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THE BEHAVIOR OF *RICKETTSIA AKARI* IN THE BODY LOUSE AFTER ARTIFICIAL INFECTION

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Rickettsialpox, belonging to the group of rickettsial diseases and characterized by skin eruptions, was first discovered in 1946 in New York and has since been reported in Boston by Pike *et al.* (1950). This disease presents a number of interesting biological problems. In an astonishingly short time the etiology and epidemiology of the disease were elucidated. The agent, *Rickettsia akari*, was cultivated in the chick embryo and its immunologic relationships established, mainly by Huebner and his associates (1946) as reviewed by the German author Kirsch (1949). The reservoir of the disease was found to be the house mouse *Mus musculus*, and the transmitter a mite of the family of *Laelaptidae*, *Allodermanyssus sanguineus* Hirst. It is not yet known whether there are other vectors. Philip and Hughes (1948) demonstrated experimentally, that the tropical rat mite *Liponyssus bacoti* Hirst can transmit *R. akari*. In one test rickettsiae were found in the nymphs of the F_1 generation, possibly by infection of the ovaries.

In the course of investigations of the behavior of various rickettsiae in arthropods we have examined the relationship of *R. akari* to the body louse. It was of special biological interest to discover whether the body louse is susceptible to these rickettsiae at all and how it reacts to the infection as compared with other rickettsiae.

MATERIAL AND METHODS

Dr. Italo Archetti from the Istituto di Sanitá Pubblica in Rome was kind enough to send me the Kaplan strain of *R. akari* from the Rocky Mountain Laboratory, Hamilton, Montana, in the form of yolk sac tissues dried in September 1949. With this material we first inoculated a series of mice intraperitoneally. As soon as there were sufficient rickettsiae in the smears of the mouse organs, we inoculated body lice (*Pediculus humanus humanus* L.) rectally by the method of Weigl and intracoelomically with infectious material from the mouse, using small glass capillaries. The intracoelomic infection was carried out only in females through the vagina. For each louse we used approximately 0.3 mg. of the infectious suspensions. The lice which withstood the infection well were kept in metal boxes and fed on the author's leg to which the boxes were strapped one hour every day. Between the blood-meals the lice were kept in an incubator at 31°C.

To follow the results, smears of the stomachs of lice rectally inoculated were made at regular intervals, and stained by the method of Giemsa. The lice intracoelomically infected were examined by means of smears of the coelomic liquid. Little droplets of the coelomic liquid can easily be obtained without

damage to the louse by amputation of a leg. After it was discovered that rickettsiae do multiply in the louse, special attention was paid to their localization in specific organs and in the cells themselves. For this purpose careful histological investigation became necessary. The stomachs were either taken out in a drop of broth and fixed, or the whole abdomen was put into the fixing solution after removing the head and extremities. We used Carnoy's fixing solution (6 parts absolute alcohol, 3 parts chloroform, 1 part glacial acetic acid). The material was treated with methylbenzoate, embedded in paraffin and then cut into longitudinal and cross sections of 3–4 μ which were stained by the moist Giemsa method and differentiated with acetone.[1]

As soon as multiplication of *R. akari* in the louse was established, the rickettsiae were again transmitted to healthy lice by injecting them rectally with suspensions of the stomachs of infected lice to make sure that the rickettsiae could be maintained in the louse by serial transfer. Altogether 6 louse passages were carried out by rectal inoculation without any essential change in the general picture, 35–40 lice being inoculated in each lot. Since the behavior of the rickettsiae after intracoelomic inoculation was rapidly revealed, only 2 series of 35 females each were infected by this method. Finally, the virulence and pathogenicity to mice of the rickettsiae from the lice were tested by intra-abdominal inoculation of stomach suspensions, coelomic liquid, and feces of the lice.

The lice would not feed on the mouse, so we were not able to infect them in the natural way by letting them feed on sick mice. We therefore inoculated 2 series of lice rectally with the blood of acutely ill mice, diluted with broth. Whether the infectious material reached the stomach via the rectum or the esophagus is perhaps immaterial, the important point being to discover whether the diluted inoculum contained enough rickettsiae to establish infection in the louse. These lice, like the others, were then nourished by feeding them on man.

BEHAVIOR OF THE STRAIN IN MICE

Mice, inoculated with the Kaplan strain of *R. akari* preserved in the form of dried yolk sac tissues, showed no clinical symptoms on the first passage, although spleen and liver preparations revealed isolated, pale rickettsiae. The second passage, initiated with spleen and liver suspensions, gave rise to clinical symptoms on the 7th day and one mouse died on the 8th day. The pathological picture was typical and did not change materially in subsequent transfers. Clinical symptoms became progressively more severe and at the fifth passage all the mice were dead by the 6th day. Smears from the organs, especially the liver, contained large numbers of rickettsiae as early as the 2nd passage. Intraperitoneal inoculations of spleen and liver suspensions were kept up for 6 passages, but beginning with the 4th passage, mice could also be infected intranasally by inhalation of spleen and liver material. These mice developed symptoms after 3 days and died as a rule on the 4th with typical pneumonitis. The lungs showed notable pathologic changes and smears contained large numbers of

[1] I am indebted to Dr. W. Behrenz for making the sections and to Miss Magdalene Kerner for her valuable technical assistance.

rickettsiae. While the strain could have been maintained in the lungs, this procedure was substituted, after 2 passages, by the inoculation of 6 to 7 day old chick embryos with organ suspensions from the 4th and 6th passages, by Cox's method. The embryos died in from 3 to 6 days, and smears from the yolk sacs contained rickettsiae in ample numbers. Thus the strain behaved normally, was quite virulent, and had evidently regained its original properties.

THE BEHAVIOR OF *R. AKARI* IN THE LOUSE

The external phenomena. Since the peritoneal smear of a mouse of the second passage contained a sufficient quantity of rickettsiae, the first series of lice was rectally inoculated with the peritoneal exudate diluted in broth. At first nothing unusual was found in the infected lice, but by the 7th day rickettsiae could be located beyond any doubt in smears of the stomachs. In later passages

FIG. 1. Smear of the stomach of a louse, rectally infected with *R. akari*. 8th day after inoculation. Epithelial cells with rickettsiae. Staining in all figures is with Giemsa. All figures except Fig. 7 are magnified 1200 times.

they could be found after 6 days but not earlier. Their number increased continuously in the following days and reached a maximum after 10 to 12 days.

The rickettsiae were rather evenly distributed in the smears (Fig. 1). Their color was darker than in the smears of the mouse. Coccoid forms and in particular plump, double rod-shaped forms predominated. Slender forms, thin and bright in the central portion, or longer chains such as are the rule after an infection with *R. prowazekii* or *R. mooseri* during the first few days, were never observed. The rickettsiae did not lie in compact and dense heaps, but were scattered and isolated. They strongly resembled *R. quintana* in size, shape, color and distribution. The staining of the poles was not noteworthy, especially at first.

After 7 days, rickettsiae appeared in the feces of the lice, but the number was relatively small throughout the test, constantly fluctuating and never reaching the quantities characteristic of *R. prowazekii* or *R. quintana*. The reasons will be discussed below.

In the first two lots of lice, none were obviously damaged by the rickettsiae.

In all, only 11 of 70 infected lice died after 8 to 15 days in one series, and 7 after 7 to 16 days in another. The death of these lice was probably caused by the rickettsial infection. The surviving lice of the first test were dissected after 17 days and at that time still contained many rickettsiae. On the 13th day 4 stomachs were emulsified in broth and rectally injected into a new lot of lice. In this passage the same phenomena reappeared as those described above: the lice had demonstrable infections after 7 days and contained large numbers of rickettsiae in the stomach. In this manner the strain was kept without incident for 6 passages over a period of 12 weeks. The experiments were interrupted after the sixth passage because the behavior of the rickettsiae and the reaction of the lice did not alter materially. In this way *R. akari* can doubtless be maintained in the louse indefinitely.

It was remarkable not only that *R. akari* was able to multiply rapidly and consistently in the lice, practically all of which became infected, but also that they caused no perceptible injury to most of the lice. Some infected lice remained

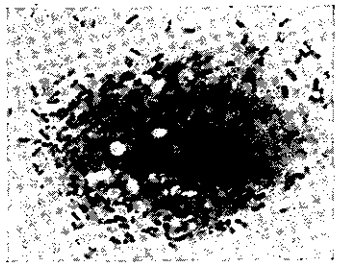

FIG. 2. Smear of the coelom liquid of a louse, intracoelomically infected with *R. akari*. 10th day after inoculation.

alive 3 or 4 weeks, i.e. their normal lifetime was not essentially shortened by the rickettsial infection.

Lice were easily infected with blood from a sick mouse and large numbers of rickettsiae were found after 9 days, although the lice were kept at room temperature for 3 days. Practically all the lice became infected and behaved in the same way as after inoculation with organ suspensions, showing little damage from the infection.

Intracoelomic injections, however, had different consequences. In earlier experiments the coelom of the body louse had proved to favor the growth of various rickettsiae (Weyer 1950). This is also true of *R. akari* which multiply rapidly and flourish under these unusual conditions (Fig. 2 and 3.) In the first experiment the lice were injected with the peritoneal exudate of a mouse; in the second, with the ground stomachs of some positive lice. After 5 to 6 days there was clear evidence of rickettsiae in the coelomic liquid. Their number did not increase remarkably during the first 8 to 10 days, but then rapid multiplication began and the whole coelom was flooded with the organisms, damaging the lice and killing them in a short time. The abdomen was swollen and frequently the lice had a reddish color. The last lice of the first lot were sacrificed after 11

days and a series of mice were successfully infected intraperitoneally with the coelomic liquid taken from four lice. In the second lot no lethal effect was observed until the 11th day, after which the coelomic liquid of both dead and living lice was densely filled with rickettsiae. The surviving lice were dissected after 14 days.

Internal phenomena and topography. The stomach smears did not indicate with absolute certainty whether the rickettsiae in the louse were intra- or extracellular (Fig. 1). While the regularity of arrangement seemed to indicate an intracellular position, the scattered distribution of the relatively dark stained rickettsiae frequently resembled the extracellular development of *R. quintana*. Also the early appearance of rickettsiae in the feces seemed to prove this. In later stages, however, a notable vacuolisation of the stomach cells was noticed

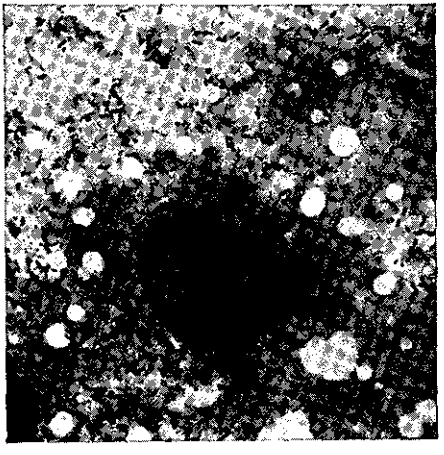

FIG. 3. Smear of the coelom liquid of a louse, intracoelomically infected with *R. akari*. 10th day after inoculation. Accumulation of rickettsiae in blood cells.

in the smears and there seemed to be rickettsiae in the vacuoles. However, their position could not be definitely determined until the lice had been examined histologically at different stages of the infection (Fig. 4 to 7).

These examinations showed that, with rectally inoculated lice, the rickettsiae multiply only in the cells of the midgut and do not invade the rest of the alimentary canal or other tissues. Unlike *R. quintana* which is found on cell surfaces and in the lumen of the stomach, the multiplication of *R. akari* is exclusively intracellular, and they are evenly distributed over the whole stomach.

Within the cells the position of the rickettsiae is irregular. They are hardly ever found in the portion of the cell toward the lumen as is the case with *R. prowazekii* or *R. mooseri*. Instead they appear beside or more often underneath the nucleus, without invading the nucleus itself. Here they do not form compact aggregations but are either scattered or assembled in small groups in the somewhat diffuse plasma. When the infection partially destroys the intercellular membranes, the rickettsiae tend to form confluent masses of more than usual

density above the basal membrane, and since almost all the cells of the stomach are eventually affected, the number of rickettsiae becomes very great. This is seen more clearly in the smears than in the sections. Vacuolization takes place in later stages and in thick smears the vacuoles may appear to contain rickettsiae, but on section are found to be practically empty (Fig. 5). In exceptional cases,

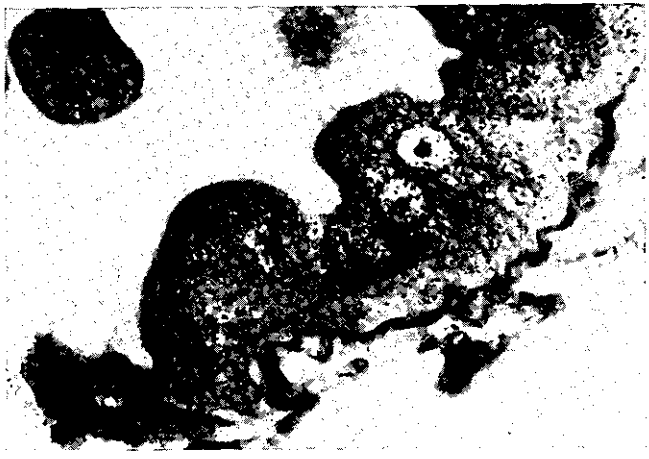

FIG. 4. Stomach section of a louse rectally infected with *R. akari*. 8th day after inoculation. The affected cells are still intact.

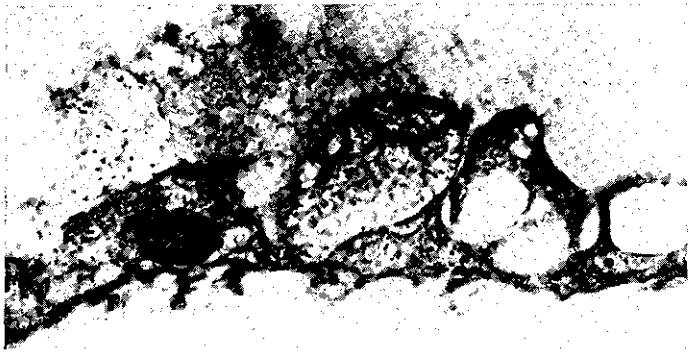

FIG. 5. Stomach section of a louse, rectally infected with *R. akari*. Vacuolization and disintegration of epithelial cells.

the intense vacuolization may cause some cells to protrude into the lumen (Fig. 6). Such infections which at the outset seem fairly harmless have, however, a definite effect on the integrity of the stomach cells and in some cases on the longevity of the lice. The cells eventually dissolve and are liquified, beginning at the base and ending with partial destruction of the epithelium (Figs. 5 and 7). In the early period of infection a few rickettsiae probably enter the lumen of the midgut but are seldom found in the feces at this time. When degeneration of the

cells takes place, free rickettsiae, single or in groups, are found in the lumen attached to fragments of cells, and it is no longer difficult to find them in the feces, although this is somewhat a matter of chance and is therefore irregular.

FIG. 6. Stomach section of a louse rectally infected with *R. akari*. 8th day after inoculation. A vacuolated cell protruding into the lumen of the stomach.

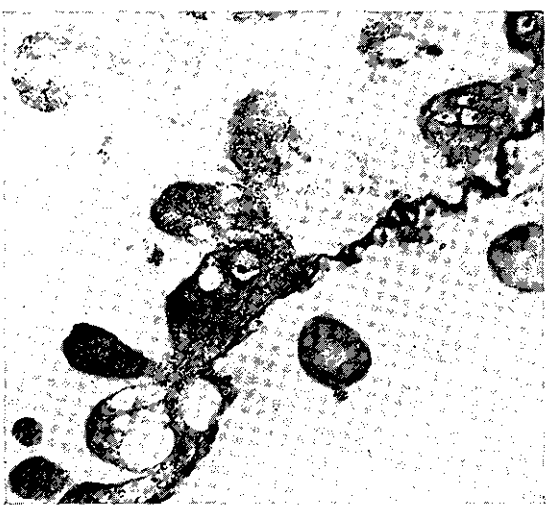

FIG. 7. Stomach section of a louse rectally infected with *R. akari*. 7th day after inoculation. Partial destruction and separation of the affected cells. Magnified 500 times.

This partial destruction of epithelium (Fig. 7) and its sequelae are unlike the phenomena occurring after infection of the louse with typhus fever rickettsiae. In the latter case, 6 to 10 days after infection, depending on the number of rickettsiae and the intensity of the multiplication, a process of destruction begins which affects almost the whole epithelium. With the rickettsiae concen-

trated in the apical pole above the nucleus in the direction of the intestinal lumen, the cells glutted with rickettsiae and blown up under the pressure pour out enormous quantities of the organisms into the lumen and perish in the act. Since the destroyed cells cannot be replaced, the stomach can no longer fulfil its physiological function. The semipermeability fails and the animals are fatally damaged. In spite of this they usually continue to feed but the blood cannot be digested. It stays in the stomach and hemoglobin enters the coelom through the damaged stomach walls. Such lice have a typical light red color which may involve only the stomach or more commonly the whole body, while the stomach of a normal louse takes on a dark red color immediately after feeding.

The behavior of the lice infected with *R. akari* is quite different. The localized loss of some intestinal cells does not produce serious consequences. The stomach continues to function and the life-span is not necessarily shortened or the vitality impaired. Feeding and reproduction take place normally. While an infection with *R. prowazekii* or *R. mooseri* is strictly limited to the stomach and does not cross this border except in some cases immediately before death, *R. akari* occasionally pass from the stomach into the coelom at an early stage. The entry is obviously through thin parts of the wall where cells have perished. Thus in a number of the infected lice one finds the rickettsiae both in the cells of the stomach and in the coelom, though seldom earlier than the 10th day, while in other lice the barrier between intestine and coelom is not crossed.

In the coelom they continue to multiply. Smears from such lice resemble those of animals which have been intracoelomically infected. In the latter, multiplication of the rickettsiae is confined to the coelom, and they do not enter the cells of the ovary as occasionally happens after artificial intracolomic infection with typhus fever rickettsiae (Weyer 1950). Other organs are not infected, except that the stomach cells are in some rare cases infected through the basal membrane. In the coelom the rickettsiae do not depend on cells for multiplication, which takes place extracellularly. Frequently one sees concentrations of rickettsiae in or on blood cells (Figs. 2 and 3), a phenomenon which can also be observed in other species of rickettsiae growing in the coelom.

More serious damage occurs if the rickettsiae in the coelom multiply in large quantities; then the lice die. The damage is probably the result either of the blocking of the blood cells or of the effects of toxins. This makes the intestine permeable although the epithelium may still be almost intact. In addition some stomach cells infected with rickettsiae from the coelom may be destroyed, a condition which manifests itself in a relatively dark reddish color of the lice which always covers the whole body. Such "red" lice live only for a few hours. In some cases the lice may die without showing the red color, although the abdomen of such animals is usually swollen. Rickettsiae entering the coelom from the intestine after rectal inoculation seem to multiply less rapidly and the lice live longer. In this case host and parasite apparently achieve a degree of mutual adaptation.

Virulence tests in the mouse. To test the virulence of *R. akari* after passage through the louse, one series of mice was injected intraperitoneally with coelomic liquid of infected lice, and another with the ground stomachs of lice of the

third and sixth passage. Feces of lice from the same lots were collected from the 10th to the 13th day after inoculation, diluted in broth and injected into mice intraperitoneally. In all cases the mice developed symptoms of disease on or after the 5th day and revealed typical pathological changes on autopsy. Smears from the organs showed rickettsiae and suspensions of mouse spleen and liver tissues transmitted the infection to other mice and to the yolk sacs of eggs. It was evident that the passage of the rickettsiae through the louse had not altered their virulence and pathogenicity for the mouse.

DISCUSSION

The body louse according to the experiments described above is a suitable and susceptible host for *R. akari*. These rickettsiae multiply undisturbed in the louse but their development is limited to the cells of the midgut and to the coelom. Other organs are not affected, and in this respect they behave practically in the same way as *R. prowazekii* or *R. mooseri*. The difference is that *R. akari* are not as numerous as *R. prowazekii*, elect a different location within the stomach cell and do not destroy the whole epithelium. They are found only irregularly in the lumen of the gut and in the feces. However the partial detachment of the cells permits the rickettsiae occasionally to enter the coelom at an early stage, which does not occur with *R. prowazekii*. This kills the lice prematurely, but unless this occurs, rectal inoculation need not shorten the life of the louse in spite of active multiplication of the rickettsiae.

R. akari can be maintained on lice by rectal infection in serial passages and their virulence and pathogenicity are not diminished in the process as may be seen from the mouse virulence tests. The feces of rectally infected lice also contain virulent rickettsiae. However, the feeding of the infected lice on man and their handling has caused no infections in our laboratory. For various reasons only artificial infections of the lice were obtained in our experiments but there is no doubt in my mind that the lice could become infected in the natural way by feeding. This was shown by the infection of the lice with fresh blood from an intraperitoneally inoculated mouse. The blood was diluted with broth so that undoubtedly less blood and fewer rickettsiae entered the stomach than after natural feeding. Nevertheless these lots of lice developed rickettsial infections in the same intensity and within the same time as after artificial infection with stomach suspensions or coelomic liquid.

Of special theoretical interest is the question why *R. akari* develops so easily and well in the louse. Is this an old or a new relationship? The question also arises whether the contact of this rickettsia with man was taking place for the first time when rickettsialpox was discovered in New York in 1946. The probable evolution of the host-parasite relationship in human rickettsial diseases has been discussed by Baker (1943) who considers it probable that the development of human rickettsial infections from what were originally arthropod diseases has taken place in steps. First the arthropod disease is transmitted to the animal which is its primary host; then man enters this arthropod-animal cycle frequently enough to make the disease endemic in human beings; eventually a

second arthropod, always a close associate of man, acquires the infection and a reciprocal transmission results which may become highly specialized and epidemic in man. Thus in typhus fever the primary vector, the flea, infects the rat and transmits the rickettsial organism sporadically to man (murine typhus); man, the secondary host, infects the louse and after a period of adaptation, virulent epidemic typhus results.

According to this hypothetical scheme, Miller (cited by Rose 1948) considers rickettsialpox as a second step. The rodent mite, *Allodermanyssus sanguineus* is the primary arthropod and vector, the mouse the primary host; the human being represents a secondary host.

"Further studies may reveal secondary arthropod vectors. If such vectors, for example the louse, are closely associated with man, the disease may move into its subsequent stages of the epidemic cycle." (Miller).

But is the body louse really the secondary arthropod host? From the relatively harmonious coexistence of *R. akari* and louse, which in our experiments permitted the survival and multiplication of the rickettsiae without materially affecting the host, one might conclude that this contact was not taking place for the first time, but had occurred at a much earlier period so that *R. akari* was already adapted to the louse.

On the other hand it is improbable that a disease like rickettsialpox with such striking symptoms should have remained unrecognized for so long. It is much more probable that *R. akari* is at least an old parasite of the mite and the mouse, and has now for the first time contacted human beings and, in our experiments, the louse. If this agent can exist both in man and in the body louse at the first contact, this may be ascribed to the fact that *R. akari* is closely related to other rickettsiae, which are already thoroughly adapted to the human being and to the louse or other vectors. In fact the complement fixation test shows that in antigenic structure there is a great similarity between *R. akari* and *R. rickettsii* and that there is a relationship between *R. akari* and *R. mooseri*.

The capacity to multiply in arthropods is a characteristic of all "genuine" rickettsiae and is a fundamental element of the definition of rickettsiae. Arthropods, probably ticks, were apparently the first hosts of rickettsiae, which have retained or acquired the ability to multiply also in other bloodsucking arthropods. The louse is an especially favorable host. We know that *R. prowazekii* and *R. mooseri* can probably develop in all bloodsucking lice, as Mooser, Varela and Pilz pointed out as early as 1934. Only recently I have shown that the crab louse, *Phthirius pubis* L. and the hog louse, *Haematopinus suis* L. are also suitable hosts for *R. prowazekii* (Weyer 1951, 1952).

Experiments of my own (Weyer 1952a, and others unpublished) have revealed that also *R. rickettsii*, *R. conorii* and even *R. (Coxiella) burnetii* can exist and multiply in the body louse. The capacity of the rickettsiae to multiply in certain tissues and cells is however not always a sign of an old relationship between host and parasite. For instance their growth in the yolk sac of the chick embryo or even in the coelom of the larva of *Tenebrio molitor* L. (Weyer 1950) proves that there are environmental conditions which are, *a priori*, suited to the biological needs of the rickettsiae and to which they do not have to adapt themselves.

To biological thinking, however, it is more plausible to suppose a close relationship between the different species of rickettsiae based on a common origin. This argument has been discussed in greater detail by Mooser (1946) among others. It is not astonishing that, in passing from primary hosts to secondary hosts, from one vector to another, etc., the properties of the rickettsiae or of the rickettsial strains have become changed in the course of time by mutation and selection, thus accounting for the different types of disease. It is not unlikely that when some strains of rickettsiae come into contact with human beings, they might produce new diseases even now. Rickettsialpox may be an example. But ultimately the old properties typical of all rickettsiae reappear under favorable conditions despite minor differences and deviations.

SUMMARY

1. *R. akari* was transmitted rectally and intracoelomically to the body louse by inoculation with peritoneal exudate of mice. In both cases the rickettsiae developed regularly and intensively in the louse. The same result was obtained when the lice were injected rectally with the blood of sick mice.

2. Multiplication of the rickettsiae was always intracellular in the stomach wall, localized particularly in the basal part of the epithelial cell; it was extracellular in the coelom liquid, from which the organisms in rare cases entered the stomach cells as well.

3. Multiplication of the rickettsiae in the stomach cells was moderate and did not deform them or cause the premature death of the louse; in the coelom, however, intense multiplication soon produced damage from which the louse died in a few days.

4. After rectal inoculation the rickettsiae invade and destroy or detach some of the stomach cells. Through the partially damaged walls they may escape into the lumen of the stomach on one side and appear in the feces, or less frequently into the coelom on the other, and by intense multiplication cause the rapid death of the louse.

5. By rectal transmission of ground stomach or coelom liquid of positive lice, *R. akari* can be maintained by serial passage in lice without losing virulence and pathogenicity for the mouse. The feces of the lice also contain virulent rickettsiae.

6. The behavior of *R. akari* in the louse can best be explained by its close relationship to other rickettsiae, if we suppose that the capacity to develop in arthropods is a fundamental characteristic of all rickettsiae and that the different species of rickettsiae all have a common origin.

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