ABSTRACT

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Emerging infectious diseases in wild animals threaten global biodiversity as well as domestic animal and human health. Their unprecedented increase in conjunction with anthropogenically induced range shifts of endemic pathogens exposes hosts to novel parasite combinations, lending urgency to research on disease dynamics in wildlife systems. In natural populations, hosts must concurrently battle infection from multiple parasite species. Host fitness, disease severity, transmission rate, host susceptibility, and effective control all depend on the dynamics of the parasite community. However, the bulk of research on infectious diseases - their transmission and fitness effects - is limited to one-host and one-parasite systems. It has become apparent that a broader perspective, which incorporates interactions among pathogens within and between hosts, is necessary to predict the outcome of co-infection. In this dissertation, I examined the role of co-infection for host immunology, host fitness, and disease dynamics. Specifically, I examined the role of two intestinal parasites, coccidia and nematodes, and two intracellular pathogens, *Brucella abortus* and *Mycobacterium tuberculosis*, in a free-ranging population of African buffalo. First, I examined coccidia and nematode infection patterns in buffalo at Hluhluwe Imfolozi Park (HIP), South Africa, to test the hypothesis that co-infection and season interact to influence the distribution and severity of infections (Chapter 2). I tested for a role of infection and co-infection on three proxies for host fitness: host pregnancy, host body condition, and parasite aggregation. By examining host fitness costs across seasons and in different demographic groups, this work showed that the effects of infection were most pronounced in the dry season and with co-infection. Then, I investigated the infection patterns, host fitness, and population level consequences of *B. abortus* infection in African buffalo of Kruger National Park (KNP), South Africa. I evaluated the use of an enzyme-linked immunosorbent assay (ELISA, IDEXX Brucellosis Serum Ab Test) for diagnosis of brucellosis, the disease caused by *B. abortus* (Chapter 3), and applied it to understand the environmental and demographic variation in the individual-level survival and fecundity costs of infection (Chapter 4). Brucellosis infection was associated with increased mortality and condition-dependent reductions in reproductive success. By summarizing these individual level consequences with a mathematical model, I explored how condition-dependent variability in vital rate parameters and disease effects translates into contrasting outcomes of brucellosis infection for population growth. Finally, I re-examined the influence of co-infection in the context of brucellosis by investigating how an emerging pathogen, *M. bovis*, the causative agent of bovine tuberculosis (bTB), alters host immunity, infection patterns, fitness costs, and the dynamics of brucellosis infection (Chapter 5). These results showed a positive association of bTB and brucellosis infections among hosts, and altered survival, susceptibility, and immunity with infection. Together, these studies demonstrate the importance of understanding the strength, direction, and context-dependency of interactions between co-infecting pathogens.