

Zeitschrift:	Acta Tropica
Herausgeber:	Schweizerisches Tropeninstitut (Basel)
Band:	18 (1961)
Heft:	3
Artikel:	Miscellanea : Arthropod vectors in relation to the reservoir mechanism of microbial agents of animal diseases
Autor:	Philip, Cornelius B.
DOI:	https://doi.org/10.5169/seals-310950

Nutzungsbedingungen

Die ETH-Bibliothek ist die Anbieterin der digitalisierten Zeitschriften auf E-Periodica. Sie besitzt keine Urheberrechte an den Zeitschriften und ist nicht verantwortlich für deren Inhalte. Die Rechte liegen in der Regel bei den Herausgebern beziehungsweise den externen Rechteinhabern. Das Veröffentlichen von Bildern in Print- und Online-Publikationen sowie auf Social Media-Kanälen oder Webseiten ist nur mit vorheriger Genehmigung der Rechteinhaber erlaubt. [Mehr erfahren](#)

Conditions d'utilisation

L'ETH Library est le fournisseur des revues numérisées. Elle ne détient aucun droit d'auteur sur les revues et n'est pas responsable de leur contenu. En règle générale, les droits sont détenus par les éditeurs ou les détenteurs de droits externes. La reproduction d'images dans des publications imprimées ou en ligne ainsi que sur des canaux de médias sociaux ou des sites web n'est autorisée qu'avec l'accord préalable des détenteurs des droits. [En savoir plus](#)

Terms of use

The ETH Library is the provider of the digitised journals. It does not own any copyrights to the journals and is not responsible for their content. The rights usually lie with the publishers or the external rights holders. Publishing images in print and online publications, as well as on social media channels or websites, is only permitted with the prior consent of the rights holders. [Find out more](#)

Download PDF: 08.02.2026

ETH-Bibliothek Zürich, E-Periodica, <https://www.e-periodica.ch>

Arthropod Vectors in Relation to the Reservoir Mechanism of Microbial Agents of Animal Diseases.*

By CORNELIUS B. PHILIP.

U.S. Department of Health, Education, and Welfare, Public Health Service,
National Institutes of Health, National Institute of Allergy and Infectious Dis-
eases, Rocky Mountain Laboratory, Hamilton, Montana, U.S.A.

The purpose of this discussion is to point out evidence that arthropod para-
sites, as well as their vertebrate hosts, may be concerned in the reservoir func-
tion in many animal disease systems and to call attention to some of the com-
plex factors involved.

Definition of the term reservoir is immediately desirable in any cogent dis-
cussion of this subject but is complicated by present imprecise concepts. The
loose use of the term as applied to both arthropod and animal hosts has been
discussed by PHILIP (1948) in relation to rickettsial disease agents. Some
writers conceive the reservoir role in arthropod-borne diseases to be played
solely by vertebrates, while in their view the invertebrate intermediate hosts
serve only as vectors. In contrast, other investigators envisage, as also a part
of the reservoir mechanism, mere long physical persistence, for example of
typhus organisms in louse and flea feces and the Q fever agent in tick feces as
causes of delayed human infection.

Still others require fulfilment of the reservoir function by biological de-
velopment or proliferation of disease agents in an arthropod vector essential
to the disease cycle (whether or not there is demonstrated transovarial passage
of the agent). The question immediately arises, must such a reservoir vector be
long-lived, and if so, how long—weeks, months, or years? For example, must
the vector in some infected stage be capable of survival through periods of low
disease incidence, or through the winter in environments with seasonal
changes?

The type of metamorphosis of the vectors can also be a factor in such
longevity; those with the incomplete type are better adapted to transstadial
transmission, e.g., triatomid bug vectors of *Trypanosoma cruzi*, than are the
higher insects with complete changes between stages. Thus, though mosquito
larvae can be artificially infected with yellow fever virus and resulting adults
are infectious (WHITMAN & ANTUNES, 1938), this appears not even remotely
possible in nature.

In a review of the literature, PHILIP & BURGDORFER (1961) considered
that the reservoir function is often a dual one involving in varying degrees
both the vertebrate and invertebrate hosts. Not uncommonly, some texts over-
simplify the subject by trying to force tabulation of "reservoir hosts" either
exclusively as animals or less often as arthropods. Instead, evidence is avail-
able that the two often supplement or complement one another in the total
reservoir system for many of the arthropod-borne zoonoses or so-called an-
thropozoonoses as well as for strictly human diseases. As data accumulate,
these interpretations of the relative importance of either agency may change,
however. For example, BLANC & CAMINOPETROS (1932) suggested revision of
the usual conception of animal reservoirs in fièvre boutonneuse by stating:
"This problem appears to be quite confused if one stops at the hypothesis that

* Read before XIth International Congress of Entomology, Vienna, Austria,
17 August, 1960. Section of Medical and Veterinary Entomology.

outside of man there is a domestic vertebrate reservoir of virus and indeed we have been able to determine the insusceptibility of most of the species of vertebrates, commensal with man, and their incapacity to infect *Rhipicephalus* gorged on them. All becomes clear on the contrary if one incriminates no longer a vertebrate but the tick itself" (translation).

Unfortunately, information is very meager as to how long infected animals may act as donors to customary vectors, subsequent to the initial infectious febrile episode. This is especially true in the case of hosts with persisting, often signless infection which PARROT & PARROT (1949) and others have considered to be in a state of so-called premunition. Can ectoparasites be infected at any intermediate time, for example on rabbits in whose bone marrow and spleen DZHANPOLADOVA (1959) found tularemia organisms 413 and 333 days after infection? FOX (1948) recovered *Rickettsia tsutsugamushi* from tissues and blood of Swiss mice 610 days after infection, also from tissues of cotton rats after 269 days, but only up to 154 days from blood, probably in part because of presence of antibodies. Though PHILIP & PARKER (1938) recovered *R. typhi* from brains of white rats after 370 days and from spleens as long as 153 days, the longest period from the blood was 31 days. For reservoir purposes, the important and mostly unanswered question in such studies is whether the usual vectors can be infected during such periods. As shown in yellow fever and the encephalitides, there is a threshold in amount of circulating virus below which mosquitoes cease to become infected. Whether an analogy to this in other diseases determines infectibility of the longer feeding Acarina we do not know. The possibility that even endoparasitic bot larvae might retain infection acquired from a host seems to have received little attention.

It is tempting to reason, however, that since body lice can become infected from recrudescence epidemic typhus cases and trench fever convalescents (WEYER, 1960; KRASNIK, 1959), then the reservoir function of some of the longer-feeding and longer-lived Acarina may be enhanced by a similar extension of the infectious period in premunized hosts. It could also be argued that the typhus reservoir concept involving man is incomplete without inclusion of lice (even though they are short-lived) in promoting typhus and trench fever outbreaks, and that tsetse flies augment African trypanosomiasis in much the same manner. This would be a necessary sequel to the philosophy of GEAR *et al.* (1952) that a community which has had an epidemic of typhus fever "remains a potential reservoir of the rickettsiae for the lifetime of the infected generation".

Thus, an important factor in the reservoir mechanism is the little-known relationship between "latent infections"¹ and vectorship of the disease. It has been recommended that the relevant term "latent period" should be avoided because of ambiguity in several disciplines (WALKER *et al.*, 1957). In plant pathology particularly (DAY, 1957) but also in medical entomology (PHILIP, 1957), the term has been used interchangeably with "extrinsic incubation period", i.e., the time elapse between acquisition of a given infection and subsequent ability of a vector to transmit. The reservoir function is naturally concerned with this period which, in certain viruses for example, may be prolonged or suppressed in vectors by low mean temperatures or low dosages.

¹ Defined as "inapparent infections which are chronic and in which a certain virus-host equilibrium is established", Symposium on Latency and Masking (WALKER *et. al.*, 1957); here DAY (1957) prefers "masked infections", a term which the Symposium recommended against, to denote "the absence of symptom expression".

The recently discovered Kyasanur Forest virus, transmitted to man, monkeys and certain domestic stock in India by *Haemaphysalis spinigera* (WORK, 1958), is an example of a widely adapted, virulent agent of a virus group having a probable reservoir in ticks. This suggests that tests for agents in other lower-primate-infesting ticks, of which HOOGSTRAAL & THEILER (1959) reported 14 species in Africa, would be rewarding. Another complex biocoenose is reported by BLANC & BRUNNEAU (1956) in the forest environs near Casablanca, Morocco, where the ectoparasites of rabbits, including ticks and fleas, were associated with agents of Q fever, murine typhus, relapsing fever, and with *Pasteurella* sp. and trypanosomes. PARKER once compared the wood tick, *Dermacentor andersoni*, to a veritable Pandora's Box causing at least five kinds of human illnesses.

In general, acarines qualify more readily as reservoirs than do the shorter-lived insects, especially the important biting fly vectors. The remarkable variations in cycles of animal disease agents adapted to transmission by different species and stages of ticks has been reviewed by NEITZ (1956). In addition to the longer existence of acarines in a given stage and generation (adult argasid ticks have lived for over 20 years, PAVLOVSKY & SKRYNNIK, 1951), they frequently transmit infectious agents through their eggs to succeeding generations. Marked adult ixodids, *D. andersoni*, were found by me to survive through two seasons free in a rigorous Montana climate and for three years as outdoor captives. However, passage to the egg stage is not unknown though rare in insects, e.g., KISSLING *et al.* (1955) reported recoveries of encephalitis virus from the ova of infected *Mansonia perturbans* and *Aedes triseriatus* and such passage of sandfly fever virus has been claimed in USSR (SABIN *et al.*, 1944). The recovery of western equine encephalitis virus from hibernating *Culex tarsalis* in a Colorado mine in late December by BLACKMORE & WINN (1956) is also of interest in the reservoir picture.

The great majority of these agents are adapted to residence in their arthropod hosts without doing them obvious harm, which facilitates the reservoir role. The thesis has been reviewed (PHILIP, 1957; WEYER, 1960) that rickettsial organisms at least may have originated as symbiotes of acarines which became pathogenetic through the parasitic habits of their arthropod hosts; a similar possibility has been discounted in the virus field (DAY, 1957). *R. prowazekii* may exhibit the most recent arthropod adaptation among rickettsiae, perhaps a secondary one as suggested by BAKER (1943), since it kills its louse host and is not passed transovarially. WEYER (1952) suggests "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 (probably not in reference to *Rickettsia sens. lat.*). *R. typhi*, apparently harmless to fleas, killed artificially-infected body lice, and did not become less harmfully adapted when passed through 17 generations (WEYER, 1960). Ticks infected with *Pasteurella tularensis* and *P. pestis* may also be adversely affected (PHILIP, 1957; PETROV, 1958). GREWAL (1957) showed that there was a high mortality, especially at time of molting, in *Rhodnius prolixus* and *Cimex lectularius* fed on laboratory animals heavily infected with *Trypanosoma rangeli*. But these instances of incomplete adaptation to residence in arthropods are infrequent and probably of relatively recent evolutionary derivation.

Another questioned aspect of the arthropod reservoir mechanism is whether this capacity for arthropod residence is predicated upon inherent proliferation of a given agent in the customary vector. Such data as are available indicate not only that animal pathogens usually multiply in the vectors, but surprisingly, that some plant viruses can as well (DAY, 1957). In spite of the close restriction of many pathogens to a specific combination of homiothermic and

poikilothermic hosts, the adaptability of the agent to abrupt alternation in the different tissue systems is remarkable. It has never been explained satisfactorily why some agents are transmissible by some species and not by others even in the same genus of parasites, and yet also propagate in a vertebrate, widely different zoologically.

While transovarial passage of the agent would enhance reservoir capacity in a given vector, it is obviously not an invariable prerequisite as illustrated by its lack in Colorado tick fever-infected *D. andersoni* (EKLUND *et al.*, in press) and in tick-borne heartwater, *Cowdria ruminantium* infection in African animals. However, DAY (1957) implies this prerequisite in stating that "obviously the tick is not an adequate reservoir of infection" in Rocky Mountain spotted fever because not all tick progeny acquire infection from parents².

The question of interference between different agents in the same vector has received very little attention but may also be important in the reservoir system. Interference in the animal host is better known, for example, between eperythrozoonosis and anaplasmosis in cattle (FOOTE *et al.*, 1957) or among certain animal viruses (HENLE, 1950). SABIN (1952) suggested not only interference with yellow fever by dengue virus in the host, but that prior dengue infection raised the minimum threshold to subsequent acquisition of yellow fever infection by the mosquito vector. Unpublished data of BURGDORFER suggest that under certain conditions there is interference between Colorado tick fever virus and *Rickettsia rickettsii* in dually infected *D. andersoni*. PRICE (1954) also showed that in ticks there could be suppression of strains of high virulence by those of low virulence of *R. rickettsii*. On the other hand, no interference occurred in the work of CHAMBERLAIN & SUDIA (1957) who demonstrated concurrent transmission by single insects with concomitant infections of both eastern and western encephalitis virus.

Another factor also related to the reservoir mechanism, particularly in ticks, is the degradation of infection resident in vectors due to subsequent blood-meals on immune hosts with circulating antibodies as, for example, recently reported by BENDA (1958) in Czechoslovakia for a decrease in amount of tick-borne encephalitis virus in *Ixodes ricinus* infected in a previous instar and fed on immune hosts. Such immune blood-meals in female ticks also adversely affected transovarial passage of the virus. Related factors in this ecosystem are reviewed by BLASKOVIC & REHACEK (1961).

Finally, the danger is pointed out, in discussing reservoir mechanisms in arthropod-borne diseases, of slanting them "homocentrically", when actually man is only an accidental intruder into most natural primary disease cycles (PHILIP, 1948). This idea of intrusion is inherent in the thesis developed by PAVLOVSKY (1955) on what he calls the "natural nidality" of diseases, principally arthropod-borne, which considers the sum of factors responsible for maintenance of various disease foci, usually with man as only an unessential visitor (PAVLOVSKY & ZASUCHIN, 1958). It became manifestly impossible to narrow this broader viewpoint of interacting factors when PHILIP & BURGDORFER (1961) initially attempted to restrict their review to a discussion of arthropods only, as reservoirs of animal disease agents.

It also becomes evident that the various considerations of arthropod vectorship complicate any attempted precise definition of the term reservoir as applied to these animal disease systems.

² Supplemental factors of natural maintenance are overlooked by him, however, which are important, viz., starting of new lines of infection by simultaneous feeding of infected and noninfected individuals on a susceptible host, and infection of ova by the male tick during fertilization (PHILIP, 1958).

References.

BAKER, A. C. (1943). The typical epidemic series. — Amer. J. Trop. Med. 23, 559-566.

BENDA, R. (1958). The common tick "*Ixodes ricinus* L." as a reservoir and vector of tick-borne encephalitis. I. Survival of the virus (strain B3) during the development of the tick under laboratory conditions. — J. Hyg. Epid. Microb. Immunol. 2, 314-330.

BLACKMORE, J. S. & WINN, J. F. (1956). A winter isolation of western equine encephalitis virus from hibernating *Culex tarsalis* Coquillett. — Proc. Soc. Exp. Biol. and Med. 91, 146-148.

BLANC, G. & CAMINOPETROS, J. (1932). Études épidémiologiques et expérimentales sur la fièvre boutonneuse, faites à l'Institut Pasteur d'Athènes. — Arch. Inst. Pasteur de Tunis 20, 343-394.

BLANC, G. & BRUNNEAU. (1956). Étude épidémio-écologique dans la forêt de Nefifik. — Arch. Inst. Pasteur Maroc. 5, 87-200.

BLASKOVIC, D. & REHACEK, J. (1961). Ticks as virus vectors in Eastern Europe. — New York: Academic Press.

CHAMBERLAIN, R. W. & SUDIA, W. D. (1957). Dual infections of eastern and western equine encephalitis viruses in *Culex tarsalis*. — J. Inf. Dis. 101, 233-236.

DAY, M. F. (1957). The relation of the arthropod-borne viruses to their invertebrate hosts. — Trans. N.Y. Acad. Sci. 19, 244-251.

DZHANPOLADOVA, V. P. (1959). Some results of microscopical detection of *Pasteurella tularensis* in the organs and tissues of guinea pigs. — J. Microbiol., Epidemiol., Immunobiol., 30, 65-66.

EKLUND, C. M., KOHLS, G. M. & KENNEDY, R. C. (1960). The ecology of Colorado tick fever: the role of transovarial transmission. Symposium, Biology of Viruses, Tick-borne Encephalitis Complex, Czechoslovakia, Oct. 11, 1960 (abstr.) (in press).

FOOTE, L. E., LEVY, H. E., TORBERT, B. J. & OGLESBY, W. T. (1957). Interference between anaplasmosis and eperythrozoonosis in splenectomized cattle. — Amer. J. Vet. Res. 18, 556-559.

FOX, J. P. (1948). Long persistence of *Rickettsia orientalis* in blood and tissues of infected animals. — J. Immunol. 59, 109-114.

GEAR, J., WOLSTENHOLME, B., HARWIN, R. & STAKES, F. H. (1952). Brill's disease (recrudescent epidemic typhus fever): its occurrence in South Africa. — S. Afr. Med. J. 26, 566-569.

GREWAL, M. S. (1957). Pathogenicity of *Trypanosoma rangeli* Tejera, 1920 in the invertebrate host. — Exp. Parasit. 6, 123-130.

HENLE, W. (1950). Interference phenomena between animal viruses: a review. — J. Immunol. 64, 203-236.

HOOGSTRAAL, H. & THEILER, G. (1959). Ticks (Ixodoidea, Ixodidae) parasitizing lower primates in Africa, Zanzibar and Madagascar. — J. Parasit. 45, 217-222.

KISSLING, R. E., CHAMBERLAIN, R. W., NELSON, D. B. & STAMM, D. B. (1955). Studies on the Northern American arthropod-borne encephalitides. VIII. Equine encephalitis in Louisiana. — Amer. J. Hyg. 62, 233-254.

KRASNIK, F. I. (1959). The epidemiological importance of patients with sporadic typhus. — Problems of Virology 4, 105-110.

NEITZ, W. O. (1956). A consolidation of our knowledge of the transmission of tick-borne diseases. — Onderstepoort J. Vet. Res. 27, 115-163.

PARROT, L. & PARROT, G. (1949). Sur la prémunition dans les rickettsioses. — Arch. Inst. Pasteur Alg. 21, 257-261.

PAVLOVSKY, E. N. (1955). [Natural nidi of human diseases and regional epidemiology] Leningrad. — Medgiz., pp. 17-26.

PAVLOVSKY, E. N. & SKRYNNIK, A. N. (1951). [Some biological peculiarities of the ticks *Ornithodoros*-transmitters of tick-borne relapsing fever.] — Dokl. Akad. Nauk SSR (N.S.) 78, 1069-1072.

PAVLOVSKY, E. N. & ZASUCHIN, D. N. (1958). Some results of investigations of natural foci of human infections. — J. Hyg., Epid., Microbiol., Immunobiol. 2, 385-394.

PETROV, V. G. (1958). [Experimental study of *Dermacentor marginatus* Sulz. and *Rhipicephalus rossicus* Jak. et K. Jak. ticks as vectors of tularemia. pp. 117-123. In: Problems of Epidemiology and Prophylaxis of Tularemia.] — Medgiz., Moscow, 187 pp. (Reprinted in English in J. Parasit. 46, 877-884, 1960).

PHILIP, C. B. (1948). The reservoirs of infection in rickettsial diseases of man. In: Rickettsial Diseases of Man, pp. 97-112 (AAAS Symposium, Wash., D.C., 247 pp.).

PHILIP, C. B. (1957). Evidence of masking and latency in the fields of insect and helminth microbiology. pp. 88-106. In: WALKER *et al.*, 1957.

PHILIP, C. B. (1958). Some epidemiological considerations in Rocky Mountain spotted fever. — Publ. Health Rep. 74, 595-600.

PHILIP, C. B. & PARKER, R. R. (1938). The persistence of the viruses of endemic (murine) typhus, Rocky Mountain spotted fever and boutonneuse fever in tissues of experimental animals. — Publ. Health Rep. 53, 1246-1251.

PHILIP, C. B. & BURGDORFER, W. (1961). The evidence of arthropod vectors in the reservoir mechanism of microbial agents in animal diseases. — Ann. Rev. Ent. 6, 391-412

PRICE, W. H. (1954). Variation in virulence of *Rickettsia rickettsii* under natural and experimental conditions. pp. 164-183. In: International Symposium, The Dynamics of Virus and Rickettsial Infections. Blakiston Co., New York.

SABIN, A. B. (1952). Research on dengue during World War II. — Amer. J. Trop. Med. Hyg. 1, 30-50.

SABIN, A. B., PHILIP, C. B. & PAUL, J. R. (1944). Phlebotomus (pappataci or sandfly) fever. — J. Amer. Med. Ass. 125, 603-606, 693-699.

WALKER, D. L., HANSON, R. P. & EVANS, A. S. (1957). Symposium on latency and masking in viral and rickettsial infections. — Minneapolis: Burgess Publ. Co., 202 pp.

WEYER, F. (1952). The behavior of *Rickettsia akari* in the body louse after artificial infection. — Amer. J. Trop. Med. Hyg. 1, 809-820.

WEYER, F. (1960). Biological relationships between lice (Anoplura) and microbial agents. — Ann. Rev. Ent. 5, 405-420.

WHITMAN, L. & ANTUNES, P. C. (1938). Studies on *Aedes aegypti* infected in larval stage with virus of yellow fever. — Proc. Soc. Exp. Biol. and Med. 37, 664-666.

WORK, T. H. (1958). Russian spring-summer virus in India. Kyasanur Forest disease. — Progress in Medical Virology 1, 248-279.