How do nomadic species and their pathogens respond to changing resource distributions?
Background: Unlike seasonally migratory species, whose movements have predictable timing and direction, nomadic animals move irregularly as they track unpredictable resources[1]. Nomadism is more widespread than previously assumed, occurring in birds, mammals, fish, and insects, and from the Arctic to Australia. Because nomadic movements influence important ecological processes, from seed dispersal to pathogen transmission[2], it is critically important that we better understand the drivers and effects of this opportunistic movement behavior.

Highly mobile animals can spread pathogens as they move across landscapes, but long-distance movements can also decrease pathogen prevalence as animals move away from infected habitats or if infected individuals cannot complete migration[3]. Though these relationships between movement and disease are well studied for migratory species, how nomadic movements affect pathogen spread and persistence is entirely unknown, despite the fact that nomadic animals can be severely impacted by pathogens (e.g. the massive die-off of the Saiga antelope in 2015)[4].

Long-distance animal movements can shift dramatically as resource distributions change[5]. I propose study how changing resource distributions alter population dynamics and pathogen persistence in nomadic animals. To do so, I will first develop a modeling framework to answer two questions: ***(Q1) How do changes in resource distributions regulate population dynamics of nomadic species?*** and ***(Q2) How do nomadic movements affect pathogen spread and persistence?*** I will then use empirical data to synthesize these ideas and ask ***(Q3) How do changes in resource distributions alter population dynamics, pathogen spread, and pathogen persistence in a nomadic waterbird, the White Ibis?*** White Ibis (*Eudocimus albus*) living in South Florida are an ideal system to study how anthropogenic changes to resources alter host movements and infection; these birds live in highly developed areas where provisioning wildlife is common and are exposed to *Salmonella*, including serovars that infect humans. By answering these questions, my research will go beyond the current paradigm of movement ecology – which has focused on identifying patterns – to uncover how animal movement affects (i) population persistence and (ii) landscape-scale pathogen dynamics. Answering these questions is critical in a world where human activities are rapidly altering animal behavior and natural landscapes.

Research plan: Migratory networks provide a powerful framework to examine population dynamics of migratory species (Fig. 1a). These models have shown that habitat loss at one site affects entire populations[6] and can identify key sites for conservation[7], but migratory networks also assume that the animal movements are highly predictable in their timing and direction, which is true for only a minority of highly mobile species. I will extend these models to include nomadic movement rules, where animals track spatial and temporal changes in resources (Fig. 1c), and will couple these networks with epidemiological models to examine how movement behavior and resource distributions interact to shape pathogen dynamics.

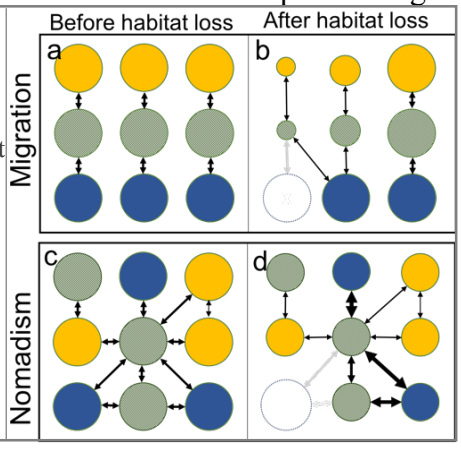

Fig. 1: Example migratory (a,b) and nomadic (c,d) networks. Colors show the season when resources are most abundant. Arrows represent movement between sites; larger nodes and thicker arrows indicate more individuals using a site or route, respectively. Open circles show habitat loss (b,d).

Q1. When habitat is lost in one season, migratory populations decline and connectivity increases[6] (Fig. 1a,b). *I hypothesize that changes in resource distributions regulate population sizes of nomadic species, but these effects are smaller than for seasonal migrants because high connectivity in nomadic networks allows nomads to better*

exploit resources available elsewhere after habitat loss. To test this hypothesis, I will develop migratory networks that include nomadic movement rules (Fig. 1c) and then simulate various resource scenarios, including both habitat loss (i.e. node elimination; Fig. 1b,d) and provisioning (i.e. node introduction). I will calculate (A) population sizes and (B) the distribution of animals across the landscape to compare how migrants and nomads respond to each resource scenario.
Q2. Migratory species often have lower infection rates than sedentary species because of migratory escape and culling[3]. *I hypothesize that nomadic species will have lower infection rates than residents because they escape infection by moving between habitats, but nomads will have higher infection rates than migrants because they move shorter distances (i.e. culling will not be strong). Further, the spatial extent of pathogen spread will be larger in nomadic than in migratory networks because nomadic networks have higher connectivity (Fig. 1c,d).* To answer this question, I will couple network models with epidemiological models. I will use the SI (Susceptible-Infected) framework, where individuals can become infected with a pathogen if they contact an infected individual within their same patch. In addition to incorporating flexible movement behavior (as in Q1), I will introduce distance-dependent culling of infected animals. At the end of a given time period, I will compare the proportion of (A) infected individuals and (B) infected patches between networks with different movement rules to determine how flexibility in movement behavior shapes pathogen dynamics.
Q3. Urban White Ibis in South Florida forage in areas where anthropogenic food is abundant. *I hypothesize that increased resource availability and predictability in urban areas leads to less movement and increased infection in nomadic White Ibis.* To synthesize Q1 and Q2, I will use GPS data from 40 White Ibis captured at 10 provisioned urban sites and 6 unprovisioned wildland sites in South Florida. With these data, I will compare movement patterns (site fidelity and movement propensity) of urban- and wild-foraging birds. I will then use these movement parameters to estimate connectivity between sites, which will allow me to examine how nomadic movement – or the loss of nomadic movement – affects *Salmonella* infection of individuals (estimated from fecal samples) and sites (from soil and water samples).
Feasibility: I have extensive experience using statistical models to analyze animal movement and my mentors at the University of Georgia are experts in infectious disease and mathematical modeling. I will have access to large amounts of empirical data on White Ibis through the UGA-based White Ibis network; this group, now in its second year, has over 10 investigators and students using field, laboratory, and computational methods to contribute data on the ecology, behavior, and diseases of White Ibis in South Florida.
Broader impacts: ***Applicability.*** Disease spread between wildlife and people is a central concern in wildlife management. In the White Ibis system, humans interact with wildlife through provisioning; understanding how this practice affects animal and human health will inform safe ways for people to engage with wild animals. ***Public outreach.*** As part of the White Ibis network, I will participate in established community engagement activities. I will also present my results at meetings of the Oconee Rivers Audubon Society (GA) and the Audubon Society of the Everglades (FL). ***Education and mentorship.*** I will create an educational module on ecological modeling to run as a workshop at local meetings of the Girls Who Code organization (Macon, GA), a club that aims to increase representation of girls and women in technology. I will also mentor undergraduate students as part of UGA's REU site, which promotes research experiences in computational disease ecology for underrepresented undergraduates.

[1]Mueller et al. 2011. *Glob. Ecol. Biogeogr.* **20**, 683–694. [2]Lundberg & Moberg 2003. *Ecosystems* **6**, 87–98. [3]Altizer et al. 2011. *Science* **331**, 296–302. [4]Milner-Gulland 2015. *Oryx* **49**, 577–578. [5]Teitelbaum et al. 2016. *Nat. Commun.* **7**, 12793. [6]Taylor & Norris 2010. *Theor. Ecol.* **3**, 65–73. [7]Taylor & Stutchbury 2015 *Ecol. Appl.* **26**, 424–437.