by any standards, especially if there are many asymptomatic cases and the treatment is unknown as with Covid-19.

Generally, $P(t)$ is the key numerical characteristic of the output of a given measure; its derivative is then the productivity. For "detection & isolation", we are going to use the following natural definition:

$$P(t) = \text{the total number of infected individuals detected till } t.$$ 

The primary measure here is testing; the number $T(t)$ of tests till $t$ is what we can really implement and control. The detection of infected people is its main purpose, but the number of tests is obviously not directly related to the number of detections, i.e. to the number of positive tests. The efficiency of testing requires solid priorities, focus on the groups with main risks, and solving quite a few problems.

Even simple mentioning problems with testing, detection and isolation is well beyond our paper. However, numerically we can use the following. During the epidemics, essentially during the stages of linear growth, which are the key for us, the number of positive tests can be mostly assumed a stable fraction of the total number of tests. When $P(t) = \gamma T(t)$ for some $\gamma$, finding $\gamma$ experimentally is not a problem.

The middle stages of epidemics mostly consist of such linear periods, though it depends. For instance, preliminary data indicate that the initial $c$ for Covid-19 is about 2, i.e. the spread is essentially quadratic before the active management begin. The measures we discuss reduce the growth to linear almost everywhere, which perfectly matches our formula $t^{c/2}$ under type (A) management or that of type (B) for sufficiently large intensity "a". As we have already mentioned, "momentum managing" is mainly present during the middle stages; not much "mathematics" is used in the beginning or at the end of epidemics.

Thus, we are going to assume that the effect of isolating infected people is approximately linear. I.e. an isolated infected individual will not transmit the disease to $(t - t_\star)$ people till the moment $t$ (now) after the isolation at $t_\star < t$. It can be $C(t - t_\star)$, but we make $C = 1$; this is a normalization matter (see below). This kind of "linearization" is very common when composing differential equations. We use that we are "near" the stage of linear growth of epidemics, though this does not mean of course that the solutions of our equations will grow linearly. Even if $c > 1$, this assumption makes sense: the number of "preventions" due to one isolation at $t_\star$ can be smaller than $(t - t_\star)^c$. 


Let the isolations occur at the moments \( \{t_i\} \) from some \( t_0 \) till \( t \) (now). Then these individuals would infect \( \sum (t - t_i) \), which sum must be therefore subtracted from \( U(t) \). Its \( t \)-derivative is exactly \( P(t) \).

Other \( P \)-functions can be used here, but this particular one has a very important feature: \textit{it does not depend on the moments of time when the infected individuals were detected}. If \( P(t) \) depends on \( \{t_i\} \), the required mathematical tools can be much more involved.

The relation resulting from our analysis is as follows:

\[
\frac{dU(t)}{dt} = c \frac{U(t)}{t} - P(t) \quad \text{for type (A).}
\]

More systematically, the minimum of \( (t - t_i) \) and the average isolation period must be taken instead of \( (t - t_i) \), but \textit{we disregard the average duration of the disease and quarantine terms throughout this paper.}

With closing factories, we count the numbers of workers there in the definition of \( P(t) \). Of course only a fraction of the workers can be infected when a factory is closed, but the assumption is that if it continues to operate, then almost everyone there can be expected to become infected. This is an obvious simplification. The \textit{exact} effect of closing (and then reopening) factories and other places where fast spread of the disease can be expected is almost impossible to estimate.

So simplifying assumptions are necessary here; there are too many factors involved with any employed measures, and they are mostly of stochastic nature. In (i), (ii) below, we will address this as follows: instead of using a specific \( P(t) \) we will ”postulate” its natural properties.

We emphasize that the measures of type (A) impact \( U(t) \) in complex ways: isolating one individual prevents a \textit{ramified} sequence of transmissions. The ”soft” measures of type (B), to be considered next, are simpler: they result in a kind of reduction of the \( c \)-coefficient.

\textbf{5. Soft measures}. These measures are different from ”hard” ones. Wearing protective masks is a key preventive measure of this kind. Social distancing considered mathematically is of similar type. Now:

\( P(t) = \text{the number of infected people who began wearing the masks before } t \text{ multiplied by the efficiency of the mask and the } c \text{-coefficient.} \)

Using the masks for the whole population, infected or not, ”simply” changes \( c \); this is not ”momentum management”. Let \( V(t) \) be the number of infected people wearing the masks. Then \( V(t) \leq U(t) \) and

\[
\frac{dU(t)}{dt} = c \frac{U(t) - V(t)}{t} + c' \frac{V(t)}{t} = c \frac{U(t)}{t} - (c - c') \frac{V(t)}{t}
\]
for \( c - c' = c(1 - c'/c) = c\kappa \), where \( \kappa \) is the mask efficiency. For instance, \( \kappa = 1 \) if \( c' = 0 \), i.e. if the mask is fully efficient for infected people who wear it. The same consideration works for \textit{social distancing}, with \( \kappa \) being the efficiency of the corresponding distance.

An instructional example is when we assume that the fraction of those wearing the masks among all infected people is fixed. If \( V(t) = \nu U(t) \) for some \( 0 \leq \nu \leq 1 \), then we have:

\[
\frac{dU(t)}{dt} = c(U(t) - \nu U(t))/t + c'\nu U(t)/t = (c - \nu(c - c'))U(t)/t.
\]

i.e. this measure results in fact in a recalculation of the \( c \)-coefficient under the proportionality assumption.

We see that the output of "soft" measures is heavily based on probabilities. However, we only need the following: the greater intensity of any measure the greater the reduction of new infections. So we practically control the efficiency of a measure by increasing or decreasing its intensity, even without knowing the exact mechanisms of its impact. This is what we are going to formalize now.

\textbf{General approach.} From now on, the \textit{intensity} of a measure or several of them will be the main control parameter. As we already discussed, the greater the intensity, the greater the number of isolated infected individuals in (A) or the number of infected people who use the masks in (B). If this dependence is of linear type, which can be expected, the corresponding coefficient of proportionality is sufficient to know. Respectively, the exact definition of the protection function \( P(t) \) is not really needed to compose the corresponding differential equations. What we really need is as follows:

\( (i) \) the usage of \( P \) reduces \( \frac{dU(t)}{dt} \), possibly with some coefficient of proportionality, by \( P(t)/t \) for mode (B), the average of \( P \) taken from \( t = 0 \), or directly by \( P(t) \) under the most aggressive mode (A);

\( (ii) \) the derivative \( \frac{dP(t)}{dt} \), the intensity, is proportional to \( U(t)/t \) under (B), the average number of infections from \( t = 0 \), or directly to \( U(t) \) in the most aggressive variant, which is (A) considered above.

Item (i) has been already discussed. Let us clarify (ii), which determines the \textit{intensity of the measures} \( \frac{dP(t)}{dt} \) we employ. It must be proportional to the current number of infections for (A) or its average, \( U(t)/t \), for (B). These are the most natural choices, practically and theoretically. Indeed, the effect of the current number of infections to our actions can be either direct or via some averages. If the averages are used, then actually there are not many mathematical choices. The key challenge is of course: which way to respond to this number leads to the end the epidemic? A mathematical answer is: mode (A) or the transitional mode \((AB)\) defined as follows.
Let us employ "hard" measures as in (A), however following less aggressive management formula for \( dP(t)/dt \) from (B). I.e. this is a combination of (i) for (A) with (ii) for (B), some transitional mode. As we will see, the epidemic will end then, but the time to "saturation" can be significantly longer than under (A).

6. Type (B) management. Let \( t_0 \) be the starting point of the management; so we can assume that \( P(t_0) = 0 \). The following normalization is somewhat convenient: \( u(t) \overset{\text{def}}{=} U(t)/U(t_0) \) and \( p(t) \overset{\text{def}}{=} P(t)/U(t_0) \), i.e. \( u(t_0) = 1 \) and \( p(t_0) = 0 \). Recall that \( U(t) \) and \( P(t) \) are the number of infections at the moment \( t \) and the corresponding value of the \( P \)-function. Here \( P \) is naturally the total of all measures in the considered mode, the sum of the corresponding \( P \).

Recall that mode (A) has no protection against stochastic fluctuations and is significantly more aggressive than (B). Philosophically, the smaller interference in natural processes the better: the results will last longer. But with epidemics, we cannot afford waiting too long.

Type (B) equations. As it was stated in Section 4, we couple (i) of type (B), which is relation (5), with (ii) for the same mode. I.e. the derivative of \( u(t) \) will be "adjusted" by \(-p(t)/t\) and, correspondingly, the rate of change of \( p \) will be taken proportional to \( u(t)/t \). This means that the impact of \( u(t) \) to \( p(t) \) and vice versa goes through the averages; i.e. the response to \( u(t) \) is not "immediate" as in (A).

Note that the exact \(-p(t)/t\) or, later, \(-p(t)\) in the equation for \( du(t)/dt \) is a matter of normalization. A possible proportionality coefficient here, if relevant, can and will be moved to the second equation.

The equations under (B) become as follows:

\[
\frac{du(t)}{dt} = c \frac{u(t)}{t} - \frac{p(t)}{t},
\]

\[
\frac{dp(t)}{dt} = a \frac{u(t)}{t}.
\]

Here \( c \) is the original intensity of the spread, the transmission coefficient, and \( a \) is the intensity of the protection measure(s). This system can be readily integrated. Substituting \( u(t) = t^r \), the roots of the characteristic equation are \( r_{1,2} = c/2 \pm \sqrt{D} \), where \( D = c^2/4 - a \).

Accordingly, when \( D \neq 0, t > 0 \):

\[
\begin{align*}
(8) \quad & u(t) = C_1 t^{r_1} + C_2 t^{r_2} \quad \text{if } D > 0 \quad \text{for constants } C_1, C_2, \\
(9) \quad & u(t) = t^\xi (C_1 \sin(\sqrt{-D} \log(t)) + C_2 \cos(\sqrt{-D} \log(t))) \quad \text{if } D < 0,
\end{align*}
\]

Here \( \xi \) is the phase shift determined by the initial conditions. If \( D = 0 \), the solution becomes the sum of exponentials.
where the constants are adjusted to ensure that \( p(t_0) = 0 \) and \( u(t_0) = 1 \). Note that a proper branch of \log \) must be chosen for \( t \) near zero.

So for \( a < c^2/4 \), the initial \( t^c \) will be reduced up to \( t_{r_1} \) with \( c/2 < r_1 < c \). When any \( a > c^2/4 \), the power growth is always of type \( t^{c/2} \). The periodicity with respect to \( \log(t) \) has the period \( 2\pi/(\sqrt{a-c^2/4}) \), which will result in the "saturation", which is at the first \( t \) such that \( du(t)/dt = 0 \), only for very large \( t \), especially comparing with \((A)\).

**Positivity of \( du/dt \).** The control parameter \( a \), the intensity, is actually not arbitrary. To see this let us invoke \( v(t) = V(t)/U(t_0) \), where \( V(t) \) is the number of infected people wearing the protective masks. It was used in the definition of \( \mathcal{P} : \mathcal{P}(t) = \kappa c V(t) \), where \( \kappa \) is the efficiency of the mask (from 0 to 1). We then have:

\[
\frac{du(t)}{dt} = c \frac{u(t)}{t} - \kappa c \frac{v(t)}{t}, \quad \frac{dv(t)}{dt} = \frac{a}{\kappa c} \frac{u(t)}{t}.
\]

Here \( v(t) \leq u(t) \) by the definition. This inequality generally provides no restriction on the derivatives. However, if \( v(t) \) is essentially proportional to \( u(t) \), there are some consequences. Let us assume that \( v(t) \sim \gamma u(t) \). Then we obtain the relation: \( a = c^2 \kappa \gamma (1 - \kappa \gamma) \). Accordingly, the maximal value of \( a \) is \( a_{\text{max}} = \frac{c^2}{4} \) at \( \kappa = \frac{1}{2\gamma} \) provided that \( 1 \geq \gamma \geq \frac{1}{2} \). This gives \( D = 0 \) for \( \gamma = 1 \), i.e. this is the case when all wear the masks of efficiency 1/2; then \( u(t) = Ct^{c/2} \), which is obvious from to the definition of \( \kappa \). We conclude that \( a \) cannot be too large.

Generally, \( du(t)/dt \geq c(1 - \kappa)u(t)/t \), and \( d\tilde{u}(t)/dt \geq 0 \) for \( \tilde{u}(t) = u(t)t^{c(\kappa - 1)} \). Thus the solutions \( u(t) \) from \((8)\) and \((9)\) grow faster than \( t^{c(1-\kappa)} \). The positivity of \( du(t)/dt \) is obvious \textit{a priori}. Practically, the range of \( t \) we consider is till \( t \approx 10 \) (about 100 days), so the \( \log(t) \)-periodicity, if present, is not a major factor.

**7. Type \((A)\) management.** The most "aggressive" model of momentum management is of type \((A)\). Mathematically, we replace the average \( p(t)/t \) in \((6)\) by \( p(t) \), and \( u(t)/t \) by \( u(t) \) in \((7)\). Actually, the first change affects the solutions greater than the second. One has:

\[
\frac{du(t)}{dt} = c \frac{u(t)}{t} - p(t),
\]
\[
\frac{dp(t)}{dt} = a u(t).
\]
Solving system (11)&(12) goes as follows:

\begin{align}
& t^2 \frac{d^2 p}{dt^2} - ct \frac{dp}{dt} + ct^2 p = 0 = t^2 \frac{d^2 u}{dt^2} - ct \frac{du}{dt} + ct^2 u + cu, \\
& u = A_1 u_1 + A_2 u_2, \ u_{1,2}(t) = t^{1/4} J_{\alpha_{1,2}}(\sqrt{at}) \text{ for } \alpha_{1,2} = \pm \frac{c-1}{2}.
\end{align}

Here the parameters $a, c$ are assumed generic, $A_{1,2}$ are undermined constants, and we use the Bessel functions of the first kind:

$$J_{\alpha}(x) = \sum_{m=0}^{\infty} \frac{(-1)^m (x/2)^{2m+\alpha}}{m!\Gamma(m + \alpha + 1)}.$$ See [Wa] (Ch.3, S 3.1). We will also need the asymptotic formula from S 7.21 there:

$$J_{\alpha}(x) \sim \sqrt{\frac{2}{\pi x}} \cos(x - \frac{\pi \alpha}{2} - \frac{\pi}{4}) \text{ for } x >> \alpha^2 - 1/4.$$ It gives that $u_{1,2}(t)$ are approximately $C t^{c/2} \cos(\sqrt{at} - \phi_{1,2})$ for some constant $C$ and $\phi_{1,2} = \pm \frac{c-1}{2} - \frac{\pi}{4}.$

**Quasi-periodicity.** We conclude that for sufficiently big $t$, the function $u(t)$ is basically:

$$u(t) \approx t^{c/2} (A \sin(\sqrt{at} + \pi c/2) + B \cos(\sqrt{at} - \pi c/2)),$$

for some constants $A, B$. So it is quasi-periodic, which means that the periodicity is up to a power function and only asymptotically; the asymptotic $t$-period is $\frac{2\pi}{\sqrt{a}}$.

In our setting, $u(t)$ always grows, so $du(t)/dt \geq 0$. The technical end of an epidemic is when $u(t)$ reaches its first maximum; $\frac{\pi}{2\sqrt{a}}$ is a reasonable estimate, but not too exact. For instance, $u(t) = t^{c/2+1/2} J_{(c-1)/2}(\sqrt{at})$ for $c = 2.2, a = 1/4$ reaches its first maximum at about $t = 4.35$, not at $\frac{\pi}{2\sqrt{a}} = \pi$; see Figure 1.

**Transitional mode.** Presumably it is applicable to model epidemic spread in the places where hard measures are applied cautiously (if any) and at later stages of epidemics, when hard measures are reduced or even abandoned. This mode is transitional mode between (A) and (B): one replaces (12) by $dp(t)/dt = au(t)/t$, but at the same time takes the equation for $du(t)/dt$ from mode (B), which is (6):

$$\frac{dw(t)}{dt} = c \frac{w(t)}{t} - p(t), \quad \frac{dp(t)}{dt} = b \frac{w(t)}{t},$$
where we replaced $u(t), a$ by $w(t), b$; the $c$ remains unchanged. It was called transitional $(AB)$–mode at the end of Section 4.

This mode basically means that we become cautious with our response to $u(t)$ by choosing the ”control formula” for $dp(t)/dt$ from $(B)$, better protected against fluctuations. However the measures we are going to employ are still of ”hard” type, as under mode $(A)$. This makes sense practically; let us see what this gives theoretically.

This system is still integrable in terms of Bessel functions; see formulas (2.20), (2.21) in [Ch]. The leading fundamental solution is

$$w_1(t) = t^{c+1/2} J_{c-1}(2\sqrt{bt}), \text{ where } c > 1.$$  

One has:

$$u_1(t) \approx t^c \frac{(\sqrt{a/2})^{(c-1)/2}}{\Gamma((c-1)/2)}, \quad w_1(t) \approx t^c \frac{(\sqrt{b/2})^{c-1}}{\Gamma(c-1)},$$

when $t$ is sufficiently close to zero. Practically, they ”almost” coincide upon the coefficient of proportionality from (18) in sufficiently large areas. This is important for the ”forecasting tool” below.

The quasi-periodicity will be now with respect to $t^{1/2}$ and the corresponding asymptotic period will be $\pi/\sqrt{a}$. I.e. the process of reaching the saturation is ”slower” than $(A)$, but still significantly faster than $(B)$, where the ”mathematical periodicity” (if any!) is in terms of $\log(t)$ in (9). This perfectly matches the qualitative description of $(AB)$ as a transition from $(B)$ to $(A)$.

Actually we have a family $(AB)_\mu$ for $-1 \leq \mu < 1$ of such modes, described by the system

$$\frac{dw(t)}{dt} = c \frac{w(t)}{t} - p(t)/t^\mu, \quad \frac{dp(t)}{dt} = b \frac{w(t)}{t}.$$  

Here the term $p(t)/t^\mu$ means that the impact of one isolation of an infected individuals grows non-linearly over time, which makes sense. Assuming that $c > 2$, the dominant solution is

$$w(t) = t^{c+1-\mu} J_{c-1-\mu} \left( \frac{2\sqrt{b}}{1-\mu} t^{(1-\mu)/2} \right), \quad w \sim t^c \text{ for } t \approx 0.$$  

We follow here [Ch]. When $\mu = -1$, $J$ depends on const$t$ and we arrive at some counterpart of $u(t)$.

8. USA, UK, Austria, Switzerland. In the range till April 14 the graphs of our solutions $u(t)$ presented in this section match surprisingly well the total number of infections for these countries starting when these
numbers begin to grow "significantly". These moments are approximately around March 16 for the USA and UK, March 13 for Switzerland, and March 7 for Austria.

Let us mention that https://ourworldindata.org/coronavirus is mostly used for the data, updated at 11:30 London time. We also use "worldometers" We will always take \( x = \text{days}/10 \).

**The USA data.** The scaling coefficient 1.7 in Figure 1 is adjusted to match Figure 2 for the United States. For the USA, we set \( y = \text{infections/100K} \), and take March 17 the beginning of the period of "significant growth". Also, \( c = 2.2, a = 0.2 \) appeared perfect.

Concerning \( c \), the transmission rate, it reflects of course the virus transmission strength, but it also depends very significantly on the way people respond to the current numbers of infections, which includes all the related information provided by the authorities in charge and mass media. It is basically around \( c = 2 \) for Covid-19, but there are some variations; say, \( c = 2.4 \) appeared more appropriate mathematically for UK and Austria, and \( c = 2.8 \) for "the world".

The parameter \( a \), the intensity of type \((A)\) measures, is 0.2 for the USA and UK, and 1/3 for Austria. The scaling coefficient, 1.7 for the USA, makes the interpretation of \( y \)--number as the total number of infections as comfortable as possible.

The red dots in all figures show the corresponding actual total numbers of infections. They perfectly match \( u(t) = 1.7t^{1.6}J_{0.6}(t\sqrt{0.2}) \) in Figure 1, which results in the following: the number of cases in the USA can be expected to reach its preliminary saturation at \( t_{\text{top}} = 4.85 \) (48.5 days from 03/17 to May 5) with \( u_{\text{top}} = 10.3482 \), i.e. with 103,482 infections (it was 60,9516 at 04/15). This is of course a projection: about 1M of total cases at the saturation point \( t_{\text{top}} \) near May 5.

Of course \( t = t_{\text{top}} \) is not the end of the spread of infection. The data from South Korea and those from some other countries demonstrate that a linear growth of the total number of cases can be expected around and after \( t_{\text{top}} \). One obvious reason is that no country is isolated from infections from other places. It can be due to reducing the "hard" and "soft" measures too quickly, which we will address below.

With this reservation, the graphs we provide and similar ones we considered for Covid-19 demonstrate that Bessel functions are a very powerful forecasting tool for almost the whole period of "intensive growth" of the spread. This is a strong confirmation of our theory.

The mode \((A)\), resulting in Bessel functions, is of course not the only force. It is always combined with mode \((B)\), and there are some
It is surprising that Bessel functions seem working very well practically for the whole period of the intensive growth. Accepting this, one can try to “capture” the parameters $c, a$ even at some early stages, before the turning point of the epidemic, which seems doable but with obvious risks involved. If $a, c$ are known, the first local maximum of the corresponding Bessel function times $t^{c/2 + 1/2}$ (or the estimate $t_{top} \approx \frac{\pi}{2 \sqrt{a}}$) gives the moment of the “saturation”, the technical end of the epidemic.

In the graphs we provide, $a$, the intensity of type $A$ measures, is 0.2 for the USA and UK, and $1/3$ for Austria. The parameter $c$, the
transmission rate, is generally around 2: \( c = 2.2 \) for the USA and 2.4 for UK and Austria. They can be mostly seen at early stages.

**Covid-19 in UK.** One of the reasons of superb match of red dots and our \( u(t) \) is that the USA consists of 50 states, and therefore the total number of infections is quite an average. Let us consider now UK for the period from March 16 till April 15.

\[
\begin{align*}
\text{total infections:} & \quad 03/16 - 04/15; \\
\text{top} & = 5.17 \text{ (May 6)} \\
\text{u} & = 16,9157 \text{ (170K)} \\
(a & = 0.2, c = 2.4)
\end{align*}
\]

*Figure 3.* \( u(t) = 2.2t^{(c+1)/2}J_{(c-1)/2} (\sqrt{at}) \) for \( c = 2.4, a = 0.2 \)

Then \( c = 2.4, a = 0.2 \) work fine, and the scaling coefficient is 2.2. The total number of cases will be now divided by 10\(K\), not by 100\(K\) as for the USA. The increase of \( c \) to \( c = 2.4 \) (very) qualitatively indicates that the "response" of the population to this kind of thread is slower in UK (by 9\%, not much) than in the USA.

Recall that if the spread of disease is not actively managed, then the growth \( \approx t^c \) can be expected, so \( c \) reflects how we, especially the infected people and those who think that they are infected, react to the numbers of infections. Providing these numbers, discussing them by the authorities in charge and in the media is of course some kind of management, but passive. By "active", we mean the \( (A), (B) \)–measures.

The expectation is now as follows: the "saturation moment" can be 5.17, i.e about 51 days after March 16, somewhere around May 6. The estimate for corresponding number of infections is about 170000, with all ifs. This is assuming that the "hard" measures will be employed at the same pace as before April 15.

**Austria:** 3/07-4/15. This is the case of almost a complete "cycle". There is some switch to a linear growth closer to the "saturation", which makes this example interesting to us. *Such a period of linear growth closer to \( t_{\text{top}} \) can be expected with all countries.* Finding \( a, c \) is
a bit challenging because of this linear trend, but modeling this whole period by Bessel function appeared quite doable.

\[ u(t) = 3 t^{(c+1)/2} J_{(c-1)/2}(\sqrt{a}t) \] for \( c = 2.4, a = 1/3 \)

**Figure 4.** \( u(t) = 3 t^{(c+1)/2} J_{(c-1)/2}(\sqrt{a}t) \) for \( c = 2.4, a = 1/3 \)

Here we start with March 07; Austria began earlier with the protection measures than UK and the USA. The parameters \( a = 0.33, c = 2.4 \) and the scaling coefficient 3 appeared the best for Austria; we divide now the number of infections by 1000, i.e. 14 in the graph corresponds to 14000 infections.

Reaching \( t_{\text{top}} \), as we discussed, is not really the end. The epidemic continues after this moment. Moreover, the period before such “saturation” is generally a union of segments corresponding to different stages. Let us comment on it using Switzerland as an example.

**Switzerland: different stages.** We focus on the period March 13- April 12. As we have already discussed, \((A)\) is supposed to dominate mostly during the final ”half” of this period toward \( t_{\text{top}} \), i.e. around the turning point and later. Since we do not discuss the combination of modes \((A), (B)\) in one system of differential equations, practically: the minimum of the \( u \)-functions obtained for these two modes dominates and is supposed to be seen in real charts.

Recall that we consider only ”total cases”, the numbers of all detected infections till \( t \), and begin at the moment when the power growth begins, which was around March 12 for Switzerland in Figure 5. I.e. \( t = 0 \) is March 12. This moment is essentially when the active measures begin; \( t_0 \) is when the first results of these measures can be clearly seen, around March 20. Practically, the initial (mostly) parabolic growth will start switching to the linear one at \( t_0 \).
The "free" spread of epidemics of quadratic type can be clearly seen in quite a few countries at early stages of Covid-19; say, in Brazil, Mexico, Sweden, Switzerland are examples. Here we use that $c \approx 2$.

Generally, mode (B) ensures a continuous transition from the "free" growth $u(t) \sim t^c$ to $u(t) \sim t^{c/2}$. This is by making $a$ for (B) from $a = 0$ to $a = c^2/4 \approx 1$. We assuming that the initial $c$ is about 2, which seems to be quite universal for Covid-19. After $a = 1$ the exponent will stabilize: $u(t) \sim t^{c/2}$ for sufficiently large $t$, even if $a$ continues to increase. For $a > 1$, the $u(t)$ given by (9) behaves somewhat differently; $\cos(\cdot)$ and $\sin(\cdot)$ contribute.

![Figure 5. Covid-19 in Switzerland](image)

Mode (A), aggressive detection and isolation of infected people and so on, begins somewhat later than (B). It takes time to increase the testing capacities and solve related problems. However, eventually it will be the main reason for $u(t)$ to "saturate".

Let us briefly comment here on changing the starting point from the absolute start of the epidemic to some $t_\star$. The equations will remain the same, but $c/t$ must be replaced by $c_\star/(t - t_\star)$ for the current $c_\star$. There can be different phases of epidemics, so this can be necessary, and not only once. Such a split of the total investment period into intervals is very common in stock markets.

9. Addressing recurrence. One of our mathematical conclusions is that managing epidemics based on (B) alone is hardly sufficient for
ending them. This can be expected: protective masks and social distancing are of limited effect compared with aggressive testing followed by prompt isolation of infected people, closing factories and so on; see Section 2. So mode (A) is the key, practically and mathematically.

Mode (AB) seems important to address the later stages, when the reduction of hard measures is considered and performed. However, it is based on hard measures too; only the way to react to the total number of infections is changed as in (B).

Mode (A) as the leading factor. The end of an epidemic can occur "naturally", due to the reduction of $c$ because of mutations of the virus, herd immunity or similar developments. In our approach, it is due to the aggressive momentum management and has the following stages.

First, the management under (A) or (B) "replaces" reasonably quickly the "natural" growth $t^c$ of the epidemic by $t^{c/2}$. The $c$–coefficient seems around 2 with Covid-19: the quadratic growth after some short periods of faster acceleration can be seen in many countries. Due to formulas (8),(9), "soft" measures alone can be sufficient to achieve the linear growth, but it will happen significantly faster with (A).

The data show, that such a linear growth can last for some time. Mode (A) becomes the key during this period and reduces the growth further to make the number of new infections approaching zero, but such an aggressive management can have side effects.

The risks of (A). Such a "technical end", almost zero new infections, mostly happens with relatively recent epidemics before the virus totally loses its strength and/or the factors like herd immunity begin to work.

The "natural" $c$–coefficient reflects both, the transmission strength of the virus and the "normal" intensity of our contacts. The second component will obviously return back to normal after the epidemic is announced finished. If the "microbiological component" is essentially the same as it was before, the recurrence of the epidemic is likely after the protection measures are removed. The recurrence of the epidemic is a sort of "cost" of our aggressive interference in its natural course.

The solutions $u(t)$ of our (A)-type equation are of quasi-periodic type for any $a > 0$. The whole quasi-periodic process cannot be observed practically, since $u(t)$, the total number of infections, can be only a growing function. However a strong mathematical tendency toward the recurrence under mode (A) is clear mathematically.

A conclusion is that we are supposed to stick to the hard measures after (or well after) the number of new infections approaches zero for preventing the recurrence of the epidemic. I.e. we are supposed to proceed "beyond" the differential equations: continue to employ the measures when there are no "momentum reasons" for this.
However it is not impossible that just the opposite can occur. The general tendency is to begin reducing the implemented hard measures as quickly as possible. It can be even on the very early signs of improvements; after the turning point (or something that looks like this point), the reduction is likely to be considered.

Let us consider some practical aspects, a combination of general scenarios and our modeling.

**The next cycle.** We will try to outline what can be expected from the viewpoint of our approach to modeling epidemics after zero levels of new infections have been successfully reached and the epidemic reached its technical $t_{\text{top}}$. First of all, some growth of $u(t)$, presumably linear, can be still expected, at least because no country is really isolated from the others. However, there are clear "domestic" reasons too. With Covid-19, such tendency can be seen in South Korea and quite a few other countries (at different stages).

It is always possible that some infected individuals or clusters of the infection are still present. Unless seasonal or other factors have undermined the strength of the virus and transmission, we can return after some pause back to equation (1), which provides an exponential growth, and then to (2), which is based on the natural reduction of our contacts due to the epidemic, where the actions of those in the risk groups are the key. After this period, (A) and (B) will begin to work.

If herd immunity was not reached, "restarting" the epidemic with few infected individuals left after the previous cycle is standard for any virus, unless it lost its strength. For the next cycle(s), our better preparedness can be expected, which will diminish $c$. Also, mode (B) (protective masks, social distancing) can be started now much faster, and mode (A) (testing etc.) is likely to be activated earlier.

Finally, let us emphasize that our differential equations describe the optimal momentum response aimed at diminishing the new infections to zero in the following sense. The main our assumption is that people and the authorities in charge constantly monitor and respond to the number of infections.

This is very similar to the ways stock markets work. Professional traders simply cannot afford not to react to any news and any change of the stock prices, even if they seem random, temporary or insignificant. Indeed, any particular event or a change of the share-price can be the beginning of a new trend. Applying this to managing epidemics, we are supposed to closely follow the data. The "flexibility" here is the usage of some average numbers as the triggers, to "protect" yourself against random fluctuations. This is exactly the "defensive" approach from (B) or (AB) versus that from mode (A).
10. Some conclusions. Under any type of momentum management, the most aggressive for (A) or the least aggressive for (B), the envelope of the \( u \)-function, describing the total number of infections, remains a power function, though with smaller exponent than the original "free" one. Namely, if \( u(t) \) is \( t^c \) without the usage of \( p(t) \), then under (A) or, correspondingly, under (B) it becomes:

\[
(21) \quad t^{c/2} \text{ for (11) & (12), or when } a > c^2/4 \text{ for (6) & (7).}
\]

For (6) & (7) upon \( a < c^2/4 \), the leading term is \( t^{c/2+\sqrt{c^2/4-a}} \), i.e. the exponent can be from \( c/2 \) to \( c \), and there is no \( \log(t) \)–periodicity.

Diminishing the exponent here is of course of importance. The original coefficient \( c \) will eventually drop without any management and "forcing" \( c \) to change to \( c/2 \) will bring us closer to the end of the epidemic. This can be related to the following feature of Covid-19. After a relatively short periods of parabolic growth of the number of infections, which corresponds to \( c \approx 2 \), the growth becomes linear almost everywhere and then it lasts. This matches (21); and see Figure 5.

**Periodicity is the key.** The main difference between (A) and (B) is the quasi-periodicity of the corresponding solutions of type (A); see (14). For type (B), the periodicity is with respect to \( \log(t) \) when \( a > c^2/4 \), which is a significantly slower process than for (A). Asymptotically, \( u(t) \) always becomes quasi-periodic for \( a \neq 0 \) due to (15) and the epidemic can be expected to end under this kind of management at about \( \frac{\pi}{2\sqrt{a}} \), more precisely, at \( t_{\text{top}} \) of the corresponding \( u(t) \). This gives a practical way of evaluating the real \( a \) on the basis of epidemic charts.

We note that the quasi-periodicity of our \( u \)-functions under momentum management of type (A) is not connected at all with the periodicity of epidemics associated with seasonal factors, biological reasons, or various delays; see e.g. [HL]. It entirely results from the active management, which is a combination of the measures employed, their effect, and our general response to the threat.

With this important reservation, the approximate reflection symmetry of \( du(t)/dt \) for \( u(t) = t^{c/2+1/2}J_{c-1/2}(\sqrt{at}) \) in the range from \( t = 0 \) to \( t_{\text{top}} \), corresponding to the first local maximum of \( u(t) \), can be interpreted as a variant of Farr’s law of epidemics. Generally, the portions of the corresponding graph before and after the turning point are supposed to be essentially symmetric to each other. This is not exactly true for \( du(t)/dt \), but close enough. See e.g. Figure 1; the turning point is at \( \max\{du(t)/dt\} \). This holds for \( w(t) \), describing the (AB)–mode.
**Main findings.** The "power law of epidemics" presented as a difference equation is the starting point of our approach to momentum management. This is different from other power laws for infectious diseases; compare e.g. with [MH]. We deduce it from the response of individuals to the epidemic data. Generally, the power growth of epidemics has solid experimental confirmations, but almost an immediate switch to a power growth and our interpretation of the exponent are not due to "general factors". They are based on "momentum risk-taking".

This is only the beginning; the main problem is to "add" here some mechanisms quickly ending epidemics and to prevent their possible recurrence. These are major challenges, biologically, psychologically, sociologically and mathematically. One can expect that this problem is well beyond the power law of epidemics itself, but we demonstrate that mathematically there is a path: Bessel functions appeared a natural link from the power law of epidemics to the "periodicity".

We provide justifications of our differential equations; they are certainly based on various simplifications, which seem reasonable to us. The "megaproblem" of understanding and managing epidemics is very ramified and interdisciplinary; significant assumptions are inevitable in any models. We model only the period when the total number of infections is significantly smaller than the whole population, but this is the essence of our approach. We also disregard the recovery process and the quarantine durations.

Making our justifications more "rigorous" is a challenge, but we think that the experimental confirmation is the key. We are of course fully aware of the statistical nature of the problem. The usage of random processes, like *Bessel processes*, is reasonable here to address the randomness around epidemics. Also, the traditional SIR and SID models can be of importance when removing some of our assumptions.

The main outcome of our modeling is that the measures of "hard type", like detecting and isolating infected people and closing the places where the spread is almost inevitable, are the key for ending an epidemic. Moreover, such measures must be employed strictly proportionally to the current numbers $u(t)$ of infections (not its derivative of any kind), which is the most aggressive momentum way to react.

Then the point of the first maximum of the corresponding Bessel function times $t^{c/2+1/2}$ is a good estimate for the duration of the epidemic, and the value at this point is an expected top number of infections. Surprisingly, this formula works extremely well for almost the whole period of extensive growth of the spread, assuming that "hard" measures of type (A) play the leading role. This is in spite of quite a few factors contributing to spread, the management of type (B) included. It seems a real discovery: see Figure 1.
Mode \((B)\) is the usage of the average \(u(t)/t\) here instead of \(u(t)\) and, especially, relying mostly on "soft" measures, like wearing protective masks. When used alone, it can significantly slow down the process of "reaching zero"; this results from our model. Though a combination of "hard measures" with the "slow response", based on \(u(t)/t\) instead of \(u(t)\), seems promising to us, which is our mode \((AB)\). It is supposed to play an important role at the later stages of the epidemic, when hard measures are reduced or even abandoned. It is still based on the "hard" measures, but the response of type \((B)\) seems a reasonable model for the action of the authorities in charge at these stages.

The risks of recurrence. This is generally a clear challenge for microbiology, epidemiology and population genetics; very much depends on the type of virus. Mathematically, we address the following aspect: the recurrence as a result of aggressive interference in a natural process. After the turning point of the epidemic, the protection measures are likely to begin being reduced or even abandoned. This must be done very cautiously to avoid the recurrence of the epidemic, as our model suggests, especially with the reduction of "hard measures". Otherwise the epidemic is quite likely to resume; mathematically, this is certain.

These are real risks. It is understandably easy to "forget" or "ignore" that the turning point and the saturation were achieved reasonably quickly mainly due to the strict hard measures imposed, and that the corresponding underlying mathematical process has a very strong tendency to become periodic. Here the mathematical quasi-periodicity can "play against us" as we approach the end of the epidemic.

The table: \((A) vs. (B)\). For convenience of the readers, let us provide a basic table presenting the \((A)\)-mode and the \((B)\)-mode with some simplifications: Figure 6. Recall that \(c\) is the initial transmission rate, \(a\) the control parameter, which is the intensity of the management, \(\kappa\) the efficiency of protective masks.

By "Detections", we mean \(P(t)\): the total number of detected and isolated infected individuals till \(t\). Practically, if the number of infections doubles, then the \((A)\)-type response is to double the rate of increase of tests (not just the number of tests). Recall that we do not subtract the closed cases and disregard similar factors.

Accordingly, by "Masks used", we mean the total number of infected people who began using the masks before \(t\). If the number of infections doubles, then the total number of masks must be doubled too under \((B)\), not the rate of change as for \((A)\). The same intensity of applying the measures as for \((B)\) is for mode \((AB)\), but now to be used for the "hard" measures like testing & detection. This will provide the periodicity with respect to \(\sqrt{t}\), which is not too different practically from the \(t\)–periodicity for \((A)\). See Sections 4 and 5.
\(U(t) : \text{total \# of all infections till moment } t\)

| Natural Course: microbiology, and sociology | None, though “c” diminishes over time “t” | Firstly, \(t^c\) for initial \(c = 2\) and then later \(c \to 1\) | The factors are mutations, \ldots, herd immunity |
|---------------------------------------------|-------------------------------------------|-------------------------------------------------|-----------------------------------------------|
| Under type A: the detection and isolation   | t-derivative of \(Detections\) is \(a \ U(t), t > t_0\) | For any \(a > 0\): \(\sim t^{c/2} \sin(pt)\) (Bessel fncts) | Approximately after \(\pi / (2\sqrt{a})\) for intensity \(a\) |
| Under type B: masks, social distancing etc. | t-derivative of \((Masks \ used) = a \ U(t)/kct\) | For \(a > c^2/4\): \(t^{c/2}\) times \(\sin(q \log(t))\) | No fast ending due to \(\log(t)\)-“periodicity” |

**Figure 6.** Two types of momentum management

11. **Toward forecasting.** The function \(u(t) = C' t^{c/2+1/2} J_{c/2-1/2}(\sqrt{at})\) matches very well the real total numbers of infections, as it was demonstrated in Figure 1 and there others. However it models mostly the initial and middle stages of the period of intensive growth of the spread, when the hard measures are coupled with the most aggressive respond to the current number of total cases. It seems essentially sufficient for forecasting if the \((A)\)-mode is employed all the way until the number of new detected infections drop almost to zero. Then \(t_{top}\), the first zero of \(du(t)/dt\) is a reasonable estimate for the ”technical end of epidemics”. South Korea, Austria, Israel and quite a few others did exactly this. Some linear growth can be expected after \(t_{top}\); one of the reasons is that no country is totally isolated. Then fluctuations, new clusters of infection and so on are quite possible during this ”linear period”. However \(t_{top}\) is really some saturation; see Figure 10.

In this section, black dots were added after the parameters of \(u(t)\) were fixed; \(w(t)\) was adjusted to match \(u(t)\) for the periods of red dots.

**The role of the \((AB)\)-mode.** A different approach to forecasting later stages is needed if reducing hard measures (if any) begins almost after the turning point, or what looks like a turning point. The growth can
be expected linear then, but the number of daily new infections can be very high. From our perspective, this means a switch from mode \((A)\) to mode \((B)\). We need to be more exact here. The hard measures are still obviously present, but the response becomes softer, of type \(B\).

For instance, if the number of new cases is essentially a constant, even uncomfortably high, the \((B)\)–response is to keep the testing-detection constant too. Furthermore, counting on the improvements with testing and better capacities for isolation and treatment, many places with potentially high risks of the spread of Covid-19 can be allowed to re-open. An argument in favor of such an approach is that people who suspect that they are infected begin more actively request help at this stage. This works in the same direction as any ”hard” measures; some countries, like Sweden, count on this.

Possibly no further modeling is needed for the countries that reached stable relatively small numbers of new daily infections. This is with usual reservations concerning new clusters of infections and similar developments. However if the new daily cases are constant but high, a different kind of modeling is required, which is \((AB)\).

Our theory provides this. The assumption is that the hard measures are still in place, but the response to the current total number of detected infections is via \(u(t)/t\) instead of \(u(t)\). The saturation will occur significantly later.

This mode, called the \((AB)\)–mode, is governed by \(w(t)\). The point \(t_{\text{TOP}}^w\) where the (first) maximum of \(w(t)\) occurs seems a reasonable upper bound for the ”technical saturation”. The prior \(t_{\text{TOP}}^u = t_{\text{TOP}}\) then is a lower bound; we obtain some forecast cone.

Actually even relatively minor deviations with \(a, c\) can lead to significant changes of \(u(t)\) over time, so we have some ”cones”. However, we have something more fundamental here. The switch to \(w(t)\) is due to a different kind of management.

When the daily numbers of new cases are high, there are significant chances of new clusters and fluctuations of the data of all kinds. So we need to model a process with many uncertainties. However, we think that this is basically no different from what we did for the middle stages, where \(u(t)\) was surprisingly efficient.

Let \(u(t) = Ct^{\frac{c+1}{2}} J_{c-1/2}(t\sqrt{a}),\quad w(t) = Dt^{\frac{c+1}{2}} J_{c-1}(2\sqrt{bt}).\) Using (18), the match \(u(t) \approx w(t)\) near \(t = 0\) gives:

\[
\frac{C}{D} \approx \left( \frac{2b}{\sqrt{a}} \right)^{\frac{c+1}{2}} \frac{\Gamma((c+1)/2)}{\Gamma(c)} \quad \text{for the } \Gamma \text{-function.}
\]

The cone for the USA. The prior \(t_{\text{TOP}}^u = t_{\text{TOP}}^w\) for \(u(t)\) was May 5, with \(a, c, C\) calculated on the bases of the data till April 16, marked by red
dots. It was under the expectations that the "hard" measures would applied as in (A) (as before April 16). The black dots represent the control period. The challenge was to understand how far $u(t)$ can be used toward the "later stages". The match was very good for quite a long period of time (the same with UK), but then there were some changes, which we attribute to the change of the management mode.

The 3 major spikes in the daily cases and other factors like de facto relaxing hard measures obviously delayed the saturation. Whatever the reasons, the switch to the (AB)-mode and $w(t)$ appeared necessary.

The current trend seems toward $w(t)$. Generally, the black dots can be expected to stay within the forecast cone, subject to all standard warnings. The cone is defined as the area between $u(t)$ extended by a constant $u(t_{\text{top}})$ for $t > t_{\text{top}}^u$ and the graph of $w(u)$ till $t_{\text{top}}^w$, which is approximately May 30, 2020. The parameters $b, D$ of $w(t)$ are calculated to ensure good match with red dots; $c$, the initial transmission rate, is the same for $u(t)$ and $w(t)$. The graphs of $u(t)$ and $w(t)$ are very close to each other in the range of red dots. The above relation for $C/D$ holds with the accuracy about 20%.

The $w$–saturation value is around 1.6M, but mostly we monitor the trend, the "derivative" of the graph of black dots, which is supposed to be close to the derivative of $w(t)$ or $u(t)$. See Figure 7. Some spikes with number of cases are inevitable; it is acceptable if the dots continue to be parallel to $u(t)$ or $w(t)$ (or in between).

UK: till June 10.

The graphs of $u(t), w(t)$ with all red-black dots available by now are in Figure 8. The expected $t_{\text{top}}^w$ is around June 9. The total number
of detected infections is expected 330K. These "predictions" will of course depend on many developments. However, there is an important argument in favor of the stability of our model: any spikes with the numbers of infections are supposed to trigger actions of authorities in charge and influence of own protection measures. This is a rationale for relatively uniform patterns of the spread of Covid-19.

Sweden: 100 days. The case of Sweden is actually to set a limit to our approach, because this country does not follow hard ways with fighting Covid-19. However it appeared that $w(t)$ works well here so far; the data follow it closely. See Figure 9. Accordingly, the forecast is that approximately after 100 days, there can be some saturation, i.e. at about June 15. Here we have of course more reservation than with other examples. However, if people readily contact the authorities with any symptoms of Covid-19, this is a "hard measure".

Israel: $u(t)$ worked. Here the usage of $u(t)$ was sufficient to model the total number of detected cases till the "saturation", which occurred almost exactly when $u(t)$ reached its maximum. The black dots demonstrate well the "linear period" after the saturation, which was essentially of the same type in South Korea, Austria and quite a few countries that went through the saturation. However we provide the forecast cone calculated as above on the basis of $w(t)$. In contrast to the USA, UK and especially Sweden, the black dots are much closer to the flat line started at the $u$-top, which was at $t = 4.5$ (April 26).

Some discussion. Recall that we consider only "total cases", the numbers of all detected infections, and begin at the moment when the "significant" growth begins, which is essentially when the active measures start. According to our theory, the match with $u(t)$ can be
expected in some interval around the turning point. Practically it holds better than this, including the beginning of the period of intensive growth. It seems that $u(t)$ gives a reasonable forecast as far as the authorities in charge follow mode (A), i.e. aggressively employ "hard" measures, even when the numbers of new cases become steady.

If the reductions of hard measures start before the new cases diminish significantly, the forecast cone between $u(t)$ and $w(t)$ can be used. Generally, $w(t)$ seems closer to "reality", especially if the reduction of hard measures on the first signs of improvements is likely.
Our restriction to the period till April 15, 2020 with fixing \( a, c \), except for Sweden, is not accidental. It was sufficient for the practical confirmations of the theory of the "middle period" of the spread presented in Figures 1, 3, and with other countries. The latest data, the black dots, provide the "real-time checks"; they were obtained after the parameters were fixed. The pandemic is far from over, but within the scope of this paper, the data we needed to claim that Bessel functions can be used for forecasting were essentially present by 04/15.

When the epidemic approaches the \( u \)--saturation, which is the first maximum of \( u(t) \) in our model, reducing and abandoning "hard" measures can be expected. If this saturation results in relatively small daily numbers of new detected infections, we would expect a further growth like \( At^{c/2} \) with some "mild" \( A \) and \( c/2 \approx 1 \). Though fluctuations can and will happen. Such growth is generally covered by our theory; it is not connected with Bessel functions. This is fully applicable to \( w(t) \), but in many cases \( u(t) \) can be sufficient.

If the daily numbers remain high, "spikes" of infections, new clusters and so on can be expected, as we can see for the USA. Then the transitional mode \( (AB) \) seems more relevant for the later stages; at least it is the most logical mathematical suggestion within our theory. Combining the corresponding \( w \)--curve with the \( u \)--curve, we arrive at the forecast cone, which seems the best we can propose by now.

Thus, generally \( w(t) \) and \( u(t) \) seem realistic upper and lower bounds for the total number of detected infections. The \( u(t) \) alone can be sufficient in countries that fight Covid-19 in aggressive ways.

On the basis of what we see, the best ways to use our curves seem as follows: (1): determine \( a, c \) when the period of linear growth seems steady, (2): update them constantly till the turning point and somewhat beyond, (3): try to adjust the intensity of the measures to stay close to \( u(t) \), (4): after the turning point determine \( b, D \) and \( w(t) \) for the "upper bound", (5): "testing-detecting" must be intensively continued well after the "saturation". The latter is needed to prevent the recurrence of the epidemic. With (3), the applicability of our models requires fast responses to new clusters of the disease, spikes with the new cases due to the reductions of the measures and so on. This is assuming that herd immunity, is not a significant factor.

Generally, our forecasting tool can serve the best if the data and the measures are as uniform and "stable" as possible. Then underreporting the number of infections, focusing on symptomatic cases, and inevitable fluctuations with the data may not influence too much the applicability of the \( u, w \)--curves.
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References.

[CJLP] R. Carrasco-Hernandez, and R. Jácome, and Y. López Vidal, and S. Ponce de León, Are RNA viruses candidate agents for the next global pandemic? A review, ILAR Journal, 58:3 (2017), 343–358.

[Ch] I. Cherednik, Artificial intelligence approach to momentum risk-taking, Preprint: arxiv 1911.08448v4 (q-fin), 2019.

[Co] S. Cobey, Modeling infectious disease dynamics, Science, 24 Apr 2020; DOI: 10.1126/science.abb5659.

[He] H. Hethcote, The mathematics of infectious diseases, SIAM Review, 42:4. (2000), 599–653.

[HL] H. Hethcote, and S. Levin, Periodicity in Epidemiological Models, In: Applied Mathematical Ecology. Biomathematics, 18, 193–211, Springer, Berlin, Heidelberg, S. Levin, T. Hallam, L. Gross (eds), 1989.

[MH] S. Meyer, and L. Held, Power-Law models for infectious disease spread, The Annals of Applied Statistics 8:3 (2014), 1612–1639.

[St] Ph. Strong, Epidemic psychology: a model, Sociology of Health & Illness 12:3 (1990), 249–259.

[Wa] G.N. Watson, A Treatise on the Theory of Bessel Functions, 2nd Edition, Cambridge University Press, Cambridge, 1944.

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