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NEW MATHEMATICAL MODELS OF THE SPREAD OF VIRAL INFECTIONS

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Abstract – *The subject and purpose of the work - the COVID-19 pandemic - has so far affected almost all countries of the world and sharply reminded of the need for further research on many aspects of viruses. An attempt to use classical approaches to describe the mechanisms of transmission and spread of this infection and the related methods of carrying out sanitary and preventive measures turned out to be ineffective. Nowadays, the creation of models of new viral infection has acquired particular relevance, allowing one to explain the features of its course observed in practice and to propose a mathematical description of the mechanism of its spread. Methods and methodology of work. The theory of probability and mathematical statistics, numerical modeling tools for solving systems of ordinary differential equations describing the spread of a viral infection are used. Results of work. A lego model for describing the structure of a compound viral infection is proposed, which makes it possible to explain the possibility of recurrent disease and the presence of several waves of it. Estimates of the probability of disease and mortality from the number of fragments of lego infection were obtained. The need to comply with special sanitary and hygienic measures to reduce the likelihood of severe disease and mortality is indicated. Using compartment models, a system of equations has been proposed to describe the dynamics of the spread of a new coronavirus infection COVID-19, which takes into account the presence of the latent period of infection, as well as the possibility of additional infection in a medical institution.*

Key words – *COVID-19 virus infection, mathematical model, mathematical statistics, numerical modeling, spread of viral infections.*

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I. INTRODUCTION

The COVID-19 pandemic is a stark reminder of the need for further research on many aspects of viruses. An attempt to use classical approaches to describe the mechanisms of transmission and spread of this infection and the related methods of carrying out sanitary and preventive measures turned out to be ineffective. It is urgent to create models of a new viral infection and a mathematical description of the mechanisms of its spread.

The models for the spread of coronavirus infection have created [1, 2], which have shown good results in assessing the restrictive measures taken. Scientists view the epidemic as a typical example of a chain reaction. One patient infects several healthy people, which in turn infects several more, and so on. The equations of the model are similar to the equations describing a chain reaction in a nuclear bomb or nuclear reactor. The model is very simple, and its solution depends on the difference between only two main parameters - the rate of infection and the rate of recovery. If the difference is positive, then the epidemic grows, if negative, then it dies out. It was alarming that the rate of infection was three to four times higher than for ordinary flu or such a dangerous disease as Ebola. At the same time, the mortality rate of 10 ... 20%, declared in the world at the initial stage of the epidemic, turned out to be quite high (for ordinary influenza, the mortality rate is less than 0.5%).

The analysis showed that in order to extinguish the epidemic, it is necessary to reduce the infection rate by at least four times. Scientists at the nuclear center in their calculations simulated a decrease in this speed, taking into account several options. The rate of infection is proportional to the proportion of non-immune people. Therefore, when 80 percent of the population is ill, the epidemic will die out on its own. An analogue of this is the burnout of fuel in a nuclear reactor, leading to its shutdown. This is acceptable, and is usually the case for epidemics with low mortality. Another way to fight the epidemic in the bud is artificial immunization. It deprives the virus of its breeding ground. Finally, it is possible to reduce the rate of infection by limiting contact between people, which has been done in a number of countries, in particular in Ukraine and Russia at the initial stage of the fight against COVID-19. But this measure is temporary, because as soon as the quarantine is lifted, the epidemic will flare up again.

The SEIRD differential model, despite its simplicity, has played an important role in understanding the basic laws of epidemic development [2]. A key addition was the inclusion of asymptomatic virus carrier factor - E in the equation. These are people who, having become infected, are asymptomatic, do not go to doctors, but continue to lead a normal life and communicate with others. It is these people who are the main channel for the spread of infection, since the identified patients with symptoms, as a rule, are immediately isolated from society and do not participate in the further spread of infection.

One of the unexpected results of the calculations was the fact that the introduction of quarantine measures for the first two to three weeks at the beginning of the epidemic has little effect on the final result, leading only to a delay in the main peak of infections in time and practically without changing its amplitude. Nevertheless, such a delay is useful in terms of mobilizing and preparing the health system for the development of the epidemic, as well as additional time for developing a vaccine, scientists say.

As numerous calculations have shown, the key in predicting the further development of the event is the question of the proportion of

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asymptomatic virus carriers. Depending on this indicator, decisions can go according to both a favorable scenario and an extremely unfavorable one. It is currently not possible to determine this coefficient directly from the simulation. Therefore, while the scientists of the nuclear center continue to monitor the development of epidemics in some regions of the world - in New York, in Lombardy, in Wuhan, in Moscow.

It can be seen that the restrictive measures taken by the authorities are having their effect, and in a number of regions the number of infections is already declining. This does not mean that after the abolition of quarantines, the epidemic cannot break out again. And recent events confirm this. Now there is another wave of increase in the incidence. But quarantines allow us to achieve two extremely important things: firstly, with a decrease in the rate of infection during quarantine, the peak load on the medical system decreases, which means more people will be saved, and secondly, quarantine delays the course of the epidemic in time, giving virologists what they need. time to study the virus, the course of the disease, methods of treatment and, finally, create a vaccine with which it will be possible to immunize the population and then declare the final victory over the epidemic. Therefore, it is so important to continue to comply with the introduced quarantine measures, no matter how difficult they seem, scientists say.

Despite the lack of detailed statistical data, including on the proportion of asymptomatic virus carriers, the constructed model of the nuclear center scientists showed good prognostic results.

One more model for the spread of COVID-19 was created [3], which can be used by the authorities to counter the second wave of the epidemic, as well as further under any epidemiological situation. According to experts, COVID-19 has a high potential mortality, victims die on average 10 years earlier than probable death from other pathologies, while not all cases of coronavirus infection are included in the statistics.

The work of these scientists involves the inclusion of a wide list of deviations in the model: asymptomatic groups that have passed and have not been tested for coronavirus, patients with a severe course of the disease and complications diagnosed as patients with pneumonia, and others. Scientists have created a meta-model, a "model of models" for the spread of COVID-19, which in its calculations relies on all previously built forecasting systems that have calculated some part of the indicators, which becomes especially significant taking into account the high probability of the onset of subsequent waves of the epidemic. The main conclusion based on the simplest first-level models for an individual city is a clear dependence of a decrease in the spread of infection on a decrease for movement of people, in particular, after the introduction of a lockdown.

The plans of the specialists are to create a multiscale model of the spread of COVID-19 (as well as any subsequent viral infection) including, in particular, the interaction of the virus and the cell, its spread throughout the body and the emergence of an immune response, vaccination, assessment of the impact of a pandemic on the economy of the region, the response of the health care system and authorities, population and other modules. Despite the obvious advantages of the considered models. Their main disadvantage is the use of classical concepts to describe the methods of spreading a new coronavirus infection, which they encountered for the first time.

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II. LEGA MODELS OF VIRAL INFECTIONS

A study published in the journal eLife [4] showed that a new viral genome could be shared between different cells and still provoke an infection. This overturns the modern understanding of viral diseases. It turns out that different parts of the viral genome can live in different cells, but at the same timework together to cause infection. This discovery undermines the traditional concept of the action of the virus in the cell, according to which the viral genome first replicates in one cell, and only then proceeds to replicate in another. In multipartite viruses, the genome is divided into several segments, each of which is enclosed in a separate viral particle. Previously, it was believed that in order to cause infection, all segments of the viral genome must move together from cell to cell. However, new research shows that this is not the case.

In the process of genome transfer, there is a high probability that the virus will lose a significant segment of it, but the remaining segment can cause infection.

Perhaps this "multicellular" lifestyle is found in different viruses. This remains to be seen in further experimentation. However, it is already clear that this discovery opens up a completely new horizon of research in virology.

Thus, a new type of coronavirus infection Covid 2019 may represent a completely new type of viral infection, both in terms of a mathematical description of the spread of the epidemic and the pathogenic effect and formation of immunity, which requires the creation of new models and methods of mathematical description [21, 22].

Consider the new Covid 2019 infection as consisting of several fragments that can exist and replicate in the body at the same time, enhancing the pathogenic effect achieved. This provides a clue to understanding some of the hitherto incomprehensible features of the new coronavirus infection. In a simplified way, the situation looks like this: a coronavirus infection is a lego, consisting of many (several) pieces, each of which can exist in the host's body for a certain time. A person who is a carrier of a piece of Lego infection, communicating with other carriers and becoming infected from them, can collect other missing pieces in his body. When the accumulated pathogenicity (both due to the accumulation of a sufficiently large fragment of the infection and its amount - the dose) can break through the host's immune barrier, then his disease will occur. The degree of the disease is the more serious, the more part of the Lego infection a person has managed to collect in his body and the higher the dose of infection received. Therefore, those who lead an active lifestyle are more likely to get seriously ill and have the opportunity to communicate with a large number of carriers of various pieces of infection, which allows them to be put together in their bodies. In addition, it is clear that being in a team of infected people (for example, in a hospital in a ward with the same patients, but who can be sick with other fragments of infection) allows due to mutual infection (collecting additional pieces of Lego in your body that your roommates have) enhance the effect of the disease. Therefore, if in a hospital the infected are kept together, then the damaging effect of the disease will be higher.

The considered hypothesis of the existence of coronavirus infection allows us to formulate conclusions that are useful in practical terms.

1. The main thing is to minimize new contacts with possible other carriers of Lego parts, so as not to give an opportunity to collect a large piece of it in your body, sufficient for serious damage to the whole body by the disease. Moreover, the moment of onset of the disease will be determined by the impossibility of resistance of the immune system to a piece of

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infection already collected in the body. It follows that people with a strong immune system may not get sick until they collect most of the lego infection in the body and then the disease breaks through the immune barrier and a serious illness begins, and the damaging factor is not so much the infection itself, but also an inadequate immune response systems of the body, which destroys the body itself and leads to its death.

For older people with chronic diseases and a weak immune system, the disease begins much earlier, when a small enough piece of infection has been collected in the body, however, by placing the sick person in the hospital with the same patients, but with other pieces of Lego infection, we help the disease to collect a larger piece of Lego and strengthen its damaging effect.

2. It is clear that such an infection can have several waves of the disease (second, third, etc., depending on the number of fragments that the infection can consist of). A person who has recovered from part of the Lego infection can then collect another part of the Lego infection from other carriers in his body and get sick again. Then he can collect the third part, and so on. However, subsequent diseases, if fewer fragments of the infection are involved in them, will be with a milder course of the disease and less severe consequences.

3. If this viral infection is man-made, and the elegance of the idea of its creation speaks precisely of this, although now they are trying to convince us of its natural character with might and main, then the creators of the infection can, apparently, throw new pieces of Lego into the human community, thus strengthening way, the pathogenicity of the disease. This explains why the Asian version of Lego that started out was less pathogenic than the European version to which pathogenicity (additional pieces of the virus) were added. But this means that the current pathogenicity of the disease may not yet be maximum. The Creator, apparently, can enhance this pathogenicity at the next iteration step (East European or South Asian). Therefore, it is hardly worth taking seriously the hypothesis that the infection is most dangerous for the elderly. Yes, at the first stage of its existence (Wuhan), the most difficult consequences were for the elderly. however, at later stages (Western European, and then Eastern European), a high pathogenicity of infection can be expected for all age groups.

4. The question remains open for how long Lego pieces can exist in an active state in the human body without causing its morbidity (since the immune system blocks them), but causing infection of others. Apparently, such a deadline is about 14 days, with an average value of 4 ... 5 days. This means that if a person who has already become infected does not contact other carriers of other pieces of infection for a time longer than the lifetime of individual pieces of infection in his body, they will die and he will stop infecting others. In addition, after that, the assembly of the Lego infection in his body will start from scratch (including those pieces that he already had, but were deactivated) and he will get sick only when he collects enough for a new one (to break through his immune barrier) a piece of infection and a large enough dose.

5. Another question is also open. Having recovered from part of the Lego infection, a person apparently develops immunity to it, but he can get sick with another part of the Lego infection if he accumulates it in his body. If he does not get sick, being a carrier of a part of the infection, and after quarantine it ceases to be active in his body, then by contacting with other carriers, he can probably collect it again and get sick from it (reinforced by other pieces).

6. Many of us already, presumably, are carriers of parts of this terrible lego. The main thing is not to allow to collect part of it, sufficient for the disease, by limiting your contacts only to those

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close and closest to your constant environment. It is especially dangerous if the part of the lego infection collected in the body will also cause the body's immune response, which will destroy it. And this happens when the body accumulates a lot of pieces of infection with a greater total pathogenicity.

7 For sick people, if the severity of the disease is not critical, it is better to be sick at home. When treating in hospitals, it is necessary to isolate various patients in boxes, excluding the possibility of their mutual infection with various lego sites. If they are not isolated from each other, then due to mutual pollination and increased pathogenicity of the disease, the probability of dying will increase sharply.

Probably, this is related to the long periods of treatment (as can be seen from the statistics) of sick people in ward. On the one hand, a person is treated, and on the other hand, they are infected with other sections of the virus at the expense of neighbors with whom he lies together in the hospital, as well as doctors who carry fragments of the infection from different patients.

8. When will the coronavirus epidemic end?

As in the classics - when about 70% ... 80% of the population are ill. However, you can get sick in different ways. One scenario - quickly and immediately. Then many people will die, but the population will quickly develop immunity.

Another scenario is to get sick with pieces of this lego and in not severe forms. To do this, it is necessary to stretch the period of infection, try to zero out already infected pieces of Lego infection through quarantine measures. To enable doctors to work out treatment technologies and, most importantly, not to overload infectious diseases hospitals, so that they do not become a source of lethal infection. It is necessary to treat seriously ill patients in quarantine boxes, which exclude the possibility of their mutual additional infection.

9. For doctors who work in hospitals, the effect of accumulating exposure to infection will also be observed, and many of them can become seriously ill and die, which once again indicates that this disease destroys not only the elderly, but also healthy young people who have accumulated a sufficient dose of infection exposure. Therefore, after a while, the personnel working in COVID hospitals must be quarantined (for 14-21 days) and after zeroing out the parts of the infection present in their bodies, they can again be used in the treatment process. The time after which they need to be quarantined is determined by the degree of protection (suits, masks, glasses), which stretches in time the accumulation of the dangerous part (fragments) of the infection and its dose.

10. For young children. They quickly gain a small patch of lego-infection, which breaks through their still weak immune system, but has minor pathogenicity, and for the most part they will easily get sick. After that, they can again accumulate another small piece of infection and again get sick, and so on, they will get sick several times, accumulating immunity to individual parts of the virus and, ultimately, to the whole disease. In serious cases, the main thing is not to let the sick child come into contact with the same sick people not from his circle of friends, who will have other pieces of the Lego virus, which can increase the damaging effect. In some Western European countries, they are trying to follow the path of using children as natural vaccinators of adults, giving them the opportunity to get sick in a relatively mild form in children's institutions.

11. The pathogenicity of this virus, like many others, can be weakened by ultraviolet radiation, so the Sun can be the savior of people - it was not in vain that the ancients prayed to the god RA. This means that by the summer the pathogenic properties of the infection will weaken, but the next wave should be expected in the fall. But by this time, a part of society will

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have been sick with pieces of infection and there is a chance that the second wave may be weaker. However, the addition of lego fragments can lead to an increase in its pathogenicity.

Rather heated discussions arise regarding the possibility of a second illness and the presence of immunity in those who have been ill. The proposed Lego model of a new coronavirus infection allows you to answer these questions.

1. About the lack of immunity to the new virus. This seems to be a widespread misconception today. This virus, as previously known, after a person has had it, gives immunity. However, a person can get sick with part of this Lego - virus and it is to her that there will be immunity. To the other part of the virus, there will be no immunity and a person can get sick again.

2. Can recovered patients who test negative for the virus get sick again after a while? - Sure. After recovery, they acquire immunity to the part of the virus that they have had. Having accumulated new parts sufficient for the disease, they will naturally also have a positive reaction to the test. In addition, the virus in the process of development can acquire additional fragments and this will lead to both an increase in its pathogenicity and an increase in the number of possible waves of the disease.

3. To protect professions that are in contact with a large number of people by the nature of their activities and can collect pieces of the Lego virus from them, it is necessary to provide for a rotational work schedule. After work, apparently for 1-2 weeks, these people need to be quarantined (self-isolation) for 1-2 weeks to reset the viral load they received during their work. Then you can reuse them. Such a regime can not only protect these people from the disease, but also prevent infection from them to people with whom they are in contact by the nature of their activities. These categories include, first of all, doctors, public transport drivers, police officers, civil servants who receive citizens, including employees of executive committees.

4. In case of a shortage of places in hospitals, it is highly undesirable to re-equip sports complexes with large halls, in particular, sports palaces, since the presence in one room of a large number of patients, with possibly different fragments of a viral infection, can lead to their mutual "pollination" to an increase in the damaging effect of the infection. If necessary, additional places must be organized in hotels on the basis of 1 local numbers. This will maximize the isolation of patients from each other. After the end of the epidemic and carrying out quarantine measures for 2-3 weeks, as well as disinfecting cleaning of the premises, they can again be used as hotels.

5. With common viral infections, the doctors treating patients are not a source of danger of infection, since all patients of the infectious diseases department have the same infection. However, in the case of this virus, patients of the infectious diseases department can get sick with various fragments of this lego-viral infection, and doctors can become a source of their mutual infection, leading to an increase in the damaging factor. It is necessary to apply stringent measures to prevent the transfer of infection by doctors from one patient to another. It is necessary to minimize contacts between patients and doctors. In China, for this, robotics was used in many operations (distributing medicines, cleaning premises, etc.). In the conditions of Ukraine, this is unrealistic, but it is necessary to minimize the number of contacts per day between patients and medical personnel, as well as to use sanitation measures for medical personnel before moving on to serving another patient.

6. It is necessary to avoid the cross-flow of the infected between different regions, since each of the regions may be characterized by the incidence of certain parts of the viral infection. With

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overflows due to the combination of various parts of the Lego - virus, pathogenicity can increase significantly.

7. Patients should be kept in separate wards to prevent the possibility of mutual enrichment of different parts of the infection. When visiting physicians, they should use preventive measures and minimize the number of visits.

8. Animals with which a person communicates can from him through contact acquire particles of the virus on the wool, and moving in public places and on the paws. Then, when licking wool and paws, the virus enters their body. Recent studies by Chinese scientists show that approximately 18% of cats in Wuhan are infected. It would be more accurate to say that a virus can exist in their body, i.e. they can be natural reservoirs of infection. Therefore, you need to carefully walk them, apply sanitization after walking with animals and be careful in dealing with them.

9. The same natural reservoirs of infections can be, apparently, both people who have been ill and vaccinated - therefore people who were not sick need to be careful when dealing with these categories.

10. Those infected from patients with severe pathologies, in particular those who have died, are more likely to get a Lego virus with a greater pathology. For the acquisition of population immunity, it is better if infections occur from patients with a mild form of the disease. Therefore, it is desirable to apply the most stringent measures of quarantine restrictions for seriously ill patients, and the lungs can act as a natural vaccination.

The main idea of the above reasoning is that the current coronavirus infection is a new type of viral infection with the code name Lego viral infections. They are characterized by the possibility of the existence of several strains with different lengths of RNA chains and, accordingly, different pathologies. In the active phase of the disease, the accumulation of various fragments by the Lego virus is possible, followed by the synthesis of longer RNA chains with greater pathology.

The main conclusions that follow from this:

- Attending physicians should treat patients with coronavirus as patients with different types of infection and, based on this, use sanitation methods when moving from patient to patient.
- When accommodating patients with coronavirus, it is necessary to proceed from the fact that they have different types of infection and try to avoid mutual infection.
- If you do not isolate patients with a mild form of the disease from each other, then this can contribute to an increase in the severity of their disease.
- For natural immunization, patients with mild forms and children who are mostly asymptomatic can be used. This approach can be used when developing strategies for removing quarantine restrictions.

2.1. The structure of various lego variants - viral infections

Let the Lego-virus infection contain at most k fragments. So for COVID-19, at the initial stage of the pandemic, it was suggested that the original fragments of the viral infection are associated with three natural carrier reservoirs: pangolins, bats and snakes. This means that each carrier gave at least 1 fragment for the infection. We will assume that each of the fragments is sufficient to infect the organism, the appearance of additional fragments leads to additional infection, which leads to an increase in the overall degree of pathogenicity of the infection. The total number of variants of various infections that can be composed of 3 fragments 7 *:* I_1, I_2, I_3, I_{12}, I_{13}, I_{23}, I_{123}. Wherein I_i means that the object is infected with the *i* fragment of a viral infection with inherent pathogenic properties. I_{ij} means that the object is infected

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with *i* and *j* fragments of a viral infection with inherent pathogenic properties, while the resulting pathogenicity will be total for both fragments. I_{123}. means that the object is infected with *1, 2 and 3* fragments of a viral infection with their inherent pathogenic properties, while the resulting pathogenicity will be total for all three fragments, i.e. maximum possible for a combination of 3 fragments. If the infection can consist of 2 fragments, then the possible combinations of fragments that the infection can consist of will be equal to 3: I_1, I_2, $I_{12,}$ i.e. infection can occur in one fragment or two at once. You can use the letter designations of the fragments: A, D, C, D ... Then, for the two-component model, the following combinations of infections are possible: AB, A, B, i.e. there are 3 of them, and three-component: ABC, AB, AC, BC, A, B, C, i.e. there are 7. For four-component models, we have the following combinations: ABCD, ABC, ABD, DCD, CDA, AB, AC, AD, BC, BD, CD, A, B, C, D, ie there are 15 of them in total. Recently there have been data on the mutation of the virus on fur farms of minks. This means that minks can be the source of 4 components of the infection. The maximum number of lego-fragments that can be mutually connected is limited by the stability of the system and will apparently be no more than 3 ... 5. With 5 components of infection, 31 variants of the disease can be expected. Obviously, the number of fragments will determine the maximum number of disease waves.

If, in the general case, the number of infection fragments is *k*, then the number of possible variants (combinations) will be:

$$K = \sum_{i=1}^{k} C_i^k \qquad (1)$$

where C_i^k – is the number of combinations of *k* elements for *i*:

$$C_i^k = \frac{i!}{k! * (i-k)!}$$

If the probability of infection by each of the fragments of infection p_i , then the probability of infection by several fragments *i* and *j at* once, taking into account the statistical independence of these events, will be determined by the product of the probabilities of infection by each of the infection fragments:

$$p_{ij} = p_i * p_j \qquad (2)$$

Similar ratios in the form of a product of the probabilities of infection with different fragments of the virus will also be for cases when the number of infecting fragments is greater than 2.

If the probabilities of infection are approximately the same for different fragments, then the resulting probabilities, as can be seen from (2), will be determined:

$$p_{ij} = p^{i+j} = p^k \quad , \qquad (3)$$

where *k* – is the number of infection fragments.

The probability of contracting an infection, which may contain 3 fragments of infection, taking into account relations (1.3), can be written:

$$P_{3\Sigma} = 3p - 3p^2 + p^3 \qquad (4a)$$

Or in general form for k - component infection:

$$P_{k\Sigma} = \sum_{i=1}^{k} (-1)^{k-1} p^i C_i^k \qquad (4б)$$

For infections consisting of a different number of components k from (4b) we have:

$$\begin{aligned} P_{2\Sigma} &= 2 * p - p^2; k = 2; \\ P_{4\Sigma} &= 4 * p - 6 * p^2 + 4 * p^3 - p^4; \\ & k = 4; \\ P_{5\Sigma} &= 5 * p - 10 * p^2 + 10 * p^3 - 5 * p^4 \\ & + p^5; k = 5; \end{aligned} \qquad (4c)$$

Using relations (4), the probabilities of infection with multicomponent infections were calculated, which are shown in Fig. 1.

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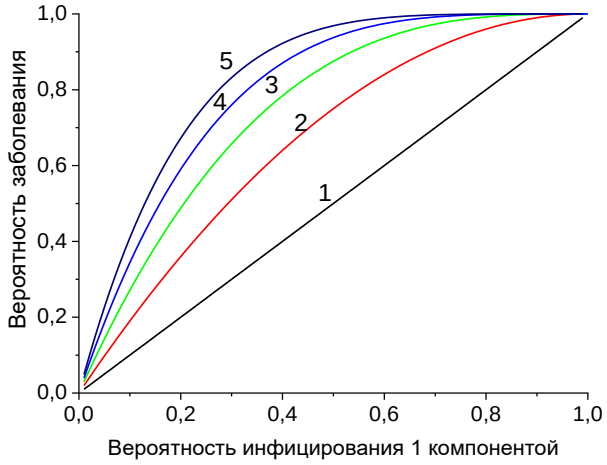

Fig. 1. Probability of disease from the probability of infection p for infections with k = 1,2,3,4,5 components

It can be seen that an increase in the number of fragments of infection leads to a rather sharp increase in the likelihood of the disease. So, with the probability of infection with 1 component of 50%, the probability of contracting 2 component infection will be 75%, and with three, four and five-component infections, respectively, 87.5%, 93.8% and 96.8%.

Along with an increase in the likelihood of the disease with an increase in the number of fragments that make up the infection, an increase in the degree of its pathogenicity will be observed, and therefore the likelihood of a severe course of the disease.

It has been established that approximately 20% of all patients are seriously ill. This means that with an overall mortality rate of 2%, the probability of death for a seriously ill person is approximately 10%.

For k fragment infection, the probability of death P_{Dk} can be written:

$$P_{Dk} = \sum_{i=1}^{k} P_I(i) P_D(i) \qquad (5)$$

where $P_I(i)$ – is the probability of infection by i fragments of infection; $P_D(i)$ - the conditional probability of a lethal outcome in case of infection with i fragments of infection.

Assuming that the conditional probability of a lethal outcome, being an increasing function of the number of infection fragments, is described by a geometric progression:

$$P_D(i) = a_0\, q^{i-4} \qquad (6)$$

where q - is the denominator of the progression, $a_0 = const$.

Consider the probabilities of infection (diseases) i fragments k fragmentinfection. Initially, it can be assumed that Covid 2019 represented a three-component k - = 3 infection. At the present time, there is reason to believe that it has transformed into a four-fragment k - = 4 infection (the so-called Indian strain), which has a greater pathogenicity, severity and rate of the disease. When considering, we will assume that the probabilities of infection by individual fragments of infection are approximately the same, i.e.

$$P(A) \approx P(B) \approx P(C) \approx p \qquad (7)$$

2.2. Three fragment virion

The disease will have the greatest severity of the course when infection occurs with all three fragments of the infection, i.e. situation described by the expression:

$$I_3 = ABC \qquad (8a)$$

probability of infection with all three 3 fragments will be:

$$P_{I3} = P(A)P(B)P(C) \approx p^3 \qquad (8б)$$

Infection with two fragments of infection and the probability of such a situation will be described by the expressions:

$$I_2 = \text{AB+AC+BC-3ABC} \qquad (9a)$$

$$P_{I2} \approx 3p^2 - 3p^3 \qquad (9б)$$

Similarly, infection with one component (disease) and its probability have the form:

$$I_1 = A + B + C - 2AB - 2BC - \\ -2AC + 3ABC \qquad (10a)$$

$$P_{I1} \approx 3p - 6p^2 + 3p^3 \qquad (10б)$$

2.3. Four fragment virion

At present, there is reason to believe that the new, so-called, Indian delta strain is enhanced by the fourth D fragment of three previously

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known ABC strains, which explains its high contagiousness and virulence.

The disease will have the greatest severity when infection occurs with all four A, B, C, D fragments of the infection, i.e. the situation described by the expression:

$$I_4 = ABCD \qquad (11a)$$

The probability of infection with all four 4 fragments will be:

$$P_{I4} = P(A)P(B)P(C)P(D) \approx p^4 \qquad (11б)$$

Infection with three fragments of infection and the probability of such a situation will be described by the expressions:

$$I_3 = ABC+ACD+BCD+DAB-4ABCD \qquad (12a)$$

$$P_{I3} \approx 4(p^3 - p^4) \qquad (12б)$$

Infection with two components (disease) and its probability are described by the expressions:

$$I_2 = AB + AC + AD + BC + \\ +DD + CD - 3ABC - 3BCD - \\ -3CDA - 3ABD + 6ABCD \qquad (13a)$$

$$P_{I2} \approx 6p^2 - 12p^3 + 6p^4 \qquad (13б)$$

Similarly, when infected with one component (disease), its probability is:

$$P_{I1} \approx 4p - 12p^2 + 12p^3 - 4p^4 \qquad (14)$$

We assume that the function describing the conditional probabilities of lethal outcome from the number of infection fragments with which a person is infected (6) is known, then using relations (5, 7-14), one can estimate the probability of lethal outcome for *k* component infection (5), and after normalization its on the probability of the disease in this type of infection (4b) to obtain the conditional probability of lethal outcome α_{dk} in relation to the sick:

$$\alpha_{dk} = \frac{N_{Dk}}{N_{Ik}} = \frac{P_{Dk}}{P_{k\Sigma}} = \frac{\sum_{i=1}^{k} P_I(i)P_D(i)}{\sum_{i=1}^{k}(-1)^{k-1}p^i C_i^k} \qquad (15)$$

where N_{Dk}, N_{Ik} – the number of deaths and cases at k fragmented infection. The results of mortality assessment (15) for various types of infections and the type of conditional probability of death (6) are shown in Fig. 2

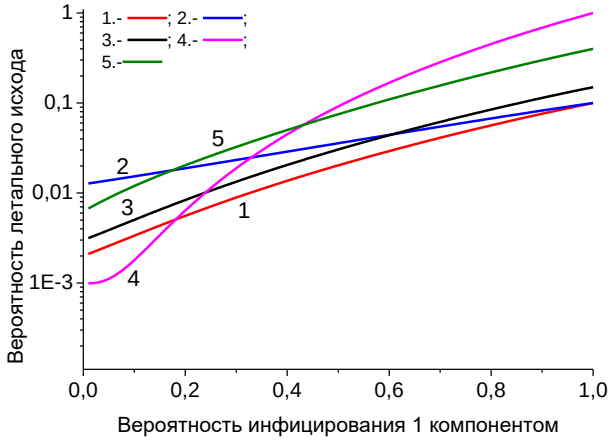

Fig. 2. The probability of death of infected k fragment infection: $k = 3$,, ; 1- a_0 =0,1; q=10; 2-1- a_0 =0,1; q=4; 3- a_0 =0,15; q=10; $k = 4$, 4- a_0 =0,1; q=10; 5- a_0 =0,1; q=4

When choosing the parameters describing the conditional probabilities of a lethal outcome, it was assumed that for a three fragment infection with a 20% probability of a severe course of the disease, approximately 2% will be lethal cases. The analysis of thein Fig. 2 dependences shownshows that with the probability of infection with 1 component of 50% ... 60% in a fairly wide range of changes in the denominator of the geometric progression, which describes the behavior of the conditional probability of lethal outcome $q = 4$ ...10 and the parameter a_0 = 0.1 ... 0, 15 for three fragment infections (Wuhan, Brazilian and British strains), the probability of death is about 2 ... 3%; which was the case with previous waves of coronavirus infection. If we assume that the new delta strain (Indian) is four-fragment, then, as can be seen from Fig. 2, with the same parameters of infection, the mortality rate will increase to 8% ... 12%, i.e. more than 4 times. At the same time, compliance with sanitary standards, as well as vaccination of the population, is becoming even more urgent. As can be seen from the analysis, an increase in the number of infection fragments leads to an increase in its contagiousness and virulence.

Until now, as a rule, the possibility of additional infection of patients with subsequent aggravation of the course of the disease in hospitals has not been considered. Since this path of development of the disease was uncharacteristic for classical viral infections. For leg viral (compound) infections or infections with a split virus, it may become dominant.

It is clear that with such a structure of a viral infection, patients with its various fragments can mutually become infected upon contact. This situation can be most acute in hospitals. For coronavirus infection, patients with various fragments of the infection. being together in the ward of a medical institution, they can transfer them to each other and mutually become infected, which will lead to an increase in the severity of the course of the disease.

If there are single-bed boxes in a medical institution, the carriers of additional infection may be medical personnel. This means that the attending medical personnel must use the same sanitation rules when serving patients. as if they were sick with various infectious diseases.

When patients are placed in single boxes and the medical staff fulfills the requirements that exclude their infection of patients, believing that the option of infection with all three fragments of an infection with three fragments is the most pathogenic, the probability of a lethal outcome will mainly be determined:

$$p_{D1} = p^3 \qquad (16)$$

In the case of double boxes, except for the situation when the patient was admitted being infected with all three fragments of Lego infection, he, having a lighter degree of pathology upon admission when infected with 1 or 2 fragments of infection, may additionally become infected from his neighbor in the box and end up with all three infection fragments.

Situations that lead to this are described by the expression:

$(ABC)_1$ situation when 1 patient is infected with all three fragments of the infection; infected with 2 fragments - $(AB)_1$, $(AC)_1$, $(BC)_1$; and when he is infected with $(A)_1$., $(B)_1$, $(C)_1$ - one fragment. Then in the first situation, when 1 patient is infected with 3 fragments, 2 patient - his roommate cannot add pathology to him. Let the probability of transmission from 2 patients to 1 be P_{21} . Let us consider the possible situations of infection of 1 patient with a viral infection with the highest pathogenicity, i.e. containing at 3 component composition all three components:

1. The first patient already has all three components:

$$C=C_1=A*B*C;\ P(C)= =P(A)*P(B)*P(C)\approx p^3 \qquad (17)$$

Then no matter how many fragments 2 patients are infected, the first will already have the maximum pathogenicity of the disease.

2. Similarly, if the second patient is infected with all the components of the infection, then it does not matter how many fragments the first patient is infected with the probability P21* p^3 he will receive all three fragments from the second patient:

$$P(C)= P(C_2)P(C_1 | C_2)=P(A)*P(B)*P(C)$$
$$P_{21}\approx P_{12} * p3 \qquad (18)$$

3. If the first patient is infected with two fragments infections, e.g. AB, then the second will be infected if C. + CA + SV, it willwith a probability of P_{21} complement the infection of the first oneto all three components. Since the situation will be similar when the first patient is infected with the other 2 fragments of infection: BC and AC. If we assume that the probabilities of infection by individual fragments are approximately the same, then the conditional probability of the second patient to be infected in this way, taking into account (4a), will be written:

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$$P(C_2|C)=\frac{3P_{21}*p3}{p^3-3p^2+3p} \quad (19)$$

4. If the first patient is infected with 1 fragment of infection, for example A or B or C, and if the second has a two-component infection with CB (or AC or AB), then in general the first patient will be infected with all three components with a probability (19).

The resulting probability of infection of 1 patient with all three fragments of infection, taking into account (17-19), will be written as:

$$p_{D2} = P(C) \approx p^3 + P_{21} * p3 + \frac{6P_{21}*p3}{p^3-3p^2+3p} \quad (20)$$

Comparing the probabilities of infection with 3 fragments for single (16) and double (20) boxes, one can estimate the degree of increase in mortality:

$$K_{D21} = 1 + P_{21} + \frac{6P_{21}}{p^3-3p^2+3p} \quad (21)$$

Fig. 3 shows the degree of increase in the incidence of three components of infection (mortality rate) from the probability of infection with one component and the probability of transmission of infection in the ward from a neighbor.

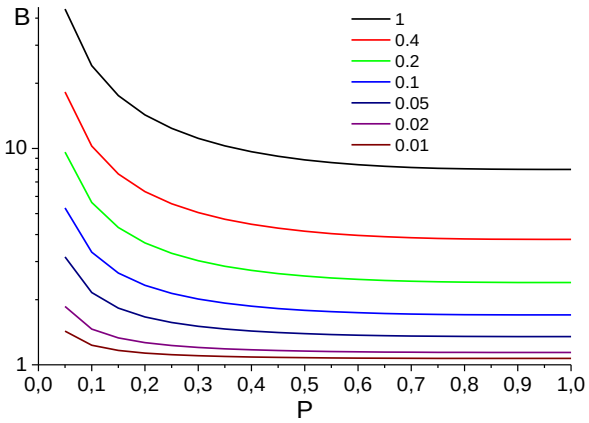

Fig. 3. Dependence of the mortality increase rate in a two-bed ward depending on the probability of infection with one component of the infection p and the probability of transmission from the second patient to the first P_{21}

The analysis shows that for bicameral boxes, the probability of infection with all three components of the infection increases significantly, and hence the mortality of the outcome. Moreover, it is the higher, the less the probability of infection with one component p and the higher the probability of mutual infection with a neighbor in the box P21.

Since the probability of mutual infection P_{21} depends on the volume of air in the ward and the time spent by the second patient. The daily consumption of air by each patient is about $22m^3$. In accordance with BC(Building Codes) [5], each bed of the infectious diseases department must have at least $8m^2$ of area, ie. about $24m^3$ air. At the same time, air exchange due to ventilation must ensure at least $50m^3$/ hour. people, and nominally $100m^3$/ hour. people In this case, the probability of infection due to the use of contaminated air will be about 0.02 ... 0.01 per day. Estimates using relation (21) show that the increase in mortality due to the presence of a neighbor in the second bed during the day will be from 1.8 ... 1.4 at p = 0.05 to 1.2 ... 1.1 at p = 0, 3. If the second patient with a high degree of pathogenicity is in the ward for 10 days (mean time to death), then the increase in mortality for the first patient will already be from 9.6 ... 5.3 with p = 0.05 to 3.0 ... 2, 0 at p = 0.3. Considering that the actual figures for air exchange may significantly differ from those required by the Building Codes and Rules downwards, it should be expected that the increase in mortality may be even greater. This means that in order to avoid a significant increase in mortality due to additional infection by neighbors in the ward, it is necessary to use single boxes. The situation will be even worse when there are 3 or more beds in the ward (box). To reduce the likelihood of additional infection of patients and aggravate the severity of the course of the disease, it is advisable to place patients with approximately the same degree of

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disease in the wards, to use the placement of members of the same family in the ward, and to the medical staff, when carrying out the treatment process, to adhere to the rules of sanitation, as in the treatment of patients with various types diseases.

Recently, the Indian strain of delta has gained significant distribution, which may turn out to be a four-fragment infection in which a new fourth fragment of infection D has appeared, and it can now consist of 4 fragments A, B, C, D. We considered the possible structure of new types of coronavirus infections in the form of a model leg of a virus-composite virus. Let us consider methods for describing the spread of this type of infection

III. COMPARTMENTAL MATHEMATICAL MODEL OF INFECTION SPREAD

Mathematical modeling of disease is a powerful tool for studying the mechanisms by which disease spreads. Epidemiological models serve as the basis for predicting and assessing the dynamics of the spread of the disease. To contain and control the epidemic, it is important to consider high-quality and adequate mathematical models of the epidemic. Currently, thanks to advances in mathematical modeling, this is a feasible task. To build a mathematical model, it is necessary to consider the process of the course of the disease. The incubation period, for Ebola, that is, the period from infection to the onset of the first symptoms ranges from 2 to 21 days [6].

To date, the following characteristics of the progression of infection in one patient are known for the COVID-2019 viral infection [4]:

Baseline reproductive number, R_0. [7]: “Our average estimate of the effective reproductive number, Re, equivalent to the base reproductive number (R_0) at the beginning of the epidemic, is 2.38 (95% CI: 2.04–2.77)”. [8]: “Our estimate of R_0 from the pooled distribution has a median value of 2.9 (95% CI: 2.1–4.5)”.

Latent period (from infection to transmission). [7]: “In addition, the median estimates of the latency and infectious periods are approximately 3.69 and 3.48 days, respectively”; see also Table 1 in article.[9]: We use the time it takes for infectivity to reach half of its peak, which occurs two days before the onset of symptoms. Since symptoms appear after five days (see "Incubation Period" below), this means a three-day latency period.

The incubation period (from infection to symptoms). [10]: “The average incubation period was estimated at 5.1 days (95% CI, 4.5 to 5.8 days), and 97.5% of those who develop symptoms will do so within 11.5 days (CI 8.2 to 15.6 days) of infection. These estimates mean that, conservatively, 101 out of every 10,000 cases (99th percentile, 482) will develop symptoms after 14 days of active monitoring or quarantine. [11]: “The average incubation period was 5.2 days (95% confidence interval (CI) 4.1 to 7.0), with the 95th percentile of distribution 12.5 days”.

Infection period. [7]: “The median estimates of the latent and infectious periods are approximately 3.69 and 3.48 days, respectively”; see also Table 1 in article.[10]: We determine the interval during which the infectivity is at least half of its maximum value (interval of half-maximal infectivity) .

Duration diseases. [12]: “According to the available preliminary data, the average time from onset to clinical recovery for mild cases is approximately 2 weeks and 3-6 weeks or more for patients with a severe or critical illness”.

Time until diagnosis. [13]: We used case data with known symptom onset dates and case confirmation and calculated the median time lag between these two dates.

Lethality. [14] - Use data from all countries with more than 50 deaths and calculate an unadjusted baseline case fatality rate for each country. The range represents the lowest and

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highest rates observed using data from[15]through April 14, 2020.

Mortality from infections. We rely on three independent approaches to assessing IFR. The first is based on data on people who have undergone extensive testing as a result of repatriation. [16]: "We get an overall IFR estimate for China of 0.66% (0.39%, 1.33%)". [17]: "Assessment of IFR from Verity et al. were adjusted for the uneven number of attacks, resulting in an overall IFR of 0.9% (95% probable range 0.4-1.4%)". [18]: - from 0.3% to 0.6% ".

The second approach is based on data collected from the Diamond Princess ship, where all passengers were checked. [19]: "We estimate the CIFR for all ages on the Diamond Princess was 1.3% (95% confidence interval (CI): 0.38-3.6)".

The third approach is based on epidemiological modeling of the time series of cases in China. [20]: "We also found that the latest IFR and IFR adjusted for lag time are estimated at 0.04% (95% CrI: 0.03–0.06%) and 0.12 % (95% CrI: 0.08–0.17%)". By combining these three methods and considering the reliability of each report, we estimate the approximate range for IFR to be ≈0.3–1.3%.

We will use them to measure the parameters of the mathematical model of the spread of infection.

Infectious diseases can have a latent or latent period of the disease. This is the period from the moment when a person is already infected until the first symptoms appear. In the case of COVID-2019, the incubation period for COVID-2019 was estimated at 5.1 days (95% CI, 4.5 to 5.8 days), and 97.5% of those who develop symptoms will do so within 11, 5 days (CI 8.2 to 15.6 days) of infection, during which the person is not contagious.

A modification of the classic SIR model - the SEIRD model [2]. - can be used as a baseline for describing the spread of an epidemic. In the SEIRD model, a situation is considered when a person is already infected, but the disease is in a latent period. These individuals will be in the latent class (from the English exposed). In the SEIRD model, the population is divided into five classes: susceptible S (t), latent (t), infected (t), immune $R(t)$, and deceased $D(t)$. The classes (t), (t) $E(t)$, $R(t)$, $D(t)$ are defined as follows:

$S(t)$ is used to denote uninfected individuals or susceptible to disease;

$I(t)$ is used to refer to infected individuals capable of spreading the disease;

$E(t)$ is used to refer to individuals whose disease is in the incubation period;

$R(t)$ is used to refer to recovered individuals;

$D(t)$is used to refer to deceased individuals.

The population is considered to be fixed at the time of the outbreak, therefore at any moment t the population is equal to N, i.e. $S(t) + E(t) + I(t) + R(t) + D(t) = constant = N$. The SEIRD model can be expressed by the following set of differential equations:

$$\begin{gathered} dS(t) / dt = -\beta S(t) I(t) / N \\ d\,E(t)\,(t) / dt = \beta(t) I(t) / N - \delta E(t) \\ dI(t) / dt = \delta E(t) - \gamma I(t) - \mu I(t) \\ dR(t) / dt = \gamma I(t) \\ dD(t) / dt = \mu I(t) \end{gathered} \quad (22)$$

where β - coefficient, which can be interpreted as the speed of contact, taking into account the likelihood of getting the disease in the event of contact of a susceptible individual with an infected person; $\gamma = 1/T$, where T – is the time of illness, the coefficient can be interpreted as the speed of recovery; $1/\delta$ - is the average time of the duration of the latent period; μ – mortality rate.

Initial data at time $t = 0$:

$$(0) = S_0 > 0,\ (0) = E_0 > 0,$$
$$(0) = I_0 > 0,\ (0) = R_0 > 0$$

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Structure of this model can be presented in the form of a block diagram in Fig. 4. The parameters are shown in Table 1.

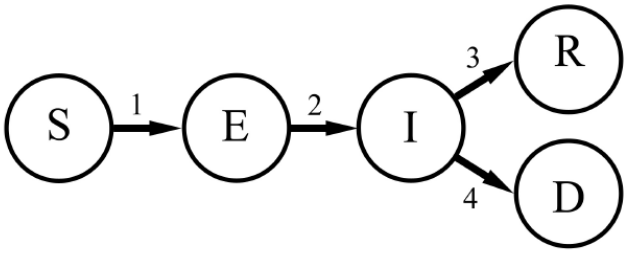

Fig. 4: General scheme of the SEIRD model: S - susceptible, E - latent, I - infected, R - refractory, D – deceased

Table 1. **Table of SEIRD model**

№	Transition	Speed of transition
1	$(S,E) \rightarrow (S-1,E+1)$	$(\beta SI)/N$
2	$(E,I) \rightarrow (E-1,I+1)$	δE
3	$(I,R) \rightarrow (I-1,R+1)$	γI
4	$(I,D) \rightarrow (I-1,D+1)$	μI

In the classic version of this model, people passing treatment in hospitals. This drawback is eliminated in the SEIHFR model. This model was proposed by scientists from the University of Pierre and Marie Curie to describe the spread of the Ebola epidemic since this model includes all groups of people involved in the epidemic. The SEIHFR model divides the population into six classes: S - susceptible (from the English susceptible), E - latent (from the English exposed), I - infected (from the English infected) H - hospitalized (from the English hospitalized), F - unburied (from the English funeral), R - immune (from the English removed).

Let us dwell in more detail on the description of the classes of the SEIHFR-model:

$S(t)$ is used to denote the number of susceptible individuals in the risk group at time t;

$E(t)$ is used to indicate the number of individuals whose disease is in the incubation period at time t;

$I(t)$is used to denote the number of infected individuals who are able to spread the disease at time t;

$H(t)$ is used to denote the number of individuals who have been hospitalized at a point in time t;

$F(t)$ is used to denote the number of individuals who have died, but not yet buried, at a point in time t;

$R(t)$is used to designate individuals who dropped out as a result of previous class recovery or death at the time t.

Note the importance of considering class F in the case of modeling the spread of Ebola. In this model: a susceptible individual S can become latent E after contact with an infected I and then move to the class of infected I after the incubation period of the disease. Some of the infected I people can be hospitalized H. For individuals in the infected class I and hospitalized H, there are two possible outcomes: individuals may die, with a chance of infecting others during funeral F, and move to the immune class R, or they may recover and move to the immune class R.

The SEIHFR model can be expressed by the following differential equations:

$$dS(t)/dt = -1/N * (\beta_I S(t)I(t) + \beta_H S(t)H(t) + \beta_F S(t)F(t));$$
$$dE(t)/dt = 1/N * (\beta_I S(t)I(t) + \beta_H S(t)H(t) + \beta_F S(t)F(t)) - \alpha E(t);$$
$$d\,I(t)/dt = \alpha(t) - (\gamma_H\theta_1 + (1-\theta_1)(1-\delta_1) + \gamma_D(1-\theta_1)\delta_1)I(t); \quad (23)$$
$$d\,H(t)/dt = \gamma_H\theta_1(t) - (\gamma_{DH}\delta 2 + \gamma_{IH}(1-\delta_2))H(t);$$
$$d\,F(t)/dt = \gamma(1-\theta_1)\delta_1 I(t) + \gamma_{DH}\delta_2 H(t) - \gamma_F F(t);$$
$$d\,R(t)/dt = \gamma(1-\theta_1)(1-\delta_1)I(t) + \gamma(1-\delta_2)H(t) + \gamma_F F(t);$$

where: β_I - is the coefficient of contact in the community; β_H - hospital contact ratio; β_F - contact coefficient during the funeral; $1/\alpha$ - average duration of the incubation period; $1/\gamma_H$ - the average duration of the period from the

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onset of the first symptoms to hospitalization; $1/\gamma_{DH}$ - the average length of the period from hospitalization to death; $1/\gamma_I$ - the average duration of the infectious period for a recovered; $1/\gamma_D$ - the average duration of the infectious period for the deceased; $1/\gamma_{IH}$ - is the average length of the period from hospitalization to recovery; $1/\gamma F$ - average duration of the period from death to burial; θ - is the proportion of hospitalizations; δ - mortality rate.

The coefficients $\theta 1$, $\delta 1$, $\delta 2$ are calculated as follows:

$$\theta 1 =(\theta(\gamma I(1-\delta 1)+\gamma D\delta 1))/(\theta(\gamma I(1-\delta 1)+ \\ +\gamma D\delta 1)+(1-\theta));$$

$$\delta 1=\delta\gamma I/(\delta\gamma I+(1-\delta)\gamma D),$$

$$\delta 2 =\delta\gamma IH/(\delta\gamma IH+(1-\delta)\gamma DH); \quad (23а)$$

At the moment of time $t = 0$ the initial conditions look as follows:

$$(0) = S_0 > 0,\ (0) = E0 > 0,\ (0) = I_0 > 0,$$

$$(0)=H_0 > 0,\ (0)=F_0 > 0,\ (0)=R_0 > 0 \quad (23б)$$

size of the population N is fixed, i.e.

$$S(t)+E(t)+I(t)+H(t)+F(t)+R(t)= \\ =constant = N. \quad (23в)$$

The structure of the model and the phases of the disease are shown in Fig. 5.

The parameters are shown in Table 2.

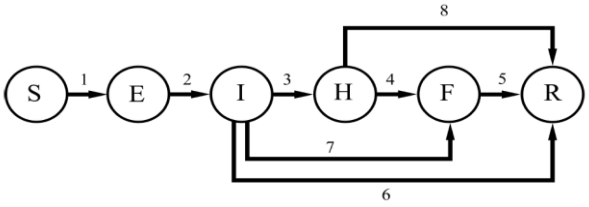

Fig. 5. General scheme of the SEIHFR-model indicating the transition number: S - susceptible, E - latent, I - infected, H - hospitalized, F - not buried, R – immune

Table 2. **SEIHFR ModelTable**

№	Transition	Speed of transition
1	$(S,E) \rightarrow (S-1,E+1)$	$(\beta_I SI+\beta_H SH+\beta_F SF)/N$
2	$(E,I) \rightarrow (E-1,I+1)$	αE
3	$(I,H) \rightarrow (I-1,H+1)$	$\gamma_H\theta_1 I$
4	$(H,F) \rightarrow (H-1,F+1)$	$\gamma_{DH}\delta_2 H$
5	$(F,R) \rightarrow (F-1,R+1)$	$\gamma_F F$
6	$(I,R) \rightarrow (I-1,R+1)$	$\gamma_I(1-\theta_1)(1-\delta_1)I$
7	$(I,F) \rightarrow (I-1,F+1)$	$\delta_1(1-\theta_1)\gamma_D I$
8	$(H,R) \rightarrow (H-1,R+1)$	$\gamma_{IH}(1-\delta_2)H$

One factor contributing to the rapid spread of Ebola in West Africa is the local funeral rites in which people have direct contact with the body of the deceased and can transmit the Ebola virus. This route of transmission is uncommon for COVID-2019. But for this infection, as was shown earlier, the path of additional infection of patients in the hospital is possible. Therefore, to describe the spread of this infection, one can use the SEIHFR model by setting the parameter values corresponding to the F state equal to zero:

Then relations (23), taking into account that. that $F(t) = dF(t)/dt = 0$ are transformed to the form:

$$dS(t)/dt= -1/N *(\beta_I(t)I(t)+\beta_H S(t)H(t);$$
$$dE(t)/dt=1/N*(\beta_I(t)I(t)+\beta_H S(t)H(t)-\alpha E(t);$$
$$dI(t)/dt= \alpha(t) -(\gamma_H\theta_1+\gamma_I(1-\theta_1)(1-\delta_1)+ \\ \gamma_D(1-\theta_1)\delta_1)I(t); \quad (24)$$
$$dH(t)/dt= \gamma_H\theta_1(t)-(\gamma_{DH}\delta 2+\gamma_{IH}(1-\delta_2))H(t);$$
$$dD(t)/dt= \gamma_D(1-\theta_1)\delta_1 I(t)+\gamma_{DH}\delta_2 H(t) =0;$$
$$dR(t)/(t)/dt=\gamma(1-\theta_1)(1-\delta_1)I(t)+\gamma_I H(1-\delta_2)H(t);$$

A slightly different approach is possible when the SEIRD model is modernized by adding the H state, i.e. by creating the SEIHRD model.

Let's dwell on the description of the SEIHRD classes - models:

(t) is used to denote the number of susceptible individuals at risk at time t;

(t) is used to denote the number of individuals whose disease is in the incubus the national period, at the moment of time t;

(t) is used to denote the number of infected individuals capable of spreading the disease at time t;

(t) is used to denote the number of individuals who have been hospitalized at a point in time t;

(t) is used to denote recovered individuals;

(t) is used to denote deceased individuals.

SEIHRD model can be expressed by the following differential equations:

$$dS(t)/dt=-1/N*(\beta_I(t)I(t)+\beta_H S(t)H(t);$$
$$dE(t)/dt=1/N*(\beta_I(t)I(t)+\beta_H S(t)H(t)-\alpha E(t);$$

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$$dI(t)/dt=\alpha(t)-(\gamma_H\theta_1+\gamma_I(1-\theta_1)(1-\delta_1)+ +(1-\theta_1)\delta_1)I(t); \quad (25)$$
$$dH(t)/dt=\gamma_H\theta_1(t)-(\gamma_{DH}\delta_2+\gamma_{IH}(1-\delta_2))H(t);$$
$$d\mathrm{D}(t)/dt=\gamma_D(1-\theta_1)\delta_1 I(t)+\gamma_{DH}\delta_2 H(t);$$
$$d(t)/dt=\gamma_I(1-\theta_1)(1-\delta_1)(t)+\gamma_I H(1-\delta_2)H(t);$$

where β_I - is the contact ratio in the community; β_H - is the contact ratio in the hospital; $1/\alpha$ - is the average duration of the incubation period; $1/\gamma_H$ - is the average duration of the period from the appearance first symptoms before hospitalization; $1/\gamma_{DH}$ - average duration of the period from hospitalization to death; $1/\gamma_I$ - average duration of infection the ionic period for the recovered person; $1/\gamma_D$ - the average duration of the infectious period for the deceased; $1/\gamma_{IH}$ - is the average length of the period from hospitalization to recovery; δ - mortality rate.

The coefficients θ_1, δ_1, δ_2 are calculated as follows:

$$\theta_1=(((1-\delta_1)+\gamma D\delta_1))/(\theta(\gamma_I(1-\delta_1)+\gamma_D\delta_1)+ +(1-\theta)\gamma_H); \quad (25a)$$
$$\delta_1=\delta\gamma_I(\delta\gamma_I+(1-\delta)),\delta_2=\delta\gamma_{IH}/(\delta\gamma_{IH}+(1-\delta)\gamma_{DH});$$

At the time = 0 the initial conditions are as follows:

$$(0)=S_0>0,(0)=E_0>0,(0)=I_0>0,$$
$$(0)=H_0>0,\mathrm{D}(0)=\mathrm{D}_0>0,R(0)=R_0>0 \quad (25б)$$

size of the population N is fixed, i.e.

$$S(t)+E(t)+I(t)+H(t)+\mathrm{D}(t)+R(t)= =constant=N. \quad (25в)$$

As can be seen from the comparison of relations (24, 25), both approaches led to the same results. The structure of the SEIHRD model and disease phases are shown in Fig. 5. The parameters are shown in Table 3.

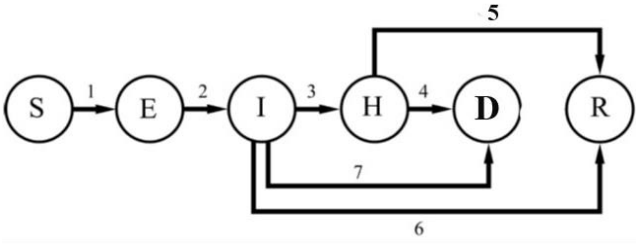

Fig. 5. General scheme of SEIHRD -model with indication of the transition number: S - susceptible, E - latent, инфициров - infected, H - hospitalized, D - deceased, R – immune

*Table 3.***Transition table of SEIHRD –model**

№	Transition	Speed of transition
1	$(S,E)\rightarrow(S-1,E+1)$	$(\beta_I SI+\beta_H SH)/N$
2	$(E,I)\rightarrow(E-1,I+1)$	αE
3	$(I,H)\rightarrow(I-1,H+1)$	$\gamma_H\theta_1 I$
4	$(H,D)\rightarrow(H-1,D+1)$	$\gamma_{DH}\delta_2 H$
5	$(H,R)\rightarrow(H-1,R+1)$	$\gamma_{IH}(1-\delta_2)H$
6	$(I,R)\rightarrow(I-1,R+1)$	$\gamma_I(1-\theta_1)(1-\delta_1)I$
7	$(I,D)\rightarrow(I-1,D+1)$	$\delta_1(1-\theta_1)\gamma_D I$

Using the MATLAB software package, the considered models were simulated using the special function ode45, which is intended for numerical integrating systems of homogeneous differential equations, as well as for modeling complex dynamical systems. The analysis showed that the SIR model and SEIRD model are too simple to simulate the spread of coronavirus infection. In turn, the SEIHFR model and the SEIHFDR model are too cumbersome and contain data that are not statistically available or are difficult to establish.

For the first time, a new model was developed and modeled, which was called the SEIHRD model. It will simulate the spread of SARS-CoV-2 (2019 nCoV) infection.

The existing compartment models do not provide modeling of infections with several waves of the disease, however, their modernization using the proposed approaches will allow modeling the spread of new coronavirus infections, predicting the process of loading the medicine system as well as the need for personnel, equipment and hospital beds in pandemics.

IV. CONCLUSIONS

1. A hypothesis is proposed about the possible structure of the new coronavirus infection Covid2019 in the form of composite leg viruses. The features of the new type of infection and the resulting requirements for sanitary and epidemic measures to prevent its spread are considered. The main thing is to consider it as a Lego infection, which can consist of several fragments, each of which, in

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combination with others, and separately can lead to a disease. The degree of pathology depends on the number of fragments that the patient has become infected with (the length of the pathogenic chain), as well as the dose received. Patients infected with various fragments can mutually infect each other, leading to an increase in the number of components of the infection and, accordingly, the degree of pathology of the disease. The attending physicians must adhere to specific sanitary and epidemiological measures and treat patients as patients with various infections in order to exclude the transmission of infection from one patient to another. The placement of patients in medical institutions should minimize the possibility of their mutual infection.

2. The fading of the pandemic will occur when the number of those who have recovered from all the fragments of infections together with the vaccinated reaches 70 ... 80%. This means that several waves of the disease are possible. Children can act as natural vaccinators, who will become infected and get sick with small fragments of infection with low pathogenicity in most cases in a mild form.

3. To prevent infection with large fragments of Lego infection, it is necessary to try to limit the circle of people in communication, try to keep it constant. For those whose work is related to communication with an unlimited circle of people, it is necessary to provide for the possibility of a rotational work schedule of up to 14 days and with the same rest interval. It is advisable to use a similar regime for risk groups (doctors, elderly people, officials who receive citizens).

To describe the spread of the epidemic of lego-viral infections such as COVID-2019, it is advisable to use fuzzy set mathematics and the modified SEIHRD compartment, model.

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НОВІ МАТЕМАТИЧНІ МОДЕЛИ РОЗПОВСЮДЖЕННЯ ВІРУСНИХ ІНФЕКЦІЙ

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Реферат *– Предмет і мета роботи – пандемія COVID-19 наразі торкнулася майже всіх країн світу та гостро нагадала про необхідність подальших досліджень багатьох аспектів вірусів. Спроба використання класичних підходів для опису механізмів передачі та поширення цієї інфекції та пов'язаних з ними методів проведення санітарно-профілактичних заходів виявилася неефективною. У наш час особливої актуальності набуло створення моделей нової вірусної інфекції, що дозволяє пояснити особливості її перебігу, що спостерігаються на практиці, і запропонувати математичний опис механізму її поширення. Методи та методика роботи: в роботі використовуються теорія ймовірності та математична статистика, засоби чисельного моделювання для розв'язування систем звичайних диференціальних рівнянь, що описують поширення вірусної інфекції. Результати роботи: Запропоновано лего модель для опису структури складної вірусної інфекції, яка дає змогу пояснити можливість рецидиву захворювання та наявність кількох його хвиль. Отримано оцінки ймовірності захворюваності та смертності від кількості фрагментів лего зараження. Вказується на необхідність дотримання спеціальних санітарно-гігієнічних заходів для зниження ймовірності тяжкого перебігу захворювання та смертності. За допомогою компартментних моделей запропоновано систему рівнянь для опису динаміки поширення нової коронавірусної інфекції COVID-19, яка враховує наявність латентного періоду інфекції, а також можливість додаткового зараження в лікувальному закладі.*

Ключові слова *– вірусна інфекція COVID-19, математична модель, математична статистика , чисельне моделювання, поширення вірусних інфекцій.*