Introduction to the Computer Modeling of the Plague Epizootic Process

Vladimir Dubyanskiy[1], Leonid Burdelov[2] and J. L. Barkley[3]
[1]Stavropol Plague Control Research Institute,
[2]M. Aikimbaev's Kazakh Science Center of Quarantine and Zoonotic Diseases,
[3]Virginia Polytechnic Institute and State University
[1]Russian Federation
[2]Republic of Kazakhstan
[3]USA

1. Introduction

Bubonic plague, caused by the bacteria, Yersinia pestis, persists as a public health problem in many parts of the world, including Central Asia and Kazakhstan. Plague is a vector-borne disease, i.e. the disease is spread by arthropod vectors that live and feed on hosts (Gage and Kosoy, 2005). Great gerbil (Rhombomys opimus Liht., 1823, Rodentia, Cricetidae) is a significant plague host in Central Asia and Kazakhstan region. Transmission happens through bites of infected fleas that earlier fed on infected hosts. This plague epizootic process study can help future plague control work.

Modeling the epizootic process of plague in great gerbil settlements allows for quantitative analyses of epizootic characteristics, which we are unable to control for in nature (Soldatkin et al, 1973). Only one such attempt of this type of modeling is known (Soldatkin et al, 1966; Soldatkin and Rudenchik, 1967; Soldatkin et al, 1973). In this study, the authors demonstrated the high potential for this model, yet it possessed many limitations and had a small work space (4,096 holes). The development of high-performance workstations, and integration of geographic information systems (GIS) and remote sensing now makes it possible to model complex, epizootic processes of plague.

We created a probabilistic, cellular automaton model of the epizootic process of plague by using all of the above-named technologies. A probabilistic, cellular automaton with Monte-Carlo, as the chosen statistical method, is the basic mechanism of the model (Etkins, 1987; Grabovskiy, 1995). It is the SEIR (susceptible, exposed, infected and removed) model with specific additions.

2. Description of the model

The model was created using Microsoft Excel 2003 with Visual Basic as the code language. Microsoft Excel has 255 columns and 65,000 rows. A user-defined, fixed size of the modeling area depends on the density of the gerbil colonies. A probabilistic, cellular automaton with Monte-Carlo, as the chosen statistical method, is the basic mechanism of the model

(Dubyanskiy V.M., Burdelov L.A. , 2010, Dubyanskiy V.M, 2010). The choice for this model type was rationalized by the following:

- Commonly-used mathematical models that rely on differential equations require input from many data sources. While, at the same time, the availability of plague surveillance source data is rather insufficient.
- Temporal models, (based on differential equations or other methods), are being used for modeling without consideration of spatial distribution. In these models, it is assumed that all events are happening at an abstract point rather than considering plague focus, landscape region, or primary square.
- If plague simulates nature, it analyzes only a static structure of plague focus or its parts.

The probabilistic, cellular automaton simulation allows the user to combine all of these rationales. The epizootic process of plague in Great gerbil settlements was a good subject for this experiment. The gerbil's burrow system represents a time-spatial, discrete unit of an epizootic process (Sedin, 1985). The data on burrow systems distribution within a plague focus was determined by GIS and remote sensing. Satellite images of the burrow systems provide a coherent picture of the colony structure (Burdelov et al., 2007; Addink et al., 2010) at many plague foci (Fig 1.).

The cell/row structure of the model's workspace allowed us to observe the proportion between burrow systems and settlements and density of colonies. The density of colonies was the main factor approximating the number of rodents. Both the density of colonies and the number of rodents are significant factors for plague prediction (Klassovskiy et al., 1978; Davis et al., 2004, 2008).

Several quantitative and qualitative relationships and characteristics between components of the plague's parasitic system were identified early on (Burdelov et al., 1984; Begon et al., 2006). Analyzing these numerous, character-combinations required that we use Monte-Carlo simulation, rather than differential equation models, to describe each specific time stamp in question. The analysis of burrow systems distribution on satellite images demonstrated that the number of nearest neighbors in the burrow system often exceeded four (von Neumann neighborhood). Hence, our model used the square (eight-cell) Moore neighborhood (Fig 2.).

The state of colonies can be categorized as "infected", "infecting", "immune", or "readied" for reinfecting. An infected colony is one that is infected, (in host, in vector or in substratum), but has not spread the plague microbe (latent period). An infecting colony is one that is infected and is spreading the plague microbe among other colonies. A colony that has been cleared of the plague microbe and is not infectious is called an immune colony. A readied colony is one which can be infected again after the immune period.

Data on the vector's and host's infection were used to the extent that it was necessary for calculating the model's parameters. An infecting agent can be transmitted to other colonies independent of the main mechanism of "host-vector-host" transmission. These processes define whether a colony is infected or not and constitutes the "black box" of the model. Nuances of an epizootic process which might be connected to specific mechanisms of plague transmission have not been reviewed.

The cycle of infection is a duration when an infected colony is being converted to an infecting colony and can spread the plague microbe among other colonies. The obligatory

scan of the model's workspace occurs at the end of each cycle of infection. The probability of transfer success is defined as one attempt of the plague microbe to transfer from an infected colony to a non-infected colony. The number of attempts required to infect neighboring colonies from one "infecting" colony during one cycle of infection is termed the coefficient of colony infection. This term was proposed by Litvin et al., (1980). However, this term, in our case, unites the coefficient of infection of a colony by Litvin and the coefficient of transmission by Soldatkin et al., (1973). The rate (or the value) of epizootic contact is the product of the successive transfer probability and the coefficient of a colony infection.

Fig. 1. Colonies of Great Gerbil in a satellite image. Each bright disc represents colonies 10-40m in diameter. The image was captured using the publicly available software Google Earth (http://earth.google.com/). Copyright 2008 DigitalGlobe; Europa Technologies.
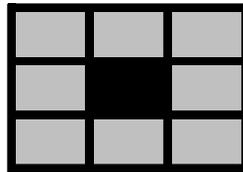

Fig. 2. Moore's environment allows using all cells in model for its greater efficiency.

$$L = C_i * C_t \qquad (1)$$

where L is a rate (or the value) of epizootic contact, C_i the coefficient of colony infection, C_t the probability of transfer success.

The relative remote drift is the possibility of more or less remote colonies infecting without nearer colonies infecting. This parameter can be regulated by the change of plague drift distance as well as by the change of plague drift realization probability. It also allows changing the speed of epizootic spread.

The structure of great gerbil settlements was reflected by altering colony-cells and non colony-cells. This allowed working with different configurations of settlements and with different densities of burrow systems. The scheme of burrow system's spatial location could be integrated into the work space of the model from a map or from a satellite image if necessary (Fig. 3). If the epizootic process was imitated, then the spatial location of burrow systems would be defined by a random-number generator. Initial results were published earlier (Dubyanskiy, Burdelov, 2008).

A   B   C
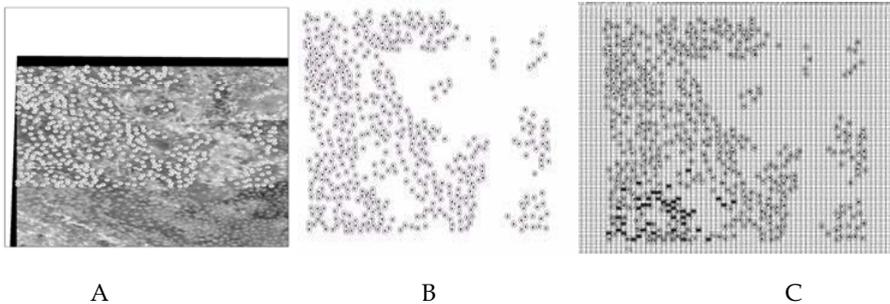
Fig. 3. Carry of a real configuration of settlement of Great Gerbil from the satellite image adhered in GIS in working space of model. A - Allocated by means of GIS colonies of Great Gerbil. B - Layer of GIS ready for carry to working space of model. C - Working space of model with developing of plague epizooty.

There are two ways of spreading the plague microbe. The "infected" colony could attempt to infect the other colony-cell as well as non colony-cell. In the latter case, the attempt of infecting is useless. However, the tendency of gerbils to migrate among colonies will spread the plague microbe to occupied colonies. The probability of transfer success is a probability that a sick flea (or sick gerbil) will be transferred from an "infected" colony to a non-infected colony and a microbiocenose of colony will be involved in the epizootic process. The coefficient of a colony infection demonstrates how often transfers occurred (i.e. a gerbil can visit non-infected colony two times or 10 times and so on).

The basic quantitative and qualitative characteristics were entered into the parameter sheet. The value of parameters can be changed before modeling, although the dynamic changing of parameters is possible. The parameters could be, for example, quantities of the "cycle of infection" for one season (or session of modeling), the duration of the cycle of infection, the density of colonies, and the probability of infecting neighbor colonies. The technical conditions of the model work were also defined. The built-in menu allows comfortably managing the modeling options.

Cellular flattened models have the edge effect. Naturally, this effect took place in our model too. It was decided that all transmission of plague microbes outside the model workspace was not consequential. In this case the model was made simpler. The model settlement looked like an isolated island of the gerbil's settlement in nature.

2.1 Basic parameters of the model

Even with the seeming simplicity of the model, the real quantity of fulfilled interrelations is rather great. For example, "infection" of a colony depends on: the duration of cycle of infection; the duration of the colony's status as infected; infectious quotient and immunity; the probability of success of transfer; the coefficient of infection of a colony; the density and distribution of colonies in the workspace of the model; and the number and arrangement of infected colonies. Considering that further behavior of a cell depends on probability and status of surrounding cells at PCA, to give the optimum and full analysis of influence of such quantities of the interconnected parameters is very difficult. Therefore, initial selection of the basic working parameters of the model were carried out in a literature search of data, including results of direct or physical modeling of epizootic process using radioactive isotopes in real settlements of the great gerbils. It is necessary to make a stipulation that the analysis of literature has shown an absence of any unequivocal quantitative characteristic of epizootic process in a required aspect.

After infection by the plague microbe, a colony becomes "infected". This period in the model lasts for 10 days where three days are the time of bacteremia and seven days are the time of flea's prohibition (block of the proventriculus) if the flea fed on a sick animal. The colony has the possibility to infect neighbors from day 11 to 50 from the beginning of its own infection. It is not infectious from day 51 to 90, and it is immune (Novikova et al., 1971; Naumov et al., 1972). The colony can be infected again after 90 days (Samsonovich et al., 1971; Rothschild et al., 1975; Rothschild, 1978).
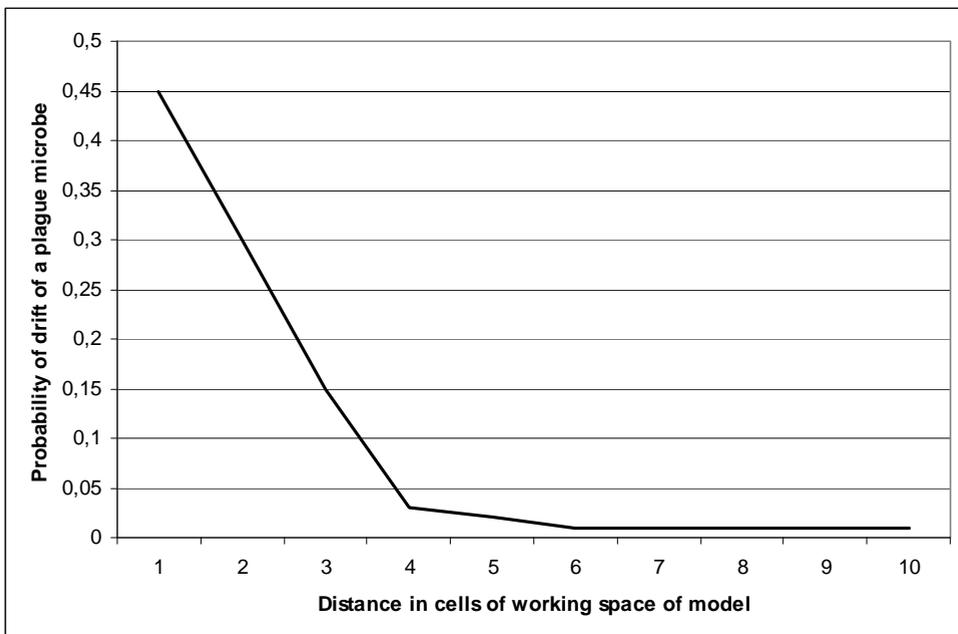

Fig. 4. Probability of drift of a plague microbe from colony to colony in working space of model during one cycle of infection.

The probabilities distribution of plague microbe drift approximately corresponds to data of direct modeling of drift of the fleas in the neighboring holes (Soldatkin, Rudenchik, 1971), and to data on movement distances of gerbils to the neighbor colonies (Alekseev, 1974; Davis et al., 2008) (Fig. 4). The probability of transfer success could be modified by the user within the limits from zero to one. The coefficient of a colony infection could be varied from one up to experimentally received or any other logically admissible value. The density, distribution, occupation, and number of a plague microbe hosts expressed through occupation were given as cell-colonies percentage, from quantity of cells in all working spaces of the model and could be changed from zero to 100. It would be desirable to emphasize that it was possible to change any parameters in the model.

The maximum drift of plague microbe in model can mark as l. Every infecting colony can infect the neighbours in a square with diagonal $2l$. The infecting colony is situated in the center of this square. The probability of contact with any colonies inside this square defined as

$$Pz = \frac{N_l - (i_1 + i_2 + R)}{N_l} \tag{2}$$

where Pz is the probability of contact with the neighbour colony, N_l is the general number of colonies in the square with $2l$ diagonal, i_1 is the infected colony, i_2 is the infecting colony, R is the immune colony.

The probability of colony infect is defined as

$$Pi = Pz * Ct \quad Ci \tag{3}$$

where Pi is the probability of colony infect, Ct is the probability of transfer success (input from experimentalist), Ci is the coefficient of colony infection (input from experimentalist).

Taking into consideration the formula (3)

$$P_l = P_Z * L \tag{4}$$

The intensity of model's epizooty is defined as

$$Y = (i_1 + i_2) / B * 100 \tag{5}$$

where Y is the intensity of epizooty, B is the general number of colonies.

The quantity of infected colonies are increasing in the beginning as Fibonacci sequence

$$F_{n+2} = F_n + F_{n+1}, n \in N \tag{6}$$

However when immune colonies appear, the complexity of the epizootic system increases. The description of epizooty by differential equations are not possibe.

The basic parameters described above would be for a case studying the influence of spatial parameters for epizootic processes. It is possible to establish a reverse problem to study the influence of temporary parameter changes at a static, spatial configuration of the settlement. In an ideal case, data can be accumulated on epizootic processes in nature and in experiments on models.

For this study, the plague spread in various settlement types of great gerbils used the following items: 1. The probability of transfer success was equal to one, 2. the coefficient of a colony infection could be varied from one up to 10, and 3. The coefficient of a colony infection was always one.

A two dimensional array of cells was used for the modeling world. The modeling square was equal to 900 ha. The experiment was successful if the epizootic process continued during 18 epochs (model's half year) 11 times without a break. It was a 0.9999 probability that the experiment was nonrandom (Rayfa, 1977). The start value of the coefficient of a colony infection was equal to one. The start density of colonies were 1.33 per ha. The density of colonies declined for 0.11 colonies per ha after each successful experiment for continuous settlements and it was increasing the distance among colonies per one cell on vertical and horizontal lines for narrow-band settlements. As a result, the density of the colonies declined exponentially. If the experiment was not successful the coefficient of a colony infection increase up per unit and recurred again.

3. Results

We gave an example of developmental epizooty in the feigned settlement of the great gerbil (Fig. 5). The goal of the experiment was an examination of qualitative likeness between modeling and natural epizootic processes. The size of the model's working field was 2x2 km. It corresponded to an area of 400 hectares with density of colonies at 2.4 per hectare. A conditional start date of epizooty was March 1st. The epizootic process was developed as a result of the distribution of a plague microbe from one infected and random established colony. The probability of transfer success and the coefficient of a colony infection were constant and equal, 0.8 and 1 respectively.
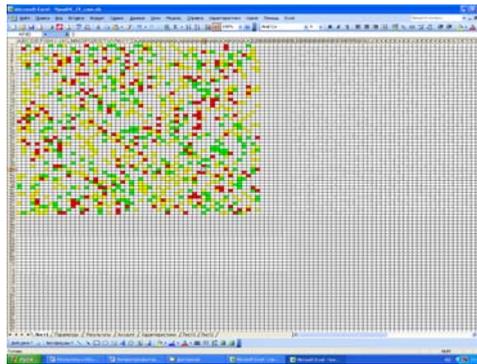
The epizooty developed slowly at the initial stage. The analysis of the modeled epizootic process showed that the critical time was the initial transfer of a plague microbe between colonies. This was also noted by Soldatkin et al. (1971). The epizootic process could proceed indefinably long if the "infected" colony transferred the plague microbe even to one "not infected" colony during its "infecting" ability. The random hit of a primary "infected" colony on a site of settlement where transfer of a plague microbe would occur raises the probability of epizootic fade-out. The epizooty will end if the transfers of plague microbe do not provide even short-term constant increase of number in the "infected" colonies (1.1 – 2 times during a minimum of 3 cycles of infection). If the start of epizooty was successful, after three months, small foci, consisting of 4 – 8 infected colonies would have been formed (Fig. 5a). The number of the infected colonies increased exponentially. On reaching a certain level of "infected" colonies, the intensity of epizootic process started varying cyclically (Fig. 6). The exponential increase of the infected colonies number was terminated by virtue of two causes. The first cause is the probabilistic character of the plague microbe transfer success from colony to colony. If two or more colonies are infected by an infected colony, the epizootic process intensity will exponentially increase. At the same time, an infected colony can be a source of infection for all neighboring colonies or infect nothing. The second cause is the relatively remote drift of the plague microbe that forms many small foci, consisting of the infected colonies (Fig. 5b). These foci are dissemination centers of epizooty. The spatial structure of the epizooty site is a complicating factor. It begins as a saturated epizooty site of "infected" and "immune" colonies, whereby the plague microbe transfers to other

"infected" and "immune" colonies in greater numbers, decreasing the probability of transferring to "non infected" colonies.
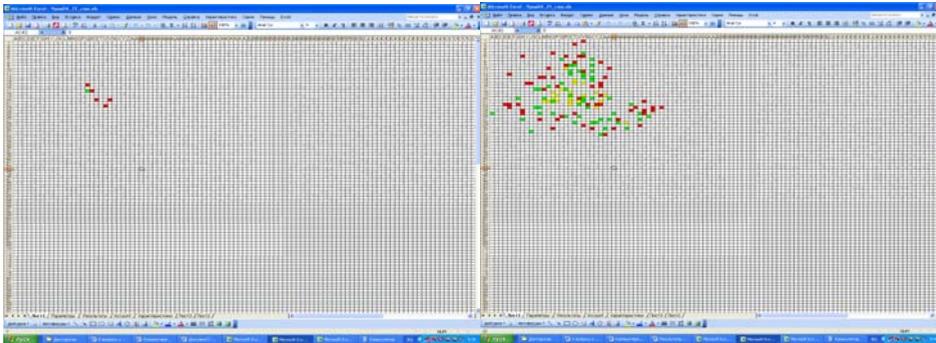
A   B

C

Fig. 5. The visual representation of epizootic process had been developed at model. A) The initial stage of the epizooty development (90 days). B) Remote drift and formation of small foci (210 days). Quantity of the "infected" colonies had started an exponential increase. C) A stage of "saturation" of the epizooty site and transition to cyclic fluctuations

Typically, two peaks of epizooty activity are registered during the year, in the spring and in the fall (21). The epizootic contact rate increases during the rut in early spring and at the end of the spring, as young gerbils explore their territory. Epizootic contact rate and process intensity decreases during the high summer temperatures as the gerbils spend less time on the surface. This also coincides with a generational replacement of the *Xenopsilla* fleas. Contact increases again during the fall when the gerbils become hyperactive, resulting in a second peak of epizooty activity. In autumn, the gerbils cache food and redistribute it among the colonies before winter. The younger generations of fleas are actively feeding, possibly transferring the plague microbe among the animals. This complex mechanism is approximated through the rate of epizooty contact within the model. The exact data about

probability of "infecting" colonies in different seasons of the year are absent in literature. Thus, for a qualitative look at the model and nature, the following conclusions were garnered from the model: the probability of a colony infection in the summer is half of that in the spring; a colony infection in the autumn is 0.15% less than in the spring; and a colony infection in the winter is three times below that in the autumn.

Numerical values of the probability of transfer success and the coefficient of colony infections were calculated earlier (Soldatkin et al, 1966, 1971). The probability of transfer success was equal to 0.8 and the coefficient of a colony infection was equal to 1.0, for spring. After entering these parameters, the picture of epizootic process in the model was appreciably changed and looked much like a natural occurrence (Fig. 7).
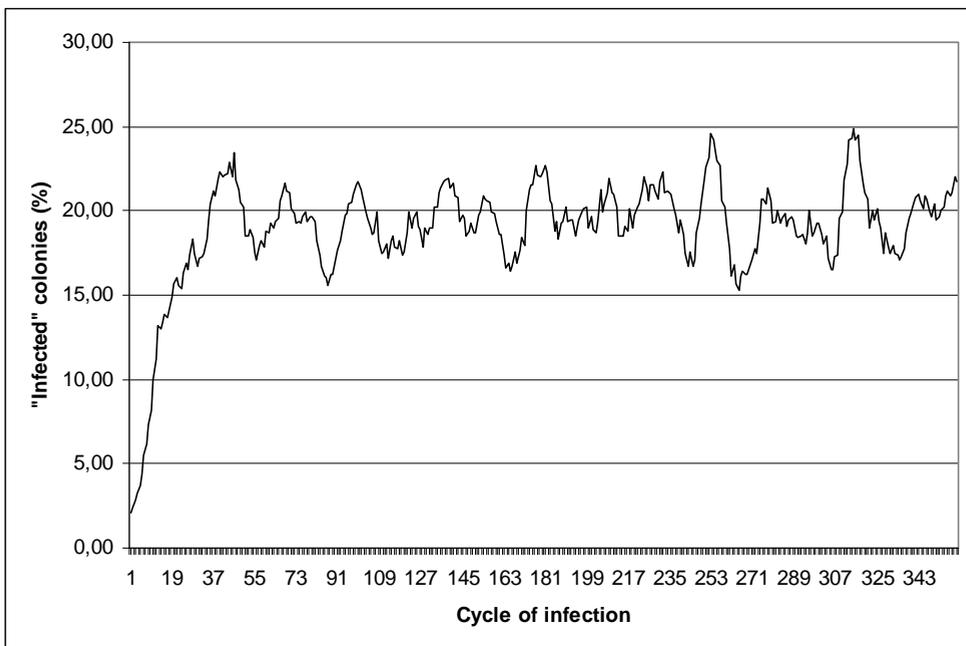

Fig. 6. Intensity of epizootic process in model

There were rare, separate "infected" colonies present in the model's space after the winter period. Those colonies were in the initial stages of the annual cycle of epizooty (Fig. 7a). There were many "immune" colonies that remained after the last epizooty in autumn. The epizooty became sharper at the end of spring when the number of plagued colonies increased, but the number of "immune" colonies decreased (Fig. 7b). In summer, the epizooty recessed and the number of "immune" colonies increased again (Fig. 7c). In autumn, the epizooty raised again as the number of plagued colonies increased and the number of "immune" colonies decreased (Fig. 7d). Thus, the annual epizootic process intensity had qualitative correspondence to a classic, twice-peak epizooty (Fig. 8) as in some natural, plague foci.

A   B

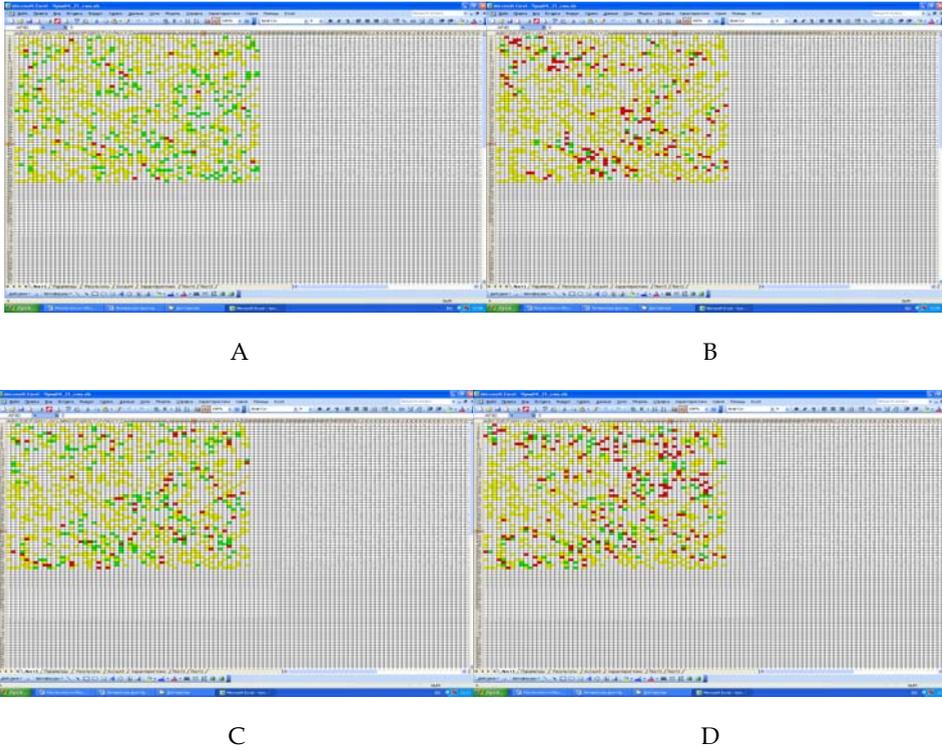C   D

Fig. 7. The epizootic process was developing with a changeable rate of epizootic contact. Intensity of epizooty in the end: A) winter; B) spring; C) summer; D) autumn
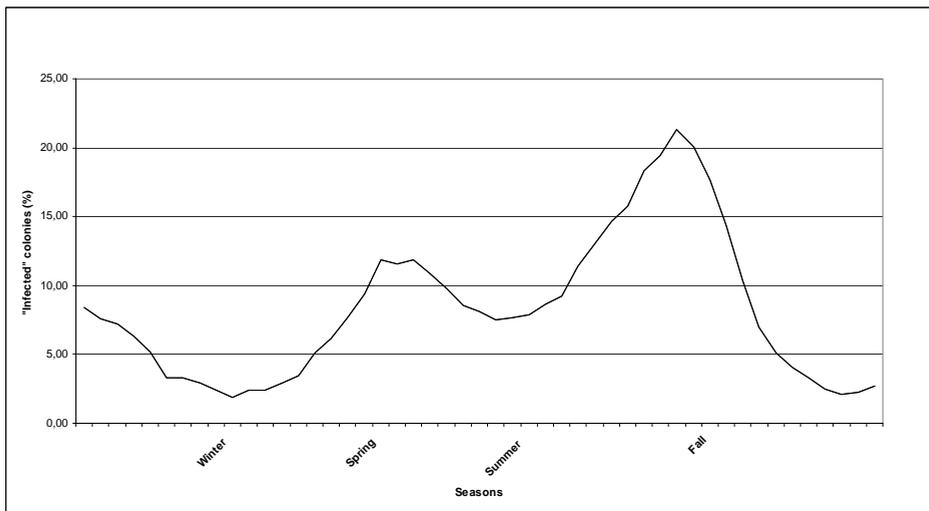

Fig. 8. The intensity of epizootic process modeling within a year

It is interesting, that when a changeable rate of epizootic contact was entered into the model's parameters, the average number of the "infected" and "immune" colonies decreased from 18.92 to 9.31 (p <0.001, Mann-Whitney U test). It was caused by the increase in colonies which were ready for a new infection. As a result, much sharper epizooty could develop. We modeled the sharper epizooty. The coefficient of colony infection was increased to two for this. The picture of epizooty is shown on Fig. 9 and Fig. 10. The epizooty intensity increased almost double in spring and in the fall. However, the number of "infected" colonies was on par with the pre-epizootic level in the winter-summer recession of epizootic activity. The speed of the epizootic spreading fluctuated from 640 to 920 meters per month in these experiments.

It is known that the colonies of the great gerbil have a non-uniform distribution on the surface. According to the distribution pattern, the groups of colonies - the settlement, may be visible as a narrow-band, a wide-band, a large patch, a small patch and continuous settlement (fig 11, 12). Some authors assumed that spatial distribution of the great gerbil settlements played a big role in the epizootic process functioning (Pole, Bibicov, 1991; Dubyanskiy et al., 1992). However that supposition was described as a quality parameter without any quantity values and was quantitatively checked up by modeling. The narrow-band settlements were presented as single colonies, arranged on an identical distance on the vertical and horizontal lines (fig 13).

A   B

C   D
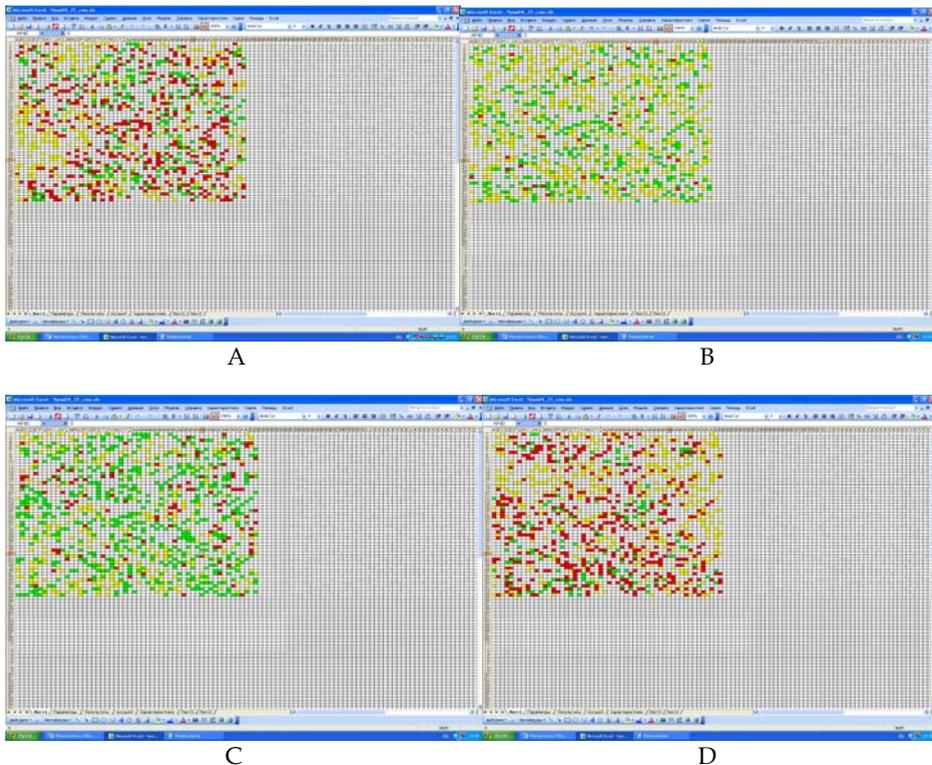
Fig. 9. The epizooty was developing with increasing of the rate of epizootic contact. Intensity эпизоотии in the end A) autumn; B) winter; C) spring; D) summer

First experimental results demonstrated a significant difference of plague spread into continuous and narrow-band settlements (table 1). When the coefficient of a colony infection is one (it is the minimum value of model) the plague can be spread from a density of 1.33 colonies per ha into the continuous settlement. At the same time a density of 0.44 colonies per ha is enough for plague epizootic into the narrow-band settlement. The difference of densities equals three. As the density declines the coefficient of a colony infection proportionally increases into the continuous settlement. A critical threshold is achieved from a density of 0.78 colonies per ha. At the following step of density (0.67 colonies per ha) the epizootic process is recommencing only from value of the coefficient of a colony infection equal 10.
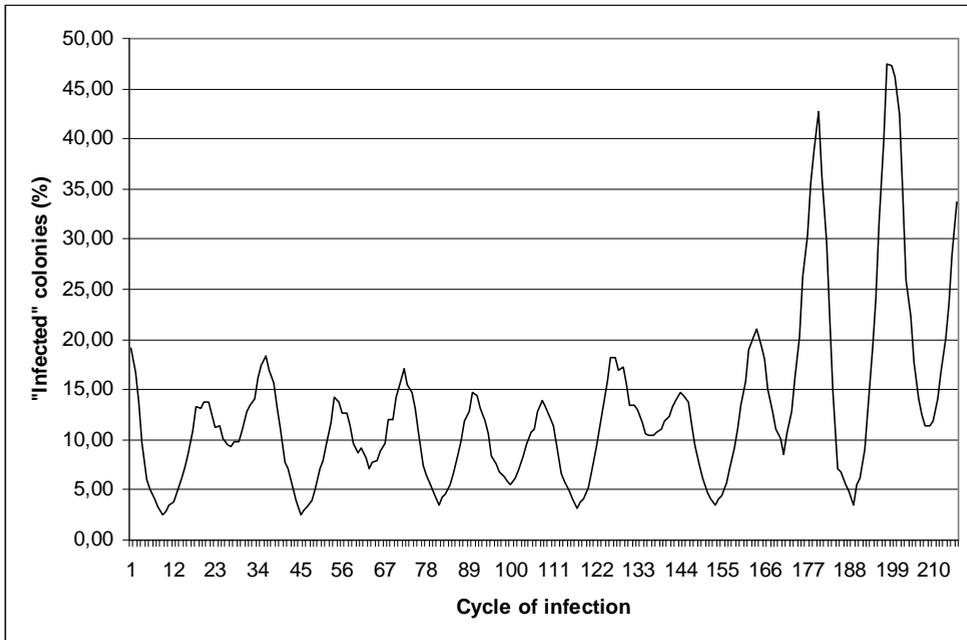

Fig. 10. The intensity of epizootic process which developed at the model with increasing of the rate of epizootic contact (the right part of curve after cycle of infection 171).

In the narrow-band settlement, the minimum limit of density was achieved at 0.11 colonies per ha, and the value of the coefficient of the colony infection was equal to eight. It is not possible to reduce the density of colonies more because of the model limits in the plague microbe drift.

Previous explanation of this phenomenon follows. As monitoring of the model shows, the colonies in the continuous settlement were aggregated to a few groups. The plague epizootic could develop under certain conditions:

1.   if the number of colonies in separate groups are enough for plague circulating only in this group;

2. if the distance among groups of colonies is not more than a limit of the relatively remote drift;
3. if the movement activity of animals is enough for connection among the groups of the colonies;
4. if the rate of epizootic contact is not the highest at the given density of colonies.

These conditions were not often established in the continuous settlement. The plague epizootic was faded by the Allee principle (Odum, 1975). It is possible to check up these items by the statistical data from table 1. The critical threshold of the colonies' density appeared when the average distance among the colonies had exceeded the maximum possibility of the plague drift. At the same time the mode of spacing demonstrated that the circulation plague microbe inside the groups of colonies was not limited by the distance. The rare connections among the colonies and groups of colonies which were situated in the plague microbe drift distance were more stable on the highest level of epizootic contact. It could be about 25% of all colonies at 0.67 colonies per ha density. It demonstrated the first quartile of the distance among colonies. Thus the plague microbe could circulate among 150 colonies in the model's space.

Fig. 11. The continuous settlement of the great gerbil. Colonies of Great Gerbil in a satellite image. Each bright disc represents colonies 10-40m in diameter. The image was captured using the publicly available software Google Earth (http://earth.google.com/). Copyright 2008 DigitalGlobe; Europa Technologies.

Fig. 12. The narrow-band settlement of the great gerbil. Colonies of Great Gerbil in a satellite image. Each bright disc represents colonies 10-40m in diameter. The image was captured using the publicly available software Google Earth (http://earth.google.com/). Copyright 2008 DigitalGlobe; Europa Technologies.

A   B

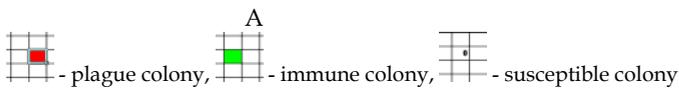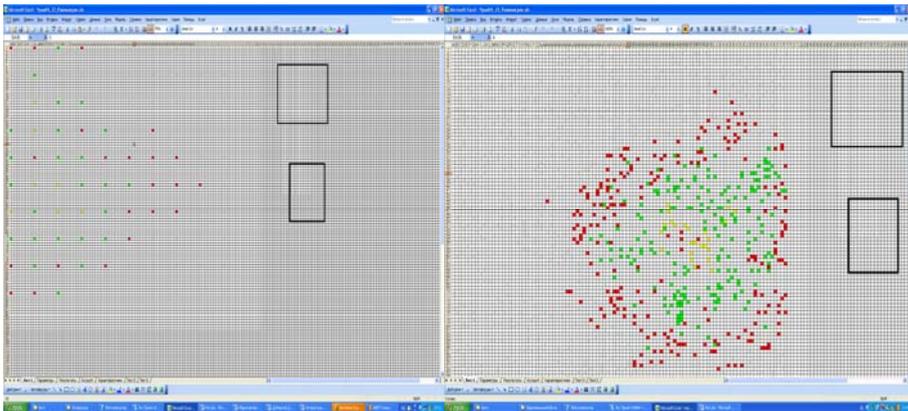 - plague colony, - immune colony, - susceptible colony

Fig. 13. The development of plague epizooty into narrow-band settlement (A) and into continuous settlement (B).

The rate of epizootic contact	Density per ha		The statistical characteristics of distance among colonies (in cells)			
	Continuous settlement	Narrow-band settlement	Average	1 quartile	Mode	Median
1	1.33	0.44	7,33	2	1	5
2	1.11	0.23	8,09	2	0	0
3	1.00	0,17	8,99	2	0	6
4	0.89	0.14	10,11	2	0	7
5	0.78	0,11	10,11	2	0	7
6	0,78	0,11	10,11	2	0	7
7	0,78	0,11	10,11	2	0	7
8	0,78	0.11	10,11	2	0	7
10	0.67	0,11	15,61	4	2	11

Table 1. Dependence of plague spread from colonies density

In the narrow-band settlement the groups of colonies were absent. The colonies were arranged in the form of a lattice and the colonies stood in the vertexes of this lattice. The distance among the colonies was not more than the limit of the relatively remote drift. Each surrounding colony contained enough numbers of colonies for supporting plague epizootic. The colonies were situated in the better places for digging holes in nature (Dubyanskiy, 1970). The migration routes of rodents were not voluntary, and arrangement depended on the settlement structure (Naumov et. al., 1972). The rodents moved along migration routes despite low density, for searching mates, dissemination of young animals, etc., increasing the epizootic contact rate (the dynamic density (Rall, 1965)). The bird-view (fig. 12) demonstrated the limited routes of migration of the animals. These routes are lying along verges of lattice. The similar direction of migration plays a big role in supporting of the plague epizootic.

There is a great difference in the intensity of epizootic processes between the narrow-band and the continuous settlements (fig. 14). The epizooty in the narrow-band settlement is more intensive as in the continuous settlement. The significant difference is represented according to Mann-Whitney U test $p <= 0.01$.

If the epizootic contact rate was variable, the intensity of the epizootic process changed according to the density of the colonies. As the epizootic process was more stable in the narrow-band settlement, this type was used for experiments in the model. When the rate of epizootic contact was to equal one the epizootic process was registered by the minimum experimental density of colonies – 0.11 per ha. However the probability of long-term epizootic process was fewer less - 0.9-0.99.

Is persistence of plague possible when the number of great gerbil is in depression? It is an important question for a problem of an interepizootic plague save solution.

It is known that great gerbils' depression, inhabited colonies are often saved as groups which were situated into better ecological niches (Marin et al., 1970; Burdelov L.A. & Burdelov V.A., 1981; Dubrovskiy et. al., 1989). It was interesting that the number of colonies in separate groups were necessary for persistent plague epizooty. The colonies were arranged as a united group in the model space. The spacing among the colonies and the level (rate) of epizootic contact changed during experiments. Results demonstrated in the table 2.

The minimum number of colonies necessary for plague persistent during the modeling year was equal to 50 with maximum value of the probability of transfer success, the coefficient of the colony infection and the spacing among the colonies. After decreasing the spacing among the colonies to eight cells, the epizootic process was developed inside a group consisting of 40 colonies, but after decreasing the spacing to seven cells, 60 colonies were required. Each decrease in the spacing among the colonies (and increasing of plague contact) the number of colonies necessary for supporting plague epizooty increased. The spatial structure of epizootic process was reminiscent of a "layer cake" (fig. 15) or "wave" of infected colonies. The external layer is a layer of infected coloniesfollowed by a layer of immune colonies.

The "readied" colonies were arranged around the first infected colony inside the "Layer cake". The drift of plague microbe from infected to "readied" colonies was difficult. The irregularity of epizootic process, which is one of the main conditions of long existing epizooty, disappeared. Thus the epizootic process degenerated. If the rate of contact decreases, the effect of "wave" is neutralized. In total the plague can persist in a group of 25 colonies when epizootic contact rate is equal to one. The biology base of this effect was confirmed in nature. When great gerbils' density was in deep depression, the movement of rodents decreased and the level of epizootic contacts decreased too (Naumov et al., 1972).
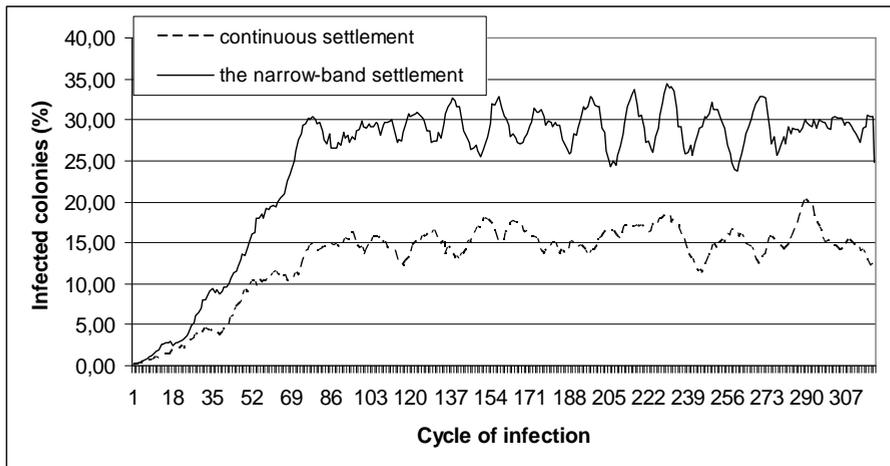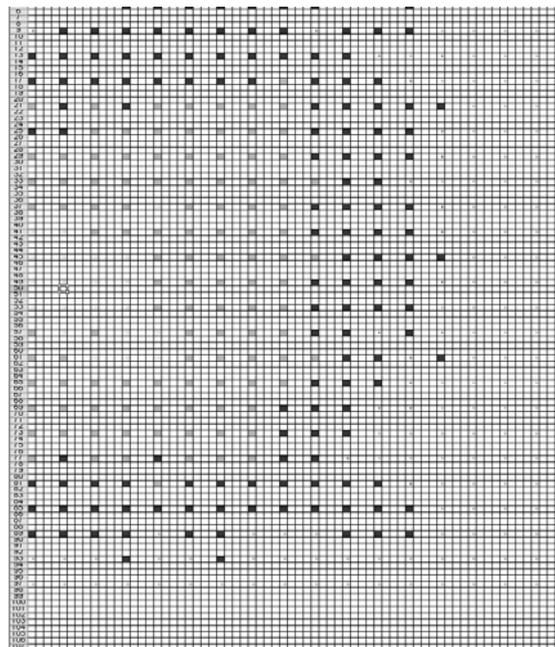

Fig. 14. The intensity of epizootic process in the different type of great gerbils' settlements in model. The density of narrow-band settlement is 0.44 colonies per ha, the density of the continuous settlement is 1.33 colonies per ha.

☐ - immune colonies, ■ - infected colonies

Fig. 15. The spatial development of epizooty at the high density of colonies and the highest epizootic contact rate.

The modelling showed that 25 colonies were enough for the plague to persist for a long time. The epizootic contact level had non-line (threshold) dependence from density and number of colonies.

The level of epizootic contact	The colonies quantity	The distance among colonies (cells)
10	50	10
10	40	8
10	60	7
10	50	6
10	70	5
10	400	4
6,48	60	4
1,2	40	2
1,14	30	2
1,0	25	0

Table 2. The level of epizootic contact, the distance among colonies and minimum of colonies quantity, when the plague persistence during 1 year period

4. Discussion

It is important to remember the following for a quality assessment of the modeling and real epizootic process. The investigation of the plague's spatial structure had shown that the small foci of plagued colonies represented pools of the neighbor colonies where there had been living sick or immune gerbils and their infested ectoparasites (Dubyanskiy, 1963; Klassovskiy et al., 1978; Rothschild, 1978; Rivkus et al., 1985). Each pool was irregularly located on the surface and separated by significant distance from other identical pools. It allowed for observing all phases of the epizootic process in a small focus at one time. Sick rodents were observed in some parts of colonies, while an increased fraction of immune rodents were observed in other parts of the colonies. Some colonies were without the plague and these were reserved for epizooty prolongation. All authors agreed on the irregularity of epizootic process in the small focus and permanent moving of epizooty in the territory of the plague focus.

The model-based process was analogous to the described-above epizooty at the small focus or at the pool of the small foci (so-called "epizooty spot"). The analogous types of colonies were present in the model. The well-defined pools of colonies (small foci) were separated from each other and non-uniformity distributed in the working space of the model. The speed of the model-based epizooty spreading coordinated well with field experiments through the marking of animals with radioisotopes and the direct observation of epizooty in nature. The cyclicity arising in the model simulated fluctuations of natural epizootic process intensities (Dubyanskiy and Burdelov, 2010) that confirmed an adequacy of the model-based process to real epizooty.

We have shown the possibility of modeling an annual epizootic cycle by changing the season's epizootic contact rate. This demonstrated the adequacy of the model in transmission theory framework. At the same time, the qualitative picture of epizootic cycle phases was simulated in the model, shown by an increase in the immune colonies' fraction after autumn or spring epizooty peaks. Even a minor increase (up to two) in the single parameter, coefficient of colony infection, was an important factor, as it led to epizooty outbreak. A doubling in the coefficient of colony infection could indicate a gerbil family's contact rate with other families as two times in 10 days instead of only one time. Our thinking, based on literature data, was that both values of the coefficient of colony infection (1 or 2) are usual for the density of a colony, which we used in the model. It demonstrates how minor fluctuations of the epizootic contact rate can lead to the strong changes in the epizootic situation. It is necessary to note that the endless epizootic process can develop even in a small model area. It can be considered as indirect acknowledgement of high survivability of a similar probabilistic process. It was correct for the real epizootic process at natural foci and it was often proved in practice.

It was quantitatively demonstrated in the big role of spatial distribution of great gerbils for plague epizootic. The various kinds of burrow systems constituting settlements, was marked by M. A. Dubyanskiy (1963, 1963a, 1970). He showed that lines of separate kinds of burrow systems played a different role in the plague spread. The modeling demonstrated spatial arrangement is very significant for plague epizootic too. It was known some plague foci had "band" settlements. These are "the foci from North desert subzone". The settlements from these foci are arranged along dry riverbeds, ravines, gullies. The plague in these foci was more intense and had more rare interepizootic periods as in the foci with the

"continuous" settlements (Aikimbaev et al., 1987). We believe the spatial kind of great gerbil settlements was a significant influence to this phenomenon.

The background epizooty of low density in the occupancy   colonies have proved the opinion that a negative result of surveillance can result not only from a real absence of plague in the focus, but also from insufficient intensity and extent of the surveillance (Dubyanskiy et al., 2007; Dubyanskiy, 2008). Culling surveillance area of plague foci is possible provided 0.1 – 0.6 colonies per ha.

When the great gerbils' density was set in the deep depression, the plague microbe was not detected. However, the plague epizooties were restarted after the gerbils' density recovered. Such "disappearing" of the plague microbe, resulted in a long discussion about routes of plague microbe conservation during interepizootic period. It was believed that the plague microbe requires specific mechanisms for conservation as a soil plague and the transmission mechanism by fleas was not effective (Akiev, 1970; Burdelov, 2001; Popov, 2002). Special works were made in Mangyshlak (Marin et al., 1970) in the South Balhash area (Burdelov L.A. & Burdelov V.A., 1981) and in Kizilkum (Dubrovskiy et al., 1989) it was shown that the occupancy colonies were arranged individually as a group, from five colonies to several. It is enough for plague microbes circulating for a long time. At the same time the most difficult to detect plague by unspecific surveillance because the groups of colonies were rare arranged on the biggest (more over 1000 square kilometers) area. Moreover the plague microbe was not conserved for every colony group. It is a cause, possibly, unregistering of epizooty and existing interepizootic periods. The modeling experiments demonstrated that the classical transmission theory can explain a complexity of plague detected during depression of gerbils and a beginnings of plague at a normal density of rodents.

The modeling allows searching numeric parameters of the epizootic process of plague. However, the simulation may appear a perspective method of forecasting plague epizootics, if applied along with permanent comparing and correcting of modeling results with the real surveillance situation.

The number of great gerbil burrow systems was nearest to natural data about quantity and distribution colonies during deep depressions of great gerbil's number. Thus, the permanent plague epizootics could occur even during deep depressions of great gerbils' number.

5. Conclusion

The model can be useful for studying the general appropriateness of the plague's epizootic process and for obtaining formerly inaccessible quantitative characteristics. It is the result of model simplicity, availability of high-performance computing workstations, model parameter input by anyone, and a large display area in the model. The model allows displaying a real and modeled structure of the rodent's settlement. Thus, it was possible to study the spatial regulation of the epizootic process depending on the density of colonies, distribution of colonies, intrasettlement migration, and so forth. Remote sensing methods and GIS can be used as a base for modeling. The basic characteristics of simulated epizootic processes qualitatively also are quantitatively close to natural analogues. It is important to emphasize the model is not constrained by species-specificity. It can be used for modeling epizootic processes among different species and for different vector-borne diseases.

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