

Calculations of the Spread of the COVID 19 epidemic in New York City based on the Analytical Model

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Abstract

A detailed description of the model for calculating epidemic spread under conditions of lockdown and mass vaccination of the population is given (ASILV model).

The proposed analytical model adequately describes the development of the epidemic in New York City. The estimates of the total number of infected persons and the seven-day incident rate made using the proposed model correlate well with the observed data in all the stages of epidemic growth.

Model calculations of the spread of the epidemic under different vaccination rates allowed an assessment of the effect of vaccination on the growth of the epidemic. Analysis of seven-day incidence curves at different vaccination intensities led to the preliminary conclusion that at vaccination rates above a minimum value, the emergence of new strains did not lead to a growing epidemic.

Keywords: COVID 19; epidemic spread; analytical model; new york city; incidence; vaccination

Introduction

Most models used to calculate the epidemic offer only numerical methods for solving. We have developed a simple and versatile analytical model [1, 2, 3, 4, 5], which enables us to quickly analyse the distribution patterns of the coronavirus epidemic.

The control calculations performed have shown a high degree of accuracy for widely varying populations, ranging from small areas of Berlin to large cities and a number of countries, such as the United Kingdom, South Africa, Germany and the United States. The correlation coefficients between the respective estimated and statistical curves reach values between 0.94 and 0.99.

The model was further developed to take into account the effects of abrupt changes in lockdown conditions and mass vaccination of the population. Comparison of the results of calculations by this modified model with data from statistical observations also shows good agreement [6, 7].

The analytical model using functional relationships between the main parameters determining the development of the epidemic makes it possible to assess the effectiveness of limiting the development of the epidemic through both lockdown and vaccination.

Despite some successes in using the proposed simple analytical model, given its great potential due to its higher speed and simplicity of application compared to currently widely used numerical models, there is a need to clarify its possible limitations, in particular related to some initial assumptions made in deriving the model equations.

Methodology

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 [6]:

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

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

Where:

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

k_0 - 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 - intensity factor of decrease in contacts of infected patients with persons who potentially can get infected by means of quarantine and other preventive measures.

v - population vaccination rate (1/day)

α - is the coefficient of vaccine effectiveness.

In the tradition of mathematical modelling in epidemiology, this model will hereafter be referred to as ASILV for short. This name underlines the main features of this model, namely:

A is an analytical model by which an analytical solution of the system of equations can be obtained,

S is the part of the population that is not yet infected, but which could become infected through contact with infected individuals,

I is the part of the population that has already been infected at time t,

L is the part of the population that is protected from infection by lockdown measures at the time in question, but which could potentially also become infected later if the lockdown conditions change,

V is the part of the population that is protected from infection by vaccination, the effectiveness of which α may vary.

Equation (1), defines the change in the number of persons potentially susceptible to the virus under conditions of lockdown and mass vaccination of the population. The denominator in the last summand of equation (1) takes into account that as the proportion of the vaccinated population $\alpha v * t$ increases, the degree of impact of vaccination on the declining epidemic increases. The coefficient of effectiveness α depends on both the type of vaccine and the number of vaccination dose (first or second). We will assume that the maximum vaccination rate will not exceed $(\alpha v * t)_{\max} \leq 0.8$, i.e. that with an 80% vaccination rate the epidemic cannot develop. This is a natural limitation of the proposed model. However, we have to take into account that some part of the population has already had the disease either explicitly or asymptotically by the time mass vaccination begins.

The solution to equation (1) is as follows:

$$S = S_0 * e^{-\lambda t} * (1 - \alpha v * t) \quad (3)$$

After substituting (3) into (2), solving the resulting equation, transformations and moving to a relative number of infections, we obtain the basic calculation equations.

For the period from outbreak to mass vaccination t_v that is

for $t \leq t_v$, when $\alpha v = 0$, the solution of equation (2) has the form:

$$i = i_0 + \frac{100}{N} * \exp \left[\frac{k}{\lambda} (1 - e^{-\lambda t}) \right] \quad (4)$$

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 :

$$K = 0,355 - 0,035 * \ln \left(\frac{1}{N} * 10^6 \right) \quad (5)$$

The K coefficient also depends on the transmissibility of the virus strain responsible for the epidemic spread during the period under

consideration. The value of the first summand in (5) was obtained for the first and second waves of the virus epidemic. For further virus strains, we assume a higher value of 0.37. In the case where the spread of infection is associated with several virus strains, the calculated dependence will be written as follows:

$$i = i_0 + \frac{100}{N} \sigma * \sum_1^n \exp \left[\frac{k_i}{\lambda} (1 - e^{-\lambda * (t - t_{i_0})}) \right] \quad (6),$$

where:

i - is the sequence number of the strain of virus affecting the intensity of the epidemic over time $t - t_{i-1}$,

K_i - the transmission rate coefficient of the new virus strain and the time of the epidemic wave associated with the new coronavirus strain

t_{i_0} - the start time of the new epidemic wave associated with the new coronavirus strain.

σ - Heaviside symbol $\sigma = 1$ when $t \geq t_i$ $\sigma = 0$ when $t < t_i$

Dependence (6) is obtained under the assumption that the two or more virus species exist independently of each other.

Under conditions of mass vaccination when $t \geq t_v$, that is, when $\alpha v > 0$:

$$i = i_0 + (i_v - i_0) * \exp \frac{k}{\lambda} \left[\left(1 - \frac{\alpha v}{\lambda} \right) * e^{-\lambda t_v} - \left(1 - \frac{\alpha v}{\lambda} - \alpha v (t - t_v) \right) * e^{-\lambda t} \right] \quad (7),$$

where $i = i_v$ at $t = t_v$

The calculations are performed first by (4) or (6) and then by (7) for the time period during which vaccination is carried out. The same equation (7) is used to calculate the spread of the epidemic under the condition of an abrupt change in vaccination rate, which was typical of many European countries, in particular Germany, for example. The model equations presented were originally given in [7].

In [7], an attempt is made to relate the model coefficient λ to the effectiveness of the lockdown condition. Let us make some specification of the relationship between this coefficient and the parameter L characterizing the level of reduction in the rate of growth of the epidemic due to lockdown

$$L = i_L / i$$

Where i_L and i are the intensity of the epidemic growth under lockdown and without lockdown, respectively. For example, if the application of lockdown reduces the maximum number of infected residents by half, then the coefficient $L = 1/2 = 0.5$. Using dependences (4) and (5) for time $t \rightarrow \infty$ we find the relation between the coefficient λ and the parameter L . The graph of this dependence is shown in Figure. 1

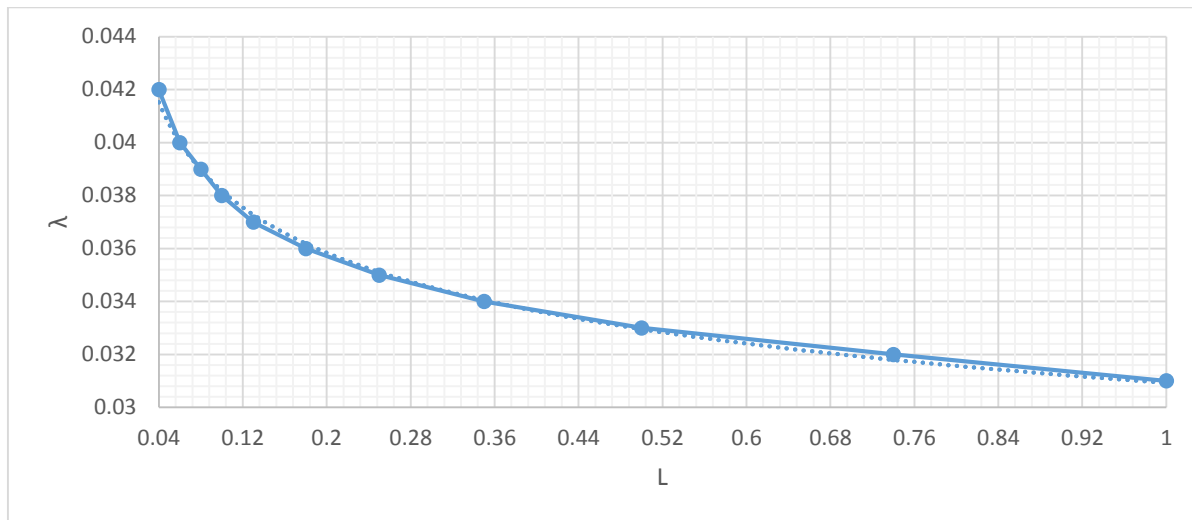


Figure 1: Dependence of the model coefficient λ on the effectiveness of the lockdown L .

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 of Europe, the coefficient $\lambda = 0.034\text{-}0.035$ 1/day, 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 1 can be approximated by the formula

$$\lambda = 0,0309*(L)^{-0,091} \quad (8)$$

Figure (1) also shows the approximation curve (8) as dashed (the correlation coefficient between the approximation curve and the calculated curve is 0.9984)

The relationship between the empirical model coefficients λ and k and the lockdown conditions, population vaccination rate, population size and strain type allow the ASILV model equations to be used not only for analysis of the current epidemic but also for operational forecasting of COVID-19 disease development.

Results

To further investigate the effectiveness of the proposed ASILV model, we use it to analyse the course of the epidemic in New York.

Calculations for the first and the beginning of the second waves of the New York epidemic using the proposed model are given in [4].

Calculations for the first wave were performed with coefficient $\lambda = 0.0345$ 1/day. The second coefficient in the calculated dependence (4) was determined by formula (5), and for New York it turned out to be $K = 0.43$ 1/day. From the graph in Fig. 1 or formula (8), we estimate that, at this coefficient λ , the growth of the epidemic in its initial stage was slowed by

the application of a lockdown by a factor of about 3.5. It should, however, be noted that the lockdown was introduced in the city with a considerable delay, on only 63 days from the start of the epidemic. At that point in time, the number of infections detected (even with low testing coverage) was already reaching around 1% of the city's population. The weekly increase in the number of infected persons in the city at that point in time exceeded 37,000, i.e. more than 5,000 people per day. Positive results from the introduction of the lockdown could not really be observed until day 77, when the rate of spread of the epidemic began to decrease. For this very early phase of the initial wave of the epidemic in New York, the corresponding coefficient $\lambda = 0.033$ 1/day was found, i.e. the epidemic slowed down by a factor of about 2. Calculations using equation (4) with a coefficient $\lambda = 0.033$ 1/day indicate that in such a situation the maximum number of infections in the city would have reached 6% of the city's population. In reality, during the first wave of the virus, the relative number of infections did not exceed 3% of the city's population [8].

A new surge of infections was recorded in most countries in mid- and late-September 2020 when a new 'wave' of the virus began to spread strongly. Analysis of the statistical data [9] revealed that the start of the new infection in New York occurred around 18 September of the previous year. This date was taken as zero for calculations of the development of the so-called "second" and subsequent waves of the epidemic. In the calculation period between its start and 4 June 2021, the date of writing, a total of more than 260 days, new virus strains emerged, lockdown and vaccination conditions changed and all these had to be taken into account when using the ASILV model to calculate the spread of the epidemic in New York. Key statistics related to the COVID-19 epidemic in New York City, used later in this paper, can be found in [9] and on the official city government website [10].

Results of epidemic spread calculations and observational data for the entire time period from the beginning of the second wave are shown in Figure 2.

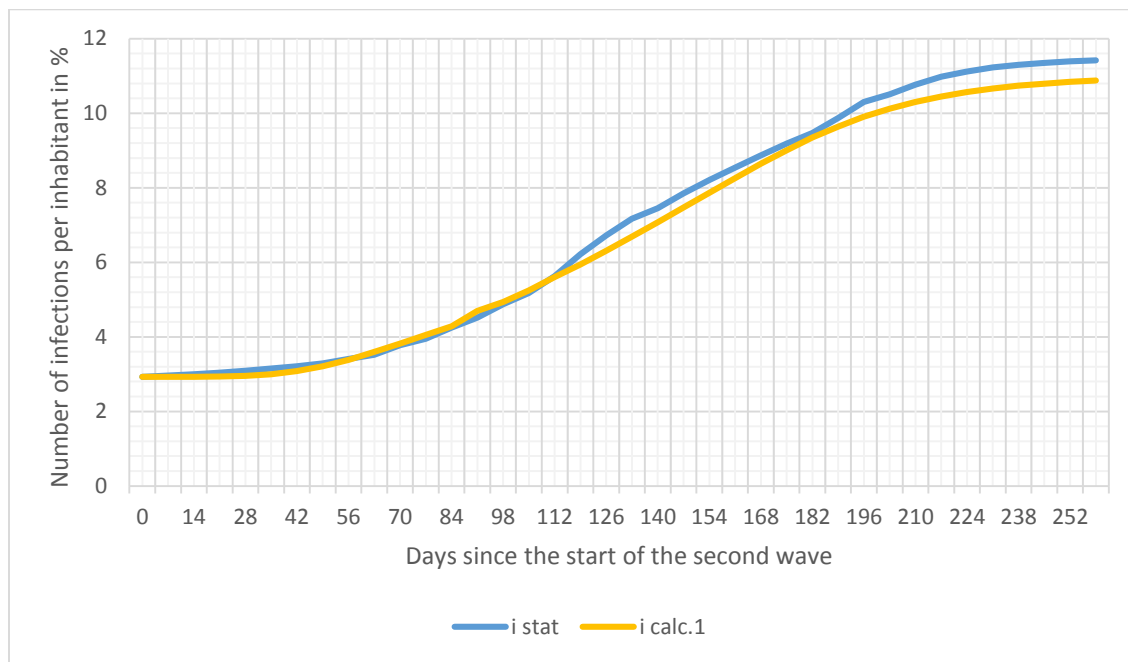


Figure 2: Development of the second and subsequent waves of the epidemic in NYC.

The calculations were performed with time interval of 1 week (from Friday until Friday of next week). In the first phase of the second wave, the virus transmission rate was assumed to be the same as for the first "wave", i.e. the value of the coefficient $K = 0.43$ 1/day was kept unchanged. As for the coefficient taking into account lockdown conditions, it was assumed to be $\lambda = 0.035$ 1/day. This value is the most typical for large European cities under standard lockdown conditions.

In general, the calculated curve at the start of the second wave satisfactorily describes the actual spread of the epidemic in the city. However, around day 60 of the outbreak (or around 15 November), according to [11], the first signs of introduction of the new virus strain into the city, identified as variant B.1.526, appeared. The main virus species determining the development of the epidemic at this period was the so-called "British" strain of B 1.1.7. The spread of this new wave of the epidemic was calculated using equation (6) with constant coefficient $\lambda = 0.035$ 1/day and a slightly increased coefficient $K = 0.45$ 1/day (allowing for increased transmission of these strains of the virus).

At the end of December and at the beginning of January, due to the Christmas holidays and the New Year, the lockdown conditions were relaxed. This has been taken into account by decreasing the coefficient λ for a short period from December 18, 2020 to January 8, 2021 (from 91 to 112 days) to a minimum value of $\lambda = 0.032$ 1/day. The same value for λ was adopted by [7] in the analysis of epidemic change for the same time period in Berlin.

From mid-January, immediately after the holidays, there is a sharp increase in the intensity of the epidemic, which was taken into account in the calculations by the introduction of a new wave of increasing infection. Simultaneously, mass vaccination of the population begins in mid-January, for the period from mid-January to early June 2021 (from 112 to 252 days after the start of the second wave of the epidemic) using equation

(7). The effective vaccination rate was calculated as averaged over the whole vaccination period:

$$\alpha v = \alpha_1 v_1 + \alpha_2 v_2 \quad (9)$$

Vaccination rates for each vaccine dose v_1 and v_2 were calculated based on the data given in [10] as the ratio of the percentage of vaccinated population to the total period of mass vaccination of the population. The BionTech-Pfizer and Moderna efficacy ratios for the first and full doses of vaccination were taken to be $\alpha_1 = 0.7$, $\alpha_2 = 0.92$ respectively [12]. By June 1 this year, over 49% were fully vaccinated, with only the first dose vaccinated about 10% of the city population. The vaccination period is about 170 days, hence the average effective vaccination rate αv is about 0.003 1/day. Model coefficients were assumed to be $\lambda = 0.035$ 1/day, $K = 0.45$ 1/day. For the final period starting January 15, 2021, the estimated spread curve shown in Fig. 2 also differs slightly from the one based on statistical data. A more detailed analysis of the data shows, however, that by late March or early April 2021 a slight increase in the intensity of the epidemic can be observed. Vaccination helps to compensate for these changes, which is why we did not need to analyse these features in our work.

Discussion

The calculation results of the proposed ASILV model agree satisfactorily with the statistical data for both the first wave of the epidemic and the subsequent waves (Figure.2). The correlation coefficient between the calculated and statistical data for the second and subsequent waves is $R = 0.9991$.

Using the standard EXCELL software, it is also possible to quickly establish an incidence rate of 7 days based on the calculated model, one of the main characteristics determining the growth of an epidemic, accepted in many countries as one of the main criteria for determining the possibility of mitigating a lockdown.

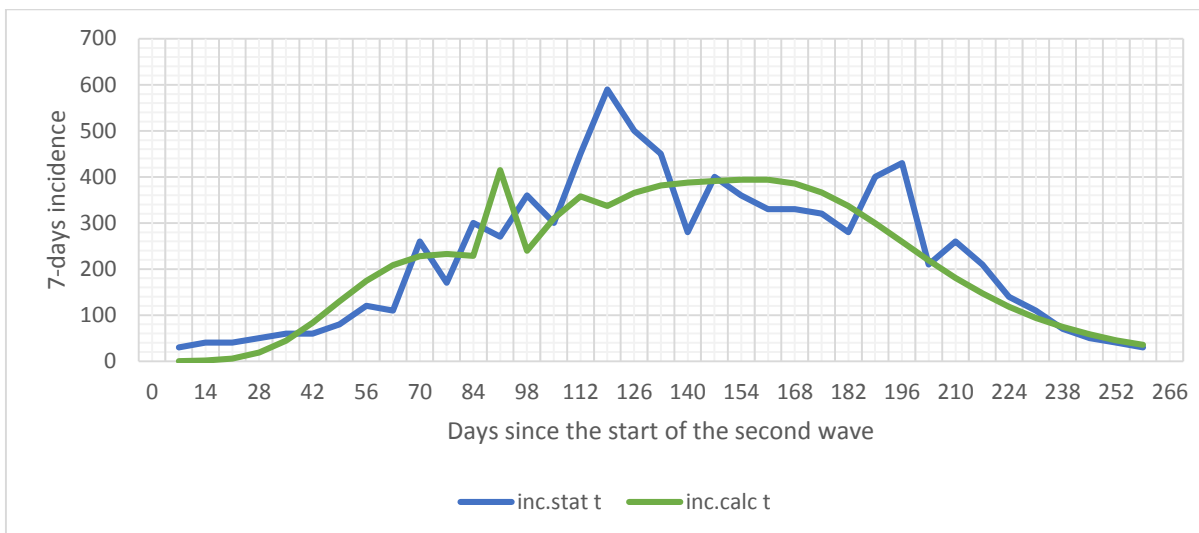


Figure 3: Incidence of epidemic growth over a seven-day period (inc.stat.- observed data , inc.calc. - calculated data)

Figure. 3 shows a comparison of the estimated and observed seven-day epidemic incidence for the second and subsequent epidemic waves (per 100,000 people). In general, the calculated values of the seven-day incidence do not differ significantly from those obtained from measurements; however, at two points in time, the deviations in both curves are striking, with growth rates from about 10 January outpacing the calculated values and reaching peak values of about 600 infected persons. In comparison, the lockdown rule in Germany can be partially relaxed when the incidence rate is kept below 25 for a prolonged period of time. A second peak in the incidence value was observed in early April, but it was neutralised to values of around 400 by vaccination. Of particular interest is the sharp rise in the epidemic after the end of the Christmas and New Year's holidays. The same sharp increase was observed in most European countries; it can be assumed that a significant weakening of the lockdown conditions during the festive period could trigger a new wave of the epidemic. That is, the weakening of the lockdown was the root cause of the new wave in the following period of time. The increase in infections may have triggered a spike in the new wave after B1.1.7 (according to the new alpha virus classification) was introduced into the USA in January. According to virologists, the virus has continued to be the most widespread strain in New York for many

months. Analysing the causes of the new waves of the epidemic is now a major challenge which will make it possible to improve the response to the epidemic.

However, mass vaccination of the population, as both observations and calculations show, can prevent an epidemic surge even when new strains of the virus emerge. It was this property of vaccination that made it possible to quickly quell the outbreak at the beginning of April, not only in New York but everywhere else where there was sufficient vaccination intensity to do so. At present we are not aware of any studies that have established this minimum intensity of vaccination to prevent new waves of the epidemic. Let us consider determining the value of this minimum vaccination intensity using the situation in New York City as an example. To do this, we perform model calculations of the spread of the epidemic at different values of the vaccination intensity. The start of vaccination and lockdown conditions are assumed to be the same as previously used in our calculations, i.e. for New York conditions. Compared to previous calculations, only the vaccination intensity is changed in the model calculations.

Figure. 4 shows the results of this model calculation.

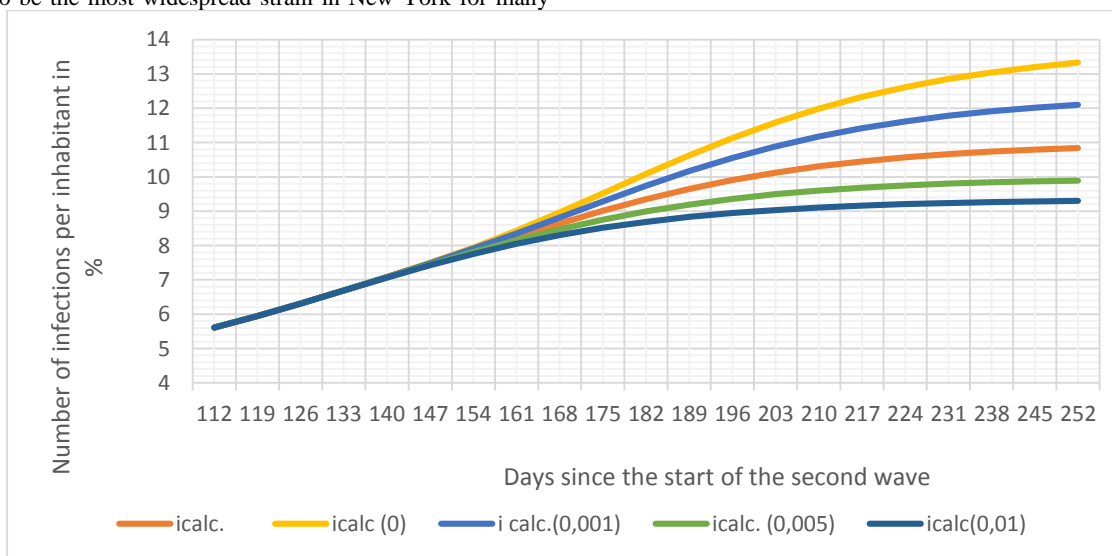


Figure 4: Effect of vaccination intensity on the spread of the epidemic

In this figure, the corresponding values of vaccination intensities are shown in brackets. The curve for which no intensity is given corresponds to the conditions of the above calculation, i.e. $\alpha v = 0.003$ 1/day. The upper

curve was calculated assuming no vaccination. As might be expected, with increasing vaccination intensity, the maximum number of infected persons decreases and the duration of the epidemic decreases.

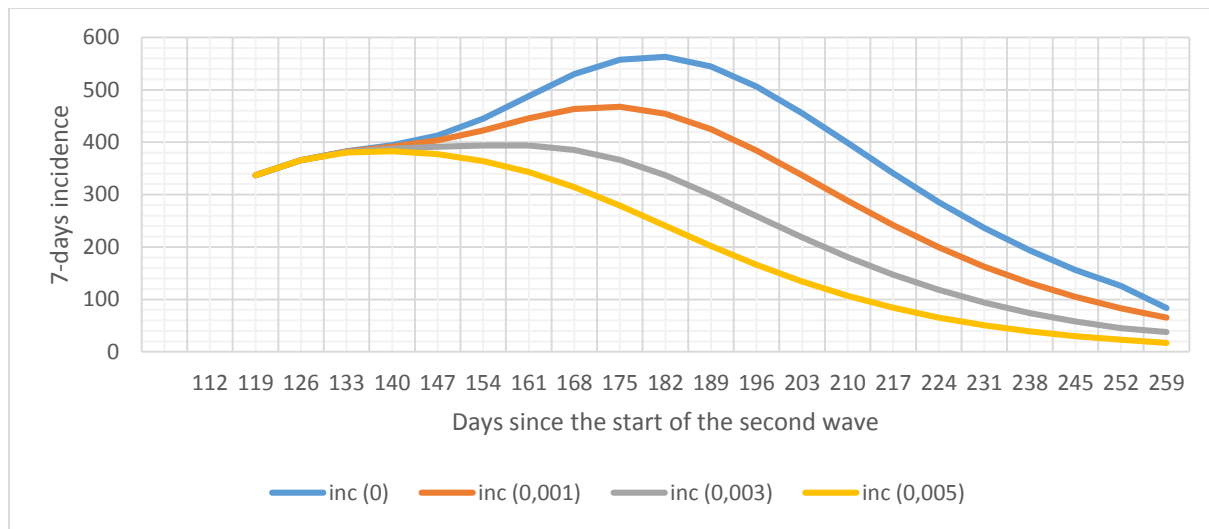


Figure 5: Effect of vaccination intensity on incidence magnitude

The effect of vaccination intensity on the spread of the epidemic can be identified more clearly by considering changes in the value of the incidence. Incidence calculations (per 100,000 inhabitants) for different vaccination intensities are presented in Figure 5.

This figure shows that without vaccination and with low vaccination intensities, the epidemic continues to develop for some time. With $\alpha v = 0.003$ 1/day and above, the magnitude of the incidence decreases immediately at the start of the new epidemic wave. With increasing vaccination time, the effect increases, so that for this or a higher value of vaccination intensity, it can be considered unlikely that an epidemic will develop with the emergence of a new strain of the virus. It is estimated, therefore, that $\alpha v \geq 0.003$ 1/day would be the minimum intensity at which an epidemic in New York City could be excluded.

An analysis of incidence data for the city of Berlin [7] provides indirect support for the assumption of a threshold minimum vaccination intensity. Although vaccination in this city had begun in mid-January, there was a steep rise in the epidemic in mid-March this year associated with the emergence and development of the "British" strain of the virus. The vaccination intensity for the period from January to April did not exceed αv of about 0.0019 1/day. It was only when the vaccination intensity in the city increased sharply to 0.0055 1/day, i.e. from mid-April onwards, that it was possible to reverse the trend of the epidemic.

Given that the lockdown conditions in New York are fairly typical for most European cities and countries ($\lambda = 0.035$ 1/day), one can take the value of the vaccination intensity obtained as the minimum αv value for the average European area. The problem of choosing this minimum value, however, needs further study and clarification.

Conclusions

1. The proposed analytical model adequately describes the development of the epidemic in New York under various lockdown conditions and under mass vaccination of the population. As in previous papers, the

control calculations are in good agreement with the observational data at all stages of the epidemic growth.

2. The incidence estimates for a seven-day period using the proposed model were in good agreement with observations, both for time periods when only the lockdown was observed and when mass vaccination was additionally administered.

3. Model simulations of epidemic spread with different vaccination rates and holding other conditions constant, allowed us to assess the impact of vaccination rate on the epidemic's development.

4. Analysis of the seven-day incidence curves at different vaccination rates gave a preliminary conclusion that when $\alpha v \geq 0.003$ 1/day, the emergence of new virus strains did not cause an increase in the epidemic.

5. Further development of the model should be performed in order to clarify the regularities of the formation of new virus strains and their influence on the development of the epidemic, including mass vaccination of the population.

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