Mathematical Models of Epidemic Dynamics to Simulate the Distribution of COVID-19

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Abstract

Mathematical modelling of the COVID-19 epidemic is based on system dynamics and SIR models, which are not considered adequate. To overcome the shortcomings of modelling, a non-classical discipline, epidemic dynamics, is proposed. The epidemic should be viewed as an open, self-replicating dynamic system in epidemic dynamics. Epidemic dynamics models are based on a dynamic system model with an extended network of inverse relationship. This non-classical approach allows the tools of non-linear and non-equilibrium dynamics to be used and models of epidemic dynamics to be represented in the form of non-linear and non-stationary differential equations. The solutions of the equations are special COVID-19 distribution functions – functions of the flows and accumulation levels of the infected and the dead. The COVID-19 distribution functions show high accuracy in approximating the statistics, demonstrating the excellent adequacy of these functions in principle. The application of COVID-19 distribution functions makes it possible to quantitatively describe the basic concepts of an epidemic to carry out comparative parametric analysis of the distribution of diseases and predict the development of an epidemic.

Keywords 1

Epidemic dynamics models, approximation, parametric analysis of epidemic distribution functions, epidemic forecasting.

1. Introduction

Mathematical modelling of epidemic processes and the search for new drugs, vaccination and preventive measures contribute to disease control. In addition, quantitative model simulations can provide comparative analysis and predictions of temporal descriptions of key epidemic categories, such as the number of people who fall ill, recover, and die. Therefore, COVID-19 prevalence models are highly demanding to match statistical data and ensure that epidemic mechanisms and underlying conceptual descriptions are adequate.

The SIR model, developed by A. Kermack and W. McKendrick in 1927-1933, is based on a scheme of epidemic transition of essential variables from one category to another: those susceptible (S) become infected (I), then recover (R). The SIR model is represented by a system of coupled first-order differential equations describing the basic concepts' time dependence. Models implementing the concept of epidemic transition have gained wide popularity and development, so the class of SIR models today also contains varieties: SIRS, SEIR, SIS, and MSEIR [1-5].

Experience with SIR models [1-5] has shown poor fit of baseline variable calculations to statistical data and poor accuracy in predicting epidemic processes. An analytical review [5] noted that such models perform poorly in heterogeneous populations, different routes of transmission and the presence of randomization factors. In our opinion, the shortcomings of SIR class models lie in the concept of epidemic transition of basic concepts, which implies the search for new concepts.

A retrospective analysis of approaches to epidemic modelling in [6] shows that a high level of complexity characterized the types of deterministic and stochastic epidemic models developed. In turn,

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they have been poorly linked to the formulation and solution of practical epidemiological problems. Therefore, their use was inefficient. The author [6] reviewed a new methodology for mathematical modelling of epidemics – "EPIDDINAMICS". This methodology is based on the method of scientific analogy in mapping the epidemic process with the process of "transfer" of matter in the equations of mathematical physics. During an epidemic, a complex self-sustaining process of "transferring" a population of a pathogen to a community of susceptible individuals is formed. The epidemic process in this concept is described by a system of non-linear partial derivative equations, very "similar" to the equations of hydrodynamics. However, analogies with hydrodynamics do not sufficiently reveal the content of the self-sustaining process of disease spread.

The nature of the COVID-19 coronavirus distribution statistics shows that the epidemic's dynamics are highly like the logistic functions. Therefore, we note the application of logistic functions to approximate a piece of given statistical information. The articles [7-10] use logistic-type models to describe the spread of COVID-19. In [7], a mathematical model of the spread of the COVID-19 coronavirus epidemic is considered using a simplified logistic model describing the increase in cases. The low accuracy of calculation results obtained in [7-8] can be attributed to the simplified representations of logistic models used for modelling.

Articles [9, 10] discuss the concept of modelling the spread of COVID-19 based on constrained growth functions. In [9], the epidemic's wave structure, represented by a set of elementary epidemic flows shifted along the time axis, is considered. However, the content of the articles does not sufficiently reveal the mechanism of epidemic development, which reduces the accuracy of epidemic forecasts. This issue can be explained by the classical methods of mathematical investigation of epidemic processes. However, a fuller description of the epidemic development mechanism requires modern non-classical methods for studying complex systems involving non-linear and non-equilibrium dynamics.

The article aims to develop mathematical models for the COVID-19 epidemic based on the non-classical approach using the methods and tools of non-linear and non-equilibrium dynamics. It allows for the presentation of epidemic dynamics models in the form of non-linear and non-stationary differential equations and dynamic models of system with an extended network of inverse relationship.

2. Models of Epidemic Dynamics

The reason for the shortcomings of epidemic modelling is the simplification and inadequacy of the mechanistic representations of system dynamics characteristic of classical mathematical modelling methodology. In classical modelling methodology, it is common to represent the object of study using the means of system dynamics [11-13] as epidemic dynamics. At the same time, an epidemic, a progressive spread of infectious disease among people capable of causing an emergency, should be seen as a complex systemic entity.

2.1. Main approaches

Epidemic dynamics is a scientific discipline in the study of disease transmission processes that views the epidemic as a complex open system, as a system dynamic viewed from a non-classical methodology.

The core of epidemic dynamics is its model. Causality in large self-regulating systems reduces to the action of a self-regulation program as a goal that ensures the reproduction of the system [Stepin]. It enables the model of epidemic dynamics to be conceived of as a procedural reproduction system. A model of epidemic dynamics in a non-classical methodology should have these properties:

- Openness means self-regulating systems are always open and exchange energy and substance with the external environment (metabolism), due to which the processes of local order and self-organization occur;
- Providing links with the environment through a network of linear and non-linear inverse relationship;
- Conditions of equilibrium as a state of crisis;
- Non-equilibrium functioning outside of the equilibrium conditions
- Reproduction of the system through positive inverse relationship.

The methods and tools of the theory of non-linear non-equilibrium dynamical systems are used to describe the epidemic dynamics model. The epidemic dynamics model is based on a dynamical system, a mathematical model of an object, process or phenomenon that neglects fluctuations and all other statistical phenomena. A dynamic system is a system with a state. In this approach, a dynamical system describes (in general) the dynamics of some process, namely, the process of a system moving from one state to another. An epidemic dynamics model consists of abstract elements representing some properties of the modelled system. The following elements are distinguished: integrator, adder, and inverse relationship chains, which link the variables – flows and quantities of accumulations.

2.2. Variable patterns in epidemic dynamics

Epidemics are the transmission of viruses from ill people to those who are healthy and susceptible to the disease. Statistics keep track of new cases – the number of people infected per day. An epidemic process is characterized by an increase in the total number of infected people, counted cumulatively at a specific date. Growth is limited by the number of people susceptible to the disease. The epidemic dynamics model derives from a dynamic system model [16], which uses a linear dynamic system [19] with inverse relationship. The main variables of the model:

- $x(t)$ - an independent variable denoting the rate, flow of infected, those number of infected per unit time;
- $X(t)$ - the dependent variable of the level of accumulation of infected (system state), denoting the total number of infected over some time.

A phenomenological inverse relationship coefficient complements the dynamic system model φ. Next model is an isolated design, usually used for linear systems

$$M = \langle x, X, \varphi \rangle,\ X = \int x dt\ ,\ \ x = X'. \tag{1}$$

To use this model for open systems, it is necessary to: increase the number of inverse relationships and relate them to the environment parameters. To extend the modelling capabilities of open systems, we use a deployed scheme in the form of multi-circuit inverse relationship. Such a detailed dynamic model of an open system provides a process description in abstract form and allows the construction of non-linear differential equations.

2.2.1. Deployment of a dynamic model

The principle of dynamic model deployment is to decompose: the total flow of infected x into partial variables and, similarly, the total phenomenological coefficient φ

$$x = \sum x_i,\ \varphi = \sum \varphi_i\ . \tag{2}$$

The complete flow of the infected as distributors of infection consists of elements. Following the decomposition principle (2), we distinguish two groups of elements based on facilitating the distribution of infection. Then the total flow of the infected equals the difference of the incumbent carriers minus the flow of loss of carriers

$$x = x^{\mp} - \tilde{x}, \tag{3}$$

where $x^{\mp}$ - streams of carriers who facilitate the distribution of the infection; $\tilde{x}$ - loss streams of carriers who counteract the distribution of the infection.

2.2.2. The flow of carriers of infection

The carrier flow (3) is also divided into two parts. One part relates to active spreaders and the other to inactive spreaders, those who have died. This approach makes it possible to distinguish between fatal cases and to consider the actual spreaders of infection. The flow of carriers is then equal to the difference of the flow of infected minus the flow of deceased

$$x^{\mp} = x^{+} - x^{-}, \tag{4}$$

where x^+ - is the flow of active carriers, reflecting new cases of infected persons in the process of spreading infection (inflow of infected persons); x^- - the flow of lethal cases (outflow of infected persons).

The flow of fatal cases is represented as a function of current infectious carriers $x^- = f(x^+)$.

2.2.3. Flow patterns

Flow models (4) describe the dependencies of the rate of infection on the number of people infected.

$$x_i = f_i(X), \quad (5)$$

These dependencies reflect the main patterns of epidemic processes. We describe the relationship between flows and the environment using phenomenological coefficients. The relationship with the environment can be linear and non-linear. For linear relationships, the flow (5) is proportional to the number of infected with the inverse relationship coefficient

$$x_j = \varphi_j X. \quad (6)$$

For non-linear relationships, the inverse coefficient is a function of the derivatives of the accumulation level variables

$$x_i = \varphi_i(X)X. \quad (7)$$

2.2.4. Patterns of carrier flows

The pattern of processes here is that the flows of carriers (4) are proportional to the number of spreaders of infection. Carrier flow patterns reflect a monotonic increase in the number of infected. We restrict the group of linear (6) relationships to two types of inverse relationships, positive and negative $\varphi^{\mp} = = \varphi^+ - \varphi^-$

1. The growth of an epidemic is mainly determined by the inflow of infected persons, which is proportional to the number of people spreading the infection

$$x^+(t) = \varphi^+ X(t), \quad (8)$$

where φ^+ is the positive inverse coefficient, a measure of the growth in the number of infected.

It follows from equation (8) that the number of people infected, and consequently the infectious flow, grows exponentially without limit $X(t) = e^{\varphi^+ t}$. This growth can be ensured if there is an unlimited number of susceptible individuals regarded as the source of the infection. However, in practice, the number of susceptible persons is limited, which poses the problem of a limited growth model.

2. The flow of deaths, which reduces the growth of the epidemic, is proportional to the number of people infected

$$x^-(t) = \varphi^- X(t), \quad (9)$$

where φ^- is the growth rate of fatal cases.

The growth rates in (8) and (9) are constant coefficients, so these carrier flow models are linear relationships. The flow of carriers (4) is then equal to the difference between the flow of infected and dead

$$x^{\mp} = (\varphi^+ - \varphi^-)X(t).$$

2.2.5. Carrier loss flows

Limit it to three types of loss and write an expression for the total flow

$$\tilde{x} = \tilde{x}_0 + \tilde{x}_1 + \tilde{x}_2. \quad (10)$$

where $\tilde{x}_i$ loss flows of certain types of carriers.

The pattern of processes here is that loss flows are proportional to the number of spreaders, but this relationship is not linear (7), and the coefficients of proportionality depend on the number of spreaders

$$\tilde{x}_i(t) = \tilde{\varphi}_i(X)X(t). \quad (11)$$

The principle behind this non-linear relationship is that loss rates are proportional to the derivatives of the number of infectious spreaders (7), so growth rates are functions that depend on the phenomenological coefficients

$$\tilde{\varphi}_i(t) = a_i\left(X^i\right)(t). \quad (12)$$

Then the total loss flow equation (10) with (11) and (12) will appear as

$$\tilde{x}(t) = a_0X^2(t) + a_1X(t)X' + a_2X(t)X''. \quad (13)$$

Consider the features of the loss components in the total loss flow equation (13) that describe the dispersal of infectious agents in the environment.

2.2.6. Zero-order loss flow models

Zero-order loss stream is a function proportional to the number (zero-order derivative) of infectious spreaders, where the coefficient of proportionality depends on the number of spreaders $\tilde{\varphi}_0(t) = = a_0X^{(0)}(t)$ (12). The flow expression is then a non-linear relationship of the form

$$\tilde{x}_0(t) = a_0X^2(t). \quad (14)$$

Zero-order flow models (14) solve the problem of limited epidemic growth, which results from the limited source of the infected population and consists of the fact that the growth of the infected population is limited to the number of persons $\hat{X}$ susceptible to the epidemic. This value can be regarded as the source of infection in the environment. The introduction of a source limits the number of infected to $X \le \hat{X}$. When the limit is reached $X = \hat{X}$, the flow value falls by leaps and bounds to zero. Then the expression for the flow of loss takes the form of a discontinuous function

$$\tilde{x}_0(t) = \begin{cases} \tilde{\varphi}_0X(t), & X(t) \le \hat{X}; \\ 0, & X(t) \ge \hat{X}. \end{cases} \quad (15)$$

A gap in the expression for the loss flow is considered a catastrophe, leading to uncertainty in the spread of infection. Given that flow gaps are not observed in practice, it is necessary to introduce a source of infection into the model (14) to exclude a catastrophe (15). To do this, consider the infected source capacity $\bar{a}_0 = \hat{X}T$ and the source action time T. Considering that $\bar{a}_0 = \frac{1}{a_0}$ we rewrite the expression for the flow (14) in the form of

$$\tilde{x}_0(t) = \frac{X^2(t)}{\hat{X}T}. \quad (16)$$

The expression for the loss flow (16) considers the source of the infected, is a continuous function of the number of infected and can be used to describe the limited growth of an epidemic.

2.2.7. First-order loss flow models

According to (12), the growth rate is a function proportional to the first-order derivative of the number of distributors $\tilde{\varphi}_1(t) = a_1X^{(1)}(t)$. It follows that the flow expression is a non-linear relationship of the form

$$\tilde{x}_1(t) = a_1X(t)X^{(1)}(t). \quad (17)$$

The proportionality factor a_1 reflects the resistive properties of the environment, which inhibit the spread of the flow of the infected.

2.2.8. Second-order loss flow models

The second-order loss rate is a function proportional to the second-order derivative of the number of distributors $\tilde{\varphi}_2(t) = a_2X^{(2)}(t)$, then the flow expression is a non-linear relationship of the form

$$\tilde{x}_2(t) = a_2X(t)X^{(2)}(t). \quad (18)$$

The proportional coefficient a_2 can be considered as the elasticity of the environment and reflects the ability to generate oscillations during flow distribution.

2.2.9. Differential equations of epidemic dynamics

Given the expressions for the elements (8), (9) and (16)-(18), we write the differential equation for the total flow of infectious agents

$$x = ((\varphi^+ - \varphi^-) - \left(a_0 X^{(0)} + a_1 X^{(1)} + a_2 X^{(2)}\right))X. \quad (19)$$

Given $x(t) = X(t)$, let us rewrite differential equation (19) for the number of infected in the standard form

$$a_2 X X'' + (1 + a_1 X)X' + \frac{X^2}{\hat{X}T} = (\varphi^+ - \varphi^-)X. \quad (20)$$

Model (20) describes epidemic dynamics as a nonlinear, non-stationary differential equation. This model reflects epidemic dynamics as a process system reproduces stably due to interaction with the environment. Equation (20) has no analytical solutions, and numerical methods are used to solve it. Solutions to equation (20) give the epidemic episode functions:

- The total infectiousness flow functions have a bell-shaped form;
- The functions of the number of infected (infection rate) are S-shaped.

The epidemic dynamics functions have two equilibrium states:

- Initial equilibrium – unstable $x(0) = 0$, $X(0)$ at $t = 0$;
- Final equilibrium – stable, $x(t) \to 0$, $X(t) \to \bar{X}$ at $t \to \infty$.

The epidemic dynamics functions vary over a range bounded by the equilibrium states, and then the epidemic processes in the range are non-equilibrium and irreversible.

Equilibrium of the system means that the growth of infected individuals stops, and the derivatives tend towards zero. We obtain the equilibrium equation from the equation of epidemic dynamics (20) with zero derivatives $X''(t) = 0$, $X'(t) = 0$ and $X(t) \neq 0$

$$\frac{\bar{X}}{\hat{X}T} = \varphi^+ - \varphi^-. \quad (21)$$

The stable equilibrium equation is a parametric equation that relates the parameters of the COVID-19 distribution functions. The epidemic threshold is described by the expression

$$\bar{X} = (\varphi^+ - \varphi^-)\hat{X}T. \quad (22)$$

In many practical cases, it is possible to restrict oneself to first-order epidemic dynamics models, which are derived from (20) at $X''(t) = 0$

$$(1 + a_1 X)X' + \frac{X^2}{\hat{X}T} = (\varphi^+ - \varphi^-)X. \quad (23)$$

In particular cases, equation (23) admits analytical solutions, but numerical finite-difference methods are generally used to solve it. The solution of finite difference equations constructed by (23) leads to discrete COVID-19 distribution functions.

2.2.10. Discrete distribution functions

In the discrete COVID-19 distribution functions, the relations of the variables are described by the discrete $X_{k+1} = X_k + x_{k+1}$, $x_{k+1} = \varphi_k^+ X_k$, where $\varphi_k^+ = (\varphi^+ - \varphi^-) \times \times \frac{1 - \frac{X_k}{\bar{X}}}{1 + a_1 X_k}$ is the equivalent variable for the growth rate of infected persons. Expressions for the discrete function of the infector flow and the number of people infected

$$x_{k+1} = (\varphi^+ - \varphi^-)\frac{1 - \frac{X_k}{\bar{X}}}{1 + a_1 X_k} X_k; \quad X_{k+1} = \left(1 + (\varphi^+ - \varphi^-)\frac{1 - \frac{X_k}{\bar{X}}}{1 + a_1 X_k}\right) X_k; \quad \bar{X} = (\varphi^+ - \varphi^-)\hat{X}T. \quad (24)$$

The discrete COVID-19 distribution functions fit the statistics well, so they are used to model the epidemic. Similarly to (24), the discrete flow and number of deaths functions are

$$y_{k+1} = (\varphi^+ - \varphi^-)\frac{1 - \frac{Y_k}{\bar{Y}}}{1 + a_1 Y_k} Y_k; \quad Y_{k+1} = \left(1 + (\varphi^+ - \varphi^-)\frac{1 - \frac{Y_k}{\bar{Y}}}{1 + a_1 Y_k}\right) Y_k; \quad \bar{Y} = (\varphi^+ - \varphi^-)\hat{Y}T. \quad (25)$$

3. Approximation of Statistical Data Covid-19 Distribution

Discrete COVID-19 distribution functions are used to analyse and predict epidemics. These functions are obtained by approximating statistical data. Two problems can be solved by approximating the statistics:

- The total flow of infected people;
- The function of the number of infected.

Ukraine [16] is chosen as an example to show how the calculated values of COVID-19 distribution functions correspond to statistical data [16].

3.1. Approximations of Data on the Number of Infected

Figure 1and Figure 2 show the correspondence between the calculated functions for the number of infected (24) and deaths (25) and the first wave statistics for April and May 2020.

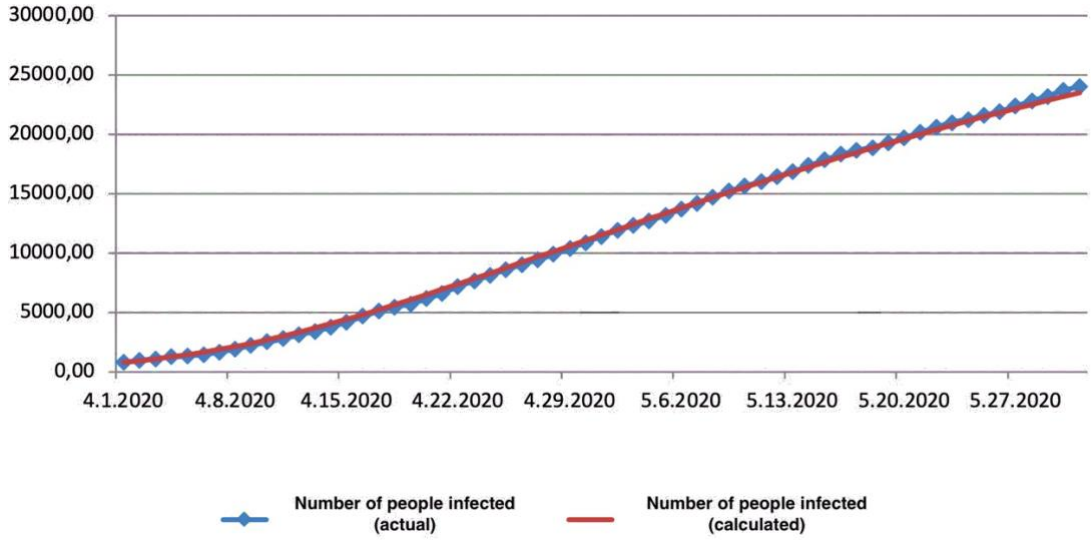

Figure 1: Correspondence of estimated numbers of COVID-19 infected with statistics for Ukraine for April and May 2020

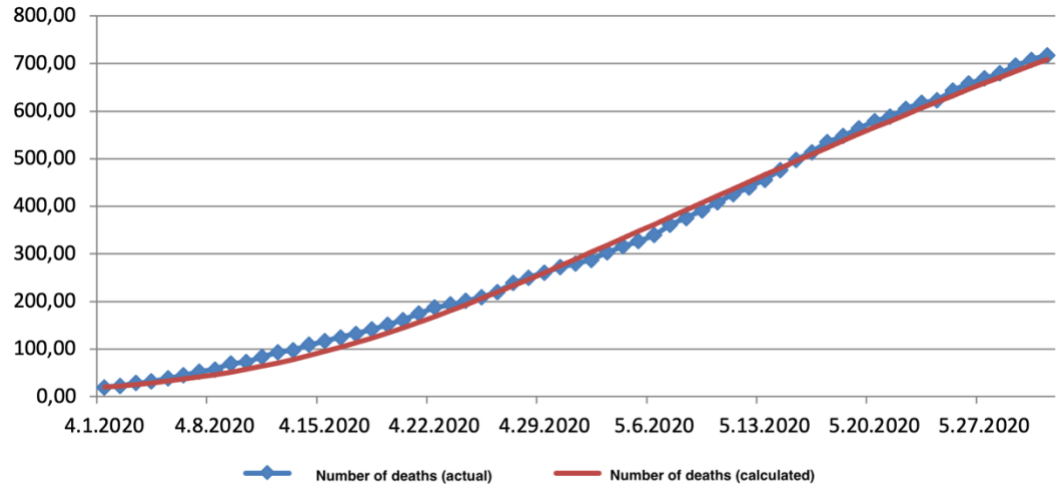

Figure 2: Correspondence between the estimated COVID-19 fatality rates and the statistical data for Ukraine for April and May 2020.

Figure 1 and Figure 2 clearly show a reasonably good agreement between the calculated and actual data, where the MAPE does not exceed 3% and 7% respectively.

3.2. Approximations of the Full Flow of Infected

Approximation of the total flow of infected persons was performed using the example of the second wave of COVID-19 spread in the time interval from 1.04 to 10.09 2020 according to the statistical data given in [17, 18]. The approximating functions have the character of episodes (27), those of completed processes, and the total flow is the sum of episodes. Figure 3 shows the results of the decomposition of the complex flow into 4 episodes, those into 4 elementary f flows.

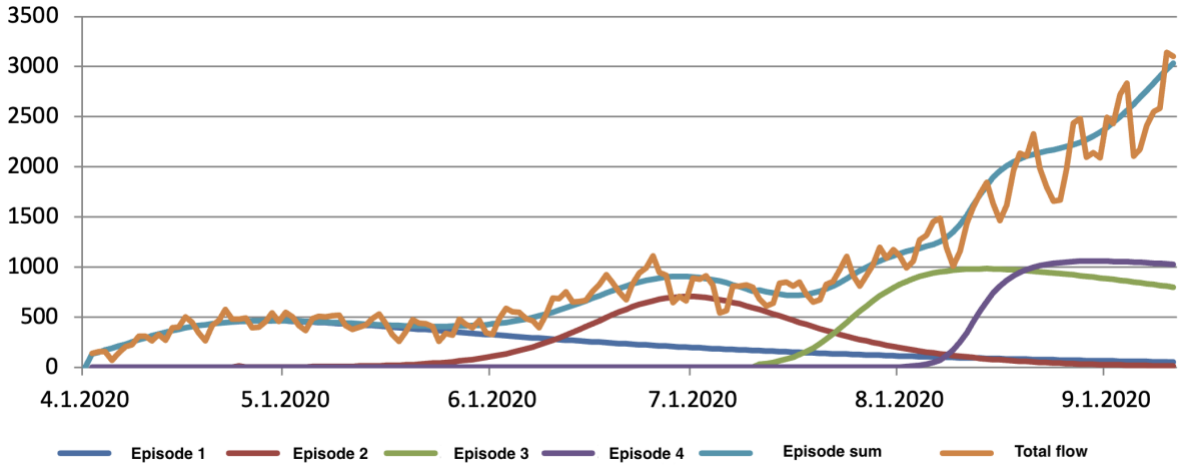

Figure 3: Correlation of estimated COVID-19 infection flows with statistical data for Ukraine for the period April - October 2020

The overall flow contains a sequence of four episodes with increasing peaks, resulting in an increase in the overall epidemic flow.

In general, such methods of epidemic dynamics correspond to the methodology of agent-based modelling, which, compared with classical models, has greater descriptive power but requires a more detailed description of epidemic categories. Agent-based models use a dynamical system representation in which details of descriptions are provided by inverse relationships.

The undoubted advantage of the method is the presentation of epidemiological dynamics models in an analytical form. This approach allows to study mathematical models and analyze the sensitivity of the result to changes in the parameters of differential equations. Also, the application of the proposed mathematical models to the modeling of epidemiological dynamics allows to take into account the nature of the studied processes of infection spread, in particular their wave nature.

4. Conclusions

Research on the history of epidemic modelling shows that the main reason for the shortcomings of mathematical modelling of epidemics (SIR, System Dynamics, “EPIDDINAMICS”) can be considered as the application of the classical methodology characteristic of simple isolated systems. Therefore, there is a problem associated with the transition of the modelling methodology from classical to non-classical positions, within which the epidemic should be considered an open self-replicating dynamic system. This shift is associated with a new scientific approach to the study of disease transmission processes, where epidemics are viewed as system dynamics from a non-classical methodology. This scientific direction is called epidemic dynamics, the core of which is a reproducible procedural system. A model of epidemic dynamics in a non-classical methodology would be open and reflect an exchange of energy and matter with the external environment to enable reproduction in a nonequilibrium functioning mode. The methods and tools of the theory of non-linear nonequilibrium dynamical systems are used to describe the model of epidemic dynamics.

The epidemic dynamics models are based on a dynamic system model with an extended feedback network. This non-classical approach allows epidemic dynamics models are represented as non-linear and non-stationary differential equations. Solutions to the equations - COVID-19 distribution functions

– are used for mathematical modelling and investigation of epidemic processes. Importantly, these functions describe elementary complete epidemic processes - "epidemic episodes". The modelling process begins by approximating epidemic statistics with discrete functions describing two "epidemic episodes" types: flows and accumulations. Next, superpositions of unrelated epidemic episodes shifted in time and described complex processes.

This approach is consistent with agent-based modelling methodology, which has greater descriptive power than classical models but requires a more detailed description of epidemic categories. The application of COVID-19 distribution functions shows high accuracy in approximating statistical data, demonstrating the excellent adequacy of these functions in principle. Applying COVID-19 distribution functions enables quantitative description and analysis of epidemic processes and reliable forecasting. Overall, applying COVID-19 distribution functions can help reduce the harm caused by a pandemic.

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