International Journal of Religion
2024
Volume: 5| Number 6 | pp. 50 – 62
ISSN: 2633-352X (Print) | ISSN: 2633-3538 (Online)
ijor.co.uk
DOI: https://doi.org/10.61707/kpwcy578

Solving a Predictive Model of Infectious Disease in Epidemiology and Epizootiology by Assigning Scores to Individuals

Jozef Kremeň[1], Peter Korim[2], Alena Nagyová[3], Oľga Dvorská[4], Daniela Takáčová[5], Mária Vargová[6], Anna Ondrejková[7], Marián Prokeš[8] and František Zigo[9]

Abstract

Every mathematical solution to a problem corresponds to a model that more or less corresponds to reality. One of the many possibilities is the solution to epidemiological problems (processes) using the susceptible-infected-removed (SIR) model. Like any model, this model also has its advantages and benefits that allow us to close such a complicated reality as the origin and course of the epidemic into simple differential equations (DE) and through the model's parameters modify it and compare it with reality. This is possible only in the case of a comprehensive understanding of reality in all its breadth and depth. This study focuses on another solution to the epidemiological or epizootiological processes based on the newly designed simulation, which assigns individuals a certain score in time intervals. In this case, the score from the simulation has a normal probability distribution with certain parameters N (μ, σ) in given time intervals. This score level also divides the given individuals into certain groups based on the given (selected) critical level. This determines whether individuals are with manifestations of the disease or without signs of the disease. In the study, the epidemic curve (EC) simulation results could be used to solve the current pandemic caused by COVID-19. It can be stated that the newly proposed model could be more suitable because it permanently confirms the agreement of experimental and expected frequencies.

Keywords: *Covid-19, Epidemiology, Epizootiology, Simulation, Modeling*

INTRODUCTION

The emergence and spread of infectious diseases have been observed and studied for many years. At present, with the spread of COVID-19, this topic may resonate much more intensely than ever before. The ability to predict how a stated disease is reported provides researchers with the opportunity to evaluate vaccination options, individual isolation, or other effective measures that have a significant impact on its further spread and mortality levels. Mathematical modeling of these processes is a tool that allows us to study the mechanisms and conditions of origin and give rise to conditions of their spread with the possibility of predicting and evaluating the strategy of their control [1]. A mathematical model is an imaginary microworld consisting of entities behaving according to precisely specified rules. The specific language of mathematics allows us to formulate

[1] Department of General Competencies, University of Veterinary Medicine and Pharmacy, Komenského 73, 041 81 Košice, Slovakia. Email: jozef.kremen@uvlf.sk (J.K.); olga.dvorska@uvlf.sk (O.D.)

[2] Department of Public Veterinary Medicine and Animal Welfare, University of Veterinary Medicine and Pharmacy, Komenského 73, 041 81 Košice, Slovakia. Email: peter.korim@uvlf.sk (P.K)

[3] Department of Public Veterinary Medicine and Animal Welfare, University of Veterinary Medicine and Pharmacy, Komenského 73, 041 81 Košice, Slovakia. Email: alena.nagyova@uvlf.sk (A.N

[4] Department of General Competencies, University of Veterinary Medicine and Pharmacy, Komenského 73, 041 81 Košice, Slovakia. Email: olga.dvorska@uvlf.sk (O.D.)

[5] Department of Public Veterinary Medicine and Animal Welfare, University of Veterinary Medicine and Pharmacy, Komenského 73, 041 81 Košice, Slovakia. Email: daniela.takacova@uvlf.sk (D.T)

[6] Department of Public Veterinary Medicine and Animal Welfare, University of Veterinary Medicine and Pharmacy, Komenského 73, 041 81 Košice, Slovakia. Email: maria.vargova@uvlf.sk (M.V.)

[7] Department of Epizootiology, Parasitology and Protection of One Health, University of Veterinary Medicine and Pharmacy, Komenského 73, 041 81 Košice, Slovakia. Email: anna.ondrejkova@uvlf.sk (A.O.)

[8] Department of Epizootiology, Parasitology and Protection of One Health, University of Veterinary Medicine and Pharmacy, Komenského 73, 041 81 Košice, Slovakia. Email: marian.prokes@uvlf.sk (M.P.)

[9] Department of Nutrition and Animal Husbandry, University of Veterinary Medicine and Pharmacy, Komenského 73, 041 81, Košice, Slovakia. Email: frantisek.zigo@uvlf.sk (F.Z.)

these rules concisely and unambiguously and helps to state clearly our assumptions, of course, with certain limitations. After creating a mathematical model, mathematical analysis, often combined with computer simulation, allows us to investigate the overall behavior of the model, thus supporting the significance of our assumptions. Such a mathematical expression of reality makes it possible to predict the future within the model and study its behavior due to changes in its parameters [1,2]. The mathematical expression of infectious disease development in a population characterizes the transmission of a pathogen from a host to susceptible individuals based on the mode of transmission of the infection. Part of this process includes the incubation period, which is the time between infection and the onset of clinical symptoms, disease duration, and acquiring immunity, either naturally or by vaccination. In describing such a model, the output may include information on the number of individuals potentially at risk during the epidemic, the duration, and the peak of emerging diseases, allowing an epidemic curve (EC) to be generated, which provides information on the number of sick individuals during the epidemic at some point in time [2]. The creation of virtual reality (mathematical model) begins the next part of the work associated with the comparison, that is, comparing to what extent the model meets the real-life requirements. Furthermore, it is necessary to define the conditions under which the given abstraction offers relevant results, either by adaptation of the model, if it allows this, or by changing and tuning the parameters of this mathematical model [3]. Generally, there are two types of epidemiological models: stochastic and deterministic models. A stochastic model is a tool for estimating the probability distribution of potential outcomes by allowing for random variables at one or more inputs over time [4-6]. It is used when there is a fluctuation in model parameters or when observing small populations. In the case of large populations, the deterministic or compartmental susceptible-infected-removed (SIR) model can be used. In this case, individuals in the population are divided into subgroups of compartments that represent certain conditions in the epidemiological process [7-9]. Movements from one class to another are expressed as derivatives, so the mathematical model is formulated by differential equations (DE). For example, for a certain disease, it is necessary to divide the population into subgroups, including those who are susceptible to the disease, those who are ill, and those who are cured or immunized. These subgroups are called compartments [10]. The solution to the course of the disease as an interaction between immunity and the disease itself is based on the assignment of a certain score. This study aims to develop a new simulation method to solve the epidemiological or epizootiological process.

METHODOLOGY

Basic Description of The Simulation Model

This principle is explained in general, and then the following examples of diseases are discussed; disease from a point source of infection, disease from a continuous source of infection, and disease from a discontinuous source of infection. It should be noted that these types of diseases used in this work were taken from the available epidemiological resources [11] to create a suitable simulation model - The Web contains information on the course and evaluation of EC representing certain types of diseases.

General Description of the Procedure

The description of this solution to the prediction of infectious diseases is presented in Excel (*see EXCEL file ECPSI, spreadsheet K*). Initially, each individual is defined, whether in the sample or in a subpopulation, by the initial immunity value from 0 to 100, which represents the initial condition or state of these individuals in the sample in terms of overall immunity (this range can be adjusted with respect to the problem, i.e., 0–1000 and 0–10,000). The initial immunity level (values of the initial immunity score) can be varied to represent the condition of a given individual in a population, that is, to express his behavior in terms of weakening or strengthening his immunity. In our example, a total of 100 individuals were generated, with a score between 40 and 100 points. Furthermore, it is possible to extend the solution and perform the stratification of the population, that is, to assign a different range of a given score to a certain group, which represents the fact that, in a different part of the population, there are individuals with different immunity levels. This state is expressed at the point of time t_0. The infectious disease represents the height of a certain negative score of as given distribution, in this case normal, which decreases the score of the respective infectious individuals at certain time points (t_1–t_{20}). In this case, a combination of several distributions is also possible, representing attacks by

several diseases, but limits would have to be set, either randomly or firmly, to determine the manifestation of the disease. Additionally, it is possible to change the size of the immune score based on the time or time jumps (points). The effect of the disease (its rate of spread in the body) in individuals could be due to the steepness of the normal distribution represented by the infection; for example, in the normal distribution, by changing the standard deviation (SD, σ), we determine the change in its steepness and the more intensive decrease in the score from individuals. Furthermore, it is necessary to maintain a balance between the scores assigned to the individuals, which represent immunity, and the scores representing infection, or their combination, and the setting of limits for determining the manifestation of the disease. The response to the onset of the disease is an increase in immunity, which is expressed by the height of the positive score of the respective distribution, in this case also normal, only with a shifted center to higher time points. It is possible to achieve a greater or lesser change in the score values at times t_1–t_{20} and thus change the course of the disease or change the overall model of the disease by changing the average of these two distributions and their SD. Essentially, it is the sum of the initial score (individual's condition), which is positive, the immunity score, which is also positive, and the infection score, which is a negative component. The last two components are shown over time by different distributions. The result is the final immunity score, which is used to assess and classify individuals into the appropriate compartments (the disease has manifested or not and resulted in death or not). Finally, the results can be represented in a graph, where the x coordinate represents the time and the y coordinate represents the number of individuals in each compartment based on the specified boundaries. The usual imaging is by means of a histogram of individuals with clinical symptoms, the so-called EC. Based on the above-mentioned description, a simple model was created in an Excel file. Figure 1 shows the generated 100 individuals with a random immunity score from 40 to 100 (see Supplementary Information-2, RANDBETWEEN, ECCSI, Excel). In the case of scores from 40 to 60, we assume a lower initial level of immunity to the disease. In this case, the maximum score is 100 points. Next, the number of time points is created; it is 20 in our case. Figure 1 shows the given fact, where a score from 40 to 100 in initial point t_0 (column C) is assigned to 100 individuals, and the timeline is divided into 20 points (columns D, E, F, etc.).

C4 f_x =RANDBETWEEN(B3;B2)

	A	B	C	D	E	F	G	H	I
1			Time						
2	Initial score	100							
3	of immunity	40	t_0	t_1	t2	t3	t4	t5	t6
4	Individuals	1	92	87	80	68	54	39	31
5		2	78	73	66	54	40	25	17
6		3	45	40	33	21	7	-8	-16
7		4	42	37	30	18	4	-11	-19
8		5	100	95	88	76	62	47	39
9		6	97	92	85	73	59	44	36
10		7	88	83	76	64	50	35	27
11		8	74	69	62	50	36	21	13
12		9	81	76	69	57	43	28	20
13		10	65	60	53	41	27	12	4

Figure 1 | Initial simulation conditions

Note: Image simulation source

The method of generating a disease is shown in Figure 2. In this case, the normal distribution N (4; 2.25) (marked in red) and the subsequent activation of the immune system with a time delay is used, which is also the normal distribution N (8; 4) (marked in blue). The position of individual distributions can be modified by changing either the mean (we determine in which time period the maximum disease or immunity will occur) or SD, where we change the rate (increase) of the disease or immunity. The sum of these two distributions (frequencies), in the case of the disease with the sign "minus," we get the result (marked in green), in which we simply calculate inappropriate time points, with a generated initial score (40–100) for each individual.

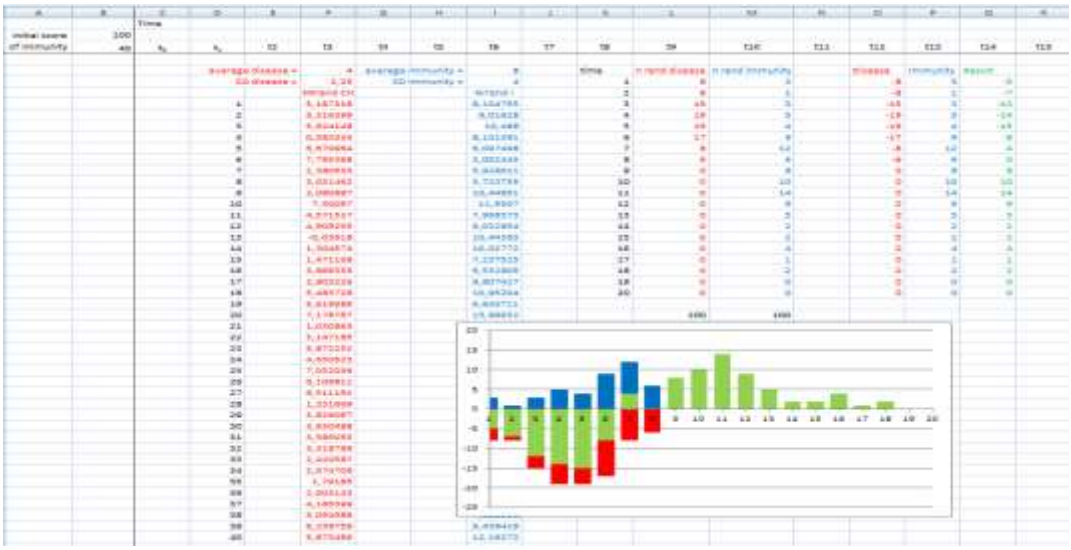

Figure 2 | Representation of the sum of given scores

Note: Image simulation source

The number of individuals and the score they achieved at each time point is shown in Figure 3. In this case, 36 subjects at time t_6 achieved a score higher than 10 (the threshold we selected to identify the clinical manifestations of the subjects); that is, approximately 36% of individuals are without clinical signs if a value of 10 is the limit for clinical manifestations. It is possible to change the given limit and thus tune up the given model for the required clinical manifestations. Since we have 100 individuals, 64 individuals are with the manifestations of the disease. Figure 3 also shows the course of the EC for a given fact, that is, the number of individuals with a score less than ten at the given time points (at time t_6, there are 64 individuals). The numerical values in the figures are not so important because after pressing the F9 or Enter key, a new simulation is performed, and the graphic outputs (frequency curves) are more important.

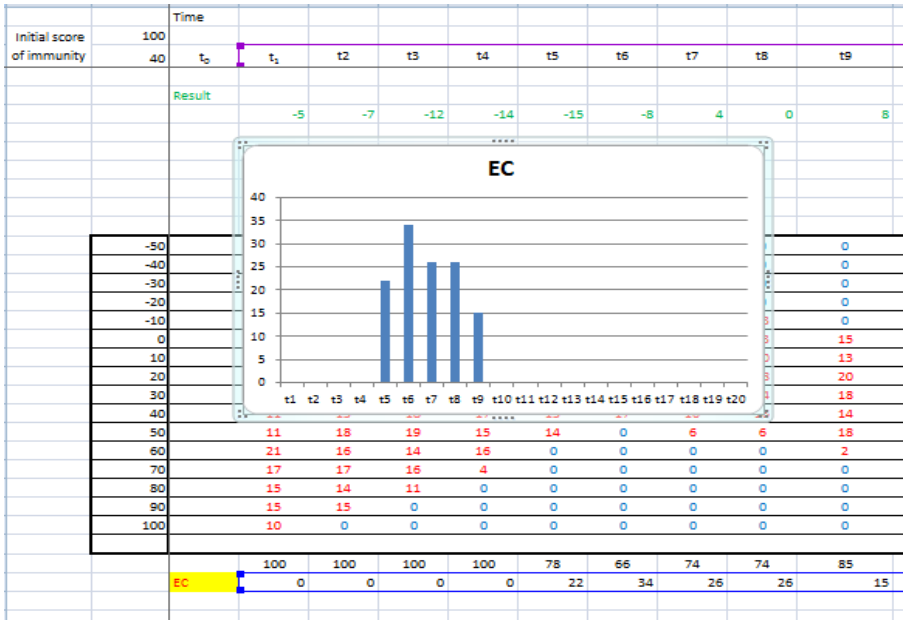

Figure 3 | Representation of the EC

Note: Image simulation source

The EC is represented in chronological time points; the *x*-axis (t_0–t_{20}) is represented by line C3: W3 and the *y*-

axis of individual scores are represented by line D108: W108. The method of calculating individual scores can also be expressed mathematically, essentially by solving the matrix R that is explained as follows:

$$\begin{bmatrix} R_{1,t_0} & R_{1,t_1} & R_{1,t_2} & \dots & R_{1,t_l} \\ R_{2,t_0} & R_{2,t_1} & R_{2,t_2} & \dots & R_{2,t_l} \\ R_{3,t_0} & R_{3,t_1} & R_{3,t_2} & \dots & R_{3,t_l} \\ \dots & \dots & \dots & R_{i,t_j} & \dots \\ R_{k,t_0} & R_{k,t_1} & R_{k,t_2} & \dots & R_{k,t_l} \end{bmatrix} = \begin{bmatrix} R_{1,t_{j-1}} \\ R_{2,t_{j-1}} \\ R_{3,t_{j-1}} \\ \dots \\ R_{k,t_{j-1}} \end{bmatrix} + \begin{bmatrix} 0 & n_{1,t_1} & n_{1,t_2} & \dots & n_{1,t_l} \\ 0 & n_{2,t_1} & n_{2,t_2} & \dots & n_{2,t_l} \\ 0 & n_{3,t_1} & n_{3,t_2} & \dots & n_{3,t_l} \\ 0 & \dots & \dots & n_{i,t_j} & \dots \\ 0 & n_{k,t_1} & n_{k,t_2} & \dots & n_{k,t_j} \end{bmatrix} + \begin{bmatrix} 0 & m_{1,t_1} & m_{1,t_2} & \dots & m_{1,t_l} \\ 0 & m_{2,t_1} & m_{2,t_2} & \dots & m_{2,t_l} \\ 0 & m_{3,t_1} & m_{3,t_2} & \dots & m_{3,t_l} \\ 0 & \dots & \dots & m_{i,t_j} & \dots \\ 0 & m_{k,t_1} & m_{k,t_2} & \dots & m_{k,t_l} \end{bmatrix}.$$

Calculating score matrix R, even individuals in t_j point is expressed mathematically by matrix notation, where the resulting score matrix (on the left side of the equation) is obtained as the sum of the column vector (R_t at time t) of the matrix R and vectors that represent both the disease and the subsequent increase in immunity (n; m). Thus, the next column of the matrix R is calculated from the previous column of the matrix R and the corresponding columns in the disease and immunity matrices. This is explained in detail as follows:

i = 1, 2, 3, ... k represents the number of individuals; in our case, k = 100.

j = 1, 2, 3, ... l represents the time distribution; in our case, l = 20.

At time t_0, we will generate Ri based on the estimation of the subpopulation state in terms of immunity, which represents the initial column vector of the score (in the matrix R, the first column), in our case randomly assigned scores in intervals of 40–100. We will calculate the following value: $R_{1,t1} = R_{1,t0} + n_{1,t1} + m_{1,t1}$ (score of the first individual at time t_1). The next value is equal to $R_{1,t2} = R_{1,t1} + n_{1,t2} + m_{1,t2}$; that is, we will calculate the next value from the previous value (score of the first individual at time t_2). When calculating, the following score must be calculated from the previous one. The values of $n_{i,tj}$ and $m_{i,tj}$ are the frequencies of the normal distribution $N_D(\mu_i; \sigma^2_{i,tj})$ (D denoting disease) and $N_I(\mu_{i,tj}; \sigma^2_{i,tj})$ (I denoting immunity), where the mean value for the disease is earlier than that for immunity. That is, the increase in the immune score is due to the action of the pathogen on the organism (in this model case). Additionally, it is possible to generate these n and m frequencies for each individual separately or together for a particular subpopulation. By the position of the mean value, in this case the peak of the disease, it is possible to determine when an individual becomes infected over time; of course, in the case of generating frequencies, for each individual separately. After calculating the scores, the number of individuals with a score lower than the selected level is expressed in the histogram, and we get a graphical presentation of the condition of individuals with clinical signs (in our case individuals with a score less than 10; the sum in line D132: W132) (Figure 3).

RESULTS AND ANALYSIS

Model Analysis - Calculation of a Suitable Model for the EC from a Point Source of Infection

In this part of the paper, attention was focused on creating a suitable model of the disease from a point source of infection using the score, which was generally explained in the previous section. The effort will be to get as close as possible to the numbers of ill individuals of a given type of disease, as shown in Figure 4, with theoretical frequencies that will be generated by the above-mentioned procedure. Generally, we aim to achieve the greatest possible agreement between the experimental and theoretical numbers of a given disease course in certain time intervals.

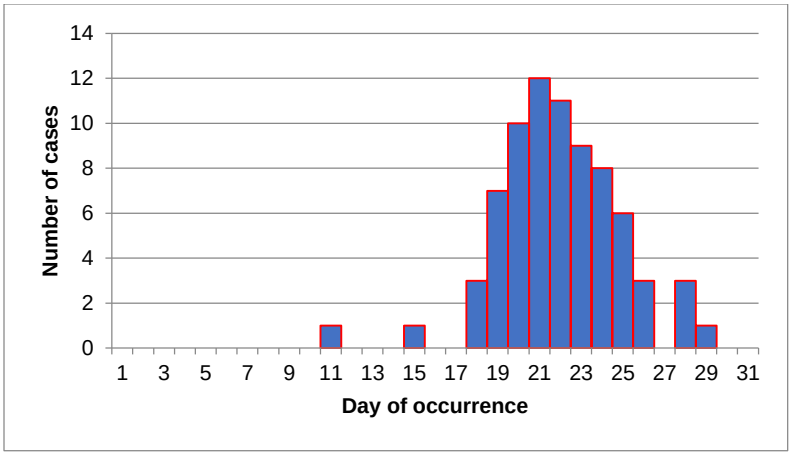

Figure 4 | Illustration of the EC

Note. Image of the EC from the Web

However, tuning this model is quite demanding and complicated because it is necessary to change 400 parameters for 100 individuals, that is, their mean values and SD of the disease and immunity and the initial values of the score or the range of this score. A suitable model was found only by an intuitive change of these parameters. The score values are entered via the RAND function. This means after pressing the F9 key in *Supplementary Information-1, Excel file ECPSI spreadsheet MS*, we get new values; that is, a new simulation and a new recalculation of the values will be performed. The total calculation sheet (calculation of the matrix R) is shown in Figure 5 – It showed the ordinal numbers of individuals are marked in red. In this case, their number is 100. The initial score representing the basic level of immunity is from 45 to 80 points. The number of time points is from t_0 t_0 t_{20}.

Initial score	80 45	Time t_0	t_1	t_2	t_3
Individuals	1	71	26	56	
	2	59	13	44	
	3	67	67	67	
	4	53	53	53	
	5	76	76	76	
	6	53	53	53	
	7	61	61	61	
	8	69	69	69	
	9	73	73	73	
	10	80	80	80	
	11	53	53	53	
	12	64	64	64	
	13	67	67	67	
	14	76	76	76	

Figure 5 | Total score calculation

Note. Image simulation source

A significant difference in the calculation of the course of immunity and disease compared to the common procedure is that the corresponding calculation of the incidence of immunity and disease for each individual was calculated separately in individual sheets of the Excel file, where their parameters were changed: both the position of immunity and disease peak and their SD. One hundred sheets were generated with different alternatively with identical parameters because we have one hundred individuals. Each sheet represents the course of the disease for each individual separately. Figure 6 documents the result of the sum of the level of immunity and disease for individual No. 1.

C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q
	t1	t2	t3	t4	t5	t6	t7	t8	t9	t10	t11	t12	t13	t14
Result	-43	28	13	2	0	0	0	0	0	0	0	0	0	0
								n rand	n rand					
average disease =		0	average immunity =		1		t	disease	immunity		Disease	Immunity	Result	
SD disease =		0,45	SD immunity =		1		1	100	57		-100	57	-43	
		NRrand CH			Nrrand I		2	0	28		0	28	28	
1		0,4083			1,691589		3	0	13		0	13	13	
2		0,732408			-0,10119		4	0	2		0	2	2	
3		0,788134			0,258635		5	0	0		0	0	0	
4		-0,06468			0,609353		6	0	0		0	0	0	
5		0,647512			1,515712		7	0	0		0	0	0	
6		0,796164			1,44033		8	0	0		0	0	0	
7		-0,74572			-0,75706		9	0	0		0	0	0	
8		0,578443			3,06756		10	0	0		0	0	0	
9		0,450866			0,175073		11	0	0		0	0	0	
10		-0,09839			2,10394		12	0	0		0	0	0	
11		-0,63311			1,13345		13	0	0		0	0	0	
12		-0,37125			-0,33174		14	0	0		0	0	0	
13		-0,54404			1,606534		15	0	0		0	0	0	
14		0,192712			1,656248		16	0	0		0	0	0	
15		-0,5543			-0,20402		17	0	0		0	0	0	

Figure 6 | Calculation of the final score for individual No. 1

Note. Image simulation source

The calculation of the final score (marked in green) for individual No. 1 is shown in Figure 6 Red shows the disease score in the normal distribution with a mean at the point zero and SD = 0.45. Blue shows the course of the immunity score at individual time points. This is the normal probability distribution with the mean at point 1 and SD = 1. The final result is in row 2 and starts from column D; in this case, the final score at time t_1 is -45 points. This value depends on equation No. 8 added to the score at time t_0, that is, to initial immunity. The course of the resulting scores for all 100 individuals is shown in Figure 7, which is a graphical representation of the table in Figure 5.

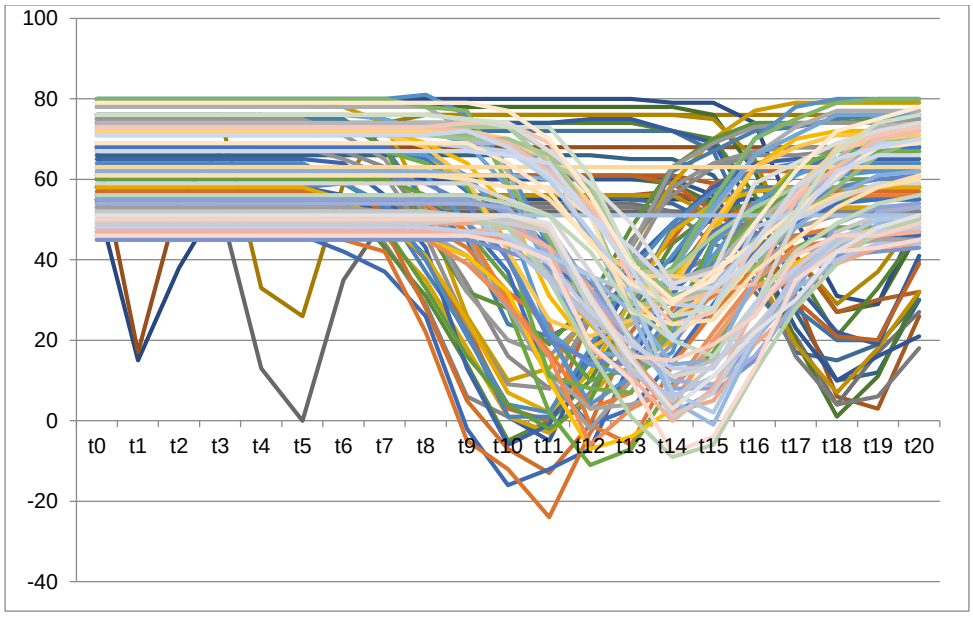

Figure 7 | Score graph for 100 individuals

Note. Image simulation source

Figure 8 shows the state of the sum of individuals according to a certain score, which in our case is in column B111: B141 and in the time intervals t_0–t_{20}. The expected numbers of the EC are obtained as the sum of

individuals who have a score level in the matrix R less than 5 (chosen for this case).

A217

	A	B	C	D	E	F
1	**Initial**	80	Time			
2	**score**	45	t_0	t_1	t_2	t_3
216						
217		-50	0	0	0	0
218		-45	0	0	0	0
219		-40	0	0	0	0
220		-35	0	0	0	0
221		-30	0	0	0	0
222		-25	0	0	0	0
223		-20	0	0	0	0
224		-15	0	0	0	0
225		-10	0	0	0	0
226		-5	0	0	0	0
227		0	0	0	0	0
228		5	0	1	0	0
229		10	0	0	0	0
230		15	0	1	0	0

Figure 8 | Number of individuals by score

Note. Image simulation source

Figure 9 shows the calculation of individuals for whom the calculated score level is less than 5. The numbers of the diseases at times t_0–t_{20} are in the line "Result."

C254 =SUM(C229:C247)

	A	B	C	D	E	F	G	H
1	**Initial**	80	Time					
2	**score**	45	t_0	t_1	t_2	t_3	t_4	t_5
251								
252								
253								
254			100	100	100	100	100	99
255		Result	0	0	0	0	0	1
256								
257								
258								

Figure 9 | Calculation of expected model frequencies

Note. Image simulation source

In this paper, the consensus of the suitability of the achieved model with the EC frequencies was calculated using the Kolmogorov–Smirnov (K–S) test. As in the case of simulating the model frequencies, the individual critical values and the value of the testing criterion are recalculated in this case. Critical values are calculated for significance levels $\alpha = 0.05$ and $\alpha = 0.01$. Figure 10 shows the calculation of the testing criterion and the critical values for a given particular simulation.

Test Kolmogorova - Smirnova							
n	75	64				D_2	0,08729
						$D_{2;0,05}$	0,231433
						$D_{2;0,01}$	0,27738

Figure 10 | Calculation of the test criterion as well as the critical values of the K–S test

Note. Image simulation source

Figure 11 It shows the experimental frequencies of the disease and the expected frequencies, which represent a theoretical model. This figure shows to what extent the empirical frequencies correspond to the given model, that is, expected frequencies in a given simulation.

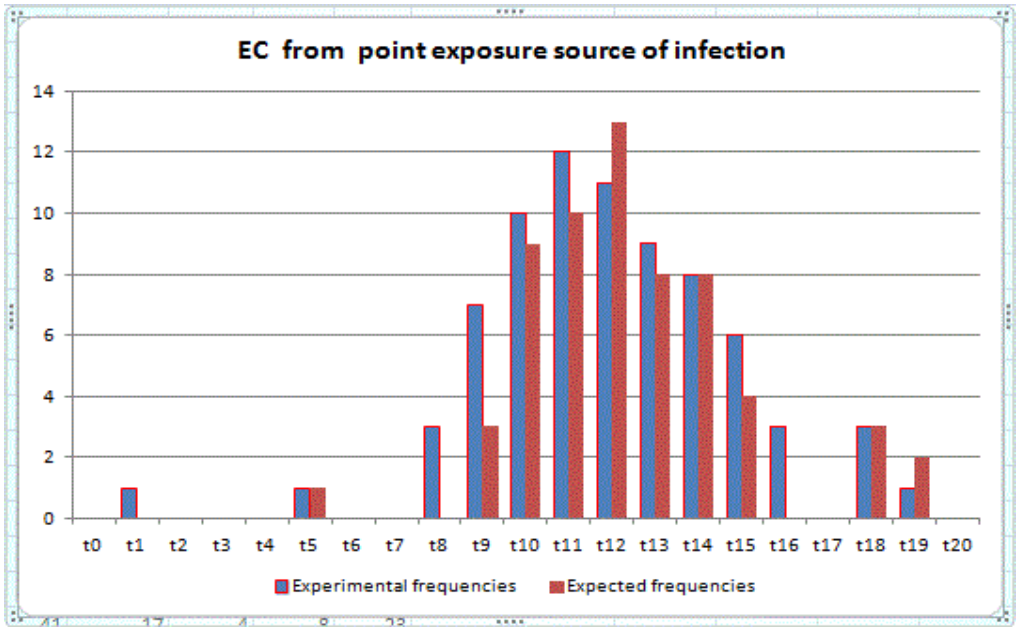

Figure 11 | Comparison of expected and experimental frequencies

Note. Image simulation source

Calculation of a Suitable Model for the EC From a Continuous Exposure Source of Infection

In this case, (Supplementary Information-2, EXCEL file ECCSI, spreadsheet MS) as in the previous one, it is a matter of achieving an agreement between the experimental frequencies, which are given by the fact (disease) and expected frequencies, calculated using the given model (Figure 12).

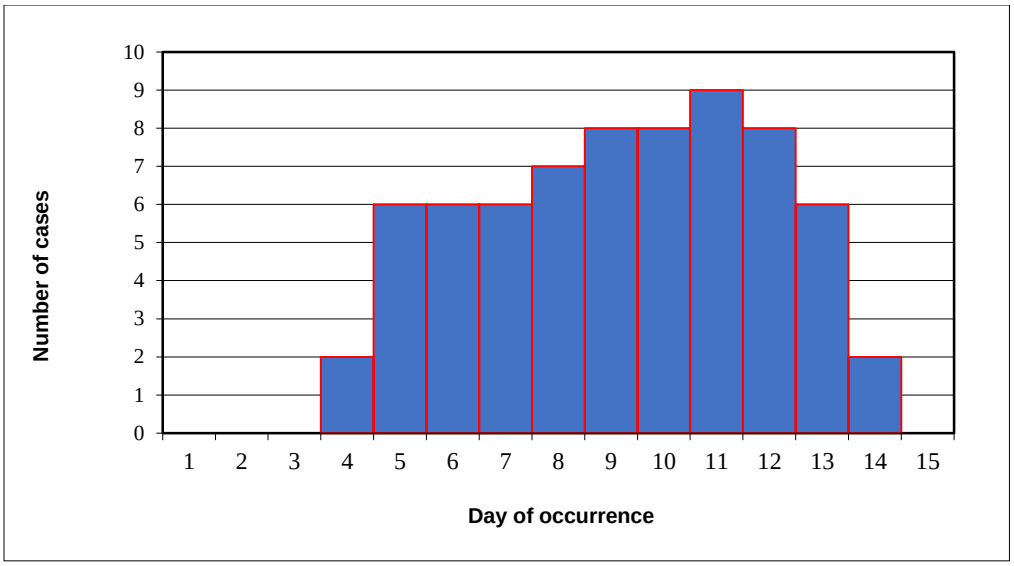

Figure 12 | EC from a continuous exposure source of infection

Note. Image of the EC from the Web

In this case, for simplicity, the procedure for calculating the expected frequencies is not explained as their calculation is essentially the same as the calculation explained in the previous example; it is just a different type of disease. The state of experimental and expected frequencies is shown in Figure 13.

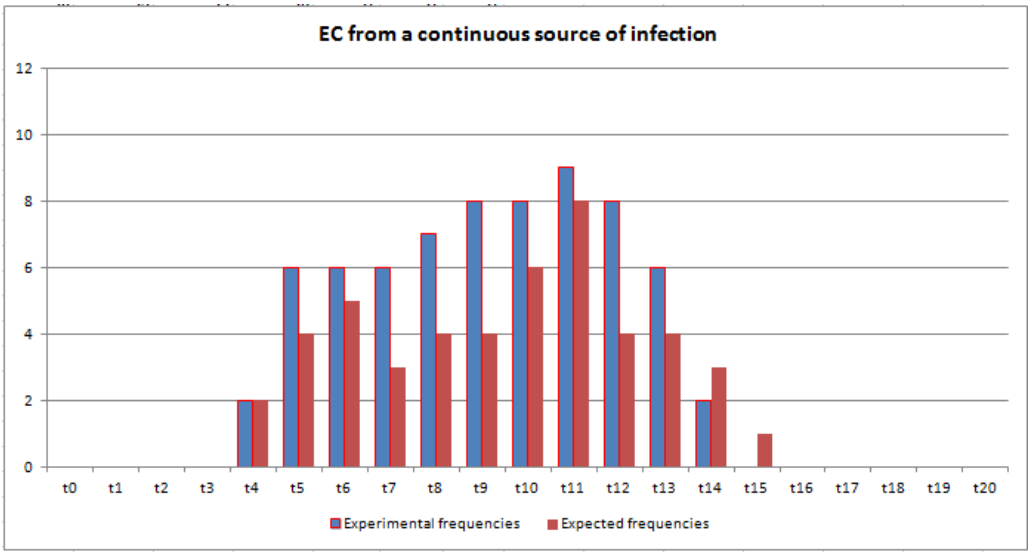

Figure 13 | Model for continuous exp. source of infection

Note. Image simulation source

Calculation of a Suitable Model for the EC From a Discontinuous Source of Infection

In the latter case, (Supplementary Information-3, EXCEL file ECNCSI, spreadsheet MS) we only mention the type of disease (Figure 14) and one of the possible solutions of the concordance of experimental and expected frequencies (Figure 15). In this case was used the time intervals t_0–t_{40}.

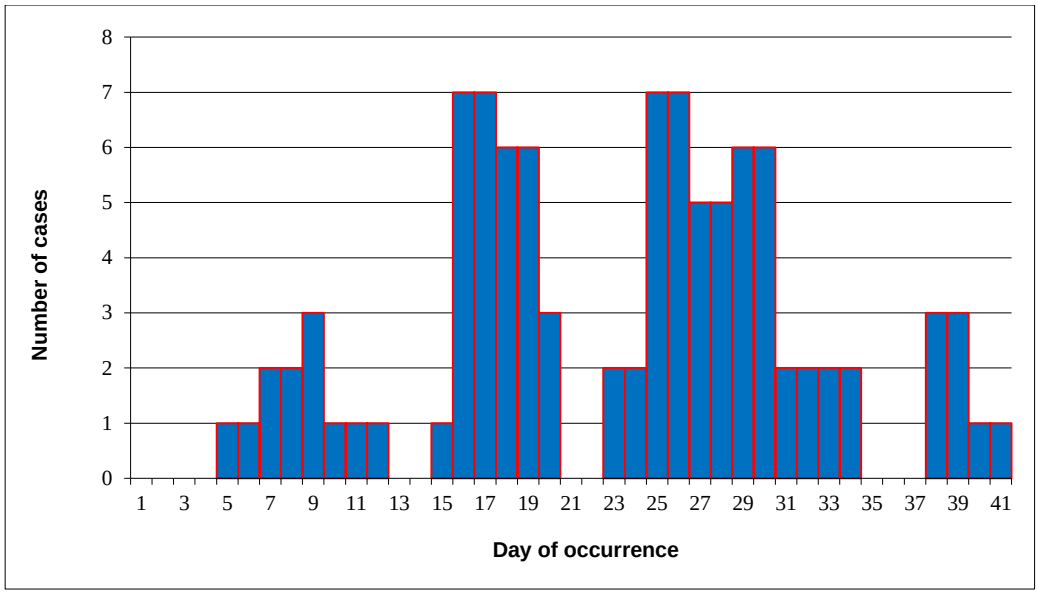

Figure 14 | EC from non-continuous exp. source of infection

Note. Image of the EC from the Web

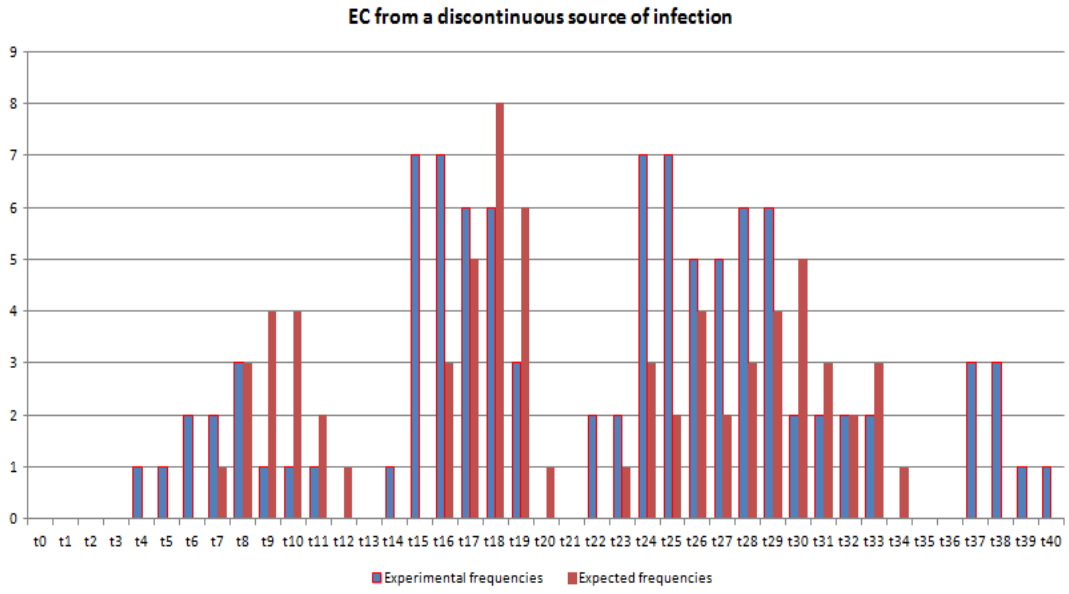

Figure 15 | Model for non-continuous exp. source of infection

Note. Image simulation source

DISCUSSION

Generally, it is necessary to compare this proposal with a reality that should be correctly understood and analyzed in depth when proposing any mathematical expression of reality. The correct number and type of parameters in the model will ensure simple and comfortable work with it in terms of predicting the extent and course of the infection and their suitability, which more or less modify it and bring it closer to reality. The validity of a mathematical model is based on deep knowledge of the origin, transmission, and causative agents of the disease (pathogens) and the whole complex of conditions that both infected and susceptible organisms must meet [1]. This is a constant work based on understanding the real world, choosing a suitable model, or

designing another model, that is, virtual reality [3]. In the above-mentioned cases, it is possible to extend the solution and perform the stratification of the population, that is, to assign a certain range of a given score to a certain group, which represents the fact that, in another part of the population, there are individuals with different levels of immunity. It is also possible to combine multiple distributions, which represent attacks by several diseases, but limits would have to be set, either randomly or firmly, to determine the manifestation of the disease. It is also possible to change the size of the immune score depending on the time or time points. The effect of the disease (its rate of spread in the body) in individuals could be stated by the steepness of the normal distribution represented by the infection; for example, in the normal distribution, by changing the SD (σ), we determine the change in its steepness and the more intensive taking of the score from the individual. Furthermore, it is necessary to maintain a balance between the scores assigned to the individuals, which represent immunity, and the scores representing infection, or their combination and the setting of limits for determining the disease manifestation. In the described examples, the model uses 400 (100 individuals' times 4 parameters for each individual) parameters that can be changed and it is possible to generate additional n that better describe the behavior of the disease through the epidemiological curve. Other types of distribution, such as t-probability distribution, F-probability distribution; Erlang's probability distribution; Chi-square probability distribution can be used to describe the disease and immunity; and much known more. Compared to the SIR model, this idea may be too complicated, but it allows the creation of a more complex model by adding, removing certain parameters, and most importantly, this idea is suitable for creating a simulation.

CONCLUSIONS

From the preliminary results of the calculation for this type of EC, it can be stated that the newly proposed model could be more suitable because it permanently confirms the agreement of experimental and expected frequencies. However, the way to solve this problem is only an introductory idea (theoretical idea), which would need to be tested in real life using a more complicated and complex mathematical and computational apparatus. In this study, the solution, in the case of functioning, could be part of a more complicated model of the disease, which includes more factors. It would be good to check if the given way of thinking works. In conclusion, it is necessary to highlight that this study presents only the basic idea of the solution. For all types of diseases, files of their solutions were created in Excel file, which are attached to the paper. After pressing the F9 key, a new simulation of values arises. The EC from a discontinuous source of infection (Figure 14) is very similar to the curve of COVID-19 at present. If this idea is appropriate, it would be good to develop this approach to dealing with the current pandemic situation.

Abbreviations: Susceptible-Infected-Removed (SIR); Differential Equations (DE); Epidem Curve (EC); standard deviation (SD, σ).

Declarations

Ethics approval and consent to participate: None needed.

Consent for publication: All authors consent to publication.

Availability of data and material: The Supplementary Material for this article can be found a EXCEL files; (Supplementary Information-1, "ECPSI", Supplementary Information-2, "ECCSI" and

Supplementary Information-3, "ECNCSI").

Competing interest: The authors declare no conflict of interest.

Funding: This research was funded by the Ministry of Education, Research, Development and Youth of the Slovak Republic with grant KEGA no. 018UVLF-4/2024.

Author contributions: Conceptualization, JK, and PK; methodology, JK, PK, AN and MP; validation, AO, MP, DT, and FZ. Analysis of results; JK, PK, AN, and FZ; data curation, OD, DT and MV; writing—original draft preparation, FZ, PK, JK; writing—review and editing, supervision, project administration and funding acquisition, FZA and JK. All authors read and approved the final manuscript.

Acknowledgement: none.

REFERENCES

Huppert A, Katriel G. Mathematical modelling and prediction in infectious disease epidemiology. Clin Microbiol Infec. 2013; 19.11: 999-1005. https://doi.org/10.1111/1469-0691.12308.

Katriel G. Epidemics with partial immunity to reinfection. Math Biosci. 2010; 228.2: 153-159. https://doi.org/10.1016/j.mbs.2010.09.003.

Alonso-Quesada S, De la Sen M, Agarwal RP, Ibeas A. An observer-based vaccination control law for an SEIR epidemic model based on feedback linearization techniques for nonlinear systems. Adv Differ Equ. 2012; 2012.1: 1-32. https://doi.org/10.1186/1687-1847-2012-161.

Kalinina EA. The most precise computations using Euler's method in standard floating-point arithmetic applied to modelling of biological systems. Comput Meth Prog Bio. 2013; 111.2: 471-479. https://doi.org/10.1016/j.cmpb.2013.04.001.

Sekiguchi M. Permanence of a discrete SIRS epidemic model with time delays. Appl Math Lett. 2010; 23.10: 1280-1285. https://doi.org/10.1016/j.aml.2010.06.013.

Shang Y. Mixed SI (R) epidemic dynamics in random graphs with general degree distributions. Appl Math Comput. 2013; 219.10: 5042-5048. https://doi.org/10.1016/j.amc.2012.11.026.

Jam, F. A., Rauf, A. S., Husnain, I., Bilal, H. Z., Yasir, A., & Mashood, M. (2014). Identify factors affecting the management of political behavior among bank staff. African Journal of Business Management, 5(23), 9896-9904.

Tien JH, Earn DJ. Multiple transmission pathways and disease dynamics in a waterborne pathogen model. Bull Math Biol. 2010; 72.6: 1506-1533. https://doi.org/10.1007/s11538-010-9507-6.

Liu J, Peng B, Zhang T. Effect of discretization on dynamical behavior of SEIR and SIR models with nonlinear incidence. Appl Math Lett. 2015; 39: 60-66. https://doi.org/10.1016/j.aml.2014.08.012.

Khan, T., Hayat, S., Hayat, H., Ali, S., Khan, S., & Ahmad, M. (2021). Population Estimation of Oncorhynchus Mykiss, Salmo Trutta and Schizothorax Plagastimous in Upper River Swat, Khyber Pakhtunkhwa, Pakistan. Mathews Journal of Veterinary Science, 5(1), 1-3.

Marinov TT, Marinova RS, Omojola J, Jackson M. Inverse problem for coefficient identification in SIR epidemic models. Comput Math Appl. 2014; 67.12: 2218-2227. https://doi.org/10.1016/j.camwa.2014.02.002.

Michelle TM. Focus On Field Epidemiology, North Carolina Center for Public Health Preparedness, The North Carolina Institute for Public Health. 2004. Available from: https://nciph.sph.unc.edu/focus/vol1/issue5/1-5EpiCurves_issue.pdf.

Wayne WL. Descriptive Epidemiology. 2017. Available from: https//sphweb.bumc.bu.edu/otlt/mph-modules/ep/ep713_descriptiveepi/index.html.

Yang Q, Jiang D, Shi N, Ji C. The ergodicity and extinction of stochastically perturbed SIR and SEIR epidemic models with saturated incidence. J Math Anal Appl. 2012; 388.1: 248-271. https://doi.org/10.1016/j.jmaa.2011.11.072.