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Research Article

General Patterns and Differences of Epidemic Spread COVID-19

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Abstract

A simple analytical model that takes into account the influence of blocking, mass vaccination and virus mutations on the intensity of COVID 19 epidemic spread is described. The results of the proposed model are in good agreement with relevant statistical data. The model uses two empirical parameters, one of which is weakly dependent on the virus strain and the other is functionally related to the blocking efficiency. Blocking efficiency depends on the behavioral characteristics of individuals, including age, and the socio-ethnic composition of the population. The psychological disposition of the population has a great influence on the development of the epidemic. In regions that are similar in their psychological and behavioral parameters, the epidemic develops in the same way. This article presents some preliminary data on the assessment of the behavioral characteristics of the population during the spread of the epidemic. The choice of settlements with homogeneous behavioral characteristics will make it possible to increase the reliability of forecast calculations of epidemic development and even to extend the results of model experimental studies conducted in small areas to the analysis of epidemics in large regions in different countries.

Key words: COVID-19; analytic model; lockdown; vaccination; mutation; age difference; psychological and behavioural characteristics; similarity theory.

1. Introduction

The widespread of the COVID epidemic globally required the analyses of disease process using mathematical modelling. Most models of various levels of detail require the application of numerical methods using special computer programs. The main applied task of models - development of forecasts of epidemic development using models of high level of detail - is complicated by the need to introduce a large number of empirical coefficients., It is important to understand that in practice, increasing the detail of models does not always lead to an increase in its reliability and accuracy of calculations, since the development of the epidemic depends largely on the behavior of the local population. Behavioral characteristics of the population determine the readiness for mass vaccination and various restrictions on the introduction of a lockdown to slow down the growth of the epidemic. Based on these considerations, we set the task of developing the simplest possible model using the minimum number of empirical coefficients. However, the model should take into account the main essential factors determining the development of the epidemic. And as in the solution of any applied problem, the criterion of the model's usefulness is the comparison of the results of calculations with real statistical data. Therefore, at all stages of model development and improvement, we sought to regularly compare calculated and statistical data. Particular attention was paid to the links between the empirical coefficients of the model and both the peculiarities of the COVIDA virus strain and the conditions for the realization of the lockdown

2. Methodology.

The accumulation of statistical data on the spread of COVID-19 in different countries allows us to significantly expand our understanding of various aspects of the emergence and course of the epidemic. More than a hundred different computational models have been proposed to describe the development of the epidemic. Some of them are based on the solution of a system of differential equations of various levels of detail, others are purely stochastic numerical models or analyses statistical data on the number and duration of contacts leading to virus transmission. All these models require the introduction of a number of coefficients for their agreement with observational data, and the number of coefficients, as a rule, increases with the complexity of the proposed models. We propose a relatively simple model that allows us to obtain analytical solutions. Let us write the initial differential equations of the epidemic model, taking into account the impact of lockdown and mass vaccination on the epidemic spread, as [5,6,7]: lockdown and mass vaccination on the epidemic spread, as [5,6,7]:

$$\frac{dS}{dt} = S(-\lambda - \frac{\alpha v}{1-\alpha v \cdot t}) \quad (1)$$

$$\frac{dI}{dt} = k_0 \cdot \frac{S \cdot I}{N} \quad (2),$$

where:

I - the number of infected persons at a given time,

$k0$ - coronavirus infection rate (1/day)

N - total population of the area under consideration,

S - the number of susceptible part of the population potentially capable of becoming infected due to contact with infected individuals,

λ - - coefficient of intensity of reduction in epidemic growth as a result of lockdown and

various factors suppressing transmission. (Reduction coefficient), (1/day)

v - population vaccination rate (1/day),

α - is the coefficient of vaccine effectiveness.

In keeping with the tradition of mathematical modelling in epidemiology, this model will hereafter be abbreviated as ASILV. This name emphasizes the main features of this model, namely: A is an analytical model that allows us to obtain an analytical solution to the system of equations; S is the fraction of the population that is not yet infected but could become infected through contact with infected individuals; I is the fraction of the population that is already infected at time t; L is the fraction of the population that is protected from infection by lockdown methods at the time in question but could potentially become infected later if isolation conditions change; V is the fraction of the population that is protected from infection by vaccination with efficacy α .

In contrast to previously developed models of this type, in our model there is no inverse relationship between the values of I and S, i.e., under lockdown conditions, the number of potentially susceptible individuals depends only on the severity of isolation. It would seem that the condition that I ≪ S under conditions of mass vaccination severely limits the application of this model. But the effect of vaccination has been found to decrease sharply with time. In addition, the emergence of new strains allows the virus to overcome the protective barriers of vaccination. Therefore, it can be considered that this computational model is also applicable in conditions of mass vaccination of the population.

For the period from outbreak to mass vaccination $t\ v$ that is for t ≤ $t\ v$, when αv = 0, the solution of equations (1) and (2) has the form

$$i = i_0 + \frac{100}{N} \cdot exp\left[\frac{k}{\lambda}(1 - e^{-\lambda t})\right] \quad (3)$$

i - is the relative number of infected persons per one inhabitant of the settlement in question, as a percentage,

i_0 - is the value of i at the initial moment of the calculation period,

k - is the transmission rate coefficient for the settlement with a population of N, which is calculated by the formula:

$$k = k_0 - \lambda \cdot \ln(\frac{1}{N} \cdot 10^6) \quad (4)$$

The coefficient k0 depends on the transmissibility of the virus strain responsible for the spread of the epidemic during the period under consideration. For the original strain, a value of k0= 0.355 was assumed. For other strains, γ times more transmissible than the original strain, the coefficient is determined by the simple formula.

$$k_0 = 0.355 + \lambda * \ln \gamma \quad (5)$$

The spread of the virus can be influenced by climatic factors [5]. The dependence of the coefficient K on temperature and UV value can be approximated using the parameter

$$W = [1 - 0,01(\theta - 6)) * (1 - 0.06(U-3)],$$

where W is the coefficient of influence of climatic parameters on the intensity of epidemic development, θ is the mean air temperature, and U is the UV index value. Accordingly, in addition to equation (4) we have:

$$Kw = k_0 + \lambda \ln W,$$

where Kw is the coefficient in equation (4), taking into account the influence of climatic factors on it. Thus, in the general case

$$k_0 = 0,355 + \lambda \ln \gamma + \lambda \ln(W), \quad (5a)$$

As a result of solving the system of equations and converting to the relative number of infections, we obtain the main calculation relation for t ≥ $t\ v$:

$$i = i_0 + (i_v - i_0) \cdot exp\{\frac{k}{\lambda} \cdot [\left(1 - \frac{\alpha v}{\lambda}\right) \cdot e^{-\lambda t_v} - (1 - \frac{\alpha v}{\lambda} - \alpha v(t - t_v))\ e^{-\lambda t}]\} \quad (6)$$

Where $i = i_v$ at $t = t_v$

i - is the relative number of infected persons per one inhabitant of the settlement in question, as a percentage,

$i0$ - is the value of i at the initial moment of the calculation period.

Taking into account the effectiveness of vaccines in preventing transmission, a relationship was derived to calculate the effective vaccination rate [7]:

$\alpha v = \alpha_1 v_1 + \alpha_2 v_2$ vaccination rates for each vaccine dose, their efficiencies are $\alpha 1$ and $\alpha 2$

$v1$ and $v2$ −vaccination rates for each vaccine dose, their efficiencies are $\alpha 1$ and $\alpha 2$ respectively.

In case the spread of infection is associated with several virus strains, we will write down the calculation dependence in the following form:

$$i = i_0 + \frac{100}{N}\ \sigma * \sum_1^n exp\left[\frac{k_i}{\lambda}\left(1 - e^{-\lambda * (t - t_{io})}\right)\right] \quad (7),$$

where,

i – the serial number of the active virus strain during the time period $t - ti-1$,

ki - the transmission rate coefficient of the new virus strain

$ti0$ - the start time of the new epidemic wave associated with the new coronavirus strain.

σ - Heaviside symbol σ = 1 при t ≥ $t\ io$ и σ = 0 при t < tio

The equation (6) is used under the conditions where an epidemic first occurs at time ti and then, at time $t\ v$, mass vaccination of the population begins to take place. However, the opposite situation is also possible, when a new strain of the virus emerges at time t N. In this case the calculations have to be carried out using the following relation:

$$i = i_{old} + \eta\ \frac{100}{N} * exp\ \frac{k}{\lambda}[\left(1 - \frac{\alpha v}{\lambda}\right) - (1 - \frac{\alpha v}{\lambda} - \alpha v(t - t_N))\ * e^{-\lambda(t - t_N)}] \quad (8),$$

where additional notations are introduced:

i_{old} - is the relative number of infected persons at the time $t\ N$ of the emergence of the new. virus variant,

i_{old} - is the relative number of infected persons at the time t_N of the emergence of the new virus variant,

η - coefficient that takes into account the effectiveness of vaccination before the emergence of a new strain:

$$\eta = 1 - \beta * \alpha_{old} / \alpha_{new} \quad ,$$

where,

$$\beta = \alpha_{old} * v * \vartheta \quad ,$$

β - is the fraction of the population that has been vaccinated by the time a new strain of virus emerges.

α_{old} and α_{new} - are the effectiveness of the vaccine for the old and new strains of the virus, respectively,

ϑ - is the time from the start of vaccination to the appearance of the new virus strain in days.

It is assumed that the effectiveness of the vaccine against the new strain of the virus is lower than against the old one, so that even if a certain population is fully vaccinated, additional infection of already vaccinated individuals is possible.

When calculating the spread of the epidemic in the selected age groups, only contacts within each group are considered. The initial equations in this case do not differ from those previously used to calculate the epidemic spread for the whole population.

Various aspects of the methodology are discussed in more detail in [1-10], depending on the specific conditions. All calculations and graphing were performed using the standard EXCEL program.

3. Results.

3.1 The factors influencing the spread of the epidemic

The main goal of our work is to identify cause-and-effect relationships between the intensity of epidemic spread and the main factors affecting this process. The lack of such relationships prevents the development of reliable and unambiguous forecasting models and forces the calculations to assume different scenarios of epidemic development., the information available so far allows us to make the first steps towards a scientifically justified choice of a scenario for the forecasting model. Some of these factors are related to the nature of the epidemic and the nature of the epidemic.

All calculations in this paper are based on the above methodology.

Initial phase of the epidemic

Let us consider, for example, the passage of the first wave in Berlin (Germany) and New York [3]. The choice of these two cities for comparison is related to the fact that, as it is commonly believed, Berlin turned out to be one of the most successful large cities, which managed to prevent active growth of the first epidemic wave, while in New York the first epidemic wave reached very high values.

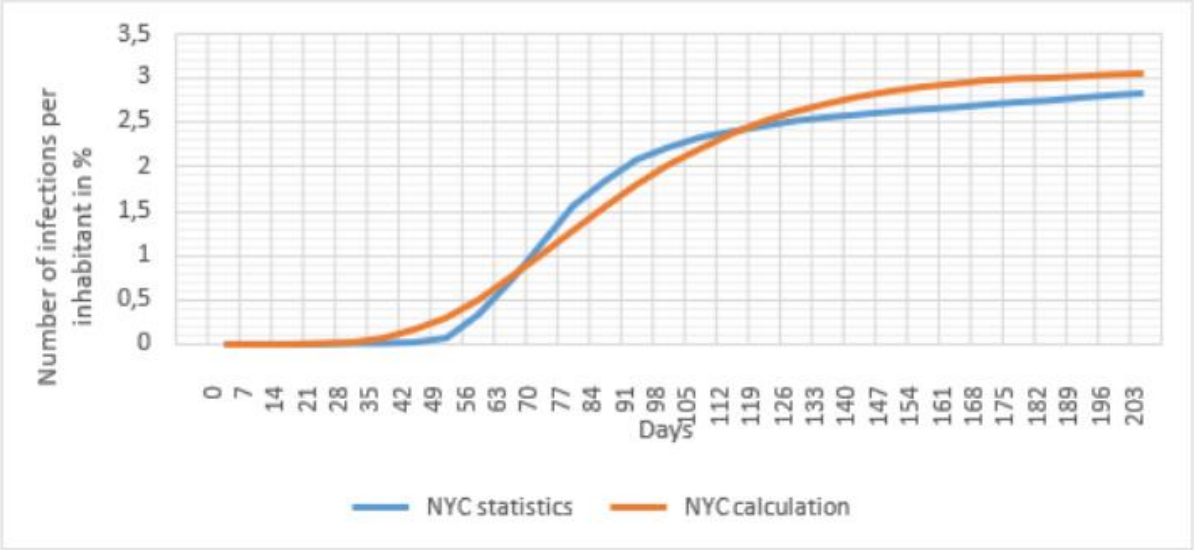


Figure 1: The passage of the first wave of the epidemic in New York City [3].

Fig.1shows a comparison of statistical data with the results of calculations for the first wave of the epidemic in New York City. The calculations were performed for the value of reduction coefficient $\lambda = 0.0345$ 1/day, the second coefficient Kr = 0.43 1/day for New York. Despite a good formal coincidence of the calculated and observed data (correlation coefficient above 0.95), we should note a number of their fundamental differences. The calculated curve much more gently in the initial stage of the epidemic, however, in the second stage of this wave, the introduction of sufficiently strict quarantine methods managed to sharply reduce the rate of further spread of the epidemic. Of particular interest is the study of the initial stage of the epidemic. First, it should be noted that quarantine in the city was introduced with a significant delay, only 63 days after the start of the epidemic. At that time, the number of detected infections, even in conditions of low testing coverage, had already reached about 1% of the city's population. The weekly increase in infected city residents by this time had reached more than 37,000 people, which is more than 5,000 people per day or 0.065% of the population. Positive results from the introduction of quarantine could actually be observed only after two weeks, when the rate of spread of the epidemic began to decline. The main conclusion that follows from the above analysis is the need to strictly control the possible occurrence of an epidemic and take the most urgent measures to eliminate it at the initial stage. As an example of such successful tactics to combat an epidemic in the early stages, consider the passage of the first wave in Berlin, Germany. The first restrictive measures to help reduce the growth of the epidemic in Berlin were taken by the city administration about a month after it began.

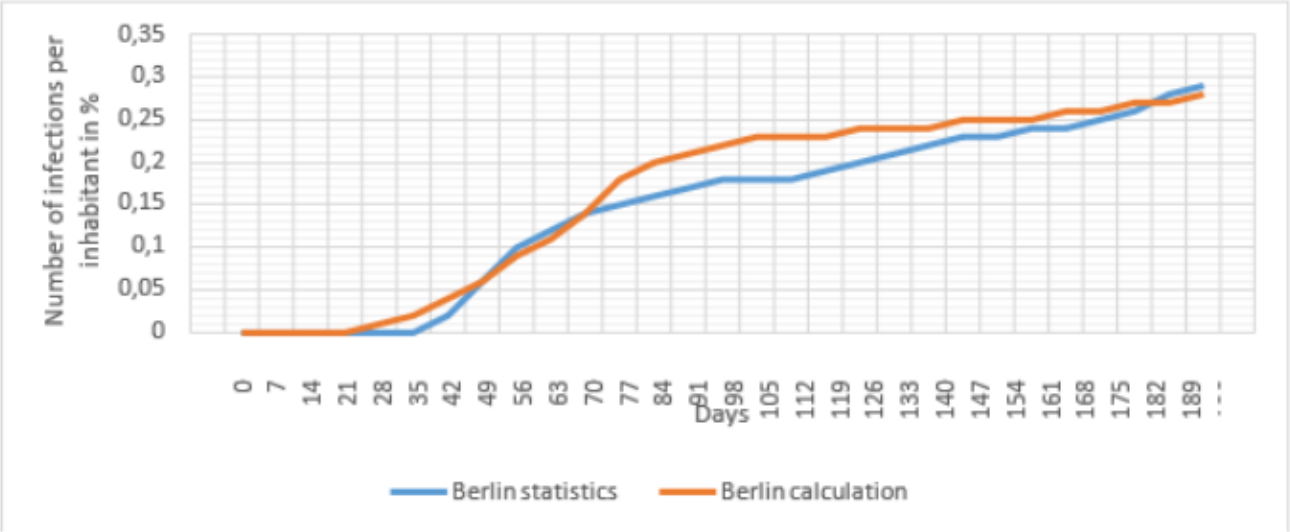


Figure 2: The passage of the first wave of the epidemic in Berlin. [3].

The relative number of people infected at the same time in Berlin, as can be seen in Fig. 2, did not exceed 0.05%. The maximum daily increase in infections during the first wave was about 200 people per day or 0.0055%, that is, the relative number of infections in Berlin in the early stages of the epidemic was about 10 times less, than in New York.

Virus mutations

An analysis of the spread of the epidemic in a number of countries shows that in the autumnwinter period of 2020 there was a sharp increase in the rate of infection. These changes are associated with the beginning of the second wave of the epidemic due to mutation of the virus and the emergence of a new strain

Figure 3: Development of the fall-winter wave of the epidemic in New York City. [3]

Figure 3 shows the development of the epidemic during the second wave of the epidemic in New York City [3]. The results of calculations and statistical data begin to deviate after about 90 days after the beginning of the second wave of the epidemic. The growth of the epidemic after the emergence of a new strain of the virus for New York City was calculated using relation (7). The coefficient λ according to the present model does not depend on the virus strain and is therefore taken as constant. A comparison of the calculation results for the third wave with statistical data is shown in Figure 4.

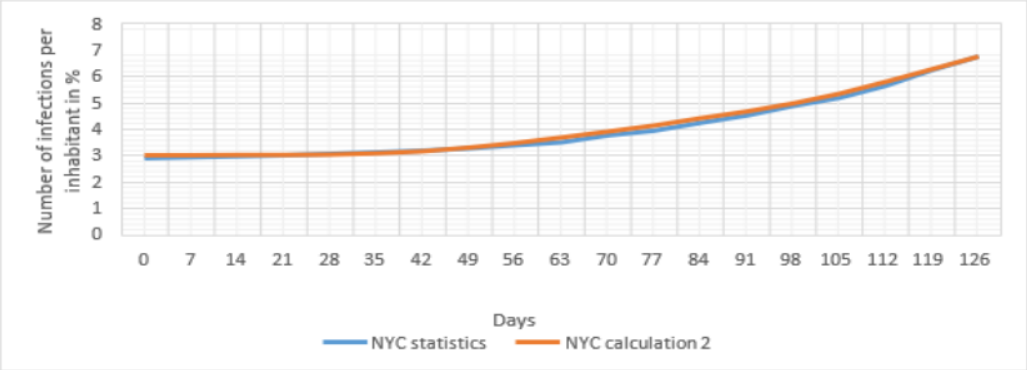


Figure 4: Epidemic spread in New York taking into account the third wave. [3]

Results of epidemic spread calculations and observational data for the entire time period from the beginning of the second wave are shown in Figure 5.

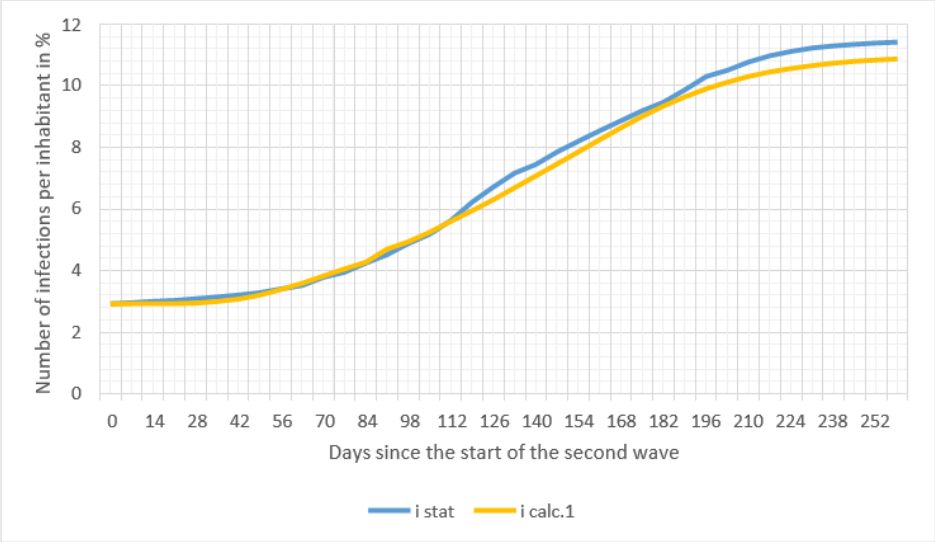
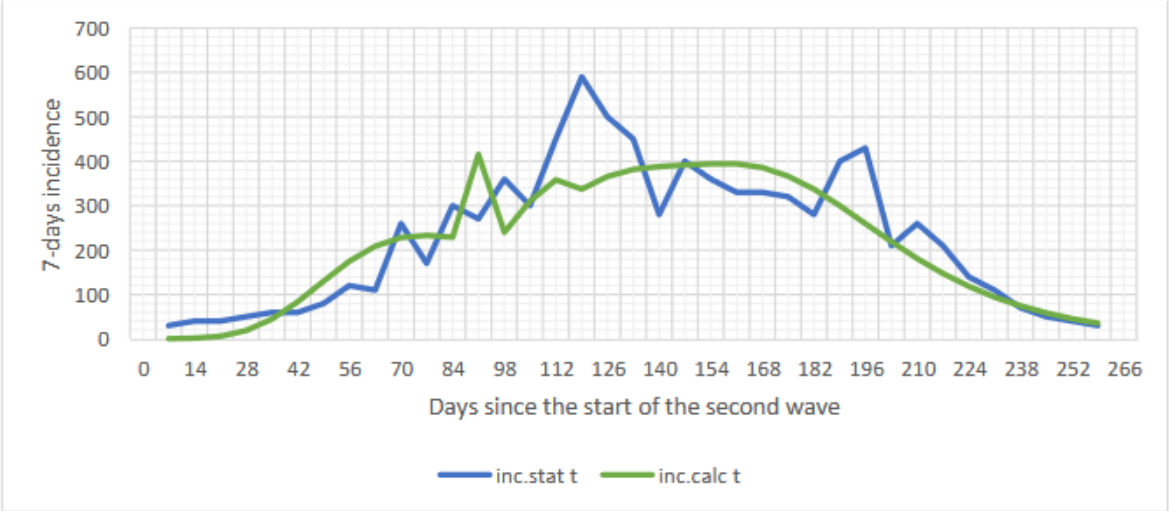


Figure 5: Development of the second and subsequent waves of the epidemic in NYC [7].

Figure 6 shows a comparison of the estimated and observed seven-day epidemic incidence for the second and subsequent epidemic waves (per 100,000 people). The 7-day incidence is taken as the difference in the intensity of infection over a 7-day period of time per 100,000 inhabitants.



(inc.stat.- observed data, inc.calc. - calculated data)

Figure 6: Incidence of epidemic growth over a seven-day period in NYC [7]

In general, the calculated values of the seven-day incidence do not differ significantly from those obtained from measurements; In general, the calculated values of the seven-day incidence do not differ significantly from those obtained from measurements;

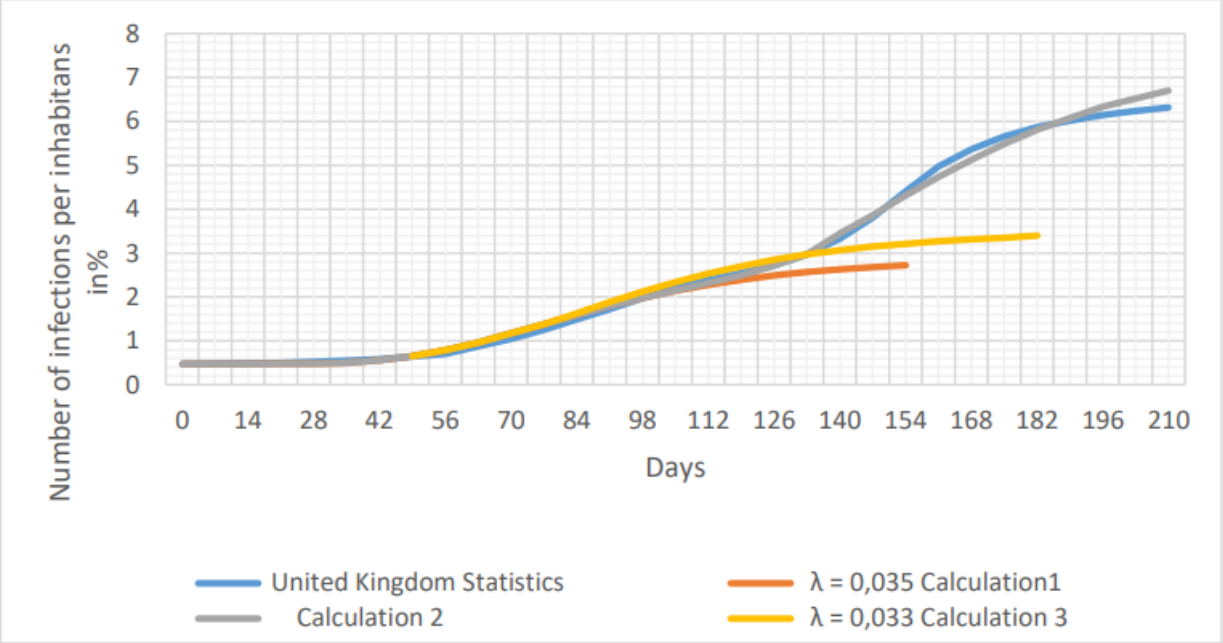


Figure 7: The spread of the epidemic in the United Kingdom [4]

Figure 7 represents statistical data and calculations for the epidemic development from the beginning of so called "second wave" in the United Kingdom [4]. Calculation 1 was performed according to the dependencies [3] and [4] for the reduction coefficient λ = 0.035 1/day Approximately 100 days after the beginning of the second wave, a new rise in the epidemic growth is observed due to the emergence of a new virus strain. Therefore, further calculation of the epidemic spread was performed according to dependence (3a) (calculation2) The coefficient characterizing the transmission rate of the virus was calculated according to ratio [4] and was equal to K = 0.5 1/day. Considering, however, that a study of this new virus species revealed its transmissibility to be markedly higher than that of the former species, this coefficient was increased to K = 0.52 1/day. The United Kingdom was chosen as an example because of the large number of studies on the characteristics of the epidemics associated with this new strain, sometimes called the "English virus" in various countries. A similar pattern was also observed in Scotland (Fig.8). The emergence of new virus strains and their impact on the spread of the epidemic is particularly clear when considering the impendence rather than the total increase in the number of infections.

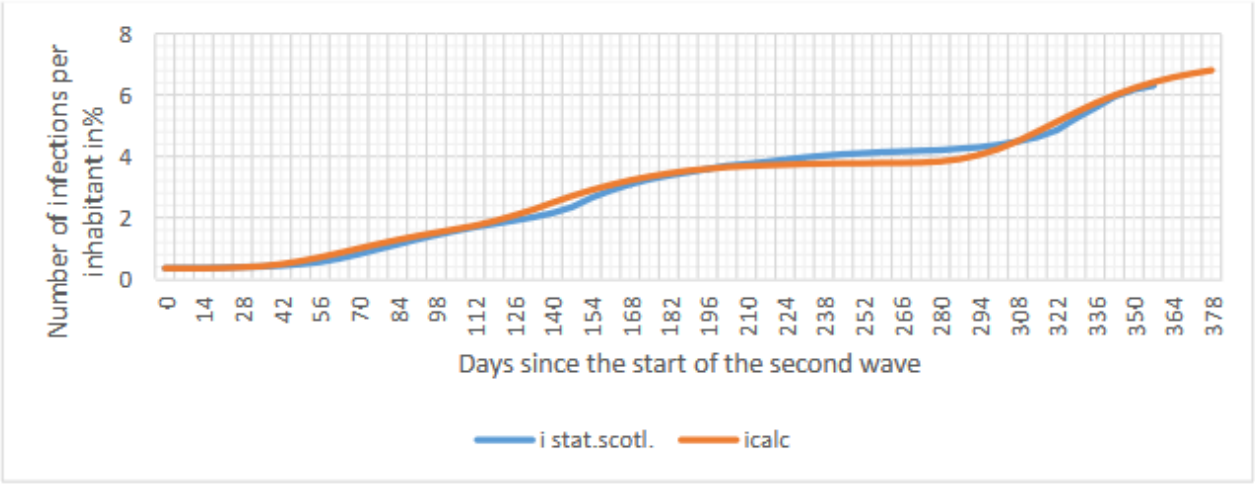


Figure 8: Development of the second and subsequent waves of the epidemic in Scotland

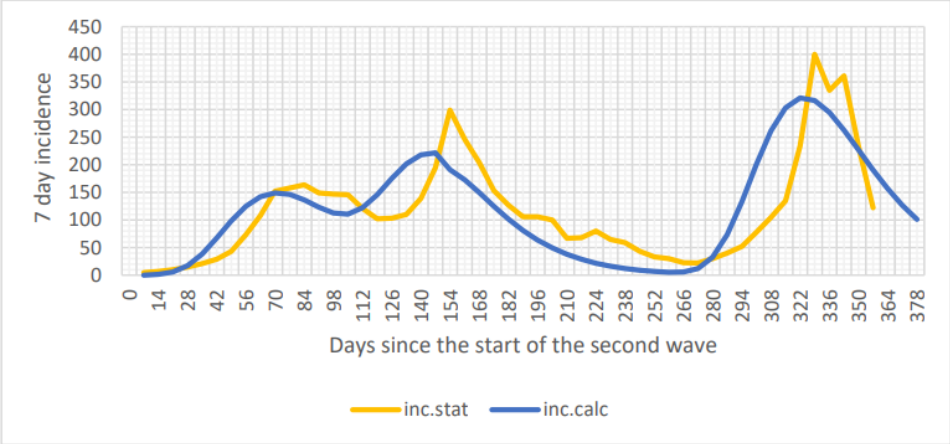


Figure 9: Seven-day epidemic incidence in Scotland.

Figures 8 and 9 show observations and calculations for the second and subsequent waves of the epidemic in Scotland [9]. Equation [7] was used for the second period, when the rate of infection growth was determined by two strains of the virus, equation [6] was used for the third period, and equation [8] was used for the fourth period. The results of calculations using the proposed method are in quite satisfactory agreement with the observed data. During the period under review, three characteristic peaks in the number of infected people can be identified.

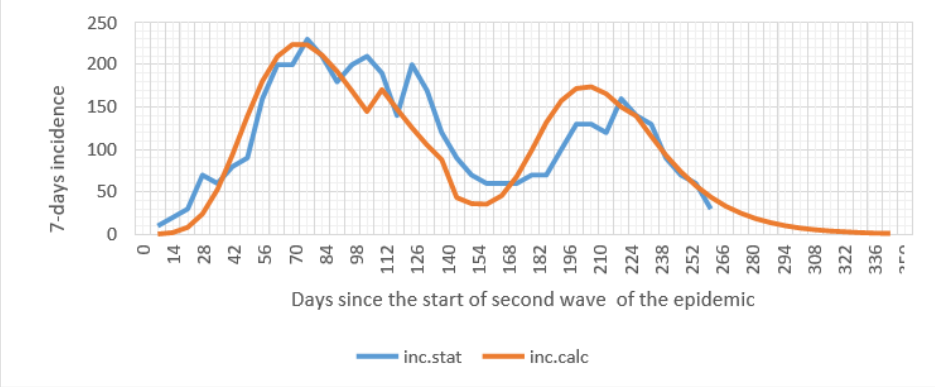


Figure 10: Incidence of epidemic growth over a seven-day period for Berlin [6].

(inc.stat.- observational data, inc.calc. - calculated data)

Fig. 10 shows a comparison of the calculated and observed values of the seven-day incident for the second and subsequent waves of the epidemic (per 100000 inhabitants) in Berlin [6]. In general, the calculated and statistical data are in good agreement with each other. The consistency of these results can be improved by shifting the calculated data to the right by about 10 days, i.e., by taking into account the "lag" effect of the response of the epidemic growth to the lockdown measures. Since abrupt changes in the epidemic growth rate are mainly associated with virus mutation and the emergence of a new strain significantly different from the previous one, the growth rate of the mutant virus variant was back- calculated

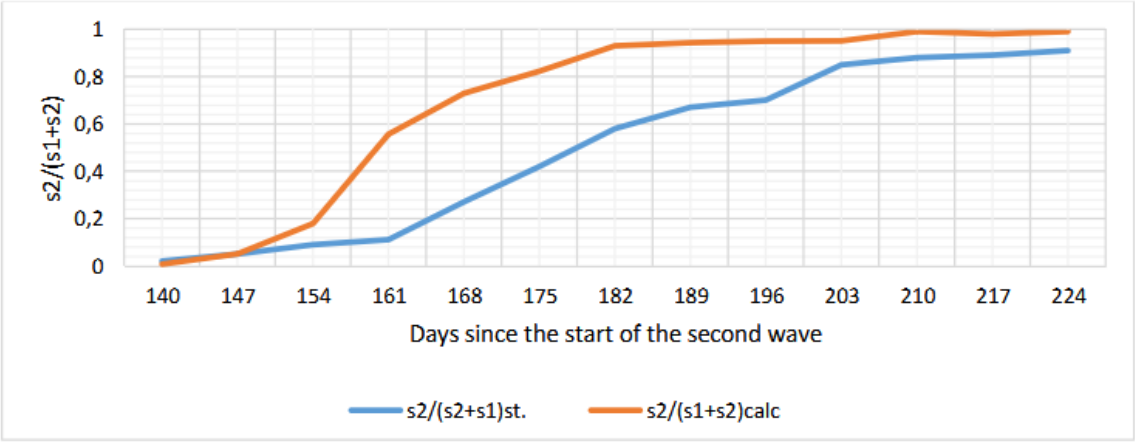


Figure.11 Growth intensity of the new virus strain in Berlin [6].

The relative number of patients infected with the new virus strain (Figure 11) was calculated as the ratio of the daily (weekly averaged) number of patients infected with the second virus strain s2 to the total daily increase in coronavirus infections (s1+s2). Observational data were obtained directly from virus sequencing. Given the time shift, the convergence of calculation and measurement results could be improved

3. Lockdown and mass vaccination

The model can be used to analyse the effectiveness of isolation and vaccination. To successfully use the proposed model for forecasting purposes, it is necessary to relate the reduction factor λ to specific lockdown actions. The effectiveness of blocking will be characterized by the parameter L [6]:

$$L = i_L / i \qquad (9)$$

where iL - and i are the epidemic growth rate with and without lockdown, respectively.

For example, if the introduction of quarantine reduces the maximum number of infected residents by half, then the coefficient L = ½ = 0.5. A graph of λ versus L is shown in Figure. 12

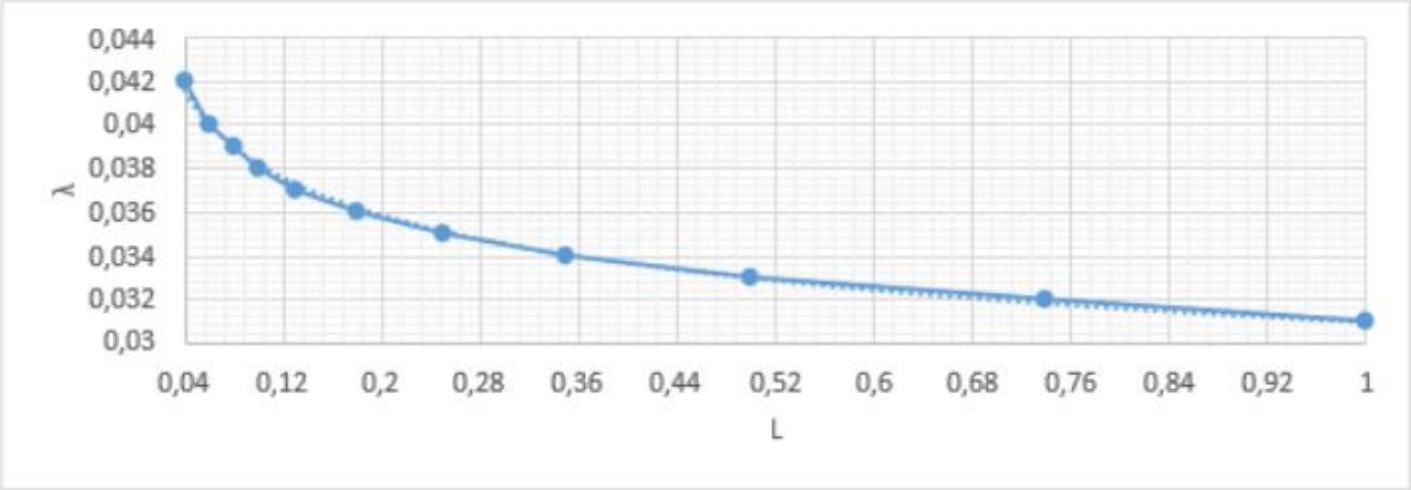


Figure 12: Dependence of the model coefficient λ on the effectiveness of the lockdown L [6].

This graph shows, in particular, that in the absence of lockdown, the coefficient λ can be assumed to be 0.031/day, and that when this coefficient is above 0.042 1/day, the epidemic wave is virtually suppressed by lockdown. However, this does not exclude the possibility of a new virus strain emerging when the lockdown conditions are relaxed. For the most characteristic lockdown conditions in most European countries, a coefficient of λ = 0.034- 0.035 1/day can be assumed; hence, the L coefficient varies between 0.2 and 0.3, which means that lockdown reduces the epidemic's growth rate by a factor of 3-5. The graph in Figure 12 can be approximated by the formula [6]:

$$\lambda = 0{,}0309*(L)\text{^}(-0{,}091) \qquad (10)$$

Figure 12 also shows the approximation curve [10] as dashed.

The main lockdown actions used to control the COVID-19 epidemic are: limitation of social contacts, implementation of recommendations to keep the distance between persons at least 1.5 meters, wearing protective masks in crowded places, compliance with hygienic norms. The dependence of the value of the coefficient L on these types of activities is presented as [6]:

$$L = (1 - N_d *d) * (1 - N_c * c) * (1 - N_m *m) \qquad (11),$$

where:

Nd, Nc, Nm - the relative number of residents who comply with the rule of physical distancing, reduction of social contacts and the prescribed mask regime, respectively, d, c, m - the effectiveness of physical distancing, reduction of social contacts and masking, respectively, in limiting the growth of the epidemic. According to approximate data [6] we can take, for example, for Germany Nd = 0, 25, Nc = 0.4, Nm = 0.75, d = 0.8, c = 0.7 and m = 0.7. By the ratio [11] we find L = 0.27. According to the ratio (10) or according to the graph Fig. 8 for these conditions λ = 0,035 1/day. This value of λ was used in calculations of virus spread in Berlin for both the first and the second wave of the epidemic. Under conditions of mass use of FFP 2 protective masks, the calculation according to formula [11] gives L = 0.165 and accordingly λ = 0.036 1/day. This value of this coefficient was used in calculations of the new epidemic wave in Berlin. Mass vaccination also significantly slows down the epidemic growth [7, 8]. Control calculations of the effect of vaccination intensity on epidemic growth were performed. The results of the calculations under different lockdown regimes for the city of Berlin are presented in Figures 13 and 14.

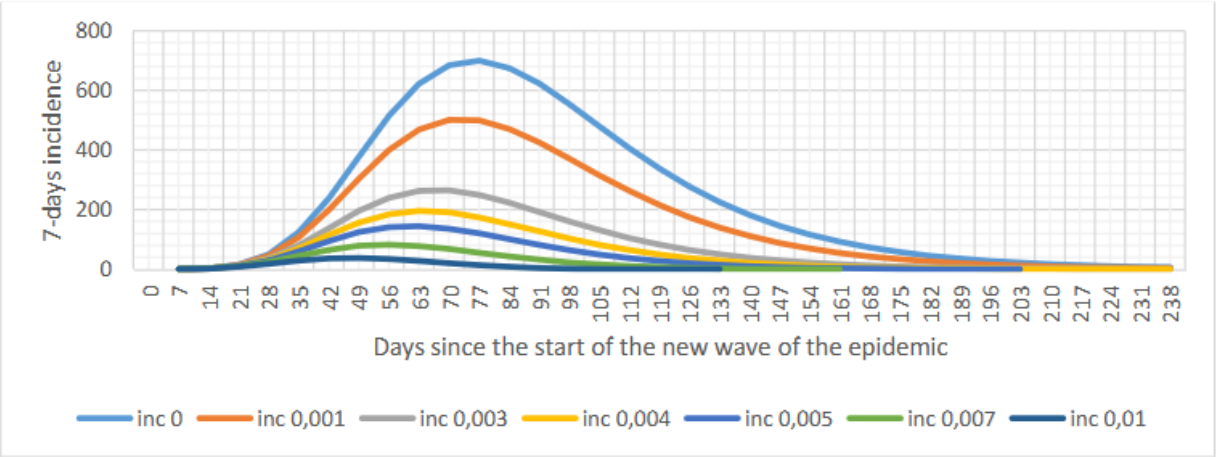


Figure 13: Seven-day incident value for the delta variant at λ= 0.035. [8]

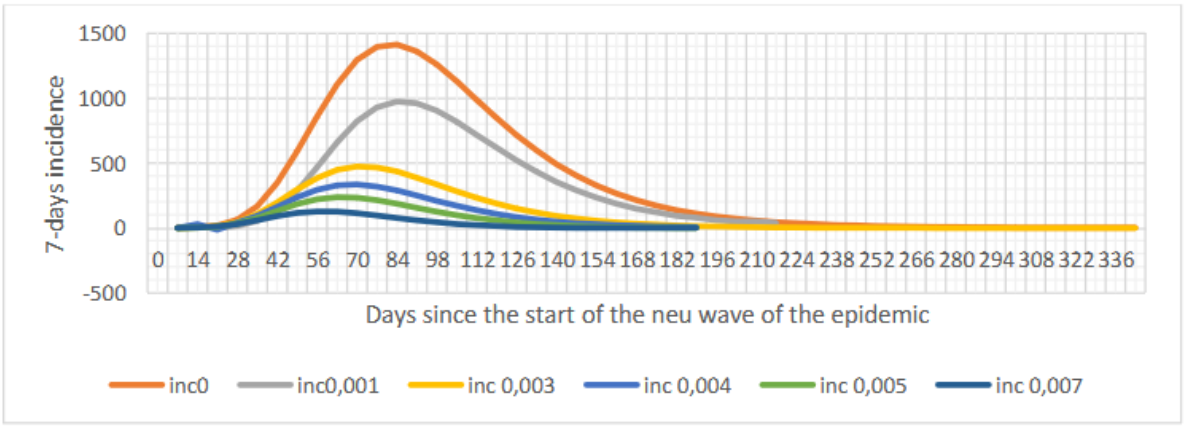


Figure 14: Seven-day incident value for the delta variant at λ= 0.033 [8]

(The graph shows vaccination rates in 1/day)

The worst-case scenario could be the emergence of new, even more contagious variants of the virus. This epidemic scenario can be prevented through isolation and fairly effective mass vaccination. One of the countries that managed to reduce the growth rate of the epidemic through mass vaccination is Israel. Vaccination began there earlier than in all European countries. The intensity of effective vaccination reaches the value αv = 0.008 1/day (for comparison: for Berlin for the same period αv = 0.001 1/day).

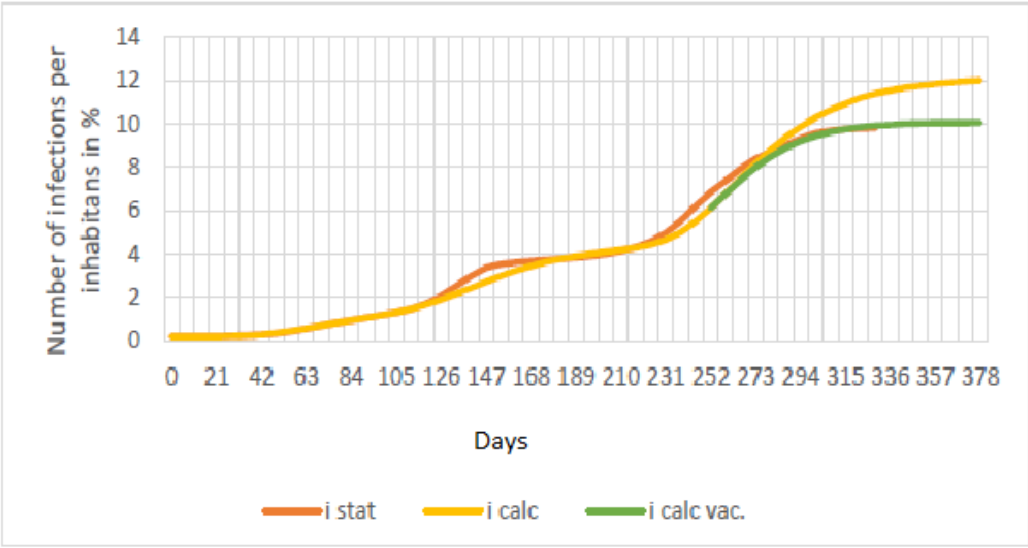


Figure15: Development of the epidemic in Israel [5]

In this figure: i stat. - observed data, i calc. - calculation without vaccination, i calc. vac. - calculation for conditions of mass vaccination of the population. Fig. 15 shows the results of calculations using dependencies (6) and (8) (with $\lambda = 0.035$ 1/day) and relevant statistics for Israel for the period May 2020 to April 2021. The calculations took into account the influence of weather conditions [5] and changes in virus activity on the coefficient k0 in accordance with formula (5a).

4. Age differences

The spread of the epidemic for different age groups was analyzed using the city of Berlin and Scotland as an example. From the general population of Berlin, 10 groups are distinguished, epidemic [8]

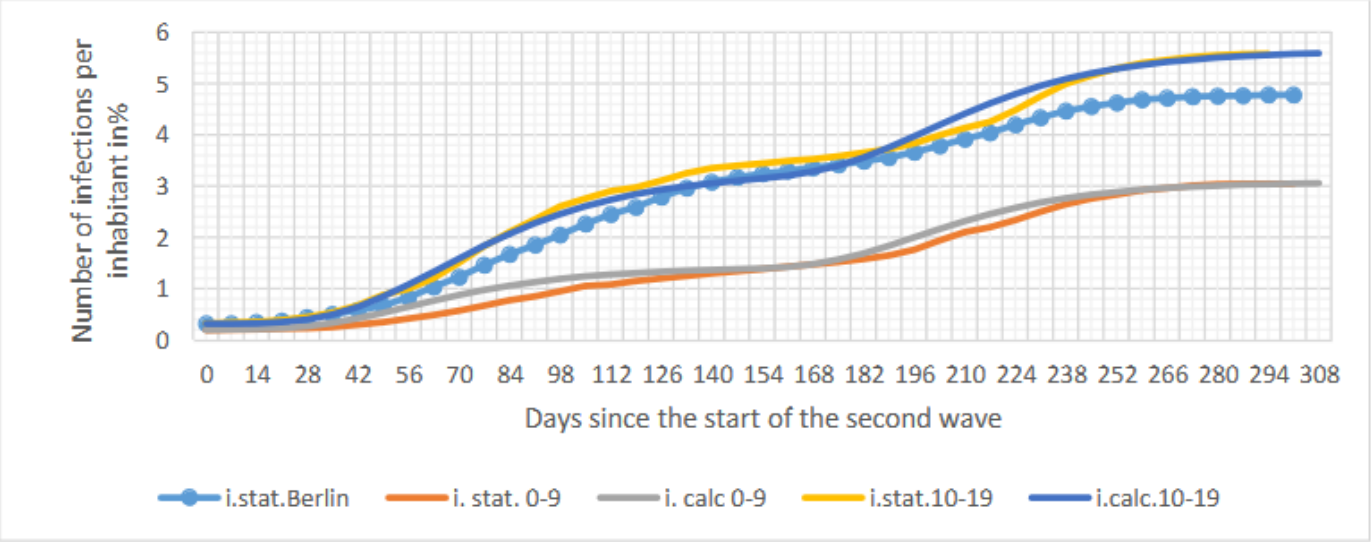


Figure 16: Epidemic prevalence in the age groups 0-9 and 10-19 years old [8]

(i.calc 0-9, i.calc 10-19 - calculation for age groups 0-9 and 10-19, respectively, i.stat.Berlin, i.stat.0-9, i.stat.10-19 - observational data for Berlin and for age groups 0-9 and 10-19, respectively),

The results of calculations according to the proposed methodology for the age groups 0-9 years and 10-19 years are presented in Figure. 16. The same graph shows the observed data both for the corresponding age groups and for Berlin as a whole [8].

Figure.17 and Fig.18 shows the comparison of calculated and observed incident values for age groups 0-9 and 10-19 years respectively. In these and subsequent graphs the following notations have been introduced:

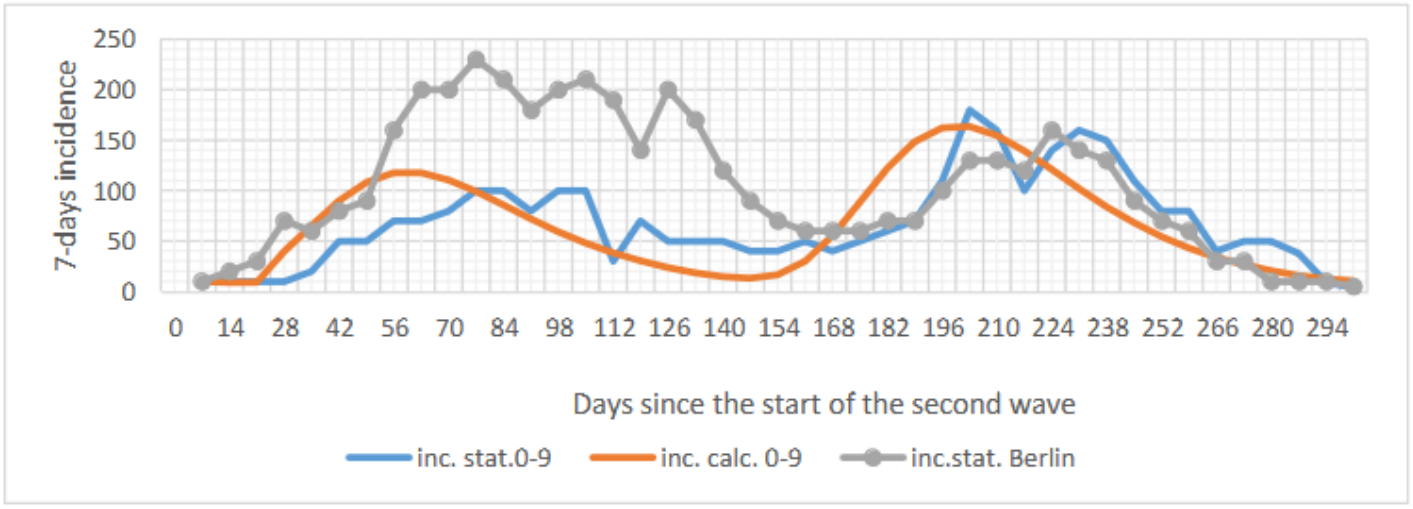


Figure 17: Seven-day incident for the 0-9 age group [8]

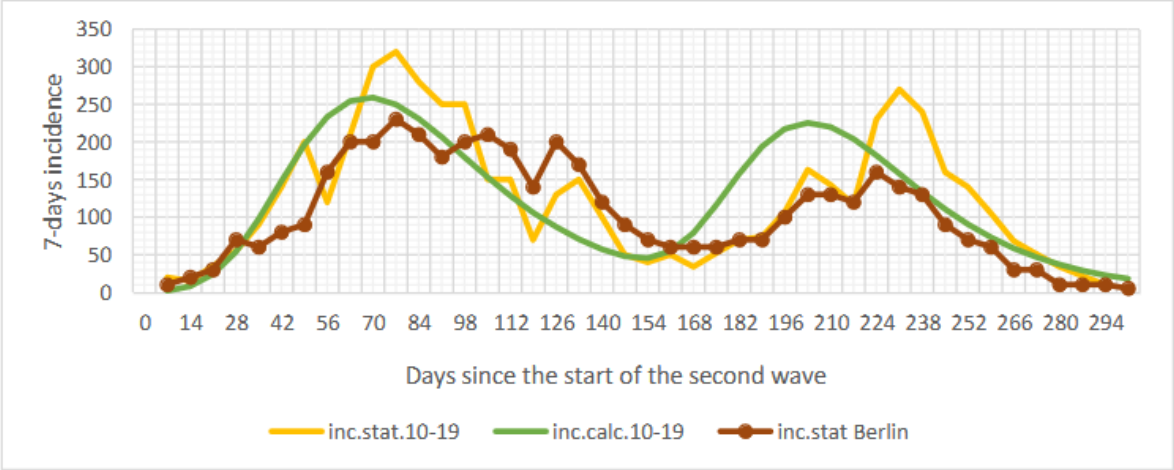


Figure 18: Seven-day incident for the 10-19 age group [8]

(inc.stat.-incident values for the indicated age groups and for Berlin from observed data, inc.calc. - inc.calc.-incident values for the specified age groups.) In general, the calculated and statistical data on incident values are qualitatively and quantitatively in quite satisfactory agreement with each other. At the same time, the intensity of the epidemic growth for the 0-9 age group is significantly lower than for both the 10-19 age group and Berlin as a whole. This can be explained both by the lower contact rate for children in the younger age group and possibly by their weaker susceptibility to viral disease. In the process of this work, calculations were performed for all ten age groups. In general, for each of the selected groups, the results of calculations are in satisfactory agreement with the observed data.

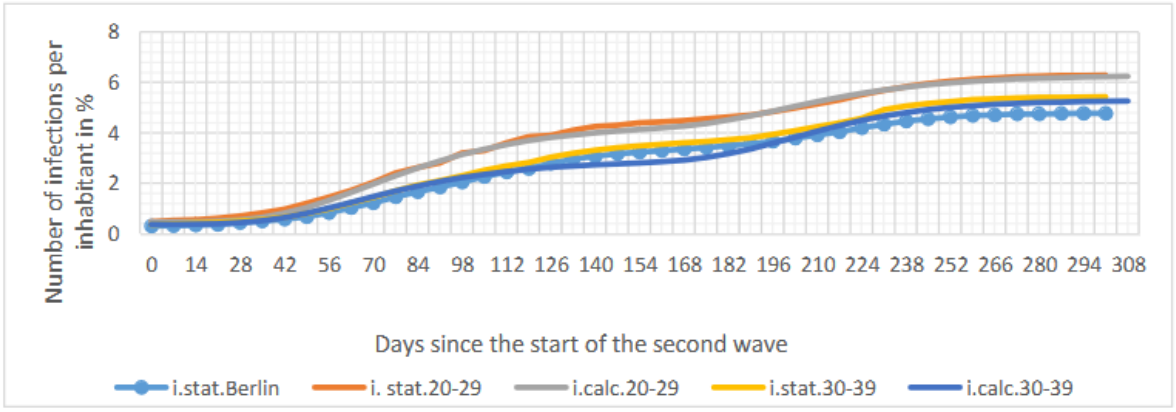


Figure 19: Epidemic distribution in the age groups 20-29 and 30-39 years old [8]

As an example, here is a graph of the spread of the virus epidemic for the age groups 20-29 and 30-39 years. (Fig.19). It should be emphasized that these two age groups are responsible for more than 35% of all infections in the city of Berlin.

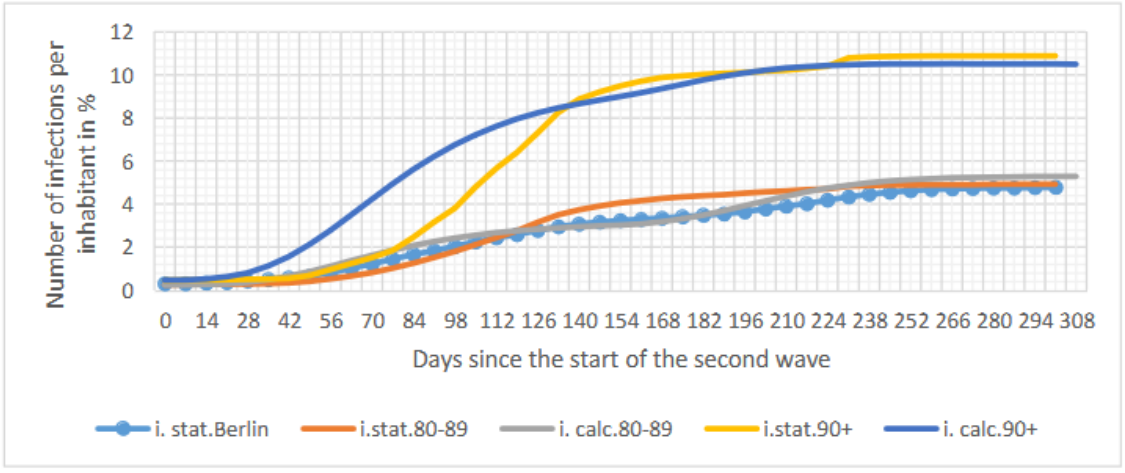


Figure 20: Epidemic spread in the 80-89 and 90+ age groups [8]

Other important age groups for the analysis are the elderly groups from 80 to 89 years of age and the 90+ age group. These groups are relatively small and they do not determine the intensity of the epidemic growth in Berlin (the number of infections in these groups does not exceed 7% of the total number of infections in the city). However, the number of infections in these groups largely determines the mortality rate from COVID disease. Figure 20 shows a comparison of the calculated results with the observed data for these age groups. The intensity of the epidemic for the 80-89 age group does not differ significantly from the average for Berlin. In contrast, the level of infection in the 90+ age group at the beginning of the epidemic is more than twice as high as the average (the reduction coefficient λ was assumed to be 0.0315 1/day). This is partly due to the fact that the majority of city residents of this age live in nursing homes, where diseases can become mass. In addition, the transmission rate for residents of this age is also higher than the average for the general population. However, it is these two age groups that have the priority right to be vaccinated, as a result of which the percentage of vaccinated residents of these age groups reaches more than 80%. As a result, starting about 200 days after the beginning of the epidemic wave, there is a sharp decline in the number of infected people, as can be seen in Figure 20 and especially in Figure 21.

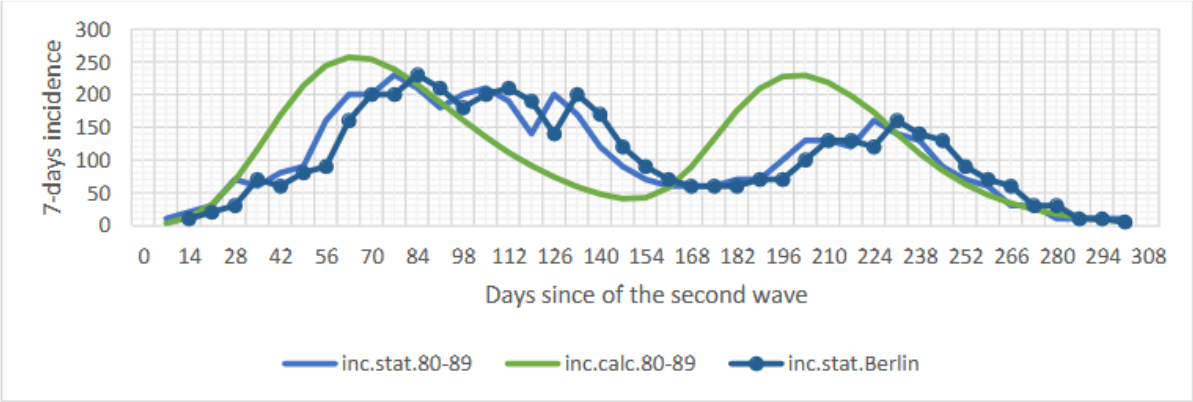


Figure 21: Seven-day incident for the 80-89 age group [8]

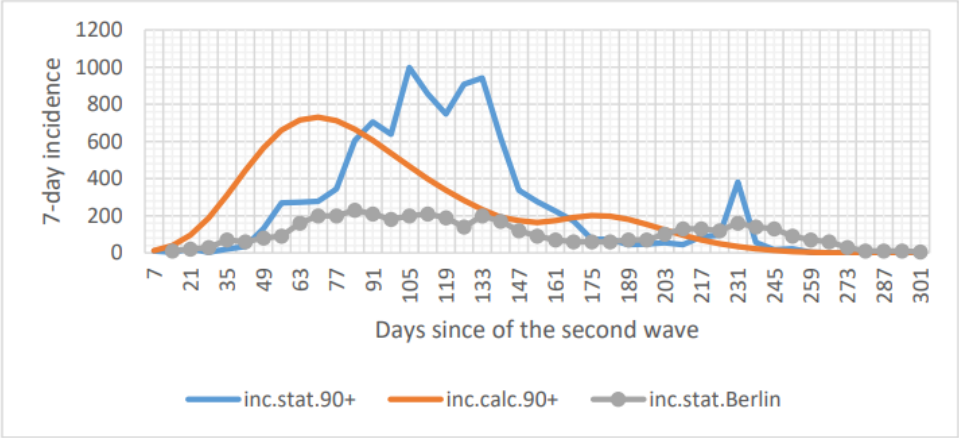


Figure 22: Seven-day incident for the 90+ age group [8]

By analyzing the graph in Figure 22, we can see that before vaccination, the infection rate in the 90+ group was about 5 times higher than the Berlin average. Only as a result of mass vaccination was it possible to extinguish the virus epidemic in this age group within a month.

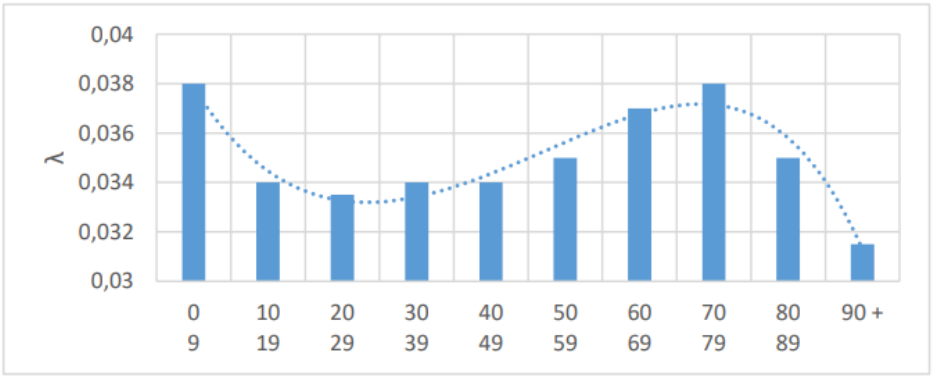


Figure 23: Values of coefficients λ for different age groups [8]

The values of the coefficients used in the model for calculations for different age groups are presented in Figure. 23. Qualitatively similar results were obtained in a study of the development of epidemic for different age groups in Scotland [9] Unfortunately, the statistical data are not harmonized across countries even in Europe. Therefore, the division into age groups in Scotland and Germany is somewhat different. As well as in Berlin, the intensity of the epidemic in children under 14 is significantly lower than in the general population, while for the 15-24 age group the relative number of infections is 1.5 times higher than for the general population in Scotland.

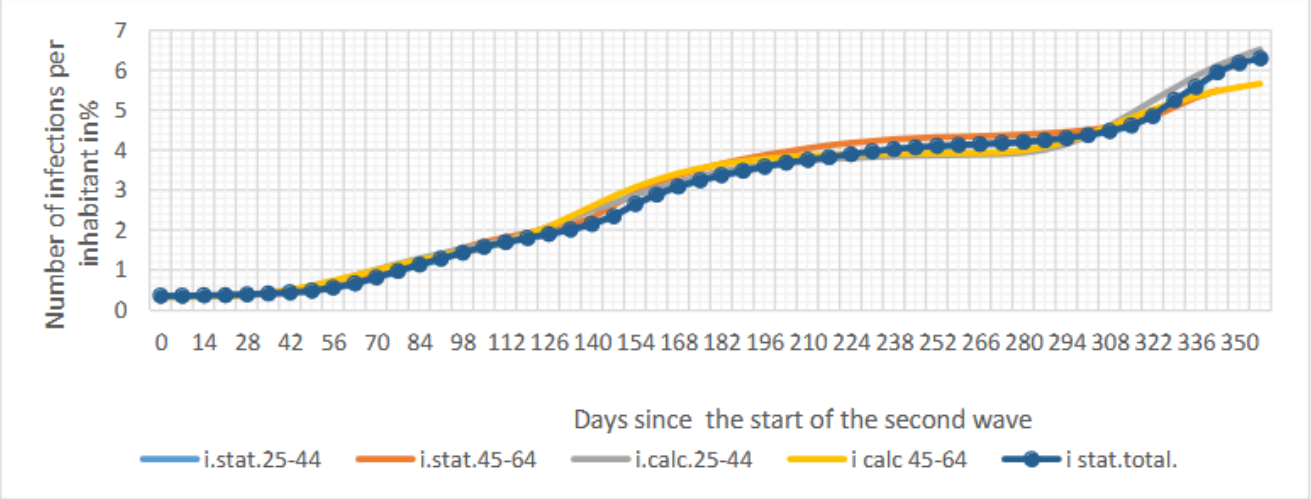


Figure 24: Epidemic development for the age groups 25-44 and 45-64 [9].

As shown in Figure 24, the intensity of the epidemic in the age groups 25-44 and 45-64 years old is almost identical and weakly different from the general population. The results for each of these age groups correspond well with the observations.

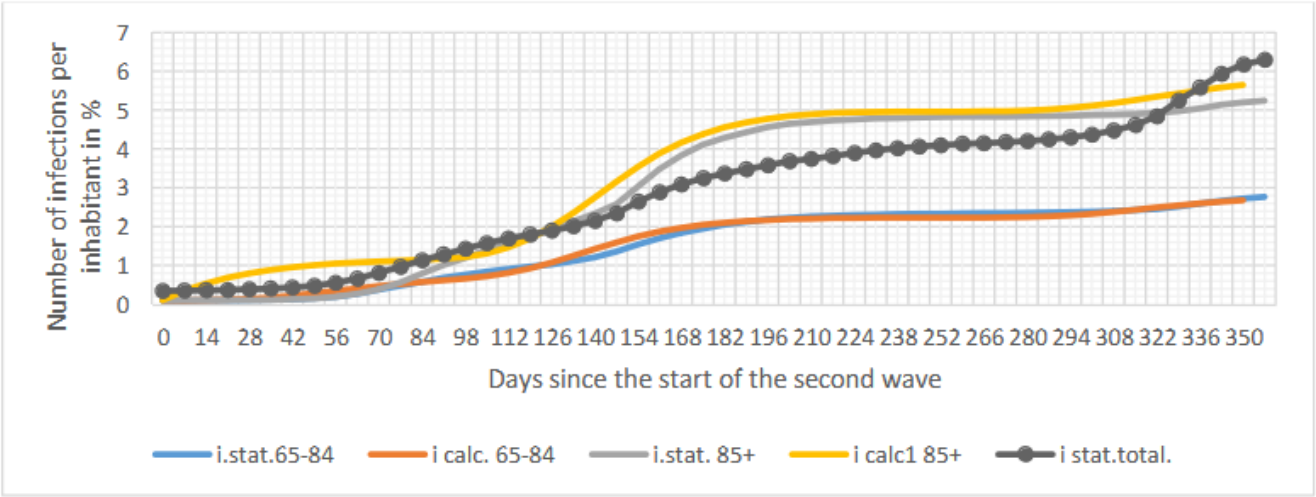


Figure 25: Development of the epidemic for the age groups 65-84 and 85+ [9].

In the age groups of the elderly aged 65 to 84 and 85+, nearly complete vaccination of this group markedly reduced the intensity of the epidemic caused by the delta variant of the virus (Figure.25). This is especially well seen when comparing the epidemic development in these age groups and in the general population in the fourth quarter of time (at t≥300 days) The general conclusion for both Berlin and Scotland is that the epidemic development in the age range 20-60 is not significantly different from the average, whereas the epidemic is less intense for the groups from about 60-85 years of age and for children under 15 years of age. For the 85+ group, the epidemic is more intense than the population average. The lower epidemic development rate for this group is largely due to active, almost total vaccination.

5. Social and ethnic background

The spread of the epidemic may be influenced by the socio-demographic characteristics of the population In Berlin, for example, before the start of mass vaccination, there was a large unevenness in the number of diseases throughout the city [1,10].

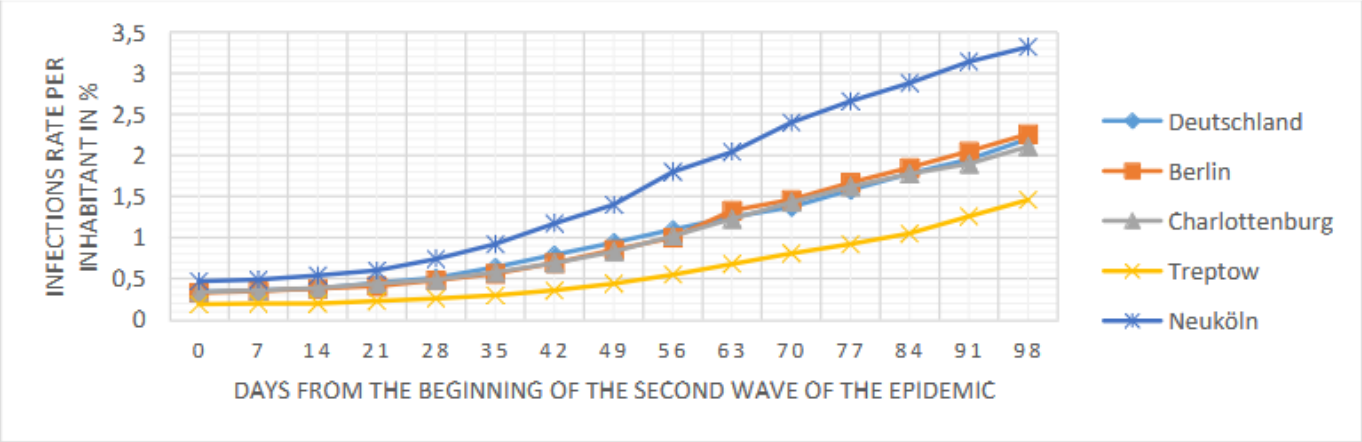


Figure 26: The depends of infection rate on foreigners from countries OIC [10]

Figure 26 shows the results of statistics on the relative increase in infections during the second wave of the epidemic in Berlin. As can be seen from this figure, there is great unevenness in the number of infected people across the city. Thus, while in some neighborhoods, such as Treptow-Kˆ penik, the maximum increase in the number of infected persons does not exceed 50 persons per day, in the Neukˆ ln it reaches 300 persons per day and considering that one of the most likely places of infection are apartments where people communicate most intensively, it can be assumed, that the growth of the epidemic should depend on the area per person, i.e., on the density of apartments. Taking into account that one of the most likely places of transmission is apartments, where people communicate most intensively. we can assume that the growth of the epidemic should depend on the area per person, i.e., on the density of apartments.

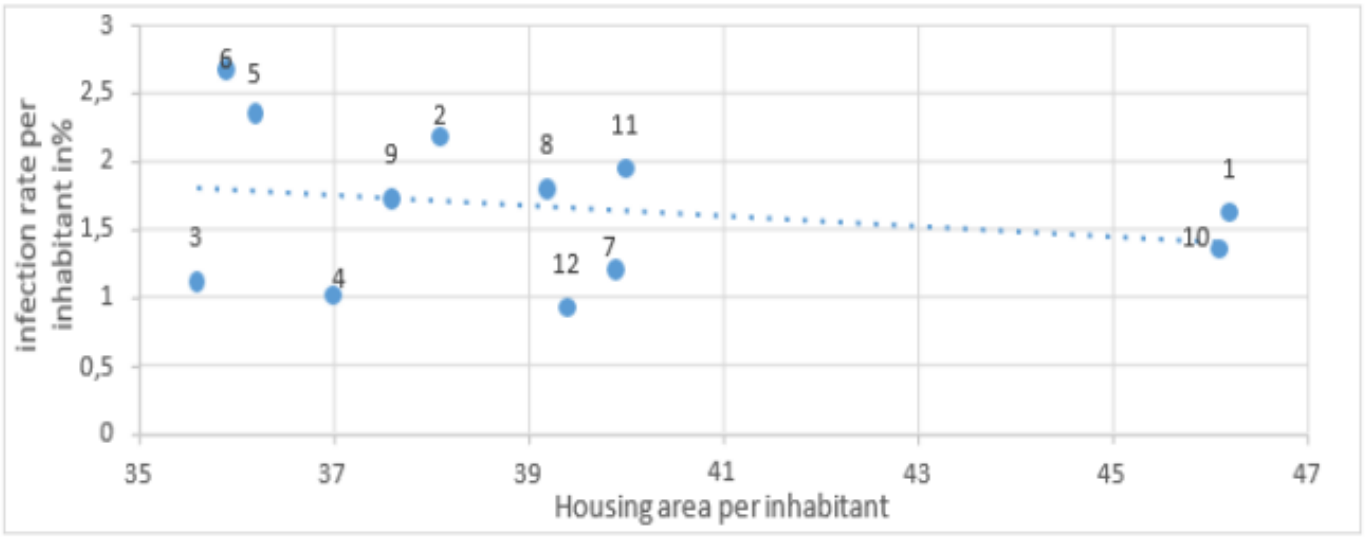


Figure 27: The depends of infection rate on the size of the living area per person [1].

Figure. 27 shows the graph of dependence of the epidemic growth intensity on this factor. Contrary to expectations, the relationship between these parameters turned out to be extremely weak, the correlation coefficient is about r = - 0.22. The correlation between the intensity of epidemic growth and the population density in Berlin districts and the relative number of emigrants was analyzed. The correlation coefficients between the intensity of the epidemic growth and these parameters are approximately 65%. But unexpectedly, the relative number of emigrants from member countries of the Organization of the Islamic Conference (OIC) (Turkey, Arab States, some African countries) in each district was found to have the maximum impact.

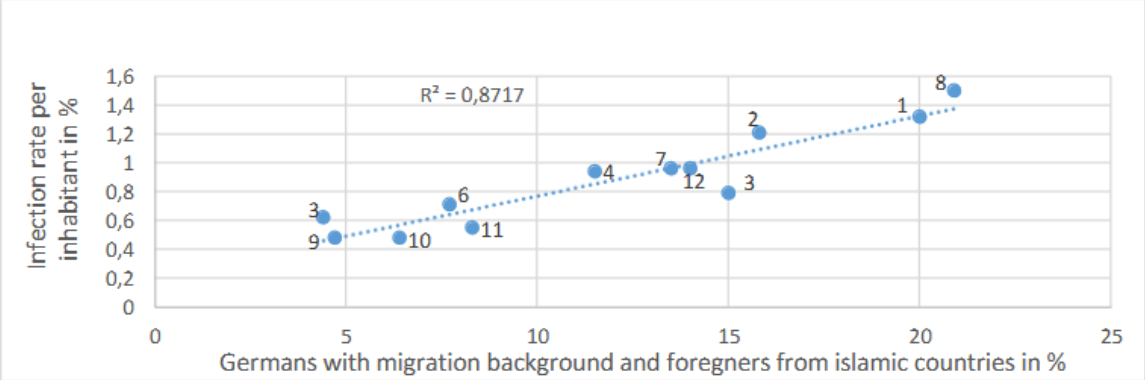


Figure 28: The depends of infection rate on foreigners from countries OIC [1].

Figure 28 shows the dependence of the number of infected people for each of the 12 districts in Berlin on foreigners from countries OIC. The high correlation coefficient (r = 0.93) shows that the connection between these parameters is not only statistically reliable, but also practically functional. It is important to emphasize that the growth curves of not only the first but also the second wave of the epidemic for the district of Charlottenburg practically coincide with the curves for Berlin and for Germany as a whole. The relative numbers of OIC residents for Charlottenburg, Berlin and Germany are 12 per cent, 11 per cent and about 10 per cent respectively whereas in the Neukˆ ln area the number is twice as high. Apparently, foreigners from member countries of Islamic countries have closer family and friendship ties, which can contribute to more intensive transmission of the virus. After mass vaccination in Berlin, differences in the intensity of epidemic growth were leveled out, although epidemic growth remained higher in areas of Berlin with a higher proportion of emigrants. This is due to the fact that the vaccination rate for the native population is higher than among emigrants.

6. Psychological and behavioral characteristics of the population

The relationship between the willingness to comply with the lockdown rules and psychological and behavioral characteristics of individuals has been studied in connection with the spread of COVID by a large number of psychologists and sociologists In [12] it is shown that individuals with more active reactions to stress or disgust more closely follow lockdown rule. An important objective indicator of the level of disgust, as well as other basic emotions - fear, anxiety or stress - can be an analysis of changes in heart rate. This physiological indicator can provide information about how quickly and well the body adapts to external influences. Heart rate variability (HRV) is the change i intervals (differences in duration) between heartbeats over time. The peaks of heart rate detected by the cardiogram are called R-spikes, and the intervals between R-spikes are called RR intervals. LF ("low frequency": 0.04-0.15 Hz) is characterized by longer changes in successive RR intervals. LF is influenced by both the sympathetic and parasympathetic systems, but the sympathetic system has a greater effect. HF ("high frequency": 0.15–0.4 Hz) represents short- term changes in successive RR intervals and is a reliable indicator of parasympathetic activation. The LF/HF ratio is often used to measure the balance in the ANS between the sympathetic (SNS) system, which is called the "fight or flight" system, suggesting that its activity favors highly aroused, active states, while the parasympathetic system (PNS) is called the system "rest and digestion." Experimental and theoretical studies were carried out by D. Below at the University of Potsdam to study the relationship between the disgust reaction and HRV characteristics [11]. The purpose of this study was to investigate whether the intensity of the disgust response could be related to heart rate variability and used for diagnostic purposes in the future. 30 students from the University of Potsdam took part in the study. The images and objects were to be rated by subjects according to their subjective level of disgust response. The results of heart rate measurements during arousal were compared with 5-minute preliminary similar measurements in a calm state (baseline). The stability coefficient B is used as a characteristic value that reflects the intensity of changes in the physiological reactions of the heart [1]. Coefficient B is calculated as the ratio of HF values during excitation and in the resting phase:

B(HF) = HF(E)/HF (Base Line)

HF(E) is HF during the excitation phase, HF (Base Line) is HF during the resting phase.

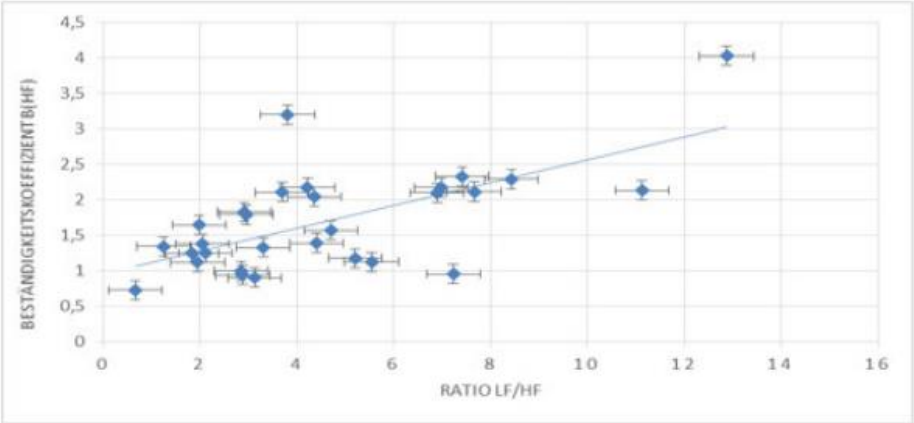


Figure 29: Dependence of B coefficient on LF/HF ratio [1].

In turn, the stability coefficient was found to be dependent on the LF/HF ratio at rest. Figure 29 shows the results of the relationship between the psychological stability coefficient B (HF) and the LF/HF ratio during the baseline measurement. The correlation coefficient between these parameters is r = 0.623, p < .01. The corresponding approximate formula for this relationship can be written as [1].

B(HF) = 1 + 0.15 LF/HF (12).

By introducing the stability factor B (HF), it is possible to predict the degree of physiological response to feelings of disgust or stress. This requires measuring the LF/HF ratio at rest, as was done in this study during the baseline measurement. Substituting this value into the formula (12), we find the expected value of the B (HF) ratio. The less this coefficient differs from 1, meaning the more stable the nervous system is, the more appropriate the response to the lockdown and vaccination compliance rules would predictably be. To increase the reliability of these findings, it is necessary to analyze in more detail the dependence of the stability coefficient B (HF) on the LF/HF ratio. In the future, it would be useful to relate the stability factor to the coefficient λ used in the model.

7. Dimensionless complexes-an indicators of the commonality of epidemic spread

The process of epidemic development, according to the developed model, is defined by three dimensionless parameters: k /λ, αv/λ and λt [10]. All variables that make up these complexes have dimensionality 1/s and characterize the intensities of individual processes: k - infection, λ - reduction of infections as a result of lockdown and αv - vaccination. Each of these processes depends on many factors, some of them can be regulated, and others cannot. The parameter k, which depends on virus strain, population size and climatic conditions, is not controllable. The other two parameters depend on the effectiveness of lockdown and vaccination respectively, i.e., they can and should be administratively regulated. From the analysis of the graphs in Fig. It follows that the spread of the epidemic in widely differing size areas is almost identical. To verify this conclusion, observations of the second wave of the epidemic in New York, Germany and Berlin were compared. These results are presented in Fig. 30. In order to exclude the influence of the previous wave, the difference between the current value of the number of infected people and their values at the time of the second wave is used. Using this technique,

As can be seen from this figure, all three curves practically coincide up to a certain point in time. Only about 90 days after the beginning of this epidemic wave, the growth of the infection in New York became significantly higher than in Berlin and Germany. This means that only the passage of the third wave of the epidemic for New York is significantly different from Berlin.

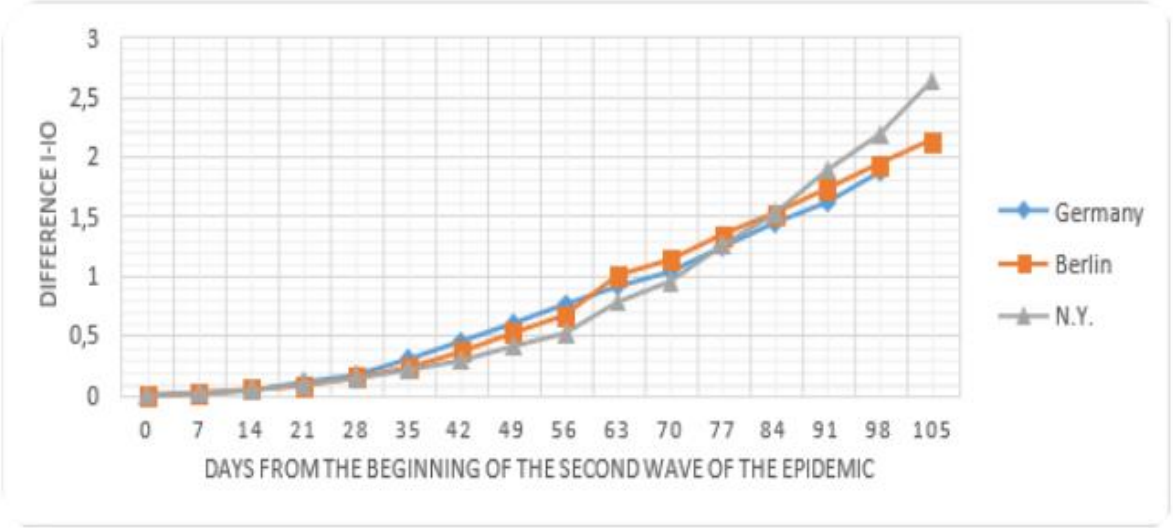


Figure 30: Changes in the number of infections compared to their numbers at the beginning of the second wave [10]

However, an important general pattern was simultaneously established: some curves of relative growth of the epidemic practically coincide. The identity of the epidemic development curves for different territories, for example Germany, Berlin and New York at certain points in time, allows us to draw a very important practical conclusion about the possibility of using similarity theory in predicting the occurrence and development of the epidemic. In particular, this will allow the use of large- scale modelling when transferring the results of a model experiment carried out in small areas (for example, the Charlottenburg district of Berlin) to much larger areas (for example, Berlin and even Germany). These similarity methods are currently widely used in hydrodynamics, thermal engineering and in solving many other physical and technological problems. With reliable prediction of the values of model coefficients, and therefore dimensionless complexes, it will be possible to widely and successfully use these methods in epidemiology.

Discussion

The comparison of a large number of calculated and statistical data confirms the possibility of successfully using the proposed simple model to calculate the spread of the COVID-19 epidemic. Significant differences in these data are found only when calculating the epidemic growth among the 90+ age group. (Figure 22). The discrepancy between the calculated and statistical data for this population group is probably due to small contacts of people within the group, which contradicts the condition of applying the model for separate age groups. For all other estimated conditions, it was found that the average statistical deviation of estimated data from those recorded in official documents does not exceed 5%. Taking into account the simplicity of the model, we can recommend its use by the administration of settlements for operational analysis and management of the epidemic without involving specialists in the field of computer modelling. Another important advantage of the model is that the obtained analytical solutions allow analyzing the separate influence of each of the determining factors on the development of the epidemic. Combining these parameters into dimensionless complexes allows for large-scale modelling of the epidemic growth process. Such modelling has been widely used in applied physics, for example, in aero-hydromechanics and heat engineering, and allowed to determine on small-scale models the performance characteristics of various real objects, such as airplanes, hydro dams or boilers of thermal power plants. With the help of dimensionless complexes in epidemiology it would be possible, for example,

to analyze the efficiency of epidemic management on the basis of studying the course of these processes in small settlements. In general, an epidemic in different localities should be identical. However, this identity is significantly disturbed due to differences in the willingness of the population to comply with the lockdown rules, which is accounted for in the model by the coefficient λ. As this coefficient increases, the rate of epidemic limitation increases. Decreasing epidemic limitation is characteristic of both some ethnic populations in the United States [13] and some immigrant communities in Europe [1]. In all likelihood, this can be explained by the lower level of trust of these population groups both in the local administration and in the prescriptions of medical authorities. The effectiveness of limiting the development of the epidemic depends on the psychological mood of the population. Significant changes in the behavior of the population can be observed in the same territories. Of course, this is largely due to the actions of the local administration to reduce the level of the disease. For example, at the beginning of the first wave of the epidemic in New York City, the necessary administrative measures to control the epidemic were not activated, resulting in high levels of disease and deaths due to virus infection. In contrast, in Berlin, all the necessary administrative resources were deployed and the first wave of the epidemic was relatively calm. As for the second wave, the situation changed dramatically. In New York, more stringent measures were taken to control compliance with the lockdown and vaccination restrictions, while in Berlin these restrictions were more leniently enforced. As a result, the incidence of the virus in Berlin increased significantly during this period and reached the level in New York (Figure. 30). The willingness of the population to comply with the requirements of lockdown and vaccination largely depends also on the psychological mood of the population. As an important indicator of such mood, we used the coefficient of psychological stability introduced by us, which is related to the activity of an individual's reaction to stress or aversion. This assumption is not confirmed by a sufficient amount of statistically independent data and requires further substantiation. The formula for the relationship between this coefficient and the physiological characteristics of the individual should also be further tested using additional data. The main conclusion for us is that, at present, any improvement of a model for predicting the spread of an epidemic must first of all objectively take into account the psychological and behavioral characteristics of the local population Psychological factors influencing avoidance mechanisms are grouped under the general term “behavioral immune system”. As shown, for example, in [14], the behavioral immune system has a strong influence on the growth of an epidemic.

An important limitation of this paper should be noted. It does not examine the consequences of waning immunity to COVID over time after vaccination. Given that COVID, like any viral disease, does not stop completely but can only decline, and the continuous mutation of the virus, it is crucial to predict possible new waves of the epidemic. The lack of such a forecast does not allow for reliable prediction of critical situations related to the COVID epidemic.

Conclusion

1. The proposed simple analytical model of ASILV accurately describes the development of the COVID19 epidemic for areas of different sizes and geographical characteristics. The model's calculation methodology takes into account the impact of lockdown and vaccination of the population, as well as the possibility that new strains of the virus may emerge due to mutation.

2. Delaying the introduction of a lockdown or mass vaccination of the population at the onset of each new wave of the epidemic leads to the risk of an uncontrollable increase in the epidemic.

3. The model uses two empirical coefficients: one related to the transmissibility of the virus strain, and the other, the reduction factor, related to the effectiveness of the lockdown. As the reduction factor increases, the growth of the epidemic slows down. The value of this coefficient depends on the age distribution of the population, the size and ethnic composition of emigration groups.

4. The reduction coefficient is functionally related to the parameter of lockdown efficiency. The effectiveness of lockdown is largely determined by the willingness to comply with social restrictions, i.e., it is closely related to psychological and behavioral characteristics of the population. To assess the possible reaction of the population to the requirements of lockdown, the coefficient of mental stability is introduced, which is objectively related to the parameters of heart rate in the unexcited state.

5. On the basis of comparison of data on epidemic development and calculations, the identity of infection spread for significantly different territories is established. This phenomenon is explained with the help of the proposed model by the allocation of nondimensional complexes responsible for the intensity of epidemic development. The application of similarity theory methods may allow in the future to extend the modeling results obtained for small regions to larger territories.

6. Some fundamentally important conclusions, such as the study of the influence of the psychological state of the population on the rate of spread of infection or suggestions on the possibility of using similarity theory methods, are based on studies in their initial stages and require further substantiation and clarification.

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