# Iceberg Model of the COVID-19 Epidemic: Can a Non-pathogenic Coronavirus Strain Function as a Universal Vaccine?

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A model of the COVID-19 epidemic is proposed, based on a separate consideration of symptomatically and asymptomatic infected. It is assumed that the main contribution to the spread of infection is due to asymptomatic *superspreaders*. Comparison with epidemic data shows that the vast majority of the world population is involved in cyclical asymptomatic reinfection, which maintains a slowly developing plateau of the symptomatic morbidity. In this sense, the COVID-19 epidemic is an iceberg, the main asymptomatic part of which has no external manifestations. In addition to the average plateau, the model explains the existence of non-seasonal epidemic waves. They arise as a result of biological correlations in the population and are described by the 3D Lotka-Volterra equations. The model allows determining the direction of viral mutations and calculating the effect of vaccination on the course of the epidemic. In the case of a stable non-pathogenic strain, the epidemic becomes completely asymptomatic. We believe that the spread of such a non-pathogenic strain and its subsequent dominance is responsible for ending the epidemic after the single wave of incidence in China. A way to stop the epidemic in the rest of the world may consist in displacing the circulating pathogenic virus with its stable non-pathogenic strain. In this approach the non-pathogenic strain plays the role of *universal vaccine*, which is insensitive to mutations of the pathogenic strains.

## I. MOTIVATION AND INTRODUCTION

The COVID-19 epidemic has divided the world into two unequal parts - China and the rest of the world.

In China, the epidemic developed in the usual way: an exponential increase in the incidence, then a pronounced maximum, then a decline and the end of the epidemic. The whole cycle took about two and a half months.

In the rest of the world, the epidemic was completely different. After a stage of exponential growth in daily morbidity in many countries, an inexplicable plateau has entered. Then the second and third waves emerged, the epidemic is currently ongoing, and it is unclear how many more waves may follow. Nothing of the kind is observed in China.

In previous works [1–3], it was shown that the asymmetric course of the epidemic with a long plateau can be explained by the presence of asymptomatic carriers of infection - *superspreaders*. However, this model did not explain the emergence of repeated waves of the epidemic. In addition, the nature of the asymptomatic state remained unclear. Finally, the question of the difference in the courses of the epidemic in China and in the rest of the world was not raised in any way. Later, these issues were raised and considered in the paper [4].

In this work, the division of the infected into asymptomatic and symptomatic is substantiated. We associate this with two types of immunity - innate and adaptive. If the innate immunity is sufficient to limit and subsequently eliminate the virus, then the person becomes the asymptomatic carrier of the virus. If the innate immunity is not enough for this, then the person becomes ill, and adaptive immunity comes into play. The choice of the type of reaction is determined by the probability $p$ with which an infected person will get sick. This probability gives an idea of the degree of pathogenicity of the virus.

With a probability $(1-p)$ innate immunity is sufficient, and the infected person remains practically healthy. Then the adaptive immunity is not activated. In this case, the infection remains in the body for a long time, which turns the carriers of the infection into *superspreaders*. In the proposed model, it is these carriers that make the main contribution to the spread of the virus.

The article is structured as follows.

In the next part, we look at the symptomatic and asymptomatic states as two modes of immune response to the virus. Response factors relevant to the two-component model are discussed here. In the third part, on the basis of these factors, the two-component model and its dynamic equations are built, the dependence of the course of the epidemic on the parameters of the model is investigated. In the fourth and fifth parts, we compare the solution with the observed course of the epidemic in the world and separately in China and determine val-

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ues of the parameters. In the sixth part the factor of strong biological correlations is considered, which leads to a sequence of incidence waves. This makes it possible to interpret the incidence waves observed during the pandemic in all countries except China. The case of stable non-pathogenic virus, which results in the simplest one-wave solution, is considered in the seventh section of the paper. We assume this case describes the epidemic in China. In the next section we consider the role of viral mutations and drift of parameters in the epidemic. In the final part, we discuss the results obtained and consider an alternative approach to ending the epidemic, not associated with mass vaccination.

## II. SYMPTOMATIC AND ASYMPTOMATIC STATES OF INFECTED

The biological characteristics of viruses and their interaction with multicellular organisms, despite numerous studies, remain poorly understood. The complexity of the problem is determined by the huge number of viruses, their constant variability, and the transition from one species of organisms to another, as happened with the virus that causes COVID-19.

To create a model for the pandemic, we used the following provisions on the interaction of the virus and the human body, which, in our opinion, do not contradict existing ideas.

1. After entering the body, the virus begins to multiply.

2. Innate/nonspecific immunity triggers nonspecific defence agents.

3. Under certain conditions, this turns out to be sufficient and equilibrium occurs at a safe level, which does not lead to the development of symptoms of infection, i.e., diseases. Since this is an equilibrium state, the system organism + virus can stay in it for a time interval $T$ without any external manifestations. It is this state that we call *asymptomatic*. In the asymptomatic state, the person is infected but not sick.

4. The number of viruses in the asymptomatic state gradually decreases. At first, for some time $T_\omega = 1/\omega$ this is enough to spread the infection. Further, during the rest time $T_\sigma = 1/\sigma$ it is not enough, and the persons do not spread. However, all the time $T = T_\omega + T_\sigma$ they remain protected from the illness by their own innate immunity, which is properly tuned by the very presence of the virus in the organism. This protection lasts as long as the virus remains in the body.

5. If the innate immunity turns out to be insufficient to establish the balance at a safe level the person becomes ill. It is this state that we call *symptomatic*. Then the adaptive immune response is activated. In this state, the main role is no longer played by the innate immunity, but by the adaptive immunity and corresponding specific antibodies.

6. The type of immune response is determined at some time point in the development of the infection, which we will call the *bifurcation point*. After it, either a symptomatic state develops (with a certain probability $p$), or an asymptomatic state (with a probability $(1-p)$). In what follows, we call this probability $p$ the pathogenicity factor.

In our work, we assume that the hidden infection is much more likely than the symptomatic one. Comparison of the results obtained in the model with the course of the COVID-19 pandemic fully confirms this assumption *a posteriori*. The transition to the latent phase is three or more orders of magnitude more probable than to the symptomatic phase, i.e. $p \sim 10^{-3}$. Therefore, we can neglect the contribution from the symptomatically infected to the spread of infection.

## III. THE TWO-COMPONENT MODEL

For a model description of the epidemic, taking into account both symptomatic and asymptomatic infected, we will introduce, along with the initial susceptible state $S$, two more pairs of states.

Asymptomatically infected people undergo states $I$ (active, *superspreader*) and $R$ (passive, temporarily unsusceptible). In these two states, the asymptomatic infected person stays for a limited time periods - respectively, $T_\omega$ and $T_\sigma$. Then they again return to their original susceptible state $S$.

Symptomatically infected persons pass the states $I_s$(sick) and $R_s$ (recovered). After that, they acquire long-term immunity and remain in the final $R_s$ state forever.

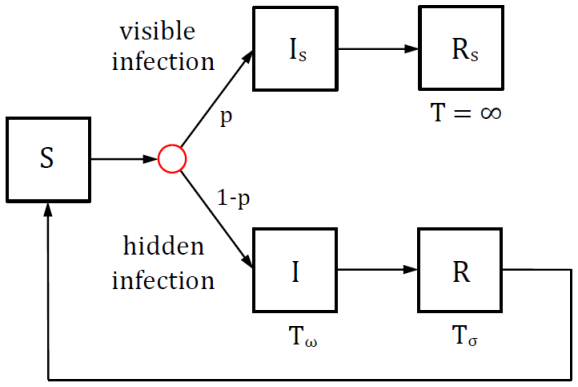


FIG. 1. Flowchart of the two-component epidemic model. Here $S$ is the share of susceptible, $I$ is the share of active asymptomatic infected, $R$ is the share of passive asymptomatic infected, $I_s$ is the share of symptomatically infected, $R_s$ is the share of those who recovered after the illness and got the long-term immunity. The model parameters: $T_\omega$ is the time of deactivation, $T_\sigma$ is the time of elimination, $p$ is the probability of visible infection, $(1-p)$ is the probability of hidden infection. The bifurcation point is shown with red circle.

The asymptomatic infection has no external manifestations and therefore is *hidden* whereas the symptomatic infection causes decease and therefore is *visible*. Thereby the epidemic has two levels - visible and hidden. Corresponding epidemic pattern represents Fig.1. The choice between two considered pathways (asymptomatic and symptomatic) occurs immediately after infection at the bifurcation point shown in Fig.1 with a red circle. Introduced epidemic variables $S, I, R, I_s$ and $R_s$ mean the proportions of the corresponding states in the population. Further, since symptomatic states do not contribute to the epidemic process, we can without loss of generality consider only the $I_s$ variable and omit the $R_s$ variable.

Now, one can reformulate the description of the epidemic process in terms of the pattern as follows.

1. Depending on the effectiveness of the nonspecific immune response, the level of infection exceeds the safety threshold and becomes dangerous (with probability $p$) or does not exceed it and remains safe (with probability$1-p$). In the first case ($I_s$ state), adaptive/specific immunity turns in play and symptoms of the disease appear. In the second case ($I$ state), this does not happen, the infected person is outwardly healthy, but the infection remains in the body in the hidden form.

2. When symptoms appear, the infected persons ($I_s$ state) become isolated and cannot spread the infection. If they recover, they acquire ideal long-term immunity and cannot be infected anymore ($R_s$ state).

3. The asymptomatic infected persons do not become isolated and can spread the infection ( $I$ state). They do it during certain time period $T_\omega$, while the level of infection in the body is sufficiently high. After that during certain time period $T_\sigma$ the level of infection is insufficient for spread but sufficient to hold the innate immunity turned on ( $R$ state). All the time $T = T_\omega + T_\sigma$ the asymptomatic infected persons (in both $I$ and $R$ states) remain protected from the symptomatic illness. After removing the virus from the body, they return to the initial susceptible state $S$. They do not acquire long-term immunity and can be reinfected.

4. Due to the appearance of long-term immunity, the transition to the symptomatic state $I_s$ is *irreversible*. On the contrary, due to the absence of the appearance of long-term immunity, the transition to the asymptomatic state $I$ is *reversible*. This means that after the asymptomatic infection $I$ the infected persons can return to their original susceptible state $S$ whereas after the symptomatic state $I_s$ can not.

A distinctive feature of the two-component model is the bifurcation point, at which it is possible to choose between the symptomatic state $I_s$ (visible infection) and the asymptomatic state $I$ (hidden infection). Another feature is the presence of a closed SIRS cycle of re-infection in the asymptomatic sector of the epidemic. The reason for the cycle is the absence of long-term immunity after the asymptomatic infection. Neither of these two features are present in the conventional SIR model, which ignores the asymptomatic infection.

The dynamic equations of the two-component model directly follow from the diagramm and have the form

$$\begin{cases} \frac{dS}{dt} = \sigma R - IS \\ \frac{dI}{dt} = (1-p)IS - \omega I \\ \frac{dR}{dt} = \omega I - \sigma R \end{cases} \tag{1}$$

$$\frac{dI_s}{dt} = pIS \tag{2}$$

Here, $\omega = 1/T_\omega$ is deactivaion rate and $\sigma = 1/T_\sigma$ is elimination rate. During transition from the symptomatic disease state $I_s$ to the recovered state $R_s$, people are isolated and do not spread the virus. Consequently, they do not contribute to the epidemic process, and the $R_s$ variable do not enter the equations at all.

The hidden (asymptomatic) sector of the epidemic described by system of equations (1) is close to the conventional SIRS model, the spread rate of the virus in these equations is taken as a unit. The only difference is factor $(1-p\ )$ in the second equation of the system which is absent in the SIRS model.

The visible (symptomatic) sector of the epidemic is described by the single equation (2). This relation expresses the simple fact that in each act of infection described by the term $IS$, the probability of symptomatic infection is $p$.

The system (1) describing the asymptomatic sector of the epidemic is completely autonomous and does not depend on the symptomatic sector represented by equation (2). Therefore, for a complete description of the epidemic, it is sufficient to obtain a solution to system (1) and use equation (2) only for direct calculation of the daily incidence. Particularly, in the limit of low pathogenicity $p \ll 1$ one can take $p = 0$ in system (1) and solve it separately. Then the found solution can be inserted into the right-hand side of equation (2), which directly gives the symptomatic daily incidence $dI_s/dt$. In what follows, we will act in this way. Comparing the results of the model with the data on the COVID-19 epidemic will show that the pathogenicity parameter $p$ is indeed small.

In the zero approximation case $p = 0$ the system (1) is reduced to the conventional SIRS model which has two equilibrium points. When the epidemic condition $\omega < 1$ is fulfilled, these two points are one unstable node $(1, 0, 0)$ and one stable focus

$$O\left(\omega, \frac{1-\omega}{\sigma+\omega}\sigma, \frac{1-\omega}{\sigma+\omega}\omega\right). \tag{3}$$

This focus determines asimptotics of the solution at $t \to \infty$ and then from (2) gives the limit value of the daily incidence $dI_s/dt$:

$$\lim_{t\to\infty}\left(\frac{dI_s}{dt}\right) = p\sigma\omega\frac{1-\omega}{\sigma+\omega}. \tag{4}$$

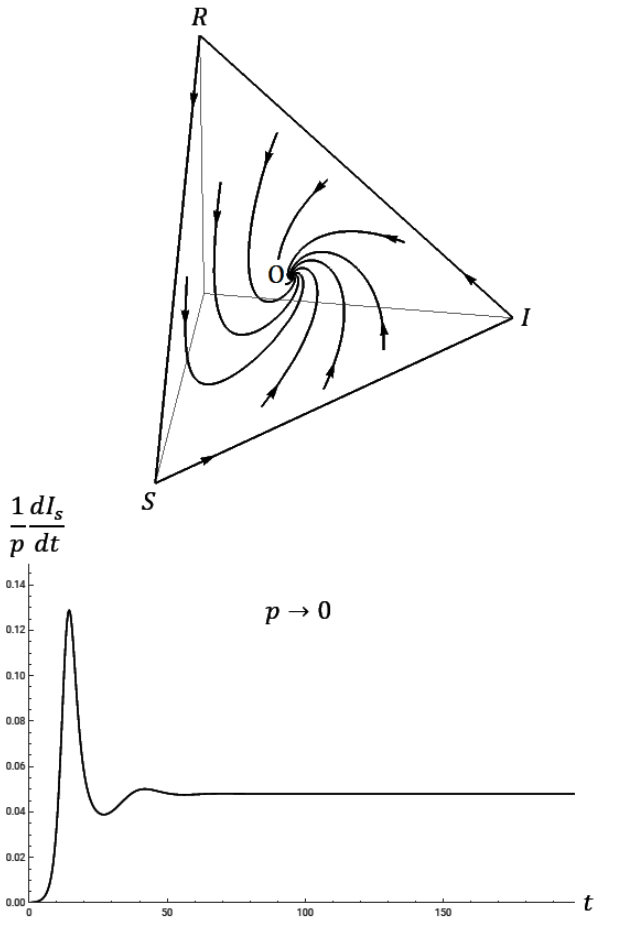


FIG. 2. Phase portrait and epidemic dependence of the symptomatic incidence $dI_s/dt$ in the low-pathogenicity limit $p \to 0$.

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This means that in the low-pathogenic limit $p \ll 1$ the daily incidence has plateau. The phase portrait of the system (1) for $p = 0$ and corresponding course of the daily incidence in the limiting case $p \to 0$ is shown in Fig.2.

In the zero approximation $p = 0$ due to identity

$$\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0, \tag{5}$$

the phase portrait of the system (1) is in plane

$$S + I + R = 1. \tag{6}$$

Accounting for $p > 0$ leads to slow overflow of the population through the symptomatically infected state $I_s$ into the recovered state $R_s$ with permanent immunity. Then conservation law (6) ceases to hold, and it should be replaced by a more general relation

$$S + I + R + I_s = 1 \tag{7}$$

which is valid for the total system (1,2). After switching on small $0 < p \ll 1$, stable focus (3) moves to the end

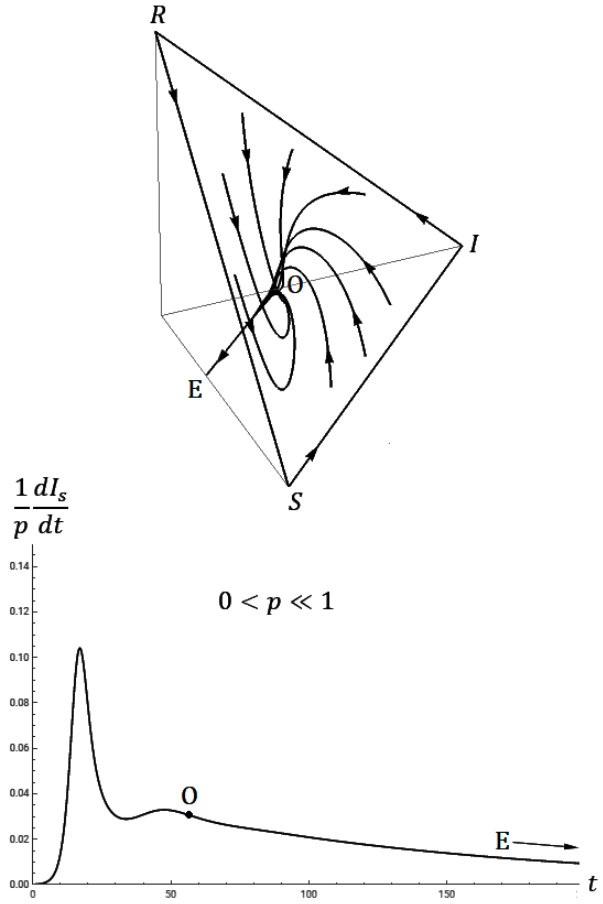


FIG. 3. Phase portrait and epidemic dependence of the symptomatic incidence $dI_s/dt$ for finite $0 < p \ll 1$. The rectilinear section $OE$ of the phase portrait at the top corresponds to the slowly decreasing plateau of daily incidence at the bottom. The plateau decreases with characteristic time $T_{tot} = 1/(p\omega)$.

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position $E(\omega, 0, 0)$. In this point

$$I_s = 1 - \omega, \quad S = \omega, \tag{8}$$

that is in the end of the epidemic the total population is divided into two parts. One part of the population has already passed the symptomatic stage $I_s$ and acquires long-term immunity. The rest of the population avoids symptomatic infection and eventually returns to the susceptible state $S$. The phase portrait of the system (1) for $0 < p \ll 1$ and corresponding course of the daily incidence is shown in Fig.3. Then in contrast to the case $p = 0$, due to nonconservation of the norm (6), the phase trajectories cease to lie in one plane.

In this case, the previous focus $O$ lying in the $S + I + R = 1$ plane becomes an entrance to a funnel. All trajectories entering the funnel asymptotically converge to the straight line segment $OE$, see Fig. 4. As follows from (8) in this segment $S = \omega$. Then the second equation of the

system (1) takes the form

$$\frac{dI}{dt} = -p\omega I, \tag{9}$$

that is $I \propto \exp(-p\omega t)$ . In accordance with (2), the daily incidence also has the same time dependence $dI_s/dt = p\omega I \propto \exp(-p\omega t)$. Therefore, the characteristic transit time of the $OE$ segment is

$$T_{tot} = \frac{1}{p\omega} = \frac{T_\omega}{p}. \tag{10}$$

This value has the meaning of the duration of the plateau, i.e. the characteristic duration of the decay of the epidemic. In words this means:

$$\boxed{\text{Duration of Epidemic} = \frac{\text{Time of Deactivation}}{\text{Pathogenicity Factor}}} \tag{11}$$

In accordance with relations (8) at the end of the epidemic, the proportion of people who got long-term immunity after the symptomatic illness is $(1-\omega)$. The rest of the population $(\omega)$ remains susceptible.

## IV. FITTING THE WORLD PANDEMIC DATA

Now we can compare the results obtained with the observed course of the COVID-19 pandemic. Since disease criteria change over time, the most accurate picture is given by daily mortality values. At the same time, the true incidence is obtained from mortality by dividing it by the fatality rate which is currently 2.2% [4].

Fitting based on first eight months of the pandemic is shown in Fig.4. This results in following values of the parameters:

$$p = 6 \cdot 10^{-3}, \quad \omega = 0.40, \quad \sigma = 0.35. \tag{12}$$

Thus, the pandemic data directly confirm the above assumption about the smallness of the pathogenicity parameter $p$, the value of which indeed is about $10^{-4}$.

The smallness of the pathogenicity parameter $p$ means that the overwhelming part of the COVID-19 epidemic is in the asymptomatic sector, i.e. is invisible. This allows the two-component COVID-19 model to be called the *iceberg model* (see Fig. 5). Here, the asymptomatic sector of the epidemic corresponds to the invisible underwater part of the iceberg, and the symptomatic sector corresponds to its visible above-water part.

In accordance with the model and its phase portrait shown in the upper part of Fig.3, the current state of the pandemic roughly corresponds to point $O$, that is, the entrance to the funnel. This corresponds to the initial portion of the plateau of the diurnal incidence, shown at the bottom of Fig.3.

According to relation (3), the current state of the world pandemic is $(0.39, 0.28, 0.31)$, that is the total population

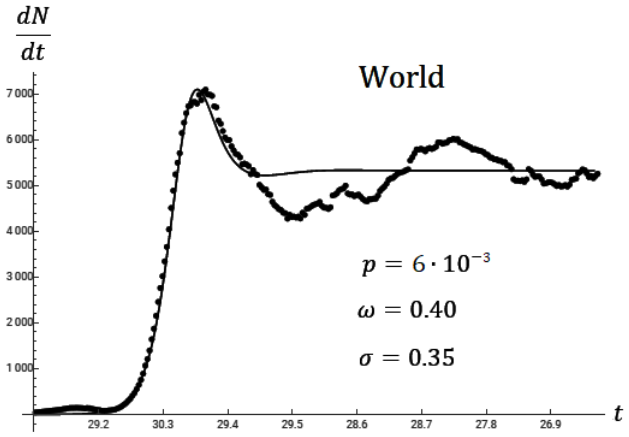


FIG. 4. Daily deaths in the COVID-19 pandemic versus time (dots) and model dependence (solid line).

of the world is approximately symmetrically divided into three very slowly changing components: susceptible $S$, asymptomatic spreaders $I$ and temporarily immunized $R$. The proportion of recovered known from the data of the pandemic is small, $I_s = 0.02$.

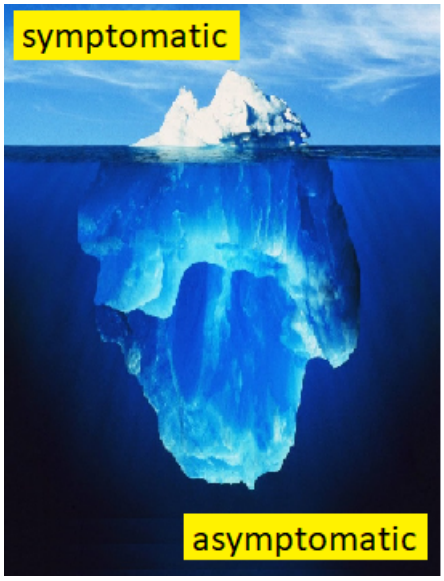


FIG. 5. Iceberg as an image of the 2-component model. The asymptomatic sector of the COVID-19 epidemic corresponds to the invisible underwater part of the iceberg whereas the symptomatic sector corresponds to its visible above-water part.

## V. FITTING THE CHINA EPIDEMIC DATA

As the course of the epidemic in China and in the rest of the world differ significantly, we fit data of the China epidemic separately. Fitting based on first four months

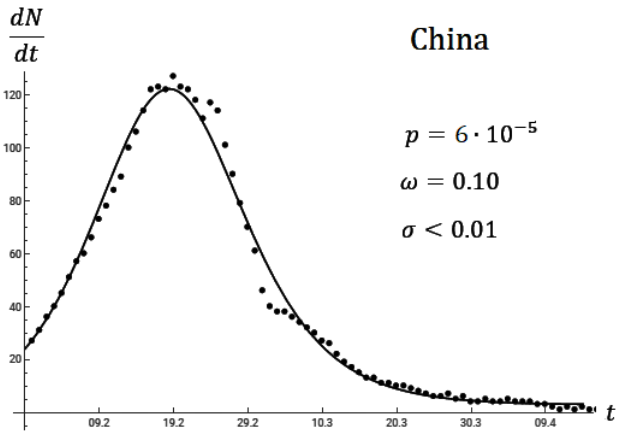


FIG. 6. Daily deaths in the COVID-19 epidemic in China versus time (dots) and model dependence (solid line).

of the epidemic in China is shown in Fig.6. This results in following values of the parameters:

$$p = 6 \cdot 10^{-5}, \quad \omega = 0.10, \quad \sigma < 0.01. \tag{13}$$

In the case of China the extremely small value of the pathogenicity parameter $p \sim 10^{-5}$ is noteworthy. In addition, the virus deactivation rate $\omega = 0.10$ and the virus elimination rate $\sigma < 0.01$ are also abnormally small. All the parameters are 1-2 orders of magnitude lower than in the rest of the world.

According to relation (3), the stationary state of the China epidemic is $(0.10, 0.00, 0.90)$, that is the absolute majority of the population is temporarily immunized, $R = 0.90$. The rest of the population is mostly susceptible, $S = 0.10$. The share of the *superspreaders* is negligible, $I < 0.01$.

Further course of the epidemic in Chine shows stable zero deaths rate. This indicates zero symptomatic incidence and therefore zero pathogenicity factor $p = 0$. Apparently, this may indicate mutation of the initial virus into completely non-pathogenic strain. In terms of phase diagrams, this kind of epidemic is described not by the iceberg model (Fig. 3), but by the conventional SIRS model (Fig. 2). This means that the epidemic reaches the equilibrium point $O$ but does not enter the funnel $OE$, and the phase portrait remains flat.

**This difference in the parameters in China and in the rest of the world suggests that a different virus strain became dominant as the epidemic spread across China compared to the rest of the world. According to the parameters found, this strain should be non-pathogenic and long-lived in the human body.**

The paper [5] already considered the hypothesis of the existence of a low pathogenic virus in the prehistory of the COVID-19 epidemic as the cause of an abnormally low incidence in China. However, there it was assumed only as a temporary factor that led to the activation of adaptive immunity in the population. On the other hand, the existence of non-pathogenic strains of coronavirus in itself is a well-known fact [6, 7], and it can be expected among the mutations of the original SARS-CoV-2 virus.

## VI. EPIDEMIC WAVES

Thus, the iceberg model (1,2) reasonably describes both the course of the epidemic in China and the time-averaged course of the pandemic in the world. In both cases, there is a plateau of daily morbidity, which is extremely low in China due to the extremely low value of the pathogenicity parameter $p$ in China.

However, this model in itself does not explain the pronounced *non-seasonal* waves of incidence which are observed both in each of the countries (except China) and in the world as a whole.

To explain the origin of the epidemic waves, it is necessary to remember that in the absence of long-term immunity, the disease becomes cyclical for each member of the population. The reason for this is cyclical re-infection which could cause also periodic course of the epidemic. Nevertheless, this does not necessarily happens, since there is a statistical averaging of the individual infection phase across the entire population.

In the previously considered iceberg model based on the SIRS-like system (1), the courses of infection in individual members of the population are statistically independent. Consequently, even if their initial states coincide, the synchronization of the epidemic process in the population is rapidly disrupted, and, therefore, the resulting fluctuations fade. For this reason, while we proceed from the SIRS-like system (1), the epidemic waves are attenuated and practically absent. This can be seen from both Fig.2 and Fig.3.

However, taking into account the strong biological correlation between the course of infection in a single individual and in the entire population as a whole completely changes the state of affairs. This leads to the stabilization of epidemic waves and makes them persistent. As it was shown in [8] account for the strong biological correlations in the population results in the change of the SIRS system (1) to the 3D Lotka-Volterra system [9, 10]

$$\begin{cases} \frac{dS}{dt} = bSR - cIS \\ \frac{dI}{dt} = (1-p)cIS - aIR \\ \frac{dR}{dt} = aIR - bSR \end{cases} \tag{14}$$

with

$$a = \frac{\sigma + \omega}{1 - \omega}, \quad b = \frac{\sigma}{\omega}, \quad c = 1. \tag{15}$$

In general case $p > 0$ the system (14) together with (2) has single equilibrium point $S = I = R = 0$, $I_s = 1$.

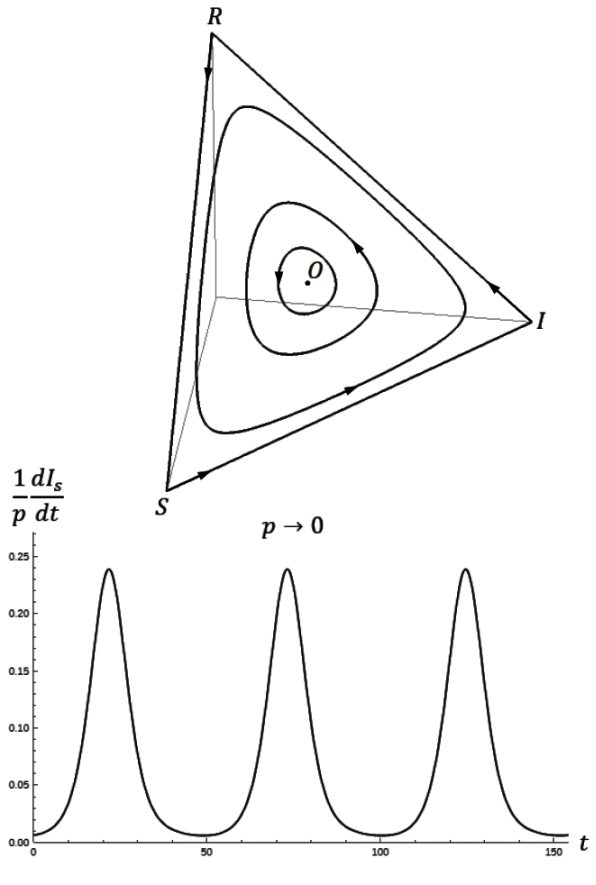


FIG. 7. Phase portrait and sustained waves of the symptomatic incidence $dI_s/dt$ with account for strong biological correlations, described by the 3D Lotka-Volterra system (14,2) taken in the low-pathogenicity limit $p \to 0$.

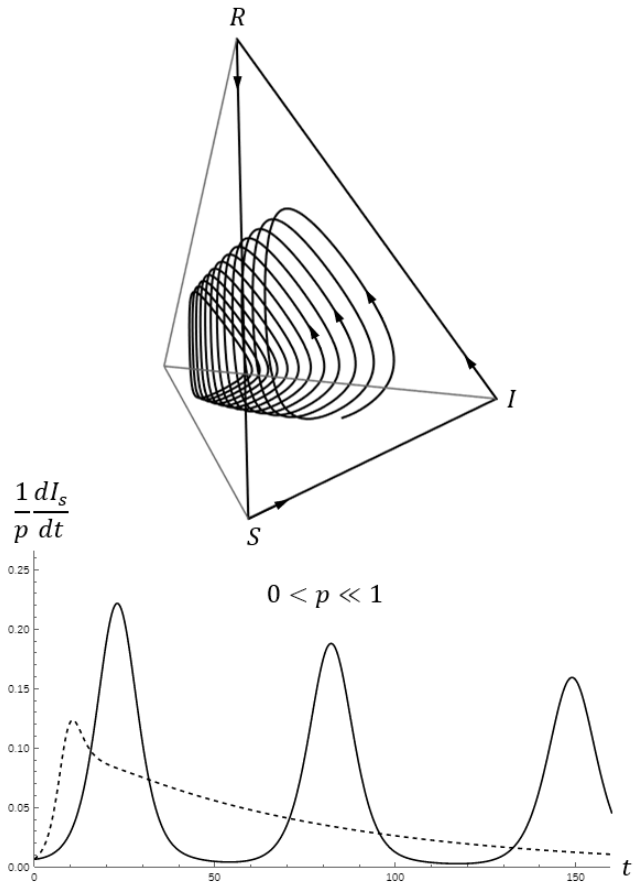


FIG. 8. One of the phase trajectories and corresponding epidemic waves in strongly correlated population described by the 3D Lotka-Volterra system (14,2) with finite pathogenicity parameter $0 < p \ll 1$. The dashed line shows the same for uncorrelated population described by the SIRS system (1,2).

In the limiting case $p \to 0$ the 3D Lotka-Volterra system (14) and the SIRS-like system (1) have the same equilibrium point $O$ from Eq.(3). Thereby the SIRS-like system (1) ignoring the correlations and the the 3D Lotka-Volterra system (14) accounting for the correlations describe the same statics of the epidemic. But their dynamics in the vicinity of the equilibrium point are different.

The phase portrait of the system (14) in the limiting case $p \to 0$ is shown in Fig.7. Unlike the SIRS-like system (1), the same equilibrium point $O$ of the system (14) is not a focus as shown in Fig.2, but a center. All phase trajectories are closed. The reason for this is that in this limiting case the system (14) has, in addition to $S + I + R = const$, one more integral of motion

$$S^a I^b R^c = const. \tag{16}$$

Therefore, this system is not dissipative. It is due to this property of a strongly correlated system (14) that epidemic waves in this system do not damp - in contrast to the uncorrelated SIRS-like system (1).

Since the cyclical course of the epidemic in the population mimics the cyclical course of the infection in the asymptomatically infected people, it can be said that strong biological correlations transform the population into a single organism.

With a nonzero pathogenicity parameter $p$, each closed trajectory turns into a helical spiral, asymptotically tending to the equilibrium point, i.e. to the origin of the coordinate system, Fig.8. It has the same meaning as the funnel in the phase portrait of the model without correlations (1,2) shown in Fig.4. The amplitude of this spiral corresponds to the amplitude of the epidemic wave. This amplitude gradually decreases as the population passes through symptomatic disease and thereby acquires long-term immunity. In the uncorrelated SIRS system (1,2), instead of the spiral, there is a thin linear funnel $OE$ of zero radius, which corresponds to a monotonically decreasing plateau without epidemic waves (dashed line in Fig.8).

Thus, taking into account strong biological correlations can explain persistent epidemic waves against the background of a monotonic plateau. In the paper [8], we showed that this factor should manifest itself precisely in

asymptomatic infections.

There is also alternative approach to explaining persistent epidemic waves, based on a fundamental and sharp limitation of the duration of the incubation period and the duration of the action of immunity [4, 11]. This factor acts in the same direction as correlations, and apparently can be considered together with them.

## VII. ONE WAVE EPIDEMIC IN CHINA

In our previous work [8], we showed that period of the epidemic waves tends to infinity when the elimination rate tends to zero, $\sigma \to 0$. Then the whole epidemic comes down to one wave, as happened in China. This corresponds to the extremely low elimination rate $\sigma < 0.01$ obtained from the China data, Eq. (13).

Along with small value of $\omega = 0.1 \ll 1$ in China, this allows us to consider the special case $\omega = \sigma = 0$. Then the asymptomatic sectors of the iceberg model both for the SIRS-like system (1) and for the 3D Lotka-Volterra system (14) have the same form

$$\begin{cases} \frac{dS}{dt} = -IS \\ \\ \frac{dI}{dt} = (1-p)IS \end{cases} \tag{17}$$

with elementary kink solution

$$S = \frac{1}{1+\exp[(1-p)(t-t_0)]}, \tag{18}$$

$$I = (1-p)(1-S). \tag{19}$$

In accordance with Eq.(2) then there is only one wave of the symptomatic incidence:

$$\frac{dI_s}{dt} = \frac{p(1-p)}{4\cosh^2\left[(1-p)(t-t_0)/2\right]}. \tag{20}$$

In our opinion, this one-wave solution shown in Fig.9 is the most important in the whole family of the wave solutions presented by Fig.8 of the previous section. We assume that this solution describes the epidemic situation in China (compare with Fig.6). The absence of repeated waves indicates a high degree of stability of the virus ($\omega, \sigma \to 0$), and an extremely low morbidity means its non-pathogenicity ($p \to 0$).

**The question of the nature of such a non-pathogenic strain requires virological research. If detected, it seems reasonable to provide its spread to displace the less stable and more pathogenic strains that are shaping the current course of the global pandemic.**

## VIII. DRIFT OF PARAMETERS

So far, we have assumed the parameters of the iceberg model to be constant. However, over time, epidemic factors change, which leads to a change in the parameters. If this change is sufficiently slow, then the same solutions nevertheless can be used, simply by replacing constant parameters with the corresponding functions of time, considering the drift of epidemic factors. The values of the parameters (12,13) found above should be interpreted as averages over a certain period and may change in the future.

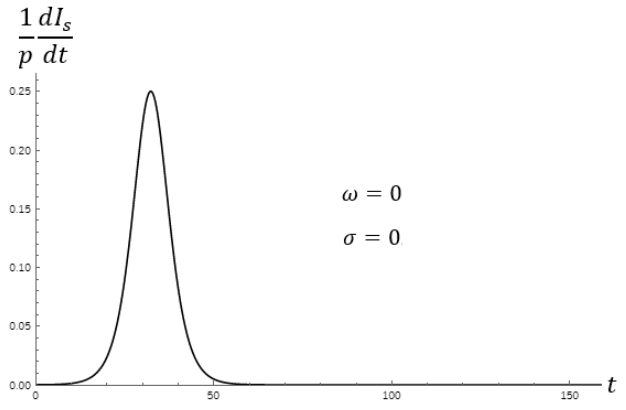


FIG. 9. Daily incidence $dI_s/dt$ in the one-wave solution (18,19) which corresponds to absolutely stable strain, $\omega = \sigma = 0$. This solution is supposed to describe the COVID-19 epidemic in China.

The factors influencing the course of the epidemic are 1)viral mutations, 2)lockdown and 3)vaccination. Below we consider all them sequentially.

1. **Viral mutations** that inevitably occur during the COVID-19 epidemic are an evolutionary mechanism that ensures the survival of coronaviruses as a species. Over time, those strains that provide the largest proportion of those infected will replicate most successfully. Since the overwhelming majority of those infected by the coronavirus are *asymptomatic*, this corresponds to the maximum value of the proportion of *asymptomatic* infected $I$.

In accordance with Eq.(3), the equilibrium value of this proportion at $t \to \infty$ is

$$I = \frac{1-\omega}{\sigma+\omega}\sigma \tag{21}$$

and tends to its maximal value $I = 1$ for $\omega \to 0$ regardless of the two other parameters $p$ and $\sigma$. This corresponds to a stable strain that remains active and can spread for a long time $T_\omega = 1/\omega \to \infty$. Therefore, the viral mutations should lead to the gradual decrease in the deactivation rate $\omega$ and, accordingly, in the increase in the proportion of the *asymptomatic* infected $I$.

Corresponding evolution of the equilibrium *symptomatic* daily incidence $dI_s/dt$ due to the mutations, as a function of parameter $\omega$ , follows from Eq.(4). This dependence is shown in Fig.10 and has maximum at $\omega = \sqrt{\sigma^2+\sigma} - \sigma$. In this graph, the Greek letters from $\alpha$ to $\delta$ shematically show successive mutations of

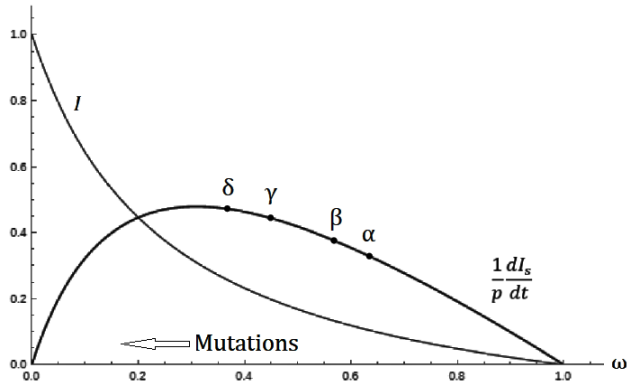


FIG. 10. Viral mutations lead to the maximal proportion of the *asymptomatic* spreaders $I$ and after maximum to zero value of the *symptomatic* daily incidence $dI_s/dt$. The two curves are described by Eqs.(4) and (21). Notations $\alpha, \beta, \gamma$ and $\delta$ mean subsequent viral mutations with decreasing deactivation rates $\omega$.

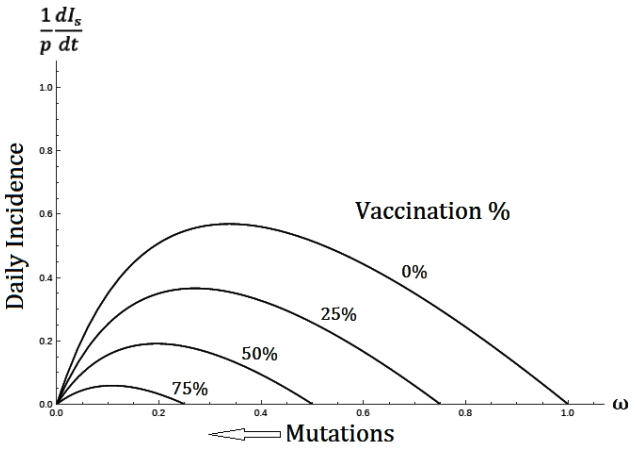


FIG. 11. Dependence of the *symptomatic* daily incidence on the viral mutations for different vaccination rates.

the COVID-19 strain.

In accordance with Fig.10 we assume that recent viral mutations from the Britain $\alpha$ strain to the Indian $\delta$ strain are responsible for the increasing in the *symptomatic* incidence, which was reported in fall 2020 and winter 2021 year. Based on these two graphs, we expect the *symptomatic* incidence to drop to zero due to subsequent mutations. At the same time, the proportion of *asymptomatic* infected should increase.

2. **Lockdown** like other quarantine measures, leads to a temporary decrease in the spread rate. The reduction can be described by the factor $Q(t) < 1$, which is equivalent to replacing the usual time $t$ with the "epidemic time" $\tau$ according to the relationship $d\tau = Q(t)dt$.

In this way, the time scale is locally stretched, and the incidence decreases with the same factor $Q(t)$. The purpose of the lockdown is to temporarily reduce the incidence. This is necessary in the context of a rapid increase in morbidity and a shortage of medical resources. The lockdown does not stop the development of the epidemic, but only stretches it over time [2].

3. **Vaccination** in the ideal case provides the emergence of adaptive immunity and transfers each vaccinated from the group $S$ to the group $R_s$ bypassing the stage of symptomatic disease $I_s$, see Fig.1. In accordance with phase portrait in Fig.3 bulk vaccination leads to accelerated progress along the same rectilinear phase trajectory $OE$ towards the same end point $E(\omega, 0, 0)$.

Let the vaccinated rate be $v$. Then this part of the population leaves the asymptomatic sector of the epidemic, and instead of the previous norm (6), the sum of the variables is $S + I + R = 1 - v$. As a result, the equilibrium point $O$ moves to a new position

$$O\left(\omega, \frac{1-v-\omega}{\sigma+\omega}\sigma, \frac{1-v-\omega}{\sigma+\omega}\omega\right) \tag{22}$$

and the equilibrium symptomatic incidence (4) at $t \to \infty$ becomes a form

$$\frac{dI_s}{dt} = p\sigma\omega\frac{1-v-\omega}{\sigma+\omega}. \tag{23}$$

This dependence of the daily incidence on the $\omega$ that is on the viral mutations for different vaccination rates $v$ is shown in Fig.11. Depending on the deactivation rate of the virus ($\omega$) the minimal required level of vaccination to stop the epidemic is $v = 1 - \omega$. On the other hand, regardless of the vaccination rate the daily incidence tends to zero when $\omega \to 0$. This is due to the increased protection from the innate immunity, supported by the long-term presence of the virus in the *asymptomatic* $R$ state.

Being considered as a function of the $\omega$ and $\sigma$ parameters this incidence has a maximum at $\omega = (1-v)/2, \sigma \to \infty$. The value of this maximum is

$$\left(\frac{dI_s}{dt}\right)_{max} = \frac{p}{4}(1-v)^2. \tag{24}$$

Its meaning is the absolute maximum of the *symptomatic* daily incidence for any mutations of the virus at given pathogenicity factor $p$ and vaccination rate $v$. This parabolic dependence on $v$ for the world pandemic is shown in Fig.12. The curve describes the dependence of the *average symptomatic* incidence on the level of vaccination $v$ and does not take into account the fluctuation effect of epidemic waves.

## IX. CONCLUSIONS

Thus, the iceberg model, considering separately asymptomatic and symptomatic infected, is able to describe both the average course of the epidemic and the epidemic waves. The model based on the conventional SIRS equations for the asymptomatic sector describes a

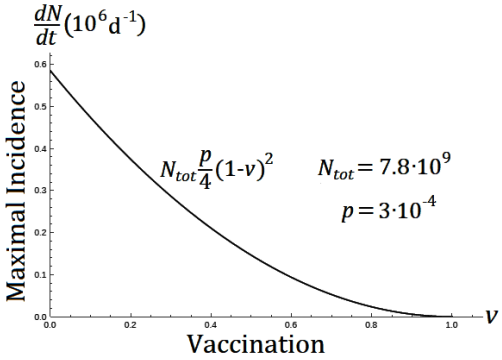


FIG. 12. Absolute maximum of the *symptomatic* daily incidence in the world as a function of the vaccination rate $v$ in accordance with Eq.(24). Corresponds to the total population of the world $7.8 \cdot 10^9$, the pathogenicity factor $p = 3 \cdot 10^{-4}$ found by fitting with the pandemic data, Eq.(12)

plateau, while accounting for strong biological correlations based on the 3D Lotka-Volterra equations results in persistent epidemic waves.

At its core, the iceberg model is phenomenological. It is based on the well-known fact that a significant proportion of those infected are asymptomatic and appear to be the main contributor to the spread of infection.

The iceberg model describes each individual infection as a fundamentally random process. The character of this process is determined soon after infection, at the point of bifurcation. After passing this point, the infected person either gets sick or becomes an asymptomatic carrier.

It is worth to note that virus infection plays a twofold role in the asymptomatic state. On the one hand, it can be transmitted further, which contributes to the development of the epidemic. On the other hand, it protects the asymptomatic infected from an immediate symptomatic infection, that is, from disease, since the innate immunity is already in a mode that keeps the infection at a safe level. During the latent period nonspecific innate immunity guarantees the same effective protection against disease as antibodies. However, after re-infection, the process may go differently and the person may get sick. Unlike protection by antibodies, the duration of which is determined by the time of their preservation, in this case the duration of protection is determined by the lifetime of the virus in the body.

In papers [13, 14] the idea of temporary protection by a virus was considered in the case of two alternating epidemics with two different viruses. In the iceberg model, there are not two different viruses, but two types of immune response to the same coronavirus. Unlike the regular flu epidemic, in which the same people carry the virus and get sick, in the current COVID-19 epidemic, some people carry the virus and others get sick.

**Apparently, a unique feature of the COVID-19 epidemic is that the level of infection that is safe for absolute majority of humans is nevertheless unsafe for the human society as a whole and leads to an epidemic spread of the symptomatic disease.** The reason for this is, on the one hand, an exceptionally low likelihood of a symptomatic course, and on the other hand, a high degree of infectiousness. The iceberg model assumes that the vast majority of infected are latent and spread the infection. However, any of them, having come out of this state and re-infected, can get sick.

Based on the presented iceberg model of the COVID-19 epidemic, we can draw the following conclusions.

1. During the COVID-19 epidemic, the vast majority of the population is infected and is in a latent asymptomatic phase. A small proportion of those infected become ill and go through the symptomatic phase.

2. The COVID-19 virus is permanently circulating in the population, which manifests itself both in the form of a plateau of incidence and in the form of waves. After elimination of the virus from the asymptomatic infected persons, they may become ill as a result of subsequent re-infection.

3. Without taking any measures, the natural duration of the epidemic can be decades. Bulk vaccinations can shorten this time, but the required vaccination rate is increased due to viral mutations. On the other hand, the mutations themselves lead to gradual decreasing the symptomatic incidence (see Fig.10) and thus suppress the epidemic.

4. **The almost complete cessation of the COVID-19 epidemic in China indirectly indicates the widespread in China of a stable, non-pathogenic strain that arose at the early stage of the epidemic due to mutation. Its presence could provide effective protection against infection by the original pathogenic virus. If such a strain were found, it would be wise to use the targeted spread of this strain to effectively end the epidemic, as has already happened in China.**

This approach is symmetric to the vaccination of humans, where there is an accelerated replacement of human immunity, which is usually carried out through disease. In this approach, there is an accelerated replacement of the virus with its non-pathogenic strain, which is usually carried out through slow mutations. Here, the non-pathogenic strain plays the role of **universal vaccine**, which is based on innate immunity and therefore insensitive to mutations of the pathogenic strains.

Since now new pathogenic strains appear and vaccination is faced with a number of problems, the proposed method may turn out to be relevant. Therefore, virological research aimed at identifying the non-pathogenic strain is advisable right now.

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