The role of social ecology in Chiropteran pathogen richness and zoonotic potential

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I, Tim C. D. Lucas, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the work.

Abstract

My research is about stuff.

It begins with a study of some stuff, and then some other stuff and things.

There is a 300-word limit on your abstract.

Acknowledgements

Acknowledge all the things!

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Chapter 1

Introductory Material

Some stuff about things. Some more things.

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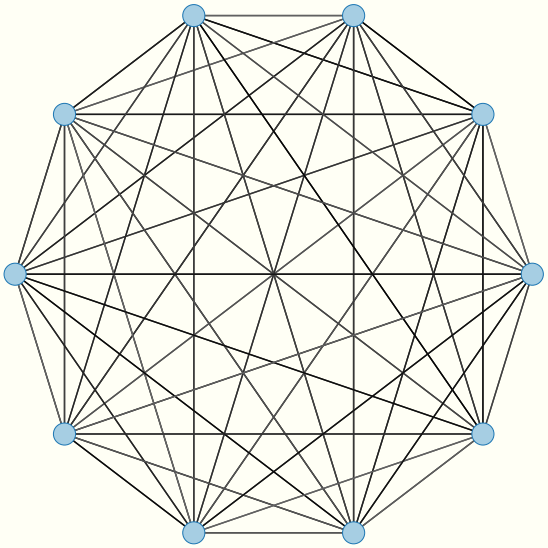
Chapter 2

Does network structure of bat populations promote viral diversity?



Abstract

One or two sentences providing a basic introduction to the field

An increasingly large fraction of emerging diseases come from animals and these diseases have a huge impact on human health. The chance that a new disease will come from any particularly wild host species increases with the diversity of pathogens in that species. However, the factors that control pathogen diversity in wild populations are still unknown.

Two to three sentences of more detailed background

Host species traits such as population density, longevity, body size and population structure have been shown to correlate with pathogen diversity. However, our mechanistic understanding of how population structure (i.e. non random contacts across the population creating barriers to disease spread) affects pathogen diversity is poor. Greater mechanistic understanding is needed to clarify the exact causal role population structure has in controlling pathogen diversity. Mechanistic models are also likely to be more robust to transferring understanding between taxa and predicting changes.

Typically it is assumed that well-connected populations promote disease spread (high R_0) and therefore promote pathogen diversity. However, if competition is strong endemic pathogens will dominate and prevent new diseases from invading and spreading. In a structured population, stochastic effects could create areas of low prevalence of the endemic disease, allowing new diseases to invade.

We consider bats as a case study as they have been implicated in a number of recent, high profile diseases such as Ebola, SARS, Hendra and Nipah. Bats have varied social structures and so the structure of populations could be one way to prioritise zoonotic disease surveillance in this group.

One sentence clearly stating the general problem (the gap)

It is unknown whether population structure allows escape from competition and therefore high diversity.

We hypothesise that low dispersal rates and a low number of connections in a metapopulation network will allow invading pathogens to establish more readily.

One sentence summarising the main result

I find that neither population connectedness nor dispersal rate affect the probability that a new pathogen will invade into a population.

Two or three sentences explaining what the main result reveals in direct comparison to what was thought to be the case previously

The common assumption that factors causing high R_0 allow new pathogens to invade and therefore increase pathogen diversity is not supported by our study. Instead we find that changes in population structure that would affect R_0 do not affect the probability of invasion of a new pathogen.

One or two sentences to put the results into a more general context.

This result means that large scale population structure does not seem to control pathogen diversity. This also implies that population structure is not a useful proxy for pathogen diversity with respect to zoonotic disease surveillance, for example in bats.

Two or three sentences to provide a broader perspective,