

## Worldwide Research on Tick Feeding in Relation to Transmission of Disease Organisms

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Much has been published on tickborne pathogens and their effects on the hosts of ticks. Arthur (1962) reviewed the relationship of these pathogens to their tick vectors, and Řeháček (1965) discussed their dissemination within vector tissues. The mode of acquisition of disease organisms by ticks while feeding on infected hosts is reasonably obvious. Portals of entry during the free-living stages of ticks may be by transstadial, transovarial, and occasionally by mating routes. However, little has been observed or published with regard to the actual transmission of pathogens *from tick to host*—a rather surprising fact, since this is obviously a key link in the vector-host relationship. Complete understanding of any tickborne disease certainly calls for a knowledge of the manner of this transmission.

Transmission may be by passive or active means. The former may be accomplished by contact with, or the ingestion of, infected ticks or tick feces by susceptible animals. Hoogstraal (1966) cited this possibility with reference to St. Louis, eastern, and western equine encephalitis viruses. It would also appear to be a possible, though not a common, route for other tickborne infections. Disease transmission by vertebrate predation of ticks seems to have received little attention.

Active transmission may be either by mechanical means, in which the pathogen is carried purely mechanically by the vector, or by biological means, involving tissues of the vector, or by both methods. According to Chamberlain and Sudia (1961), it is theoretically possible for any bloodsucking arthropod to be a mechanical vector. As such, no intimate relationship between the organisms and host is involved, inasmuch as the pathogen need only be transported and transferred through the agency of some surface of the vector—usually its mouthparts, although fecal contamination, concomitant to tick feeding, is also a frequent source of transmission. Multiple feeders, such as the argasids and many male ixodids, presumably lend themselves to mouthpart contamination, and tick feces in general may contain varying supplies of infectious organisms, depending on little understood factors which may activate or inactivate them or release them during the vector's feeding (thus, as Day (1955) noted for lice, fecal transmission may also be of a biological nature). Řeháček (1965) cited the observation of viable *Coxiella burneti* rickettsiae in tick feces for up to 65 days; Philip (1948) extended this period up to 586 days for feces of the Rocky Mountain wood tick, *Dermacentor andersoni* Stiles. Philip and Burgdorfer (1961) noted that *Brucella abortus* can be shed likewise for longer than 3 months.

Other diseases shown to be capable of mechanical transmission include: *Anaplasma marginale*, by *D. andersoni* (Anthony et al. 1964), Central European tickborne encephalitis (Benda 1958), and tularaemia (Francis 1927).

Transmission from vector to host by biological means, on the other hand, demands a specialized presentation of pathogens within the tick to routes that are favorable for their transfer to host tissues. The manner by which they reach these points of exit has attracted much speculation and was discussed at length by Řeháček (1965), who assumed that in biological transmission receptors and inhibitors within the gut wall play a major role in the threshold of infectivity. Other concepts (Philip 1963, Řeháček 1965) include the transport of rickettsiae by hemocytes to the salivary glands, the possible transformation of protozoans and spirochetes to submicroscopic filterable units during their migration through tick tissues, and predilection (organotropism) such as *Borrelia duttoni* shows toward certain tick organs. Similarly, Pavlovsky and Solov'yev (1963) showed that the virus of human Russian spring-summer encephalitis has an affinity for a tick's salivary glands, which serve as "gateways" of exit. Riek (1964) regarded a specialized development of *Babesia bigemina* in the salivary glands of the southern cattle tick, *Boophilus microplus* (Canestrini), as being a mature infective form, ready for liberation in salivary fluids.

It is sufficient to say here that the pathogens to be transmitted are believed to end up in salivary secretions, or, in argasid ticks, also in the coxal fluid. In the latter, particularly where viruses and spirochetes are concerned, entry into the host may take place through the neighboring tickbite puncture and possibly through the host's skin. The pathogen direction is thus one-way, and is not complex. Philip (1963) cited the danger of *Brucella abortus* infection from coxal fluid and Řeháček (1965) suggested that experimental transmissions of West Nile and yellow fever by *Ornithodorini* might be by this means. Relapsing fever may be transmitted in the coxal fluid of *Ornithodoros moubata* (Murray), although transmission in saliva is more common in the ornithodorid ticks (Varma 1956). Pathogen transmission to the host during and by direct involvement of the vector's feeding is more commonplace, and yet has been the least observed. This is because the heavily sclerotized walls of the tick capitulum are such that both direct and histological observations of the mechanism and function of the feeding apparatus are difficult, and the nature of tick attachment, with its mouthparts buried