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l'existence, comme celle des symbiontes des arthropodes, dépend étroitement de la présence de cellules vivantes.

Summary.

The history of the separation of murine typhus from classic epidemic typhus is presented. Contrary to the opinion of NICOLLE and his school (50) there is only one murine typhus. A differential diagnosis between murine typhus and classic typhus in a given case can only be made by laboratory methods. Petechiae as well as the extension of the rash to the palms of the hands and the soles of the feet considered by NICOLLE and his pupils (19, 66) as specific phenomena of murine typhus are more frequently observed in classic typhus than in the murine variety. The terms "Endemic Typhus of the Southeastern US", "Mexican Typhus", "Manchurian Typhus", "Shop Typhus" or "Urban Form of Tropical Typhus" are synonymous with the term murine typhus and should be dropped.

The hypothesis of the murine origin of classic typhus is discussed. Contrary to the claims of NICOLLE and GIROUD (58) *R. mooseri* develops in *Pediculus* exactly as does *R. prowazeki* and there is no difference between the two with respect to their multiplication in fleas (29, 40, 41). There is regular and solid postinfectious cross-immunity between murine strains and strains of classic typhus (30, 36, 60). Antigenic differences between the two types of strains have, however, been disclosed by cross-immunisation experiments with dead *Rickettsiae* (60). These differences are quantitative, not qualitative because with sufficiently concentrated vaccines not only homologous but also heterologous immunisation can be obtained (70, 97, 102, 103). The scrotal lesion of the guinea pig and the presence of *Rickettsiae* in smears from the tunica vaginalis at first considered pathognomonic for murine strains can also be observed in some strains of classic typhus. This holds true especially for NICOLLE's Tunisian strain of classic typhus (29, 38). It has generally been accepted that strains of classic typhus cannot be transferred in series in white mice. This does not hold true either for all strains of classic typhus. NICOLLE's strain for instance has been transferred through 22 consecutive white mice (90). The infections were, however, inapparent. The same strain accidentally contaminated with the virus of infectious ectromelia ran through 86 consecutive mouse transfers (90). Peritoneal smears from these mice regularly showed very large numbers of *Rickettsiae*. Whereas murine strains are very pathogenic for white mice, strains of classic typhus cause a lethal rickettsial peritonitis only when a very heavy dose of a pneumonic lung is inoculated, for instance 10^{-1} of

a lung, whereas 10^{-10} - 10^{-14} of a pneumonic lung of the murine strain may still cause a fatal infection. The differences between the two types of strains are therefore merely quantitative also in this respect. Since, however, these quantitative differences between murine and classic strains—heterologous protection with vaccines, scrotal phenomenon in the guinea pig, pathogenicity for white mice, serial transfer in white mice and rats—are regularly observed, they must be deemed specific. This specificity therefore is manifested by the disproportionality which the two types of strains exhibit with respect to their pathogenicity and serological activity. The assumption that classic typhus derives from the murine variety remains a hypothesis as long as artificial conversion of the murine type of strain into the classic type has not been accomplished in the laboratory.

The discovery of murine typhus has given a great impetus not only to research in typhus but also to the study of the other rickettsial diseases. A very abundant literature has appeared on the subject since 1931. A good deal thereof has brought forth scarcely more than redescriptions of what had already been published in Mexico and in the United States of America. The unexpected activity in experimental research in typhus and related diseases was due to the fact that murine strains offer ideal opportunities for the study of the causative organism of a rickettsial disease (8 b, 8 c). The controversy about the significance of the “rickettsia bodies” could definitely be closed as the peritoneal smear permitted the demonstration of the causative agent also in the tick born rickettsioses (116, 123, 140) as well as in tsutsugamushi (115). The way was such opened for large scale production of preventive vaccines. It was in the course of the studies on typhus in Mexico that NICOLLE’s famous hypothesis of the role of inapparent human infections for the preservation of the agent of typhus during the interepidemic period could first be disproved (43). Those who reject the hypothesis of the murine origin of classic typhus (29) have now the choice between ZINSSER’s hypothesis (11, 21) of late relapses in man and the assumption of Polish authors (153, 154) that dried feces of lice carry the infection over the interepidemic periods. Since the span of life of *Pulex* is not affected by the invasion of its gut with *Rickettsia prowazeki*, fleas may play a role in the preservation of the germ of typhus outside of the human body. Many authors classify the *Rickettsiae* among the viruses, others assign them a position intermediate between the viruses and the bacteria. There is no doubt in the mind of other authors (8 c, 38) that the *Rickettsiae* are true bacteria, which, like the intracellular symbionts of the arthropods, are strictly dependent on living cells for their existence.