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L. Bolzoni, Rosà R., Cagnacci F., Rizzoli A.

Fondazione Edmund Mach, Research and Innovation Centre, via Mach 1, San Michele all'Adige, Tn

Tick-borne encephalitis (TBE) is an emerging zoonotic disease reported in several European and Asiatic countries with complex transmission routes that involve various host species. Understanding and quantifying the contribution of the different hosts involved in the TBE virus (TBEV) cycle is crucial to estimating the threshold conditions for virus emergence and spread. Some hosts, such as rodents, act as both feeding host for ticks and reservoir of the infection. Other species, such as deer, provide important sources of blood for feeding ticks, but they do not support TBEV transmission, acting instead as dead-end (i.e. incompetent) hosts. The aim of this study was to estimate the contribution of the main ungulate tick hosts to the pattern of tick infestation on rodents, and to TBEV occurrence in rodents and questing adult ticks. In the empirical study, we compared areas where endemic human TBE occurs, with control sites having no reported human TBE cases. In these six sample sites located in Italy and Slovakia, we assessed deer density using the pellet group count-plot sampling technique, collected questing ticks by dragging, live-trapped rodents (primarily *Apodemus flavicollis* and *Myodes glareolus*) and counted ticks feeding on rodents. Both rodents and adult ticks were screened for TBEV infection. TBEV infection in ticks and rodents was positively associated with the number of co-feeding ticks on rodents and negatively correlated with deer density. We hypothesize that the negative relationship between deer density and TBEV occurrence could be attributed to deer (non competent hosts) diverting questing ticks from rodents (competent hosts). In fact, we observed that, after an initial increase, the number of ticks feeding on rodents reaches a peak for an intermediate value of deer density and then decreases. In order to validate and interpret in a robust theoretical framework the empirical findings regarding the effect of deer density on tick infestation on rodents and TBEV occurrence, we introduce an eco-epidemiological model to explore the dynamics of tick population and TBEV infection. Model results show hump-shaped relationships between deer density and both feeding tick on rodents and the basic reproduction number for TBEV. This suggests that deer may act as tick amplifiers, but may also divert tick bites from competent hosts, thus diluting pathogen transmission. However, our model shows that the mechanism responsible for the dilution effect is more complex than the simple reduction of tick burden on competent hosts. In fact, while the number of feeding ticks on rodents may increase with deer density, the proportion of blood meals on competent compared to incompetent hosts may decrease, triggering infection decline. As a consequence, using just the number of ticks per rodent as a TBE risk predictor could be misleading if competent hosts share habitat with incompetent hosts.