

INTERRELATIONS BETWEEN MAMMALS AND PATHOGENS OF SOME BACTERIAL DISEASES*)

T. N. DUNAEVA

The Gamaleya Institute of Epidemiology and Microbiology, the USSR Academy of Medical Sciences, Moscow

Abstract. Various interrelations of mammals with some bacterial pathogens are analyzed. Plague and tularemia are the most thoroughly investigated infectious diseases in this respect. The importance of a different degree of susceptibility and sensitivity to infection for its development in mammalian reservoirs is underlined.

Many bacterial diseases occur among mammals in nature. The evaluation of the epizootiological and epidemiological importance of the animals cannot be reliable without elucidating interrelations between the host and the pathogen, which determine the pathogenesis of the disease and the possibility of transmitting the pathogen to other animals, mammal species, vectors and human beings.

Detailed studies on the relationship of mammals to diseases were carried out in the research on plague and tularemia and served as a basis for taking definite viewpoints on the nature of interrelations between the host and the pathogen. The studies of tularemia were used as a model for working out the methodology of experimental research and for formulating the concepts characterizing the animal-infection relationship. Three types of the relationship of mammals to tularemia were established and the species involved were divided into groups differing in their degree of susceptibility and sensitivity to infection: I — highly susceptible and highly sensitive species; II — highly susceptible species of low sensitivity; and III — practically insensitive species of low susceptibility (Olsuf'ev et al. 1950, Dunaeva 1964). This classification has been also adopted for other infectious diseases.

The degree of susceptibility is determined by the size of the smallest infectious dose or of the smallest complete dose causing the disease when a definite way of infection is used. Highly susceptible species get infected when minimal doses of pathogen, i.e. single bacteria, are introduced parenterally. Animals of low susceptibility get infected only when considerable doses, sometimes exceeding thousands and millions of microbes, are introduced. The sensitivity of animals is characterized by the gravity of the disease development and by its outcome. When determining the degree of infectious sensitivity different indices are used: the size of a complete lethal dose DCL (tularemia), $DL_{50\%}$, the percentage of the animals perished, the time of death (plague), the duration of pathogen-carrying period (leptospirosis), the index of the distribution of bacilli in organs (brucellosis), and others.

Highly susceptible and highly sensitive species do not possess effective protective mechanisms and any infectious dose causes a lethal issue in them. This type

*) Presented at the First International Theriological Congress in Moscow, June 6—12, 1974.

Table 1. Lethality of highly sensitive animals experimentally infected (subcutaneously) with pathogens of some infectious diseases. After: Antsiferov and Brikman 1969, Biryukova 1960, Vasilev et al. 1960, Dunaeva 1967, 1964, Mkrtchyan et al. 1963, Aikim-baev 1967.

Doses	Tularemia				Listeriosis				Plague			
	<i>Arvicola terrestris</i>	<i>Lagurus lagurus</i>	<i>Lemus timidus</i>	<i>Marmota baibacina</i>	<i>Lagurus lagurus</i>	<i>Meriones meridianus rogniorum</i>	<i>Meriones tamarensis</i>	<i>Lagurus lagurus</i>	<i>Citellus citellus xanthopygmnus</i>	<i>Meriones meridianus hepularis</i>	<i>Rhabomys opimus</i>	<i>Spermophilopsis leptodactylus</i>
1 · 10 ⁻¹	154/114	7-13	126/62	7-13	7/6	7-12	7-10	& 18*	8/8	11-25	—	—
1 · 10 ⁰	46/46	6-11	20/20	7-8	18/18	7-10	7-10	—	8/8	12-17	3/2	—
1 · 10 ¹	32/32	6-10	20/20	6-8	11/11	7-10	8/8	—	8/8	19/18	7-8	7/5
1 · 10 ²	—	—	—	4/4	6-7	—	8/8	9-19	21/21	2-8	28/28	3-8
1 · 10 ³	—	—	—	—	—	—	8/8	5-16	19/18**	3-6	20/20	3-5
1 · 10 ⁴	—	—	—	—	—	—	8/8	9-12	21/21	3-5	102/102	1-5
1 · 10 ⁵	—	—	—	—	—	—	8/8	6-11	36/36	3-5	7/7	2-3
											14/14	2-3

* Spontaneous infection with pseudotuberculosis was found (Dunaeva 1969).

** Spontaneous infection in the surviving *Lagurus* with streptococcus was found.

First column — the numerator indicates the number of infected, the denominator indicates the number of dead animals. Second column — time of death in days.

Table 2. Lethality of animals of low sensitivity experimentally infected (subcutaneously) with pathogens of some infectious diseases. After: Abushev 1962; Akhundov 1969; Busoedova and Antipova 1962; Dunaeva 1964; Dunaeva and Pshenichnaya 1953; Volkov and Vasilev 1960; Yermilov 1967; Tsibulevskaya 1960

Doses	Tularemia				Listeriosis				Plague			
	<i>Apodemus agrarius</i>	<i>Citellus pygmaeus</i>	<i>Dryomys nitedula</i>	<i>Hemicheirus auritus</i>	<i>Apodemus agrarius</i>	<i>Meriones ligatus</i>	<i>Apodemus agrarius</i>	<i>Meriones meridianus hepularis</i>	<i>Rhabomys opimus</i>	<i>Spermophilopsis leptodactylus</i>	<i>Rhabomys opimus</i>	<i>Spermophilopsis leptodactylus</i>
1 · 10 ⁰	20/-	—	10/2*	11-14	5/-	2/1	—	—	10/-	7/-	—	—
1 · 10 ¹	20/-	—	10/2*	5-13	5/-	2/-	—	—	10/2	5/1	12/2	—
1 · 10 ²	20/1	8	—	—	5/-	—	—	—	10/3	6/1	12/2	—
1 · 10 ³	10/-	—	36/1	9	5/-	2/1	—	—	10/3	7/2	12/3	30/-
1 · 10 ⁴	10/-	—	36/11	8-12	5/-	2/-	20/1	3/2	10/4	7/3	12/3	30/1
1 · 10 ⁵	15/3	7-11	36/16	5-17	—	2/-	20/-	3/-	10/4	7/2	12/3	3/-
1 · 10 ⁶	15/12	4-8	36/22	6-16	5/1	3/-	20/1	3/1	10/4	7/2	12/6	30/1
1 · 10 ⁷	15/12	4-7	36/28	5-12	4/1	2/-	20/5	3/2	10/10	7/4	12/7	30/3
1 · 10 ⁸	24/24	2-5	36/34	3-13	4/4	2/1	20/13	6/6	10/10	7/7	12/7	30/5
1 · 10 ⁹	8/8	2-3	36/36	2-5	4/4	—	20/16	—	10/10	—	12/9	30/5

* The susliks died of mixed infection (Dunaeva and Pshenichnaya 1953).

** The virulence of the strain for white mice: DCL — 10 microbe cells (Yermilov 1967).

** The virulence of the strain for white mice: DCL — 1,000 microbe cells (Novikov 1972).

First column — the numerator indicates the number of infected, the denominator indicates the number of dead animals. Second column — time of death in days.

of interrelations is characteristic of many species of rodents, hares, insectivores when they are infected with tularemia (Dunaeva 1963), but it occurs less often with other diseases such as plague, listeriosis, salmonelloses (Biryukova 1960, Vasilev et al. 1960, Kucheruk 1965, Holdenried and Quan 1956, Stagg et al. 1956, Seeliger 1955). An acute development of disease, the death of the animals soon after infection, intensive septicemia and bacteremia are typical. The intensity of septicemia does not depend on the infectious dose with the species which are highly sensitive to tularemia. The same appears to be observed with plague as well (Dunaeva 1954, Malafeeva 1959, Kletz et al. 1959, Aliev and Zakutinskaya 1961).

Several species highly sensitive to tularemia are known (*Allocricetus curtatus*, *Marmota baibacina*, *M. caudata*, guinea-pigs), that are characterized by some peculiarities of pathogenesis, indicative of some protective reactions in response to the development of infection. A lesser intensity of septicemia depending on the infectious dose as well as a prolonged development of disease when small doses have been applied are characteristic of them. Antibodies to tularemia microbes are found in the animals which have died later than 13—15 days after infection.

Species of low sensitivity have the same degree of susceptibility but possess a genetically determined ability to form specific protective immunoreactions which ensure the recovery of animals under certain conditions of interrelation between pathogenic properties and the dose of pathogen. This type of interrelation is common among rodents infected with plague as well as with some other infectious diseases such as tularemia, erysipeloid, listeriosis, pasteurellosis and others.

The gravity of the disease development, the intensity of septicemia, the death of the animals depend on the size of the infectious dose. When sublethal doses are used for infection, an immunity is formed that is accompanied by bacteria-carrying at first and later becomes sterile. Tularemia and plague bacteria are regularly found in animals of low sensitivity up to the 15th—20th day after infection. The pathogen is later detected in an insignificant number of animals and is localized in lymph nodes or abscesses, and in lungs of some species. Generalization of tularemia infection has not been observed (Dunaeva 1964). The virulence of the tularemia microbe cultures isolated from immune animals is not different from that of the initial one. Cases of infection generalization at a later date were sometimes observed with plague. Changes in the virulence of some subcultures isolated from immune animals were established; this is associated with the loss of the ability to synthesize fraction 1 (Petrunina 1951, Levina 1960, Punskey et al. 1972).

Infection of practically insensitive animals of low susceptibility with small doses does not cause the disease while after massive doses the infection is usually symptomless. The animals do not die even after being infected with tremendous doses of pathogens amounting to tens of milliards of bacteria. The poor ability of the pathogens to adapt themselves to the environment and their rapid elimination from the organism seem to be associated with the high activity of non-specific protective reactions (cellular and humoral) which ensure a rapid trapping and digestion of microbes. Such a type of relationship to tularemia infection has been established for carnivores of the families Mustelidae, Canidae, Felidae (Křivinka 1939, Dunaeva 1954, Girard 1955) and for ungulates (Yershova 1964, Valtonen 1969). Experimental studies of the pathogenesis of any infectious diseases in the species of this group have not received much attention due to their insignificant role in epizootiology. Such studies would be useful to reveal the mechanism of natural immunity manifestation.

The character of the animal-infection relationship is revealed only in the interaction with a definite pathogen. The same animal species reacts differently to different infections and even to the infection with strains of one microbe species, but possessing

various properties. For example, rabbits (*Oryctolagus cuniculus*) and American wild rabbits—cottontails (*Sylvilagus floridanus*) show a low sensitivity to tularemia caused by the strains of *Francisella tularensis holarktika*; in this case DCL equals 100 million—1 milliard microbes. But when infected with nearctic strains (*F. tularensis nearctica*) the animals die after a dose of a single microbe cell (Dunaeva 1970). Considerable lability of the antigenic structure of plague microbes determines the selective virulence of the strains, which have lost one or another virulence determinant, in respect to different species of laboratory and wild animals (Burrows and Bacon 1956, Burrows 1963, Peisakhis et al. 1969, Kudinova 1972).

The determination of the stability of the character of the host-pathogen interrelations is an important question disputable to a certain degree. The studies on tularemia have shown that the adherence to the group of either high or low sensitivity animals is a feature typical of the species, which is not subject to considerable fluctuations depending on the sex or age of the animals, nor on the seasonal changes in their physiological state including such drastic changes as hibernation. This peculiarity of tularemia is determined by the stability of the virulent properties of the pathogen, its high invasive capability ensuring a rapid development of infection in animals. A high immunogeneity of the tularemia microbe naturally causes the development of acquired immunity in the species of low sensitivity.

Many researchers are of an opinion that there is a considerable variability of the relationship of rodents to plague. Statements have been even made that within the population of one species there are animals of high, low and practically no sensitivity to the infection, i.e. there are animals possessing different types of relationship with the pathogen (Malafeeva 1959, Kalabukhov 1959, 1969, Rall 1958).

An analysis of the data found in literature, however, shows that no considerable changes are observed in the course of the infection process in the non-hibernating highly sensitive species in case of plague. *Mus musculus*, *Microtus minutus*, *Lagurus lagurus*, *Meriones tamarii*, *M. liticus* and *M. meridianus* preserve their high sensitivity throughout the seasons (Biryukova 1960, Vasilev et al. 1960, Yolkin 1960, Mamed-Zade et al. 1959).

The assertions of some authors about the seasonal shifts of infectious sensitivity to plague of non-hibernating species of low sensitivity have not been confirmed by experiments carried out on a higher methodical level and with a large number of animals. Thus in the experiments of Lavrentev et al. (1967) on *Meriones meridianus heptneri* 400 animals were infected in spring and 300 animals in autumn with same dose of 10 million microbe cells, the mortality rate being 52 and 57 per cent respectively. In the experiments of Novikov (1972) *Rhabdomys opimus* were infected with plague bacteria doses ranging from one cell to one milliard microbe cells in spring and in autumn. Of the 210 animals in each batch 11.4 and 9.0 per cent died. No changes in the infection sensitivity of great gerbils were observed when they were infected in different seasons by the bites of "blocked" fleas (Rivkus et al. 1974). No seasonal differences in the sensitivity to plague have been observed in *Apodemus agrarius*. The findings of Tinker and Kalabukhov (1934) concerning different infectious sensitivity of young and adult *Citellus pygmaeus* to plague have not been confirmed (Valkov 1965, Shiryaev et al. 1965, Akiev et al. 1972).

With species of low sensitivity the gravity of the clinical symptoms of the disease and the latter's outcome depend on the interrelation between pathogenic properties and dose of the pathogen on the one hand and the macroorganism's immunological preparedness on the other hand. Therefore, it is natural to observe some fluctuation in the gravity of the disease development and in the range of incomplete lethal doses which cause some animals to die while others recover. The time of death of animals, the period of bacteria-

carriage may be different in various animals because they depend both on the speed and on the level of development of acquired immunity. The factors which suppress the organism's immunological activity (inferior nourishment, pregnancy, some intercurrent infections) aggravate the development of the disease and may cause the animal's death, an increase in the septicemia intensity and prolongation of the bacteria-carrying period even when the animal is infected with a small dose. And vice versa, an increase in the animals' immunological reactivity contributes to their recovery (Aisely 1956, Dunaeva 1955, Kletz et al. 1959, Kalabukhov 1969).

Different manifestations of immunoreactions typical of the species do not give any grounds for considering some animals as highly sensitive and others with low sensitivity, i.e. showing a different type of relationship with the pathogen. As stated above, the difference between the types of relationship with the pathogen lies in the fact that when highly sensitive species are in the normal (original) immunological state they lack specific protective reactions while species of low sensitivity are able to form specific acquired immunity.

The immunological reactivity typical of the species can be changed under the influence of factors directly affecting the immunogenesis mechanisms, primarily when immunization has been done with weakened bacteria of the same type or when the infection has been caused by sublethal doses of virulent strains (in species with low sensitivity). Deviations from the typical course of plague or tularemia can be also caused by previous infections of the animals with pathogens of other diseases, such as listeriosis, salmonellosis, brucellosis, pseudotuberculosis and others (Aparin 1959, Domoradsky et al. 1960, 1962, Gubina and Dunaeva 1963, Dunaeva 1970, 1972, Owen and Larson 1956, Henderson 1960).

When species of high sensitivity to tularemia are subjected to mixed infection, a prolonged period before death and a decrease in the septicemia intensity are observed, which is caused by intensification of the phagocytic activity of cellular elements. Rare cases of recovery of guinea-pigs from tularemia have been observed, the bacteria-carrying period being as long as 54—110 days. This is indicative of the weak development of specific immunoreactions. In species of low sensitivity to tularemia the stimulation of non-specific protective mechanisms, such as phagocytosis, production of plasmatic cells, etc., caused by antigenic irritations creates favourable conditions for a speedy manifestation of specific immunoreactions. This ensures the survival of some animals when infected with complete lethal doses. Rapid elimination of bacteria from the organs is observed in the survived animals.

The manifestation of unspecific immunity is more pronounced in cases of plague than tularemia including highly sensitive species. Cases of survival of white mice, guinea-pigs previously infected with some pathogens, including conditionally pathogenic, have been observed more frequently. Apparently a decreased adaptation of plague bacteria to the environment is observed when the general immunity mechanisms (primarily phagocytosis) are activated, and some animals do not become infected (Goldfarb et al. 1972, Korobkov et al. 1972).

The relationship of hibernating rodents to plague undergoes rapid seasonal changes. Without going into a detailed analysis of this phenomenon which has been studied by many authors I shall note that when awake, different hibernating species of susliks (*Citellus pygmaeus*, *C. fulvus*, *C. citellus*) and marmots (*Marmota baibacina*, *M. caudata*) are highly sensitive to plague and die of acute infection when infected with small infectious doses. But even at this time cases of prolonged duration of the disease with fatal outcome, or survival of some animals are observed. Only in some of the survived animals the past plague infection can be demonstrated by isolating the pathogen or by serological tests. The resistance to infection is increased in the period of preparation for

hibernation, and during hibernation of the animals. Some of the animals perish soon after they wake up in spring when generalization of plague occurs (Gaisky 1944, Makarov and Makarova 1957, Peisakhis 1958, 1963 and others). In the greater number of the survived animals bacteriological or serological methods do not reveal that they have had the plague. Some authors presume that in some animals the incubation period of the disease is prolonged due to unfavourable conditions for the pathogen's accumulation in the organism and due to the low body temperature of the animals. In others the infection does not develop due to the poor adaptation of the microbes to the environment in the animals during hibernation (Peisakhis and Surmina 1961, Dobrokhотова 1965, Kalabukhov 1969).

Here it is necessary to stress the importance of phenotypic changeability typical of the plague pathogen which readily changes its antigenic structure and aggressive properties under the influence of the environment. The combination of the changes in the physiological state and reactivity of the macroorganism on the one hand, and the properties of the pathogen on the other, determine the peculiar features of the development of plague in hibernating rodents.

The ways of disease transmission. The effective transmission of pathogen from a sick animal to a healthy one, either directly or through vectors, depends on the threshold of susceptibility corresponding to any natural way of infection and on the disease pathogenesis responsible for the intensity of the accumulation of bacteria in the infected material such as the animals' organs, excretions, blood. The susceptibility threshold differs with different ways of the pathogen's penetration. Experiments with tularemia, plague, listeriosis and some other diseases have shown that microbes readily adapt themselves to the environment when they are introduced sub- or intracutaneously, which corresponds to the infection transmitted by blood-sucking arthropods. In this case the infectious dose amounts to several bacteria. The susceptibility threshold is somewhat lower with aspirational or conjunctival penetration of the microbe, the infectious dose being 100—1,000 bacteria. In the populations of wild animals in nature there are no conditions which can make aspirational infection possible. Conjunctival infection with tularemia is possible when the animals get in contaminated water (Dunaeva 1959). Alimentary infection occurs when massive doses of pathogen amounting to 10—100 million microbe cells are swallowed (Korobkova 1928, Dunaeva 1954, Ogneva 1964, Burdo 1965, Adamov et al. 1969).

Low susceptibility of animals to alimentary infection is compensated for some infectious diseases by longer periods of bacteria elimination with the sick animals' excretions and by bacteria accumulation in the environment due to their ability for prolonged survival and even for multiplication (*Listeria*, *Erysipelothrix*, *Leptospira*). In the case of tularemia alimentary way of transmission is strictly dependent on the intensity of the accumulation of bacteria in the sick animals' excretions and regularly occurs only in highly sensitive species. The same regularity applies to other infectious diseases such as listeriosis and plague (Nikanorov 1925, Volosovitz 1959, Ogneva 1964).

The infection of vectors is determined by the bacteriemia in the animals which are infection donors. Further existence of the microbe in the vector and the peculiarities of the excretion mechanism determine the effective transmission of the disease. These interrelations have been studied in detail in cases of plague and tularemia which have their specific vectors, the fleas and ixodid ticks.

All phases of the development of ixodid ticks are capable of accepting the tularemia pathogen while feeding on sick animals; of transmitting bacteria during metamorphosis and of maintaining them for life. The multiplication of bacteria in ticks occurs during their feeding on animals. During metamorphosis the number of bacteria in ticks is

reduced 100—1,000 times. The transmission is brought by tick bite and by contamination with intensively infected tick excrements. While feeding on animals with weak bacteriemia hungry ticks (after moulting) may become completely free of the pathogen or the degree of their infection is insufficient for the bacteria to penetrate the haemolymph or salivary glands (Olsuf'ev and Petrov 1967). This feature determines the main role which highly sensitive animals play in infecting the ticks because when the latter feed on animals of low sensitivity they obtain a small number of bacteria and become free of them during metamorphosis (Petrov and Dunaeva 1955, Aikimbaev et al. 1967). Tularemia bacteria do not multiply in fleas and are rather rapidly eliminated along with excrements.

In case of plague other interrelations are formed with some species of fleas (genera *Xenopsylla*, *Ceratophyllus*, *Neopsylla*, *Oropsylla*) parasitizing the main hosts i.e. susliks, marmots, gerbils. In plague-infected animals, even of high sensitivity, bacteriemia is not so intensive as in case of tularemia. The number of plague bacteria in one milliliter of the blood of infected white mice ranges from 10 thousand to 10 million (Tiflov 1960), while in case of tularemia it amounts to 100 million — 1 milliard. Sixty plague bacilli have been counted under microscope in a blood smear of white mice, 90—120 bacilli have been observed in the blood smear of highly sensitive *Meriones libycus*, while in that of *Rhombomys opimus* infected with massive doses less than 20 bacilli have been found (Bibikova et al. 1967, Bibikova 1968). The bacteria multiply in fleas and are harboured by them for a long period. Their multiplication continues even when the fleas have been fed no additional blood (Alutin and Sorokina 1970). The flea obtains from single to several thousand bacteria with the blood of sick animals. The bacteria multiply in the stomach and forestomach blocking the latter and obstructing its valvular action. Regurgitation of the infected blood into the bite wound occurs when "blocked" fleas attempt to suck the blood of animal recipients (Ioff 1941).

Thus, contrary to tularemia where the principal factor is the intensity of bacteriemia in animal donors, in case of plague the effective transmission depends more on the conditions of accumulation and localization of the pathogen in the vector. Therefore the mammal species less sensitive to plague and with insignificant bacteriemia have been able to become the main hosts of the disease. There is no doubt that highly sensitive species are of great importance in infecting the fleas. As observed both in experiments and in field studies (Mikulin et al. 1959, Osolinker and Soletsky 1965). It is possible that plague cannot be maintained for a long time in the population of highly sensitive animals not only because the animals quickly die off but also because a large number of their fleas perish as well, due to massive infection and to the ensuing blockage in a great number of specimens.

The above materials show that the interrelations between warm-blooded hosts and pathogens represent a complicated problem in which general regular and peculiar features caused by specific characteristics of both partners may be outlined.

ВЗАИМООТНОШЕНИЯ МЛЕКОПИТАЮЩИХ И ВОЗБУДИТЕЛЕЙ НЕКОТОРЫХ БАКТЕРИАЛЬНЫХ ИНФЕКЦИЙ

Т. Н. Дунаева

Резюме. Даётся анализ разных взаимоотношений между млекопитающими и возбудителями некоторых бактериальных инфекций. Наиболее широко в этом отношении изучены чума и туляремия. Выдвинуто значение разной степени восприимчивости и чувствительности к инфекции для развития болезни у млекопитающих-резервуаров.

REFERENCES

Abushev F. A., Trudy Azerb. protivochum. stantsii 3: 308–319, 1962. — Adamov A. K., Naumov A. V., Shmerkevich D. L., Problemy osobo opasnykh infektsiy 5: 114–116, 1969. — Aikimbaev M. A., Tularemiya v Kazakhstane. Diss. dokt., Alma-Ata 1967. — Akiev A. K., Bratkova M. N. et al., Problemy osobo opasnykh infektsiy, 5: 101–105, 1972. — Alutin I. M., Sorokina L. Ya., Sb. Perenoschiki osobo opasnykh infektsiy i borba s nimi. Stavropol, pp. 15–21, 1970. — Aparin G. P., Izv. irkutsk. gos. nauchn. issled. protivochum. inst. Sibiri i Dalnego Vostoka 21: 178–180, 1959. — Bibikova V. A., Vzaimootnosheniya perenoschika i vozbuditela bolezni pri chumce. Diss. dokt. Alma-Ata, 1968. — Bibikova V. A., Kozachenko A. I. et al., Materialy V. nauchnoi konf. protivochum. uchrezhd. Sredney Azii i Kazakhstana, pp. 270–272, 1967. — Biryukova E. S., Trudy nauchn. issled. protivochum. inst. Kavkaza i Zakavkazyia 4: 84–94, 1960. — Burrows T., Ergebn. Microbiol. Immunitäts 37: 59–113, 1963. — Burrows T., Bacon G., British. J. Exper. Pathol. 39: 278–291, 1958. — Busoedova N. M., Antipyeva O. A., Doklady Irkutsk. protivochum. inst. 3: 112–114, 1962. — Dobrokhотова N. D., In: Epidemiologiya i epizootologiya osobo opasnykh infektsii, Moskva, pp. 245–249, 1965. — Domoradskiy I. V., Vozbuditeli pasterellezov i blizkikh k nim zabolеваний. Moskva, 1971. — Dunaeva T. N., Eksperimentalnye issledovaniya tularemii dikkh zhivotnykh (gryzunov, nasekomoyadnykh i khishchchnykh) i ich znachenie v izuchenii prirodnnykh ochagov. Diss. dokt., Moskva, 1964. — Dunaeva T. N., Zool. zh. 42: 727–742, 1963. — Dunaeva T. N., In: Prirodnaia ochagovost, epidemiologiya i profilaktika tulyaremii. Moskva, pp. 22–37, 1970. — Dunaeva T. N., In: Itogi razvitiya ucheniya o prirodnaya ochagovosti bolezney cheloveka i dalneishie zadachi. Moskva, pp. 88–107, 1972. — Elkin Yu. M., Trudy nauchn. issled. protivochum. inst. Kavkaza i Zakavkazyia 4: 95–107, 1960. — Elkin Yu. M., Vasilev N. V., Ibid 115–122. — Ermilov A. P., Medvedevskii V. I., Kamennova L. S., Materialy V. nauchnoi konf. protivochum. uchrezhd. Sredney Azii i Kazakhstana, pp. 284–285, 1967. — Ershova L. S., Kleshchi *Ornithodoros lahorensis* i *O. papillipes* kak khraniteli i perenoschiki tularemijnogo mikroba. Diss. kand. Alma-Ata. — Gaiskiy N. A., Izv. Irkutsk. nauch. issled. protivochum. inst. Sibiri i Dalnego Vostoka, vol. 5, 1944. — Girard G., Bull. Acad. Veter. 28: 103–112, 1955. — Goldfarb L. M., Gerasimenko R. T. et al., Problemy osobo opasnykh infektsiy 5: 114–117, 1972. — Golovacheva V. Ya. Doklady Irkutskogo protivochum. inst. 4: 202–207, 1962. — Henderson D., Bacterial Reviews 24: 21, 1960. — Holdenried R., Quan S., Publ. Health Rep. Wash. 71: 10, 1956. — Ioff I. G., Voprosy ekologii blokh i svyaz s ikh epidemiol. znacheniem. Pyatigorsk, 1941. — Kalabukhov N. I., Sb. Prirodnaia ochagovost i epidem, osobo opasnykh infekts. zabolеваний. Saratov, pp. 372–386. — Kalabukhov N. I., Periodicheskie izmen. v organizme gryzunov. Leningrad, 1969. — Kartman L., Prince F., Quan S., Starc H., Ann. N. Y. Acad. Sci. 70: 3, 1958. — Klets E. I., Khrustselevsky V. P. et al., Izv. Irkutsk. nauchn. issled. protivochum. inst. Sibiri i Dalnego Vostoka 14: 3–18, 1957. — Klets E. I., Shechekunova E. I., Padalko Z. F., Ibid 21: 92–97, 1959. — Křivinka J., Spisy vysoké školy zvěrolékařské, Brno, 131–261, 1939. — Kudinova T. P., Problemy osobo opasnykh infektsiy 5: 118–121, 1972. — Kucheruk V. V., Fauna i ekologiya gryzunov 7: 5–85, 1965. — Lavrentov A. F., Kranousova N. V. et al., Materialy V. nauchn. konf. protivochum. uchrezhdennii Sredney Azii i Kazakhstana, pp. 289–290, 1967. — Lovina A. A., Voprosy prirod. ochagovosti i epizootol. chumy v Turkmenii. Ashkhabad, pp. 241–270, 1960. — Makarov N. I., Makarova E. P., Izv. Irkutsk. nauchn. issled. protivochum. inst. Sibiri i Dalnego Vostoka: 83–87, 1957. — Malafeeva L. S., Trudy inst. "Mikrob" 3: 235–241, 1959. — Mamed-Zade U. A., Mikhailova R. S., Petrosyan E. A., Prirod. ochag. i epidemiol. osobo opasnykh infektsion. zabolev.. Saratov, pp. 387–398, 1959. — Mikulin M. A., Zagniborodova E. N. et al., Voprosy prirod. ochagovosti i epizootol. chumy v Turkmenii. Ashkhabad, pp. 22–49, 1960. — Mkrtchyan S. A., Vartanyan A. A. et al., Trudy Armyanskoy protivochum. stantsii 2: 23–41, 1963. — Novikov G. S., Problemy osobo opasnykh infektsii 1: 91–99, 1972. — Olsufev N. G., Dunaeva T. N., In: Tularemiya pp. 96–206, 1960. — Olsufev N. G., Dunaeva T. N., J. Hyg., Epidem., Mierobiol., Immunol. 5: 409–422, 1961. — Olsufev N. G., Petrov V. G., In: Biolog. vzaimootn. krovosos. chlenistonogikh s vozbud. bolezney cheloveka. Moskva, pp. 200–218, 1967. — Osolinker B. E., Soletskii G. K., In: Epidemiologiya i epizootologiya osobo opasnykh infektsiy. Moskva, pp. 342–349, 1965. — Owen C., Larson C., J. Exper. Med. 193: 753–763, 1956. — Peysakhis L. A., Trudy Sredne-Aziatskogo nauchn. issled. protivochum. inst. 4: 81–99, 1958. — Peysakhis L. A., Surmin V. M., In: Surki, ekologiya, ektoparazity, prirodnaia ochagovost chumy. Alma-Ata, pp. 173–176, 1961. — Petrunina O. M., Trudy Sredne-Aziatskogo nauchn. issled. protivochum. inst. 1: 17–25, 1951. — Rall Yu. M., Prirod. ochag. i epizootol. chumy. Moskva 1965. — Seeliger H., Listerioze. Leipzig 1955. — Shiryaev D. T., Semenov H. M. et al., In: Epidemiologiya i epizootologiya osobo opasnykh infektsiy, pp. 103–111, 1965. — Stagg G., Tanner W., Lavender H., J. Inf. Dis. 99: 34–37, 1956. — Tiflov V. E., Trudy nauch. issled. protivochum. inst. Kavkaza i Zakavkazyia 4: 15–35,

1960. Tsibulevskaya F. S., Voprosy prirod. ochagovosti i epizootologii chumy v Turkmenii. Ashkhabad 277-279, 1960. — Valkov B. G., Sbornik nauchnykh rabot Elistinskoy protivochum. stantsii 2: 147-164, 1961. — Valtonen M., Suomen Elainlaakarilehti 75: 497, 1969. — Vasilev N. V., Elkin Yu. M., Zyuzin A. S., Trudy nauchn. issled. protivochum. inst. Kavkaza i Zakavkazyia 4: 123-126, 1960. — Volosovits I. F., Nauchn. konf. protivochum. uchrezhd. Kazakhstana i Sred. Azii. Alma-Ata, 29-30, 1959.

Received 1 April 1974.

T.N.D., The Gamaleya Institute of Epidemiology and Microbiology, USSR Academy of Medical Sciences, Moscow, USSR

FOLIA PARASITOLOGICA (PRAHA) 22: 161, 1975.

The 50th birthday of Associate Professor PhMr Jaroslav Vošta C.Sc.

On December 24, 1974, Associate Professor PhMr Jaroslav Vošta, C.Sc. celebrated his 50th birthday. Dr. Vošta is Head of the Department of Microbiology, Chair of Biology, Faculty of Economy, Agricultural College, České Budějovice. He was born in Tábor, southern Bohemia where he went to primary and secondary school. In March 1943, shortly before the end of his last secondary school term, he was prosecuted by the Nazis together with his schoolmates and other young Czech patriots and sent to Nazi Germany for forced labour. For his uncompromising, antinazi attitude he soon became an active member of the illegal antinazi movement and, on these grounds, was arrested in 1944 and imprisoned at the concentration camp of Dachau, where he remained until the end of the war. After the liberation of Czechoslovakia and soon after his return home, he started his practical training in pharmacology at a dispensary and, in 1947, after finishing his practice, he started to study pharmacology at the Faculty of Natural Sciences, Charles University, Prague. He graduated in 1949 and was employed at the District Department of Hygiene and Epidemiology, České Budějovice. After passing a post-graduate course in parasitology in Prague, he was made Head of this Department. His profound interest in parasitology brought Dr. Vošta in close contact with academician O. Jírovec, under whose leadership he worked on his PhD thesis; in 1959, he was awarded the scientific degree of Candidate of Biological Sciences (PhD). In 1964, he joined the Agricultural College in České Budějovice, where, a year

later, he was named Associate Professor in the field of microbiology. His particular interest in leptospirosis dates back to his work on the PhD thesis, when he studied the natural foci and reservoirs of this infection in the Tábor district. He has remained faithful to this field of research up to the present. He has added to his studies those on problems concerned with the diagnosis of leptospirosis in the laboratory, the effect of Leptospirae on tissue culture, and a number of other questions. Many of his papers deal with intestinal parasites recorded from collectives of children, and the therapy of helminthic and protozoan infection of man. In recent years, he has added to his extensive research activities studies on problems of cattle leptospirosis designed mainly to the purpose of finding effective control measures against this economically important zoonosis. His academic activities have greatly been acknowledged, his list of publications comprises more than 60 papers in Czechoslovak and foreign periodicals.

Dr. Vošta is liked by his collaborators and by everybody coming into contact with him for his kind and modest nature, and for his readiness to help, where help is required. He is extremely fond of nature, loves Tábor, his home town and southern Bohemia, but mainly his work and people.

We congratulate Dr. Vošta on the occasion of his 50th birthday, wishing him sound health, personal happiness, success in his work, and to keep up his unconquerable, optimistic outlook on life.

Dr. Z. Šebek C.Sc.