Extrinsic and Intrinsic Factors Influencing Parasite Burden in the Big Brown Bat (*Eptesicus fuscus*)

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Introduction

- In most host-pathogen systems, the key feature of transmission dynamics and rate of spread of a parasite or disease is the variation among hosts in the numbers of parasites they harbor and the factors controlling infection
- Pathogens are typically aggregated across their host populations: relatively few individuals are heavily infected, while most potential hosts suffer light infections or remain uninfected
- Aggregation among hosts may be generated by variation among individuals in their exposure to parasite infective stages and by variation in susceptibility once an infectious agent has been encountered
- Host heterogeneity in parasite burden has a strong influence on the establishment and subsequent spread of pathogens
- Understanding the basis of this variation is critical to understanding transmission dynamics and the development of effective mitigation strategies

Objective

To determine the relative influence of various intrinsic and extrinsic factors on parasite burden in *E. fuscus*

- Model: Big brown bat, *Eptesicus fuscus* and its intestinal helminths
- Intrinsic factors: Sex, age, body condition, immunocompetence
- Extrinsic factor: Colony of origin

Methods

Bat captures

- Six colonies in SW Michigan and N Indiana (USA) sampled June-August, 2008
- Captured 47 females and 32 males, including 48 adults and 31 juveniles
- Sex, age, mass, forearm length recorded
- Body condition calculated as (forearm length / mass) x 100

Functional immunocompetence

- Bacterial killing assays (BKA)[1] performed with *Staphylococcus aureus* and *Escherichia coli*
- Hemolysis/Hemagglutination assays performed with rabbit red blood cells assess relative activity of complement, native antibodies (NAbs)

Helminth burden

- Intestinal parasites collected, counted, stained, and identified
- Data analyzed via structural equation modeling [3]

1. Trematodes were dominant form of parasite encountered

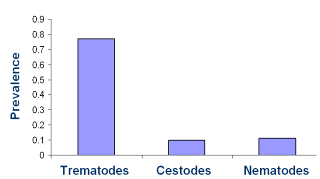

Figure 1. Prevalence of intestinal parasitic taxa among all hosts.

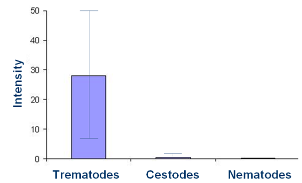

Figure 2. Mean intensity of intestinal parasitic taxa among all hosts.

2. Parasite aggregation among hosts in sample population

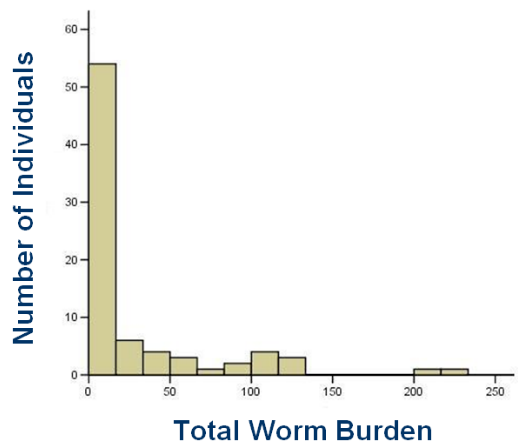

Figure 3. Frequency of helminth infection among all hosts.

3. Model of host susceptibility

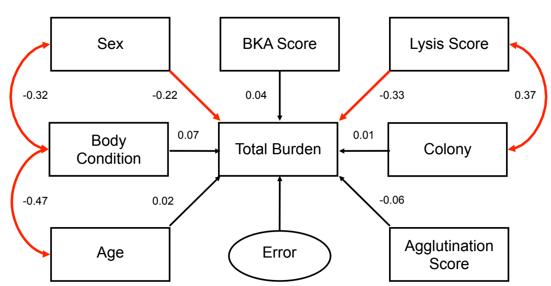

Figure 5. Predictors of total helminth burden as modeled with structural equation modelling. Significant (p<0.05) standardized weights noted in red. Overall model: χ^2 =20.445 , p = 0.004

Two significant predictors

- Females have more helminths
- Low lytic activity indicates higher helminth burdens

Three significant interactions

- Females have better body condition than males
- Adults have better body condition than juveniles
- Lysis score varies among colonies

Conclusions

1. Parasitism was female-biased

- Not a result of inter-sexual differences in immunocompetence
- Higher activity of females may lead to increased exposure to intermediate hosts
- Females had better body condition, and may promoted higher parasite survival

2. Individual variation in immune response was critical for predicting infection risk

- Individuals with low lytic activity are most heavily parasitized
- These individuals may drive transmission of helminths to intermediate hosts and promote parasite persistence

3. Immune function varied among individuals from different colonies

- Suggests a heritable component to immunocompetence, or local adaptation to geographically variable parasite component communities

Future Research

- Expand sampling spatially to include additional colonies in other regions to examine patterns of local adaptation
- Examine year-to-year variation in parasite burdens and the factors predicting host susceptibility
- Include additional factors in our model that may influence parasite burden, such as neutral and immunogenetic diversity, hormone levels, measures of stress, and different components of immune function
- Test our model with experimental infections

References

[1] Millet, S., J. Bennett, K.A. Lee, M. Hau, and K.C. Klasing. 2007. Quantifying and comparing constitutive immunity across avian species. *Developmental and Comparative Immunology* 31: 188-201

[2] Matson K.D., R.E. Ricklefs, and K.C. Klasing. 2005. A hemolysis–hemagglutination assay for characterizing constitutive innate humoral immunity in wild and domestic birds. *Developmental and Comparative Immunology* 29: 275–286

[3] AMOS. 2008. SPSS, Inc. Chicago, IL

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