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Hybrid SIRS model of infection spread

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Abstract. *Purpose* of this work is to build a model of the infection spread in the form of a system of differential equations that takes into account the inertial nature of the transfer of infection between individuals. *Methods.* The paper presents a theoretical and numerical study of the structure of the phase space of the system of ordinary differential equations of the mean field model. *Results.* A modified SIRS model of epidemic spread is constructed in the form of a system of ordinary differential equations of the third order. It differs from standard models by considering the inertial nature of the infection transmission process between individuals of the population, which is realized by introducing a «carrier agent» into the model. The model does not take into account the influence of the disease on the population size, while population density is regarded as a parameter influencing the course of the epidemic. The dynamics of the model shows a good qualitative correspondence with a variety of phenomena observed in the evolution of diseases. *Discussion.* The suggested complication of the standard SIRS model by adding to it an equation for the dynamics of the pathogen of infection presents prospects for its specification via more precise adjustment to specific diseases, as well as taking into account the heterogeneity in the distribution of individuals and the pathogen in space. Further modification of the model can go through complicating the function which defines the probability of infection, generation and inactivation of the pathogen, the influence of climatic factors, as well as by means of transition to spatially distributed systems, for example, networks of probabilistic cellular automata.

Keywords: population dynamics, SIRS model, dynamical systems.

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Introduction

Methods of nonlinear dynamics allow us to study natural phenomena by constructing and analyzing simple (qualitative) mathematical models. Qualitative models, unlike simulation models, do not pretend to predict the detailed behavior of the simulated system under specific conditions. However, they allow us to understand the nature of the observed phenomena, to identify their patterns and internal mechanisms, as well as to determine by what parameters or influences their

characteristics can be changed. One of the areas of application of qualitative models is the study of the spread of epidemics of infectious diseases in biological populations [1–4].

Mathematical modeling of epidemics is one of the sections of population dynamics [5], used both in biology and in other sciences, for example, sociology. Various mathematical methods are used to predict the course of epidemics: time series analysis [6], regression [7] and autoregressive [8] models, grids of cellular automata [9–12], artificial neural networks [13–15] and others.

Classical models of the spread of infections are systems of ordinary differential equations. The most famous of them is the SIRS model, proposed in the 1920s Kermack and McKendrick [16]. In the SIRS model, the population is divided into groups of healthy and susceptible (S— **S**usceptible), infectious (I— **I**nfectious) and recovered (R— **R**ecovered) individuals and systems of equations are constructed that determine the law of change the relative number of individuals in each of the groups, based on the assumption of random and the uniform distribution of individuals in the population. Such a system is called *the mean-field model*. At the same time, the processes of infection of individuals are described like collisions of ideal gas particles in statistical physics.

The basis of this approach is the assumption of Hamer [17] that the rate of spread of the epidemic depends on the frequency of contacts between susceptible and infected individuals. The frequency of contacts is determined by the product of the population densities of susceptible and infected individuals in the population. This approach, despite its obvious simplicity and clarity, does not always adequately describe the real processes of infection, which may be characterized by a certain non-locality and inertia. Inertia of biological processes it can be taken into account by introducing a delay time into the model, that is, by using equations with a delayed argument.

One of the first such approach was applied in 1948 in the work of Hutchison [18]. In the future, delayed equations were used in a variety of works, an overview of which can be found in [19, 20]. However, lag models are systems with an infinite number of degrees of freedom, which makes it difficult to analyze them.

A one more possible approach is the introduction of an additional equation and an additional variable that describe the inertia mechanism. It is this method that is proposed in the work.

In this paper, a modification of the SIRS model is proposed, in which the transmission of infection occurs indirectly, due to interaction with the carrier agent. Viruses, bacteria or parasites can act as such an agent[1]. This approach is especially relevant for predicting the spread of respiratory viral infections, in which the agent causing infection is extremely mobile and relatively long-lived. Therefore, the act of infection can occur far (in time and space) from infected individuals. Such a model is a model of interaction between two systems: a population of individuals and a population of viruses, each of which lives according to its own laws. By individuals we will understand isolated particles whose state changes in a discrete way, and by viruses — an external field that affects individuals and leads to a change in their state, that is, infection.

1. Modified two-component SIRS model of the spread of infectious diseases

Infectious diseases have common features:

- the disease spreads through infection, the source of which is a previously infected individual (I), and the recipient is a susceptible individual (S). As a result, the susceptible individual becomes ill and becomes the source of subsequent infections itself: $S \to I$;
- the ill individual is cured with time, while getting immunity to subsequent infections (R): $I \to R$;

[1] In the future, we will conditionally call all such intermediary agents viruses

- acquired immunity is lost with time, the individual returns to its original state: $R \to S$.

Thus, in the evolution of each individual, we observe a cyclic chain of transformations between a discrete and a finite set of states $S \to I \to R \to S$. Hence the name of this model — *SIRS*.

In the standard *SIRS*-model, the act of infection is described as the result of local contact of individuals S and I: $S + I \to 2I$. However, in practice, infection can occur without direct interaction of individuals. In this paper, we propose exactly such an infection scheme based on «exchange interaction» individuals with viral particles. In this scheme, an infected individual (I) acts as a generator of viruses (V), which then, due to diffusion or mixing, spread through the habitat, infecting susceptible individuals (S): $S \overset{V}{\to} I$. Viruses in this scheme are intermediaries between a ill individual and a susceptible one. Thus, instead of the standard *SIRS*-model, we propose a two-component (individuals + viruses) model: $S \overset{V}{\to} I \to R \to S$.

In the proposed model, the dynamics of viral particles is fundamentally different from the behavior of individuals in the population. Individuals of a population are objects with a discrete set of states $\{S, I, R\}$. Transitions between them are random events and are characterized by their probability values (P_k). Each of the individuals requires a certain habitat. Therefore, the number of individuals in a given area is always limited to some maximum number N. Viral particles can accumulate indefinitely at every point in space. Therefore , their number can take arbitrary positive values. They are also able to move in the process of diffusion. Thus, the interaction of individuals with viruses is some analogy of the interaction of particles with a field. The described transformations that occurred in each elementary cell of space during the time Δt can be represented as the following scheme (Fig. 1):

$$\begin{aligned} I &\to I + \nu V, \\ S + \eta V &\overset{P(\eta)}{\to} I + \eta V, \\ I &\overset{P_2}{\to} R, \\ R &\overset{P_3}{\to} S, \\ V &\to (1 - \mu)\, V, \end{aligned} \tag{1}$$

where the letters above the arrows indicate the probabilities of the corresponding transitions.

We briefly describe the sequence of operations of the scheme (1).

1. An infected individual (I) generates ν of virus particles (V).
2. A susceptible individual (S) becomes infected upon contact with η viruses with a probability of $P(\eta)$.
3. An infected individual (I) recovers with a probability of P_2 and becomes immune (R).
4. An immune individual (R) loses immunity with probability P_3 and returns to the receptive state (S).

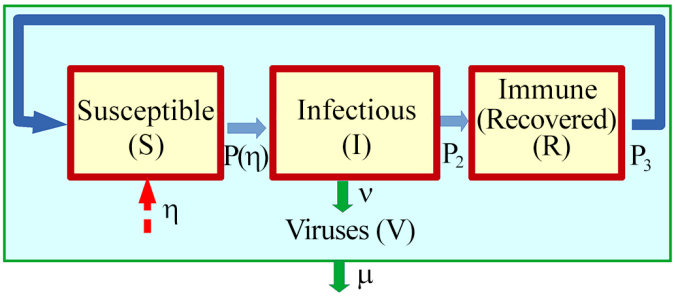

Fig. 1. Diagram of transitions between states of the SIRS+V model: solid lines indicate the direction of transitions; a dashed line indicates the influence of viruses on the transition between states of individuals

5. Inactivation of μ viruses.

All transformations are unidirectional. The order of changing the states of individuals is rigidly deterministic. At the same time, the transformations themselves and the moments of time at which they occur are random. The dynamics of this model is determined by both the parameters and the choice of the function $P(\eta)$.

When considering the spread of the epidemic, the main interest is not the dynamics of individuals, but the change in their number N_k ($k \in \{S, I, R\}$). The total population size of $N_S + N_I + N_R$ is limited in size by some maximum allowable value of N, which we will call the maximum capacity of the population. The value of N is due to some common resource (the amount of food, the area of the territory, etc., etc.), restraining unlimited population growth. The ratio of the total population to the maximum possible is the relative population

$$C = \frac{N_S + N_I + N_R}{N},$$

taking a value from zero to one. Since the total number of particles in the scheme (1) does not change, C is a parameter. The value of the parameter C is an important factor determining the course of infectious processes in the population.

2. The mean-field model

In the mean-field approximation for the scheme (1), it is possible to compose a system of equations governing the change in the number of individuals in each of the states N_k ($k \in \{S, I, R\}$) and the number of viruses N_V for a small interval Δt. We introduce as variables the relative population densities $k = N_k/N$ and, passing to the limit $\Delta t \to 0$, write down a system of ordinary differential equations

$$\begin{aligned} \dot{i} &= P(v)\,(C - i - r) - P_2 i, \\ \dot{r} &= P_2 i - P_3 r, \\ \dot{v} &= \nu i - \mu v \end{aligned} \tag{2}$$

(it is taken into account here that $s + i + r = C$). To determine the type of the function $P(v)$, it is natural to assume that it should monotonically increase with the concentration of viruses from $P(0) = 0$ to $P(\infty) = 1$. As such a function, we choose $P(v) = (1 - (1 - P_1)^v)$, where $P_1 \in [0 : 1[$ — probability of catching «with a single» portion of viruses[2]. Denoting $\alpha = -\ln(1 - P_1)$, you can write the probability function in a more convenient form: $P(v) = 1 - \exp(-\alpha v)$. In the equation (2), you can reduce the number of independent parameters by entering a new variable: $z = \alpha v$, and a new parameter: $\sigma = \alpha\nu$. As a result , we get

$$\begin{aligned} \dot{i} &= (C - i - r)\,(1 - \exp(-z)) - P_2 i, \\ \dot{r} &= P_2 i - P_3 r, \\ \dot{z} &= \sigma i - \mu z. \end{aligned} \tag{3}$$

The equation (3) will be the basis for analyzing the behavior of the system (1). It follows from the conditions of the problem that all variables and parameters (3) are non-negative numbers; in addition, the variables i and r are summarily bounded from above: $i + r \leqslant C \leqslant 1$.

[2]The choice of this dependence is determined by the fact that the probability of not getting infected by v portions of viruses will be Q^v, where Q is— probability to stay healthy after contact with one portion of viruses.

3. Stationary solutions

Let's analyze the stationary solutions of the system (3)in the range of acceptable values of parameters and variables. The limiting trajectories of the system (3) are two equilibrium states: trivial $E_0 = (0, 0, 0)$ and nontrivial

$$E_1 = (i_0, Ai_0, Bi_0), \tag{4}$$

where the notation is used: $A = P_2/P_3$, $B = \sigma/\mu$, and the value i_0 is defined as the root of the transcendental equation

$$i_0 = \frac{1}{1+A}\left(C - \frac{P_2 i_0}{1 - \exp(-Bi_0)}\right). \tag{5}$$

The point E_0 corresponds to the case of complete recovery of the population. Its stability is determined by the roots of the characteristic equation

$$\begin{aligned} \lambda_1 &= -P_3, \\ \lambda_{2,3} &= \frac{-(P_2+\mu) \pm \sqrt{(P_2-\mu)^2 + 4\sigma C}}{2}, \end{aligned}$$

which, by virtue of the non-negativity of all the quantities included in the formula, are real. Depending on the ratio between $d = BC$ and P_2, it can be either a stable node (at $P_2/d > 1$) or a saddle node (at $P_2/d < 1$). Bifurcation condition for E_0

$$P_2 = d \tag{6}$$

simultaneously corresponds to the passage of the point E_1 through the origin, at which it becomes stable. Thus, for any parameter values in the phase space (3) there is only one stable fixed point. For $P_2 > d$, this is the point E_0 while for $P_2 < d$ this is the point E_1. In the first case, the population is not susceptible to infection and any accidental penetration of infection fades in time; in the second case, if there is an initial infection, an epidemic occurs.

It can be seen from the expression (6) that the ratio d/P_2 plays a decisive role in epidemic processes. It is the product of several parameters: $\alpha\nu C\tau_v\tau_2$, where $\tau_v = \mu^{-1}$ — average virus viability time, $\tau_2 = P_2^{-1}$ — the average duration of the disease (the so-called «infection period»). What is the biological meaning of this factor? If the average concentration of viruses generated by one infected individual is $\nu\tau_v$, and the proportion of susceptible individuals is close to 100%, then the value of d determines the average rate of infections produced by one infected individual at the initial stage of the epidemic. Multiply it by the average period of infection τ_2 and we get that d/P_2 represents a well-known characteristic in epidemic modeling — *basic reproduction index*, usually denoted as R_0. Thus, the condition of loss of stability by the equilibrium state E_0 in the equation (3) is completely consistent with the condition of epidemic occurrence known in epidemiology $R_0 > 1$.

4. The established level of the disease in the population

Next, consider the system ((3) at $P_2 < d$, that is, under conditions of disease development. In this case, the coordinates of the stable point E_1 determine the establishment of a dynamic equilibrium between the number of ill and healthy individuals. Usually of interest is the relative

number of ill individuals in the population $X_0 = i_0/C$, which characterizes the level of the disease. It is determined by the transcendental equation

$$X_0 = \frac{1}{1+A}\left(1 - \frac{P_2 X_0}{1 - \exp\left(-dX_0\right)}\right). \tag{7}$$

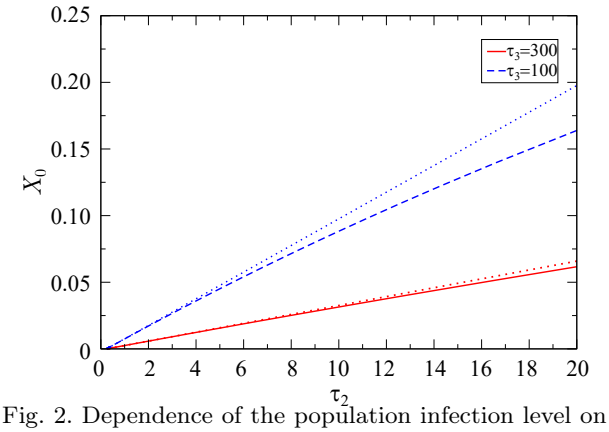

Fig. 2. Dependence of the population infection level on infection period τ_2 with long-term immunity; $d = 4.16$

We will conduct a qualitative evaluation of the formula (7) for typical values of the parameters included in it. The value in parentheses is less than one, and the disease level is limited from above by the multiplier $(1+A)^{-1}$. For most infections, the average duration of immunity $\tau_3 = P_3^{-1}$ is many times longer than the duration of the disease itself τ_2 [4].

Therefore, A — is a large enough number[3]. It follows that: (a) the unit in the denominator (7) can be neglected and (b) the value of X_0 does not exceed a few percent. Therefore, with not too large values of the factor d, the exponent in the denominator can be linearized: $\exp\left(-dX_0\right) \simeq 1 - dX_0$. Then the formula for X_0 will be significantly simplified

$$X_0 \simeq \frac{1}{A}\left(1 - \frac{1}{R_0}\right). \tag{8}$$

Here $1 - R_0^{-1}$ — the established level of the disease in the population that would exist there in the absence of immunity. As can be seen from the formula (8), the presence of immunity reduces the average level of patients by A times.

It should be noted that the values A and R_0 are not independent, since both contain the parameter P_2. To eliminate this ambiguity , the formula (8) can be rewritten as

[3]For example, for most respiratory viral infections, the average infection period is one to two weeks, and immunity can persist for a year or more.

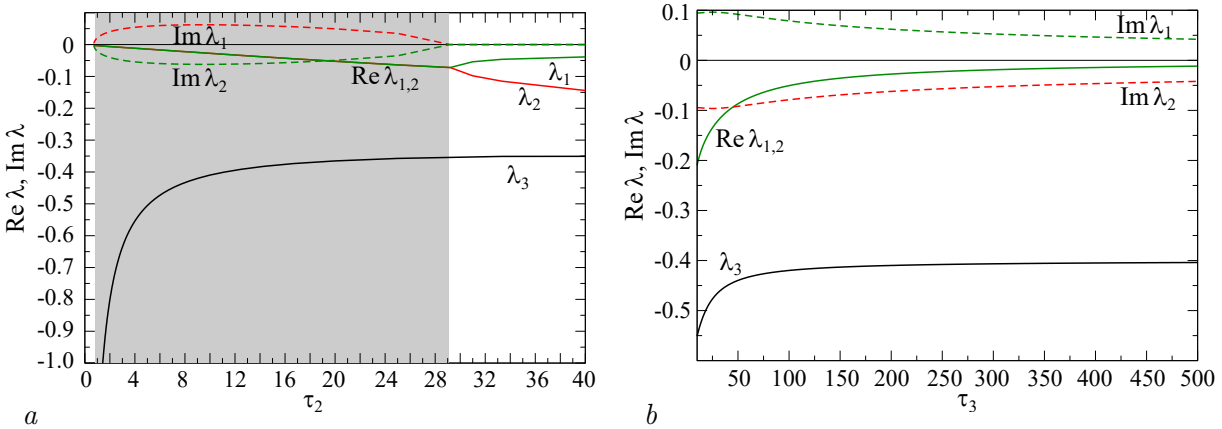

Fig. 3. Dependence of the eigenvalues of the equilibrium state E_1: a — on τ_2 at $\tau_3 = 200$ and b — from τ_3 at $\tau_2 = 10$; the other parameters are fixed: $\sigma = 0.7$, $\mu = 0.3$, $C = 0.6$. The solid curves correspond to real values, dashed lines correspond to imaginary ones; gray region on the fig. a marks the zone where E_1 is the focus

$$X_0 \simeq \frac{1}{\tau_3}\left(\tau_2 - \frac{1}{d}\right). \tag{9}$$

In this case, as can be seen from (9), the established level of disease in the population is determined by the action of two factors, one of which is τ_3^{-1}, and the second is— exceeding the duration of the infectious period over its critical value d^{-1}. In general, the presence of long-term immunity ($\tau_3 \gg 1$) protects the population from high levels of illness. Other factors constraining X_0 are equally a decrease in the infection period τ_2 and an increase in the inverse factor d^{-1}.

Fig. 2 shows graphs $X_0(\tau_2)$ for long immunity intervals $\tau_3 \sim 100$, calculated according to the full formula (7) and its simplified version (9); the latter are represented by dotted lines. We see that even with large $d \simeq 4$, if the average infection period does not last too long ($\tau_2 \leqslant 10$), both formulas give similar values. Therefore, for many diseases, the ratio (9) can be a good approximation.

5. Initial stage of the disease

The coordinates of the point E_1 determine the level of infection in the population at long times, that is, at $t \to \infty$. However, in practice, it is often important to know how the disease will develop at the begining stage after the initial infection. Therefore, we are also interested in the transition process from an arbitrary initial state in the vicinity of the origin to a stable point E_1. The transition process is determined by the structure of the phase space in the neighborhood of E_1, that is , the type of this equilibrium state. It can be determined based on the eigenvalues of the λ_{1-3} Jacobian of the system (3):

$$\begin{bmatrix} \exp\left(-\frac{\sigma}{\mu}i_0\right) - P_2 - 1 & \exp\left(-\frac{\sigma}{\mu}i_0\right) - 1 & \left(C - \left(1 + \frac{P_2}{P_3}\right)i_0\right)\exp\left(-\frac{\sigma}{\mu}i_0\right) \\ P_2 & -P_3 & 0 \\ \sigma & 0 & -\mu \end{bmatrix}.$$

The analytical form of eigenvalues is quite cumbersome, so we will use numerical calculations. Let's choose the parameter values that were used in the previous section: $C = 0.6$, $\sigma = 0.7$,$\mu = 0.3$, $\tau_2 \sim 10$, $\tau_3 \sim 100$ and plot the dependence of the eigenvalues on the average period infections τ_2 (Fig. 3, *a*) and the average duration of immunity τ_3 (Fig. 3, *b*)

As can be seen from the graphs, in the considered parameter area, the point E_1 has two complex conjugate eigenvalues λ_1 and λ_2 and one real — λ_3. At the same time, the real parts of $\lambda_{1,2}$ are very small and monotonically decrease with increasing duration of immunity. Thus, E_1 represents a stable focus. While approaching this point, the trajectory will make many turns, showing at first significant fluctuations in the number of cases, gradually decreasing in amplitude.Time-series of this transition the process is determined by the initial conditions. Let's choose as such a point near the origin: $i_0 = 0.001$, $r_0 = 0$ and $z_0 = 0$, which corresponds to the situation of penetration of several infected individuals into an initially healthy population. A typical variant of the transition process observed in this case is shown in Fig. 4 (line marked with circles). Here we see an extremely rapid increase of the number of infected immediately after the start, followed by an equally rapid decline to almost zero. This is followed by a second peak of a significantly smaller magnitude, after then the trajectory reaches a level close to E_1. From the point of view of population dynamics, this behavior can be interpreted as a decaying sequence of *infection waves*, in which the infection level of the population shows a sequence of sharp peaks before the epidemic reaches a dynamic equilibrium characterized by a low level of infection. In this case, the peak of infection at the very beginning of the disease affects more than a third part

of the population. The large magnitude of this peak is determined by the fact that at this stage immunity in the population has not yet been formed.

Thus, despite the relatively small established stationary level of infection, the number of infected at certain points in time can reach significant values. With a severe course of the disease, accompanied by probable fatal outcomes, the presence of a high peak of the first wave of infection can lead to serious consequences for the population.

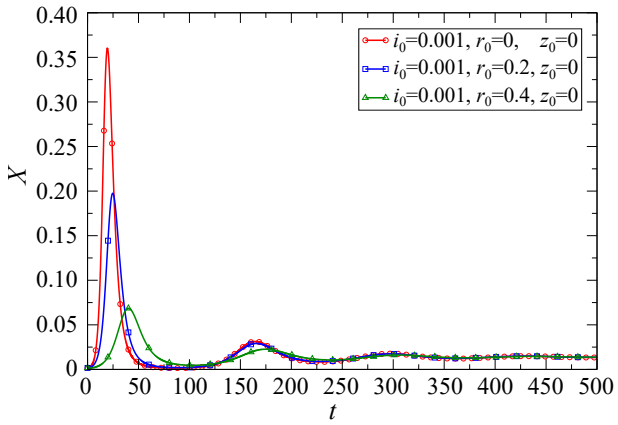

Fig. 4. Population infection levels $X(t)$ at initial stage of the epidemic under different initial conditions; the parameter values are: $\tau_2 = 5$, $\tau_3 = 300$, $\sigma = 0.7$, $\mu = 0.3$, $C = 0.6$

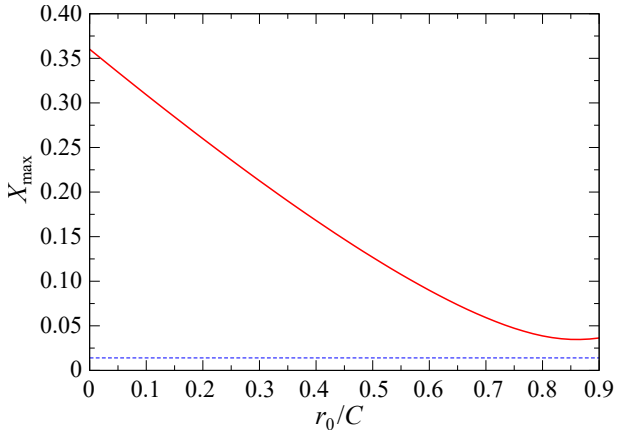

Fig. 5. Dependence of maximum value of infected relative number on the level of formed immunity available in the population at the time of infection. Dashed line marks the infection level corresponding to the steady state (X_0)

Under other initial conditions, the dynamics of infection may look different. If, at the time of initial infection, a sufficient number of individuals with immunity are already present in the population (for example, due to pre-vaccination), the transition process becomes smoother. In this case, the increase in the number of infected is slower, and the maximum level of infection is significantly lower. At $r_0 = 0.2$ (which is 33% of the population) at the peak of infection, the number of infected is about 0.2. At $r_0 = 0.4$ (67% of the population size) — less than 0.075 (fig. 4). In general, the dependence of the maximum number of infected on the level of immune individuals available at the time of infection is shown in Fig. 5. As can be seen from the graph, at the level of immune individuals from zero to about 60%, the magnitude of the infection peak decreases with the growth of r_0 almost linearly, after which it becomes more gentle and, starting from the level of 80%, stabilization occurs around the value of $X_{max} \simeq 0.075$. With other parameter values, the quantitative values will be different, but the qualitative the type of this dependency is preserved.

5.1. The influence of seasonal factors on the course of the disease. In the previous sections, an autonomous model of infection spread was considered, where all parameters are constant numbers. However, the processes associated with infection depend on environmental conditions, which for most climatic zones change periodically due to seasonal factors. Therefore, an autonomous model is insufficient for them.

Let's try to modify the system (3) to take into account the influence of changing conditions. The change of seasons will be to lead to periodic modulation of the equation parameters around some average values, which may affect seasonal fluctuations in the number of infected. Of all the parameters used in (3), the most sensitive to the influence of external factors is the rate of inactivation of viruses μ, the inverse value of which (τ_v) determines the time during which viral particles can remain active outside the infected individuals. This interval significantly depends on the temperature and humidity of the environment, as well as on a number of other factors

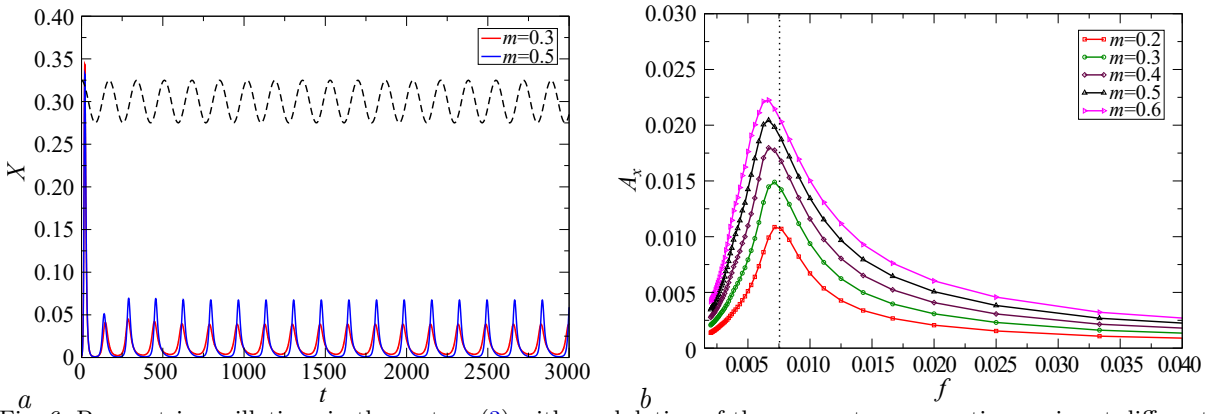

Fig. 6. Parametric oscillations in the system (3) with modulation of the parameter μ: *a* — time-series at different modulation indices at $T = 170$ and *b* — a family of resonance curves for different m; dashed line on the fig. *a* indicates the modulation of the parameter. Parameter values are: $C = 0.6$, $\mu_0 = 0.3$, $\sigma = 0.7$ (color online)

(the amount of solar radiation, average wind intensity, etc., etc.). To take into account seasonal factors, we assume that the parameter μ in the equation (3) modulated by a harmonic function of time around the mean value of μ_0 according to the law: $\mu(t) = \mu_0\,(1 + mF\,(\cos\,(2\pi ft)))$, where f is the frequency, and m is the modulation index; the function F defines the influence of external factors on the parameter μ, it is selected so that $|F(x)| \leqslant 1$ when $|x| \leqslant 1$.

Let's first consider the case of linear dependence: $F(x) = x$. Let's choose the average value of the parameter $\mu_0 = 0.3$. In the presence of parameter modulation, we observe periodic oscillations of the infection level $X(t)$ in the form of a regular sequence of sharp infection peaks, the type of which is shown in Fig. 6, *a*. The oscillation period is equal to the period of the modulation function $T = f^{-1}$. As a result, the disease periodically reaches high infection levels at peaks and drops to almost zero in the intervals between them. The amplitude of steady-state oscillations A_x evidently grows with the growth of the modulation coefficient. As for its dependence on the modulation frequency f, it has a resonant character. This can be seen from the resonance curves plotted in Fig. 6, *b*. At low modulation indices, the resonant frequency is close to the self frequency of the oscillator, defined as $\mathrm{Im}\,(\lambda_{1,2})/(2\pi)$ (the latter is shown in the figure by the dotted line). With an increase in the modulation depth, a slight shift of the resonant frequency towards the lower frequencies is observed.

In addition to oscillation with the frequency of external parametric action , at some values of the parameters in the oscillator, a doubling of the oscillations period is observed. In this case,

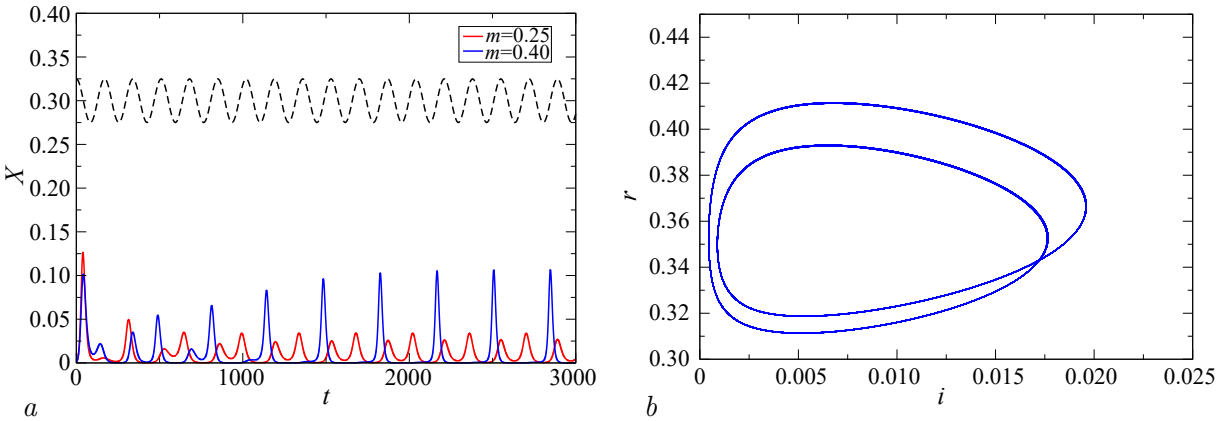

Fig. 7. Doubling of the oscillation period: *a* — time-series $X(t)$ and *b* — the phase portrait in variables $i-r$; the dashed line on the fig. *a* indicates the modulation of the parameter. Parameter values are: $C = 0.6$, $\mu_0 = 0.6$, $\sigma = 0.5$, $T = 300$ (color online)

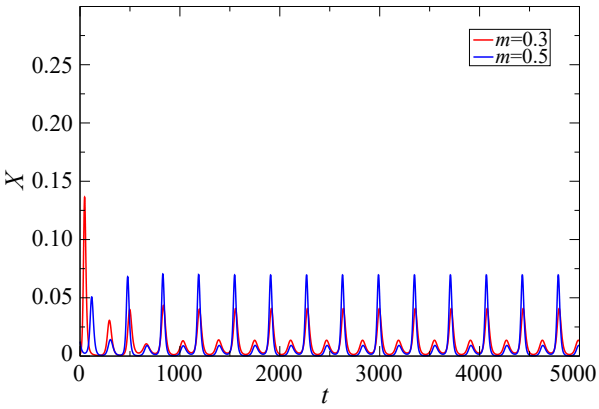

Fig. 8. Time-series of parametric oscillations with nonlinear dependence of the inactivation parameter: $F(x) = x^2 + 0.5x - 0.5$; $\sigma = 0.5$, $\mu_0 = 0.6$, $C = 0.6$ (color online)

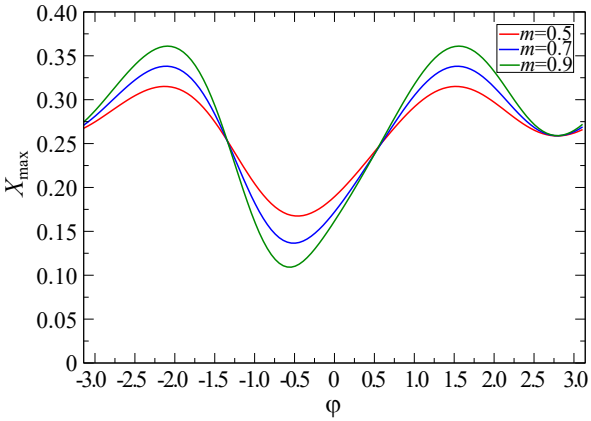

Fig. 9. Dependence of maximum infection on initial phase of the parameter modulation function (color online)

the oscillation period doubles in comparison with the modulation period (Fig. 7, *a*), and the phase portrait takes the form of a two-turn limit cycle (Fig. 7, *b*). This type of oscillations means in practice the presence of outbreaks of the disease with a two-year interval. A similar dynamic is indeed observed for some diseases. For example, it was noted for the disease measles before the introduction of mass vaccination [21].

The dependence of the rate of inactivation of viruses μ on environmental conditions can also be expressed by a nonlinear function $F(x)$. In this case , peaks at harmonics multiples of the base frequency can be expected in the spectrum of fluctuations in the number of cases. For example, if we choose a dependence in the form of a quadratic polynomial $F(x) = ax^2 + bx + c$, then the disease maxima appear twice during the period. This type of oscillation is shown in Fig. 8. It corresponds to the well-known phenomenon of seasonal outbreaks of respiratory viral infections.

A non-autonomous model is not invariant to time. Therefore, the choice of the moment of initial infection can have a significant impact on the processes in it. The development of the epidemic can occur in different ways, depending on what time of year the initial infection occurred. To take this factor into account, we introduce the initial phase (φ) into the law of parameter modulation: $\mu(t) = \mu_0 (1 + mF(\cos(2\pi ft + \varphi)))$ and we will measure the level of the disease at the peak of the epidemic $X_{\max}$ depending on φ. The results of calculations confirm the assumption about the importance of the moment of initial infection on the course of the epidemic. A typical type of dependency is shown in Fig. 9. As can be seen from the graph, the magnitude of the peak of infection can vary several times depending on how favorable the moment of initial infection penetration into the population turned out to be for the spread of the epidemic .

Conclusion

The proposed modified SIRS model demonstrates the dynamics characteristic of the development of epidemic processes in natural populations. If we do not take into account the periodic changes in conditions caused by climatic factors, then in the presence of an initial infection in the community, a sequence of several attenuating «waves» of infection occurs, which reduces to a small average steady-state level. At the same time, the amplitude of the first wave

can reach very large values comparable to the size of the entire populations. Taking into account climatic factors leads to recurrent outbreaks of the disease, which can occur both with twice the frequency (seasonal outbreaks of respiratory infections) and with half the frequency of exposure (biennial outbreaks of certain infections). The period of these processes is determined by the type of function describing the influence of climatic factors on the rate of inactivation of viruses in the environment. Specification of the type of this function is possible by taking into account biological factors that characterize the behavior of the causative agent of infection outside the body of an infected individual. The system under consideration describes many characteristic phenomena observed during the development of epidemics. At the same time, it is rather approximate, since it does not take into account the specific features of diseases, the possible movement of individuals and the pathogen in space. Modification of the model may follow the path of clarifying the type of function $P(\mathbf{v})$, for example, taking into account the threshold nature of infection observed for some diseases; using a more realistic (nonlinear) equation describing the generation of viruses by a diseased individual; clarifying the type of function modulations $F(x)$. Such refinements are an adjustment of the model to a specific type of disease and take into account its biological features, that is, they correspond to the transition from qualitative modeling to simulation. Another direction of modification of the proposed model may be the transition from a system of ordinary differential equations to lattices of probabilistic cellular automata. Such an approach will make it possible to naturally take into account the stochastic nature of epidemiological processes and consider the impact of unevenness in distribution on these processes individuals and viruses by habitat and the impact of their movement processes.

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