
Health, zoonotic pathogens and parasites

Back to the future: pathogens as drivers of vole population cycles

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Already in the early era of quantitative population ecology, Charles Elton suggested diseases to regulate animal numbers. The refutation of this disease hypothesis in the 1950s has however caused pathogens getting low or almost no attention during the following period of “vole wars” where different schools of rodent researchers were competing to identify the likely misleading single factor driving cyclic dynamics of especially microtine rodents. Predation and food availability have been commonly proposed as the primary drivers. However, the predation hypothesis remains controversial, and interactions between multiple drivers are poorly understood. In addition, the emergence of new analytical techniques in recent decades has finally opened new cross-disciplinary research options which have increased our understanding of the distribution and ecology of rodent-borne pathogens fundamentally. The gained insights motivate us to revisit and revive the disease hypothesis. Indeed, there is mounting evidence that certain pathogens play an important role in the fitness of rodents. Here, we therefore revisit the disease hypothesis by studying and using a 42-year time series of field data, the role of food availability (i.e., seeds of Scots pine [*Pinus sylvestris*] and Norway spruce [*Picea alba*]), a common zoonotic pathogen (*Orthohantavirus puumalaense*), and predation by a specialist predator, for the population dynamics of bank voles (*Clethrionomys glareolus*).

We show that the interaction between pathogen infection and predation drives population cycles in bank voles. The vole, pathogen, and predator populations cycle synchronously. Dynamic modelling of the data revealed that vole cyclicity emerged only when both pathogen and predator were considered together, with the pathogen spreading efficiently in the vole population during winter, resulting in reduced reproduction in the following summer, and the predator likely increasing predation on infected voles. Our results demonstrate that pathogens play a critical role in vole population dynamics, suggesting that the disease hypothesis has been prematurely dismissed. The findings provide a basis for further exploration of multi-factor models in other cyclic species or for bank voles in other regions, examining the role of pathogens either alone or combined with other factors.