Superspreading as a Regular Factor of the COVID-19 Pandemic

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An impact of superspreading on the course of the COVID-19 epidemic is considered. A two-component model of the epidemic has been developed, in which all infected are divided in two groups. They are asymptomatic superspreaders spreading the infection and sensitive persons which can only get infection. Once infected the sensitive exhibit clear symptoms and become isolated. Taking into account both factors allows find the numbers of superspreaders and sensitive persons. It is shown, that the ratio of increment of the number of daily cases in the beginning of the epidemic and decrement at the end of the epidemic is equal to the ratio of numbers of the superspreaders and sensitive persons. In this way, on the base of data from 12 countries and territories, the share of asymptomatic among all infected persons is found to be $(17 \pm 3)\%$. Specific measures to limit the epidemical incidence are proposed. The possibility of an allergic component in the disease is discussed.

I. INTRODUCTION

The COVID-19 pandemic has a number of differences from previous flu epidemics.

1. One of its main features is the existence of active asymptomatic carriers of the virus - superspreaders [1]. They cause a significant contribution to the spread of the virus because once infected do not sick and therefore are not isolated. Such carriers can be detected by random testing or by testing of contacts of already infected persons (which, of course, is more effective).

2. Another feature of the COVID-19 pandemic is the significantly lower number of cases (about $0.2-2\%$) than the normal flu epidemic (10 – 20\%). This suggests that only a small fraction of people are sensitive to the SARS CoV-2 virus that is responsible for the current pandemic. As in the case of the superspreaders, they have also been successful in reproducing the virus, but this is now accompanied by a noticeable immune response and thus a disease.

The aim of this work is to take into account the impact on the spread of the COVID-19 epidemic of both factors.

1. The presence of a limited number of people who are susceptible and insensitive to the virus and who, once infected, become asymptomatic carriers and spreaders of the virus;

2. The presence of a limited number of people who are both susceptible to and sensitive to the virus and who, once infected, exhibit a strong immune response and get sick.

This approach treats superspreaders as not as an exotic factor leading to separate outbreaks of the virus, but as a regular element in the dynamics of the epidemic. It is this element that defines the main features of the development of the COVID-19 pandemic.

The article is structured as follows.

The second part classifies those infected by the nature and intensity of the immune response, which is the basis for the further proposed epidemiological model.

In the third section, a corresponding two-component model of the epidemic is constructed and its analytical solution is found.

The fourth part compares the found results with observed epidemics in a number of countries and territories.

The final part summarizes the application of the model and discusses the possibilities for limiting the incidence resulting from this model.

II. IMMUNOLOGICAL CLASSIFICATION OF INFECTED

From an epidemic point of view, the mobility of infected persons is the most important factor, as only the mobile infected persons can transmit the infection. It is therefore reasonable to divide those infected into two broad groups by degree of mobility. Since mobility, in turn, is determined by the presence of pronounced symptoms of disease that are linked to immune response, such separation must be based precisely on the nature and degree of the immune response.

During the COVID-19 pandemic, a significant amount of data was collected describing the nature and diversity of the human immune response to the virus. In this section, we will use part of this data to limit and give an immunologically accurate definition of the two main groups of the model - superspreaders and sensitive.

The basis of the definition is the appearance of antibodies to the virus in the mucous membrane (IgA immunoglobulin), as well as in the blood (IgG immunoglobulin). Tests taken in persons with different degrees of severity, from asymptomatic to acute, show the following pattern [2].

1. For asymptotically infected persons, both types of antibodies are largely absent. This fact points to the category of superspreaders and indirectly explains the large number of viruses in such carriers, resulting in numerous cases under certain circumstances.

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NOTE: This preprint reports new research that has not been certified by peer review and should not be used to guide clinical practice.
in accordance with classification in Table 1, the isolated, and unable to transmit the infection. Thereby sensitive persons in this model are included in the group supersensitive. These infected people should also be classified as supersensitive, as they are usually hospitalized and anyway isolated. Unlike those who have experienced the disease in mild form, we will further refer this part of the category sensitive to the subcategory supersensitive. This separation does not affect the proposed two-component model, which is concerned only with the fact that the carrier is isolated.

In this way, one can give an immunologically precise definition of the groups superspreaders and sensitive. The first includes only asymptomatic carriers, which are not subject to any isolation and spread the infection. The second is symptomatic, isolated and therefore unable to spread the infection. This division is presented in the Table 1. The sensitive group has a subgroup supersensitive. It includes those infected who exhibit the strongest immune response, including antibodies in both the mucous membrane and the blood.

From an epidemic perspective, sensitive and supersensitive groups are equivalent, as in both cases the infected persons are isolated and unable to spread the infection further.

Based on the three types of reaction considered, two of them are polarly opposite: superspreaders and supersensitive. In the case of superspreaders, the immune response is completely absent. In the case of supersensitive, it is the maximum, see Fig. 1. The rest of the infected, referred to as sensitive, occupy the equatorial (intermediate) position in the figure.

III. THE TWO-COMPONENT MODEL OF THE EPIDEMIC

Consider an epidemic model involving only two types of potential carriers. These are superspreaders and sensitive persons. The superspreader, once infected, remains asymptomatic. He does not get sick and begins to spread the infection. Sensitive persons, been infected get sick, isolated, and unable to transmit the infection. Thereby in accordance with classification in Table 1, the supersensitive persons in this model are included in the group of sensitive.

Unlike the conventional one-component model of the epidemic, where anyone infected continues the chain of infection, in this model only superspreaders have this capability. The sensitive persons play a passive role only. They can get the infection but not spread it. Thus, the epidemic is two-component. The two components of the epidemic, sensitive and superspreaders, are progressing in parallel. The latter has a unilateral impact on the former, while the former cannot influence the latter.

Let the full number of the sensitive persons be $N_1$, of which $n_1$ are infected by time $t$. The total number of the superspreaders is $N_2$, of which $n_2$ are infected by time $t$. Then the dynamics of the epidemic is described by a system of equations

$$\frac{dn_1}{dt} = g \left( 1 - \frac{n_1}{N_1} \right) n_2, \quad (1)$$

$$\frac{dn_2}{dt} = g \left( 1 - \frac{n_2}{N_2} \right) n_2. \quad (2)$$

where $g$ is the spread rate. Its meaning is the average number of people who can be infected by one average superspreader in one day. The definitions of all variables and parameters of the model are given in Table 2, and the scheme of interaction between the groups of the superspreaders and sensitive leading to the dynamic system of equations (1, 2), is presented in Fig.2.

After introducing new dimensionless variables

$$\frac{n_1}{N_1} = s_1, \quad (3)$$

$$\frac{n_2}{N_2} = s_2, \quad (4)$$

$$gt = \tau, \quad (5)$$

Table 1: Definition of two groups (superspreaders and sensitive/supersensitive) by the type of immune reaction

|             | Virus | IgA | IgG | Mobility |
|-------------|-------|-----|-----|----------|
| superspreader | +     | -   | -   | free     |
| sensitive   | +     | +   | -   | isolated |
| supersensitive | +     | +   | +   | isolated |
Table 2: Definitions of model variables and parameters

| Variables and parameters | Definition |
|--------------------------|------------|
| \( n_1 \)               | Current number of infected sensitive persons |
| \( n_2 \)               | Current number of infected superspreaders |
| \( N_1 \)               | The total number of sensitive persons |
| \( N_2 \)               | The total number of superspreaders |
| \( t \)                 | Time (in days) |
| \( g \)                 | Spread rate (in 1/day) |

Fig. 2: Scheme of the two-component epidemic model

The initial system of equations (1, 2) acquires a form of

\[
\begin{align*}
\frac{ds_1}{d\tau} &= \alpha (1 - s_1) s_2, \\
\frac{ds_2}{d\tau} &= (1 - s_2) s_2,
\end{align*}
\]

(6, 7)

where

\[
\alpha = \frac{N_2}{N_1}
\]

(8)

is a Relative Number of Superspreaders (RNS).

The resulting system of equations (6, 7) has an exact solution

\[
\begin{align*}
s_1 &= 1 - \left[1 + s (e^\tau - 1) \right]^{-\alpha}, \\
s_2 &= s s + (1 - s) e^{-\tau},
\end{align*}
\]

(9, 10)

that meets the initial conditions

\[
\begin{align*}
s_1(0) &= 0, \\
s_2(0) &= s.
\end{align*}
\]

(11, 12)

The given solution corresponds to the initial state, in which there is already a non-zero number of infected superspreaders, \( n_2 = sN_2 \), but no sick persons yet, \( n_1 = 0 \).

The empirical value observed is the daily number of cases \( \frac{dn_1}{dt} \), which according to the solution (9) depends on time as follows (see Appendix A):

\[
\frac{dn_1}{dt} = gN_1 \frac{\alpha se^{\alpha t}}{[1 + s (e^{\alpha t} - 1)]^{1+\alpha}}.
\]

(13)

Fig. 3: Dependence of the daily number of cases on time at different values of \( \alpha = N_2/N_1 \)

An important property of this solution is the asymmetry of its exponential asymptotic at the beginning and end of the epidemic, i.e. for \( t \to 0 \) and for \( t \to \infty \). At the beginning of the epidemic, it behaves as

\[
\frac{dn_1}{dt} \propto e^{gt} \quad (t \to 0),
\]

(14)

and at the end of the epidemic as

\[
\frac{dn_1}{dt} \propto e^{-\alpha gt} \quad (t \to \infty).
\]

(15)

Three graphs in Fig.3 demonstrate the growth of this asymmetry as the Relative Number of Superspreaders \( \alpha = N_2/N_1 \) decreases. The smaller the value of the parameter, the slower the daily number of cases decreases. This is quite natural, because the decline occurs by exhausting the fraction of susceptible in the \( N_1 \). The speed of this process in the two-component epidemic model is determined by the number of superspreaders, which tends to \( N_2 \) towards the end of the epidemic. As follows from the asymptotic (14, 15), the ratio of the exponential decrement at the end of the epidemic and the increment at its beginning directly gives the parameter \( \alpha = N_2/N_1 \), i.e. the Relative Number of Superspreaders. On the other hand, the asymptotic value of the total number of cases at the end of the epidemic is \( N_1 \), because the superspreaders do not get sick. From here one can immediately get \( N_2 = \alpha N_1 \) - the total number of superspreaders capable of spreading the virus during the epidemic.

It is important that from this simple calculation falls out the spread rate \( g \), which is unknown in advance and different for different countries and localities. In general, all four parameters \( (N_1, N_2, s, g) \) are uniquely determined by the two asymptotics of the epidemic, or by its initial asymptotic and the position of the maximum point.

The analytical solution (9, 10) enables to find the exact position of the maximum point for the daily number of cases, as is done in Appendix A. If the spread rate constant \( g \) increases, the maximum is reached earlier. The
increase in the number of superspreaders, expressed in the growth of the parameter $\alpha = N_2/N_1$, has the same effect, as shown in Fig.3. Comparing the course of two epidemics, the Spanish flu in 1918, and the current COVID-19 epidemic, shown in Fig.4, exhibits a marked difference between them in the final phase of the decline. In the case of COVID-19, it occurs much more slowly. The two-component epidemic model can explain this by assuming a relatively small fraction of superspreaders, $N_2/N_1 \ll 1$, as shown by the third graph in Fig.3.

In the proposed model, the virus first undergoes exponentially rapid spread among superspreaders and sequential activation, which means infection, most of them. Then activated superspreaders continue to spread the virus among the sensitive persons. The number of spreaders at the point of maximum is already saturated, and the base for further spreading of the virus among sensitive is also gradually being exhausted. Therefore, the epidemic is gradually subsiding.

### IV. ANALYSIS OF THE EPIDEMIC IN DIFFERENT COUNTRIES AND STATES.

Next, consider the course of the epidemic in a number of countries and states where it has already peaked and is expected to end soon. In each case, we fit the empirical daily number of cases by the result (13) of the two-component model.

Fig.5 shows the results of this fitting for 12 countries and states: Austria, Belgium, Canada, France, Germany, Italy, Netherlands, New Jersey and New York, Spain, Switzerland and the United Kingdom. The empirical values are shown by points, and the model result is shown by a red line. In each case both the full number of the sensitive persons $N_1$ and the full number of the superspreaders $N_2$ were found. The parameters following from this fitting are summarized in Table 3.

In all cases shown in Fig.5, the exponential growth of daily morbidity at the start of the epidemic is much faster than its further decline. The reason for this is that in all cases the Relative Number of Superspreaders $\alpha = N_2/N_1$ is less than one. However, as can be found from Tab.3, the absolute number of the superspreaders $N_2$ is by no means small. The share of this group is equal to (17±3)% of the total number of infected $(N_1 + N_2)$.

The very narrow range of values of the parameter $\alpha$, mainly from 0.2 to 0.3, is noteworthy, while the incidence varies much more widely - from 0.2% to 2%.

### Table 3: Model parameters for some countries and states.

| Country/State | $N_1$ (M) | $N_2$ (M) | $100\% \cdot N_2/N_1$ | $N_1$ | $N_2$ | $g$ (1/d) |
|---------------|-----------|-----------|-----------------------|-------|-------|----------|
| Austria       | 9,000,000 | 16,000    | 0.18%                 | 5,000 | 5,1 | 0.29    |
| Belgium       | 9,000,000 | 60,000    | 0.67%                 | 19,000| 32   | 0.16    |
| Canada        | 38,000,000| 120,000   | 0.32%                 | 35,000| 29   | 0.10    |
| France        | 65,000,000| 155,000   | 0.24%                 | 35,000| 23   | 0.21    |
| Germany       | 84,000,000| 185,000   | 0.22%                 | 45,000| 24   | 0.24    |
| Italy         | 60,000,000| 250,000   | 0.42%                 | 50,000| 20   | 0.20    |
| Netherlands   | 17,000,000| 50,000    | 0.29%                 | 16,000| 32   | 0.15    |
| New Jersey    | 9,000,000 | 185,000   | 2.06%                 | 35,000| 19   | 0.18    |
| New York      | 19,000,000| 420,000   | 2.21%                 | 50,000| 21   | 0.22    |
| Spain         | 47,000,000| 300,000   | 0.64%                 | 40,000| 13   | 0.27    |
| Switzerland   | 9,000,000 | 30,000    | 0.33%                 | 9,000 | 20   | 0.25    |
| UK            | 60,000,000| 350,000   | 0.51%                 | 65,000| 18   | 0.14    |
V. CONCLUSIONS AND DISCUSSION

Thus, despite all its simplicity and schematicity, the proposed two-component model gives a plausible description of the course of the COVID-19 epidemic in all considered cases. Extending the model by taking into account the deactivation of superspreaders over time (see Appendix B) does not, however, lead to any qualitative change in the results.

The proposed model is based on two specific types of virus carriers. They are

1) superspreaders, in which the infection results in active reproduction of viruses without appreciable immune response and any symptoms of the disease, and

2) sensitive persons, in which infection leads to the reproduction of viruses, and to a noticeable, and in the case of supersensitive often to an acute immune response. It is this reaction that manifests itself as the terminal course of the disease.

The rest of the population is neutral in relation to the virus - in most cases the virus appears not to be reproducible in significant amounts in cells. It should be noted that this is the absolute majority, currently 99.87% of the total population of the Earth.

The comparison of possible variants of the body’s immune response to the COVID-19 pathogen presented in the Table 1 raises the question of which of these variants is adequate. There are two possible answers to this question:

1) The immune response is generally adequate to the degree of damage that the virus causes to the cell. In the case of superspreaders, the virus for some reason does not cause significant damage to the cells, and therefore there is no immune response. In the case of supersensitive, the virus causes significant damage, which causes a strong immune response and leads to the terminal course of the disease.

2) The immune response is largely inadequate to the degree of damage that the virus causes or may cause to the cell. In the case of superspreaders, the immune system ignores the appearance and reproduction of the virus and the person remains virtually healthy. In the case of supersensitive, a strong immune response occurs and the person is exposed to the disease in an acute form.

Both approaches describe the same infection with the same result, the difference between them is in the interpretation of the nature of the immune response.

In the first case, it is considered as an adequate response to the virus. In the second case, the immune response is interpreted as an inadequate allergic reaction to the very presence of the virus, or to the products of its activity. And then the allergic component plays a significant role in the course of the disease.

In the first case, a viral infection is the cause and the immune response is a consequence of the disease. In the second case, the viral infection is a virus agent that provokes an inadequate response of the immune system.

Fig. 5: Comparison of course of COVID-19 epidemic in 12 countries and territories (points) with two-component model (red line)
The answer to this question determines what measures can reduce the incidence.

In the first case, it is necessary to activate a specific immune response through a proper vaccination. The target group of the vaccination here is superspreaders. Then the reduction of incidence is achieved by reduction of the number of the superspreaders via transferring them to an intermediate group of sensitive.

In the second case, the aim of a proper vaccination is to reduce a specific immune response of the supersensitive. Then the target group of the vaccination is supersensitive, and the reduction of incidence and lethality is achieved by transferring the supersensitive to the more safe intermediate group of sensitive.

And in both cases it seems reasonable to take preventive measures to early identify superspreaders and supersensitive, followed by limiting their contacts.

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Appendix A

As follows from formula (10) of the main text for \( s_1 \) ist derivative is

\[
\frac{ds_1}{d\tau} = \frac{\alpha s e^\tau}{[1 + s (e^\tau - 1)]^{1+\alpha}}.
\]

(A1)

After replacing the variables (3-5), this gives formula (13) for the daily number of cases.

To find the point of the maximum, we write out the logarithm of \( ds_1/d\tau \) and turn to zero its derivative:

\[
\ln \left( \frac{ds_1}{d\tau} \right) = \ln (\alpha s) + \tau - (1 + \alpha) \ln [1 + s (e^\tau - 1)],
\]

(A2)

\[
\frac{d}{d\tau} \left( \frac{ds_1}{d\tau} \right) = 1 - \frac{(1 + \alpha) s e^\tau}{1 + s (e^\tau - 1)} = 0.
\]

(A3)

This directly follows the point of maximum:

\[
\tau_{\text{max}} = \ln \left[ \frac{1}{\alpha} \left( \frac{1}{s} - 1 \right) \right]
\]

(A4)

and the maximum number of daily cases

\[
\left( \frac{ds_1}{d\tau} \right)_{\text{max}} = \left( \frac{\alpha}{1 + \alpha} \right)^{1+\alpha} (1 - s)^{-\alpha}. \quad \text{(A5)}
\]

Taking into account the replacement of variables (3-5), this gives the point of the maximum \( t_{\text{max}} \) and its peak value:

\[
t_{\text{max}} = \frac{1}{g} \ln \left[ \frac{1}{\alpha} \left( \frac{1}{s} - 1 \right) \right], \quad \text{(A6)}
\]

\[
\left( \frac{dn_1}{dt} \right)_{\text{max}} = gN_1 \left( \frac{\alpha}{1 + \alpha} \right)^{1+\alpha} (1 - s)^{-\alpha}. \quad \text{(A7)}
\]

Appendix B

Consider an extended two-component model in which superspreaders are deactivated for the time of \( 1/\mu \). Then the system of equations (6, 7) takes the form

\[
\frac{ds_1}{d\tau} = \alpha (1 - s_1) s_2, \quad \text{(B1)}
\]

\[
\frac{ds_2}{d\tau} = (1 - s_2) s_2 - \mu s_2, \quad \text{(B2)}
\]

Its exact solution is completely similar to the solution (9, 10)

\[
s_1 = 1 - \left[ 1 + \frac{s}{1 - \mu} (e^\tau - 1) \right]^{-\alpha(1-\mu)}, \quad \text{(B3)}
\]

\[
s_2 = \frac{(1 - \mu) s}{s + (1 - \mu - s) e^{-\tau}}. \quad \text{(B4)}
\]

It differs from the previously found solution at \( \mu = 0 \) by only two substitutions

\[
s \rightarrow \frac{s}{1 - \mu}, \quad \alpha \rightarrow (1 - \mu) \alpha. \quad \text{(B5)}
\]

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