How do bats maintain infections? Mathematical modelling to disentangle mechanisms driving the temporal cycles of infection in Australian bats

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Zoonotic pathogens, (pathogens that can be transmitted from animals to humans), represent an increasing public health concern. Cross-species transmission can only occur when particular conditions of reservoir host ecology, infection dynamics and species distributions align, making spillovers (the transmission of a pathogen from a vertebrate animal to a human) at the same time a global public health concern and a complex and poorly understood phenomenon. Pathogen circulation in reservoir host populations is one of the factors contributing to spillover risk, therefore, understanding the mechanisms driving infection dynamics can help us in preventing spillovers to occur.
In Australia, the recent changes in land use caused a rapid modification in bat behaviour that coincided with the emergence of Hendra virus (HeV). HeV is maintained in wildlife by bats of the Pteropus genus (flying foxes) and can be transmitted from bats to horses and from horses to humans. Bats do not show marked signs of infection, but in humans and horses the symptomatology is very severe, with a high fatality rate (57% and 80% respectively), pointing out the need to avoid HeV spillovers. HeV spillovers usually occur in winter and often coincide with an increase in viral shedding in urines from bats, suggesting a temporal trend in pathogen circulation in reservoir host populations. Several factors can contribute to drive the observed temporal dynamics, and different maintenance routes have been theoretically hypothesized to explain the cyclicity of infection.
To disentangle the mechanisms driving the pathogen dynamics in reservoir host, we applied a set of mathematical models to an historical dataset collected in Boonah (Queensland, Australia) between July 2013 and June 2014. We used a system of four differential equations (SEIR model type) to simulate infection dynamics in the host population, and we included time dependent parameters to simulate the seasonality of infection and periodicity of births.
We showed that the sole population dynamic of bats, with the introduction in the system of susceptible new-borns, can not explain the annual cycles of HeV shedding, and either a waning of the immunity in adult bats or the development of a chronic infection with cyclic viral reactivation must be hypothesized to explain the observed infection dynamics. Our results support the hypothesis that cyclicity in HeV shedding can be driven by environmental stressors, like winter food shortages, that can affect bat immune response to pathogens. These findings evidence the need to reduce anthropogenic stressors that might exacerbate HeV shedding from bats. Furthermore, given the adaptability of the modelling framework, the method proposed can be applied to other infections/bat species.