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ENZOOTOGENESIS OF RODENT PLAGUE

Abstract. The article discusses the micro-focal structure of the plague infection enzootia. A hypothesis of enzootogenesis based on the "bottleneck" effect in population genetics is proposed. The phenomenon of preservation of the causative agent of the plague in soil amoeba during the inter enzootic period is discussed. The role of fleas in the transmission of a microbe is recognized as local. However, their significance is monopolistic in the epidemiology of the plague in bubonic form.

Key words: plague, micro-foci, landscape-epizootological region, enzootogenesis, population genetics, "bottleneck" effect, soil amoeba, flea.

Introduction. In the XIX century, it became clear that the plague infection or the causative agent of the plague is a bipolar microbe that is localized in certain territories. Later, in the 30s of the last century, Y.N. Pavlovsky formulated for the first time the concept of the natural focality of human and animal diseases as a separate section of the geographical landscape, together with a set of donor-hosts and vectors of the pathogen [7]. By hosts, he meant warm-blooded animals, and invertebrates were vectors. Wu-Lien-Teh polemicized this doctrine, expressing a certain skepticism regarding the phenomenon of the natural focus of the plague. His priority was the physiological state of rodents. In particular, the author believed that if rodents had absolute immunity, then there would have been no foci of plague infection [19].

In the post-Soviet space, the plague is the most studied. Over time, the natural foci of plague turned into an official administrative structure, a kind of natural and technogenic conglomerate formed as a product of normative decisions to streamline ongoing anti-epidemiological and preventive measures, taking into account the regional natural and geographical factor.

Hypotheses and theories of the phenomenon of natural foci of plague. Over the past decades, the spatial and biocenotic structures of enzootic territories have been studied. However, the deeper the epizootic and epidemic process was studied, the more questions arose. And the main one is where the plague microbe comes from, where it disappears and how it reappears. As a result, there are many hypotheses and theories trying to explain the phenomenon of focal plague. The main ones are:

- "nomadic" plague, characterized by continuous "waves" of epizootics and the remnants of the small foci of infections as sources of new epizootics[13];
- micro-focal plague, in the form of minimal areas, where the microbe is autonomously stored. For the first time substantiated by the types of marmot settlements [14];
- "telluric" plague in which there is a long-term preservation of the pathogen in virulent form in the substrate of rodent burrows[16,12];
- plague of blood-sucking ticks stored in the body of ixodic and other groups of arthropods [2];
- non-transmissible plague, excluding the role of fleas in the transmission of the microbe with intense, diffuse epizootics [10];
- latent plague associated with prolonged inter-epizootic periods, excluding the occurrence of epizootics[1];
- plague in the L-form, when periodically the R-form of the microbe is transformed into an inactive L-form [6];
- "bird" plague - transfer of the plague microbe from the active natural foci of infection by different types of birds [5].

It should be noted that currently there are about 30 different hypotheses and theories regarding the mechanism of enzootia of the studied infection.

Micro-focal structure of the plague. The enzootia of the rodent plague of Eurasia actually exists in the form of micro foci. In the landscape-ecological and epizootological aspect, it is located on the most optimal sites, where, due to the diversity of natural complexes, favorable conditions are created for the survival of rodent populations. Their settlements are the oldest, with a high density of holes, and the number of animals is stable [4]. In the populations, the classical course of the long-term dynamics of the number of small ground squirrel, midday, tamarix and great gerbils is expressed with differentiated cycles from depression to rise, peak and decline. And this is especially important for plague enzootogenesis.

Regarding the poorly studied micro-foci of plague in Karakum and Kopetdag (Turkmenistan), it should be noted that in general epizootic activity is low here and is similar to Southern Kyzylkum [8].

Of the 18 landscape ecological areas (LEAs) of the Karakum desert plague focus, the most active are the Zaunguz and Central Karakum with an epizootic index of 0.5. Slightly lower, 0.2 - in the Western Karakum and Sarykamysh cavity. In other LEAs, epizootics are extremely rare, with an epizootic index of about 0.1. And three of them - the Tedzhen-Murgaba interfluve, the Badkhyz highlands and the Karabil upland, with a total area of about 31.0 million hectares with different species of gerbils, are marked by the lack of registration of the plague microbe. A very low epizootic activity characterizes the Kopetdag desert focus, in two LEAs of which the corresponding index is below 0.1. In general, in the southern subzone of deserts, the micro-focality of plague is poorly studied. Presumably in Karakum there can be no more than two of them.

Table-Micro foci of plague in Kazakhstan and Central Asia

Name of the foci	Epizootic Index	Micro foci	Square, thousand ha	Long-term average abundance of the main source of infection
Ground squirrels foci of plague (small ground squirrel) Ural-Wilsky steppe foci	0,28	South Chelkar	80,0	High, 20 spc/ha
Gerbils foci of plague (small gerbils) Volga-Ural foci (great gerbil)	0,28	Becketai-Kamysh-Samarian	150,0	Average, 4-8 spc/ha
Ural-Emba desert foci	0,86-0,34	North-East Caspian	740,0	Average,5-10 spc/ha
		Lower Wilsky	370,0	Average,5-10 spc/ha
		Lower Embinsky	580,0	Average,5-10 spc/ha
Predustjurt desert foci	0,68	Ushkan	90,0	Low, 2-3 spc/ha
Ustjurt desert foci	0,40	Akzhigit	320,0	Low, 3-5 spc/ha
Mangyshlak desert foci	0,57	Mountain Mangyshlak	55,0	Low, 2-3 spc/ha
Aral-Karakum desert foci	0,38	Northeast Karakum	620,0	Low, 2-3 spc/ha
		Central Karakum	180,0	Low, 3 spc/ha
Aryskum-Daryalyktakyr desert foci	0,43	West Daryalykta	40,0	Low, 3-4 spc/ha
Karakum desert foci	0,1-0,5	Zaunguz-Karakum	?	Unstable, 6-18 spc/ha
		Central Karakum	?	Unstable, 6-18 spc/ha
Kyzylkum desert foci	0,24	Central Kyzylkum	250,0	Unstable
Moyynkum desert foci	0,40-0,70	Moyynkum-Saksauldalia	300,0	Low and average, from 3-5 to 10 spc/ha
Taukum desert foci	0,50	West Taukum	200,0	High 10-12 spc/ha

Notes. 1 - Epizootic index - the ratio of the number of years during which plague epizootics were recorded to the number of years of studies.

2 - In the Karakum desert foci, the mean annual abundance of large midday gerbils is given; unstable number, i.e. population is exposed to a deep depression.

It should be emphasized that the microcenters of great and are two-component: in the first of them there are areas with the most intense epizootic processes that surround areas with less intense epizootics. This can explain the stability of the functioning of the micro foci as a self-regulating ecosystem.

It must be noted that over time, under the influence of environmental and anthropogenic factors, the micro foci of the plague can change their sizes, move in space, or even completely disappear. So, in the last, more than 10 years, the Salt-marsh LEA of the Ural-Emba desert center has lost activity. Since 2004, it gradually disintegrated the role of continuous settlements of the large gerbil into separate fragments, often not related to each other [9]. According to the oral report of F.A.Sarayev, plague epizootics are still not recorded. The reason most likely lies in global warming. The average long-term air temperature in the long-term aspect of the Northern Hemisphere began to increase noticeably, starting from the 80s of the twentieth century. At the beginning of the XXI century, it reached an absolute maximum for 120 years of observation. And over the past 30 years, average air anomalies have gradually increased from 0.17 to more than 0.64 ° C [20]. Or almost 3.7 times.

Recent space images of the Ural-Emba interfluve and the Aral Sea Karakum from Google Earth show the most noticeable ecological destruction of the first region (light spots) and the development of similar processes in the second (Fig. 1). At the same time, plague epizootics in the Aral Karakum also started to decline, although in the active Central Karakum LEA in 2018, 5 strains from the great gerbil and 9 from fleas were isolated according to the Aralsea antiplague service.

A B

Figure 1 - Space images of the territories of the Ural-Emba (A) and Aral-Karakum desert foci of plague. The Salt-marsh and Central-Karakum LEAs are outlined

Hypothesis of endootogenesis of rodent plague. The hypothesis is based on animal population genetics [15]. The source of the causative agent of infection becomes an edificator species, the most numerous in natural complexes according to the law of Baitanayev's maximum in ecology [16, 3]. This law, in contrast to the Liebig minimum law, reflects the biology features of the mammalian fauna of different landscapes. The high abundance of the background species in the phase of the peak of abundance negatively affects the biology and ecology of the population. The stressful situation from overconsolidation causes disturbances in the genome. Then, when there is a decrease and depression in numbers, conditions for imbreeding arise, which leads to depletion of the gene pool. Since absolute genetic diversity is possible in the most numerous populations of free random crossing. As a result, there is an increase in homozygosity, changes in the frequency of genes appear, and the tendency is fixed at the loci of only one allele. A number of alleles drop out, for example, those responsible for the body's immunity against various infections.

Another significant concept, in addition to the above source of infection, is the host of the pathogenic microbe, which is inherent in the soil amoeba. The plague microbe is stored in soil amoeba cysts, which become protective reservoirs for the plague pathogen. This happens in rodent settlements, where the microbe is stored in cysts during the interepizootic period. A model of the mechanism of enzootogenesis is presented in Figure 2.

The hypothetical scheme of the members of the epizootic triad, taking into account the new one, therefore should look like this: rodent-microbe-amoeba. A flea falls out of it, which, obviously, is not the

cause of spontaneous, "explosive" plague epizootics in large areas. The flea factor is local, acting within a limited number of holes. Therefore, the place of a flea on a global scale belongs to the soil amoeba.

The significance of fleas is monopolistic in bubonic plague in the epidemiology of the studied infection. A flea is ineffective as a carrier in epizootics of a wide scale, but is most significant in the epidemiology of human bubonic plague.

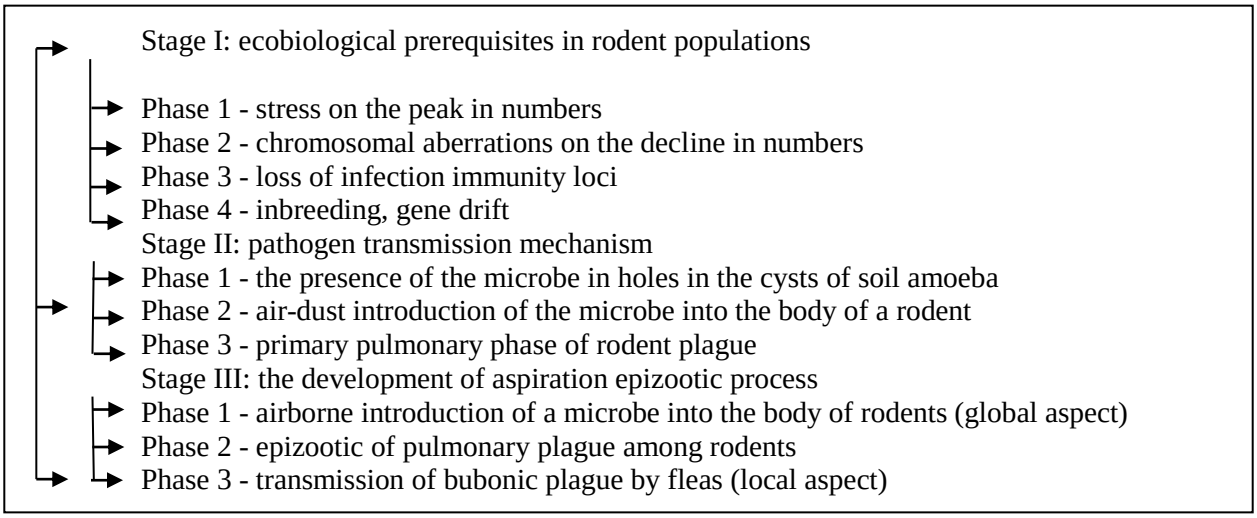

Figure 2 - Generalized scheme of the mechanism of plague enzootogenesis

Enizootogenesis is realized in three stages: from ecobiological prerequisites in rodent populations to the microbial transmission mechanism and the aspiration development of the epizootic process. Moreover, the primary infection of rodents with the pulmonary form of the disease. This can explain the explosive nature of the spilled, intense epizootics of the plague. And the transmission of it by fleas is less important.

After the publication of the hypothesis of enzootogenesis, the first articles related to this problem appeared. For example, in one of them the method of variation statistics considers the genetic component of the sensitivity of great gerbils to the plague pathogen [11]. And in the other - the authors sequenced the gene of the great gerbil from Western China, with its more than 96% coverage. The genetic variation of the MHCLL locus, which manifests itself at the population level to determine the importance of gene duplication in the resistance of the great gerbil to the plague microbe, was studied [18].

Conclusion. Studies of the genetic diversity of rodent sources of plague infection are at early stage. The full implementation of the main provisions of the hypothesis of enzootogenesis will make it possible to uncover the real causes of the disappearance and occurrence of the plague and to carry out reorganization on the basis of innovative technologies.

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КЕМІРГІШТЕР ОБА АУРУЫНЫҢ ЭНЗООТОГЕНЕЗІ

Аннотация.Мақалада оба жұқпалы ауруының шағын ошақтық құрылымы қарастырылған."Бөтелке құсығының" асері негізіндегі энзоотогенез гипотезасы ұсынылған. Эпизоотия аралығы кезенде микроб топырақты амебада сақталу ерекшелігі талқыланған. Трансмиссивтік табыс ету саласындағы бүргенің рөлі жергілікті болып табылады. Бірақ бубондық оба эпидемиологиясында олардын маңыздылығы монополиялық.

Түйін сөздер: оба, шағын ошағы, ланшафттық-эпизоотологиялық ауданы,энзоотогенез, популяциялық генетика,"бөтелке құсығының" әсері, топырақ амебасы,бүрге.

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ЭНЗООТОГЕНЕЗ ЧУМЫ ГРЫЗУНОВ

Аннотация. Рассматривается микроочаговая структура энзоотии чумной инфекции. Предлагается гипотеза энзоотогенеза на основе эффекта «бутылочного горлышка» в популяционной генетике. Обсуждается феномен сохранения возбудителя чумы в почвенной амебе в межэнзоотический период. Роль блох в трансмиссивной передаче микроба признается локальной. Однако их значимость монопольна в эпидемиологии чумы при возникновении ее бубонной формы.

Ключевые слова: чума, микроочаг, ландшафто-эпизоотологический район, энзоотогенез, популяционная генетика, эффект «бутылочного горлышка», почвенная амеба, блоха.

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