How Disease Risks Can Impact the Evolution of Social Behaviors and Emergent Population Organization

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Abstract Individuals living in social groups are susceptible to disease spread through their social networks. The network's structure including group stability, clustering, and an individual's behavior and affiliation choice all have some impact on the effect of disease spread. Moreover, under certain scenarios, a social group may change its own structure to suppress the transmission of infectious disease. While many studies have focused on how different network structures shape the disease dynamics, relatively few have directly considered the equally important

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evolutionary question of how disease dynamics shape the success of social systems. In this paper, we summarize the relevant mathematical and biological literature on evolutionary theory and population network structure to discuss what is known about how the synergistic effects of network-based epidemiology of infection and social behavior can shape the evolution of social behaviors and the population structures that emerge from them. We close by discussing open questions, including how these insights may shift when instead considering macro-parasites as the infection spreading on the network.

1 Introduction

In the last 15 years, significant progress has been made in studying how social contact patterns influence the epidemiological dynamics of infectious disease circulating in host populations. Similarly, the past decade of research in animal sociality has greatly expanded our understanding of the emergence of population structure from individual behaviors. An active area of research is determining and understanding the links between a group's organizational structure and the population's success and evolutionary fitness. Advances in this area have produced the diverse and intricate pieces required to begin asking questions about the impact of infection risks on how social systems may have evolved, but efforts to integrate them have only just recently surfaced.

In this paper, we begin in Sect. 2 with a review of our current knowledge in the evolution of sociality under disease constraints. Section 3 describes existing models which try to explain how organizational structures can emerge from individual behavioral dynamics. In Sect. 4, we discuss the use of evolutionary dynamics to study the fitness of a population with pathogens. We conclude with Sect. 5 where we highlight the current gaps in our knowledge that need to be filled before further questions can begin to be addressed.

2 Motivation: Multilevel Selection and Social Behavior

2.1 Review of Evolutionary Theory

The most fundamental question in considering the evolution of any trait is how the trait impacts the fitness of the individual who expresses it. In general, the fitness consequences of a given trait depend on other traits the individual possesses or expresses. For example, a muscle trait that would enable faster running might have little impact on the fitness of an individual who also carries a trait that causes joint stiffness [25]. Fitness also depends on the environment in which the individual lives. For example, a trait that improves the ability to safely digest the leaves of a particular plant, despite its strong chemical defenses, may not play a strong role in the fitness

of the individual if the environment is diverse or rich enough [1, 76]. If competition for food from other species who do not carry that trait decimates all other resources, the ability to digest the plant in question, although originally only a minor food source for the individual, may become critical to that individual [76]. Beyond the diverse array of factors influencing the direct impact to survival and reproductive success of the individuals who express the trait, we must also consider any impacts of the trait on their family members. This insight extends the benefit of a trait to include fitness consequences to those whose reproduction indirectly benefits the evolutionary fitness of the individual in question, by increasing the likelihood of the survival and reproduction of others who share their genes [31]. However, the concept of indirect fitness consequences can be extended beyond the inclusion of familiar ties.

In the 1970s, evolutionary theorists began to consider "group selection," a concept that extends the idea of evolutionary fitness from focusing solely on an individual to instead also consider groups of individuals. This allowed the consideration of how the traits (i.e., actions) an individual exhibits might benefit or compromise the survival and reproduction of the social group in which they live, even if they are unrelated genetically. The resulting insights provided discussion of potential synergy achieved by groups of unrelated individuals all displaying the same traits to improve the outcome for the group as a whole [80]. While there are many potential benefits of collaborating in social groups such as increased resource discovery and distributed predation risk [9, 64], there are also associated costs that include attraction of predators, reduced or misallocated shares of resources, and exposure to infectious diseases [39]. During the early twentieth century, many researchers argued that groups could be evolutionarily successful, even if they were made up of relatively unsuccessful individuals [81]. This argument is fundamentally flawed since any genes that cause individuals to suffer a net fitness penalty should decrease in representation over generations [24], thus group selection theory was abandoned for a time [81]. Luckily, more recent studies have revisited the idea that traits can make an impact at scales larger than the individual [82]. These efforts, now termed "multilevel selection," reflect the idea that individuals can benefit from participating in successful groups, but that group benefit must be reflected in benefit to all of the individuals who participate, since individuals are the ones who must pass along their genes to enable the next generation and the propagation of the successful group trait [82]. Formalizing this realization allowed for a renaissance in the study of how evolution may have shaped the transition from solitary to social species [41, 72, 83]. Making frequent use of game theory, elegant studies considered the possible mechanisms of selection that could favor both isolated instances and ongoing cooperation among individuals both within and across generations [54]. Only more recently, however, has the field begun to consider how evolutionary forces might have shaped particular types of traits governing social interactions in already social species [3, 7, 20, 73].

Among the risks of social living, infectious diseases can pose one of the greatest challenges for quantifying the impact to individual fitness. Predicting how many individuals in a population are likely to be exposed to a pathogen is itself an

entire field of applied mathematics [2, 15]. Outcomes from infection may also be influenced by social processes [65]. The evolutionary literature has traditionally considered infectious diseases as a constraint on the success of large groups [8, 42, 69]. Recent advances in the study of networks have allowed exploration into how vulnerable particular social structures may be to different types of infectious disease risks [19, 84]. While the majority of scientific literature has focused on how network structure affects disease risk, network theory also critically enables us to ask questions about how selection due to infections might act on the structure of social groups, and therefore on the individual behavioral social traits that govern their emergence [36]. Are there types of social interactions that yield successful groups while minimizing risks of infection? What types of organizational success are compromised by mitigating transmission risks? Are certain strategies for group success fundamentally compromised if disease-driven mortality alters the underlying social network too drastically? These questions are only now becoming accessible as the tools of game theory, network theory, and complex adaptive systems are maturing.

2.2 Review of Organizational Success of Static Network Structure in Populations

While many studies have focused on the role of social behaviors that generate interaction networks in directly affecting individual fitness [43, 44], fewer within the context of animal social behavior have focused directly on the benefits of the emergent structure of the social system itself [13, 18]. This is not, however, to say that the benefits of particular types of network structures have gone uninvestigated. In the broader field of network science, applied across other disciplines, many studies have investigated questions such as how network structure supports the functions of collaborative decision-making, how structure effects the efficiency of information diffusion, how structure influences the emergence of subgroup behaviors such as violence or radicalization, how coordination structure among network subunits enables higher-order computation or function, and many more [17, 23, 27, 28, 46, 74]. Beyond the network structure itself, biological literature has also looked at related questions such as social stability and efficient social collaborations. Many studies, without focusing on the explicit social network structure, have shown that the fitness of individuals in a population is strongly tied to social stability over time [45, 78]. Social position has been linked to individual fitness across a diversity of taxa [16, 21, 63, 77]. Further, the efficiency of social groups at decision-making or collective actions has been shown to have strong fitness consequences [12, 62]. Together, these studies strongly support the idea that particular social structures that emerge from the synergy of individual behaviors can have a strong evolutionary impact on individuals in social species.

3 The Evolution of Emergent Population Structure from Dynamic Social Behaviors

A number of models have investigated how organizational structure can emerge from individual behavioral dynamics. The idea is based on the notion that individuals of any species may observe the affiliations of others and, therefore, naturally quantify the "desirability" of others. In this type of social network, individuals make decisions regarding social choices and associations based on some measure of the popularity of those around them. There are a number of examples of this in emergent social structures such as individual body size [56] or predator-attracting color displays [61]. The effects of these dynamics are explored through models of social network theory where mathematical metrics are used to quantify the relative importance of nodes and the organization of the network. Network centrality theory is used to identify the most important individuals in a social network as well as to characterize the properties of the network as a whole. The fitness of individuals and the network as a whole are determined by a choice of centrality measure.

Although there are hundreds of different network centrality measures, we focus on three that are frequently used in the research of social networks and evolutionary biology: in-degree, betweenness, and closeness (see Fig. 1). The in-degree of node *A*, sometimes called the popularity of *A*, is the number of adjacent nodes that are directed into node *A*. (Note: In order to capture the asymmetric nature of social interactions, for example, grooming and being groomed, we use arcs (i.e., arrows)

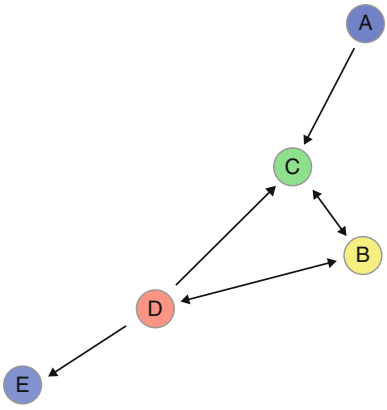

Fig. 1 Network Centrality and Social Groups. This social network depicts individuals (nodes) A through E interacting through directed social affiliations (edges). When individuals seek to modify their affiliations, the preferences they display will influence which nodes are the most significant to the group. Here, the green node (C) displays the node with largest in-degree value (e.g., most popular individual), the yellow node (B) displays the node with largest betweenness value, and the red node (D) displays the node with largest closeness value in the network

rather than bidirectional edges in our network.) Closeness describes a measure of the average path length between node *A* and each of the other nodes in the graph, while betweenness provides a mathematical compromise between degree and closeness. Betweenness of a node *A* measures the percentage of shortest paths from one node in the network to another node in which node *A* is included. While this metric is used extensively in the analysis of networks, there is no known biological species which functions based on betweenness. This centrality measure was chosen specifically to be a metric well-studied in network science that does not have a biological equivalent, but does indicate path efficiency in global organizational function.

Fefferman and Hock have extensively explored the effects of choice mechanisms such as in-degree, closeness, and betweenness on societal structures [20, 34]. In the case where social connections are made at random, one can infer how social groups may have organized before taking into account an individual's social preference. In experiments where social affiliations are based on socially derived measures of individual fitness that can be approximated by centrality, they observed how more complex social preferences may have evolved from an initially simple set of individual-level choices. Fefferman and Ng [20] demonstrated that an individual's fitness impacts the evolution of a group's social structure.

In addition to the experiments driven uniquely by centrality metric-based affiliation strategies, a number of other choices might influence both individual behavior and overall social organization. Various papers have considered the impact of factors such as kin selection, cases in which not all individuals in the population used the same metric for affiliation preference, and cases in which individuals defy established social norms [20, 35, 53]. In this last case, these individuals, known as "rule breakers," showed an impact on the evolution of more general social behaviors, but these impacts were dependent on the underlying social preferences [35]. With regard to kin selection, Greening and Fefferman [29] studied the evolutionary trade-off between inclusion of family groups and the ability of a population to sustain a well-organized social structure. They showed that there may be trade-offs between benefits an individual accrues due to participating in family units versus those due to participating in a well-organized society; the magnitude of these trade-offs may be dependent on the expected size of the family relative to the broader society. For example, simulations showed that when using betweenness as a centrality measure rule breakers enjoyed an increase in success while conventional individuals received less. This model indicated that rule-breaking behavior can transform convention-based societies, even before direct impacts on individuals can be measured.

Allowing each individual to select their own affiliation preference may also play a role in the overall organization of a group. Fefferman investigated this idea by developing a model that allowed each individual to choose the centrality measure with which they would choose whom they would like to associate [53]. Rather than assigning fixed affiliation preferences, individuals were allowed a "learning period" where they compared their individual success using each of the three centrality metrics mentioned. Based on the outcome of this learning period, individuals chose a strategy in which to continue interacting with the larger organization. Fefferman explained that, in many scenarios, allowing individuals to self-select an affiliation

strategy leads to higher organizational stability for the group (as measured by the variance of the group-level centrality over time) than in cases where an individual's strategy was predetermined.

4 Disease Risks in Social Networks

When studying disease risks in a population, the classical technique in mathematical biology is to use a system of ordinary differential equations describing the time evolution of the number of susceptible (S), infected (I), and recovered (R) individuals (where N is the total population). By introducing parameters that describe the rates of transmission, death, and recovery, one can write down a system that has the following form:

$$\begin{aligned}\frac{dS}{dt} &= bN - \lambda IS - dS\\ \frac{dI}{dt} &= \lambda IS - gI - dI\\ \frac{dR}{dt} &= gI - dR\end{aligned}$$

where b is the birth rate, λ is the rate of transmission, d is the natural death rate, and g is the rate of recovery from infection into an immune state. While this model can provide a powerful analytical framework to explore disease transmission, it is fairly simple and does not account for the specific details of the mechanics and progression of infection. Furthermore, this model is based on the key assumption that the population is well-mixed, i.e., any individual is equally likely to encounter any other individual in the system. Thus, the social network structure of a population is not taken into account. For further explanation on SIR models and networks, see [40].

Social networks are often complex and have high levels of heterogeneity—some nodes have many connections while others have only a few. There are several examples of using social network analysis to study complex systems like airline transportation networks [11], computer viruses [57], HIV epidemics [52], and spread of diseases under immunization [59]. In particular, there is growing interest in using mathematical analysis to consider pathogen spread in various social network structures. In general, we assume that the larger the social group, the higher the risk each individual has in acquiring an infectious disease. This assumption is reasonable since an increase in infected individuals can lead to an increase in the prevalence that they will infect an additional individual. However, other studies show that the issue is more complex. One such study hypothesizes that by subdividing a group into subgroups the ability for the disease to spread among all individuals in a population may be bottlenecked, thus reducing the prevalence of infectious disease in the social group [55]. In addition, some studies have demonstrated that population-level disease outcomes are sensitive not only to

the epidemiological parameters that describe the disease but also to the topological structure of the population's contact network [22]. Analyzing both epidemiological and structural variations of a network increases the understanding of the extent of population vulnerability to, and timing of, outbreaks of an infectious disease. For example, scale-free networks [5] do not experience spread of infection with very low transmission probabilities [49], and heterogeneous networks can actually be more robust against infection than their homogeneous counterparts [47]. Additional explorations of disease spread on social networks can be found in [10, 40, 58, 75].

The disease burden of a population changes based not only on the social organization of the group but also on the transmission rate of the pathogen [36]. Hock and Fefferman suggest that a group may change its social structure to reduce the prevalence of a pathogen (although this may leave the social group susceptible to other pathogens). Even small net benefits of a social organization may boost the ability for individuals to establish immunocompetence. Thus, the social structure of the group may itself play a role in the spread of infectious diseases.

While much insight can be gained by analyzing disease spread on static networks where individuals have fixed social connections, in practice, individuals within a group tend to interact dynamically to create new and remove old associations on a constant basis. In fact, an extensive body of work suggests that pathogen infection in dynamic networks where individuals constantly change associations differ from the static case [4, 19, 48, 67]. In Fig. 2, we depict an example network of four individuals evolving in time. At each step, individuals stochastically modify their network connections. Theoretical analysis has shown that even after a population reached a stable network structure and remained static, the disease load was significantly different than a network that continued to shift dynamically [19]. In particular, the authors argued that under certain types of social organization the disease load may be smaller in dynamic networks compared to static networks. However, a study of disease spread in badger populations argued that dynamic social networks may

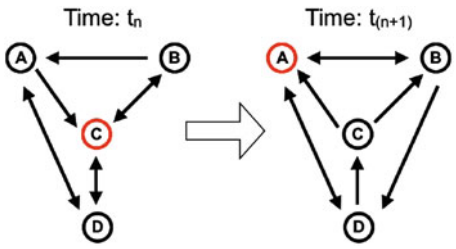

Fig. 2 Dynamic Social Network. We show an example dynamic social network. In this model, each individual in the social group chooses 2 other individuals with whom to interact (outgoing arrow). At each time step, every node in the graph modifies their network by: dropping one existing outgoing edge, adding one new outgoing edge. As the network evolves, the importance/significance of an individual in the network may change. For example, at time t_n, node C (red) has the largest in-degree value (e.g., is the most popular individual) but at $t_{(n+1)}$, node A (red) the largest in-degree value

actually increase the spread of infection [79]. Thus, when studying pathogen spread in social networks, one must be cautious to consider the network centrality measures discussed in previous sections (degree, closeness, and betweenness) to quantify the dynamics and level of heterogeneity in the network in order to properly characterize pathogen spread.

Dynamic network models such as those just described are capable of independently simulating both social choice and pathogen transmission, demonstrating that emergent social organizations could help protect populations from the spread of pathogens. In particular, the authors of [36] hypothesized that, under certain scenarios, increased complexity of social network structure could have evolved in response to pathogen pressure rather than in spite of it.

Ongoing research has shown that the complexity of emergent network structures resulting from ongoing social dynamics can significantly impact patterns in the circulation of infection [33]. However, there are many open questions in the field, including what kinds of social structures provide protection against disease and whether increased complexity of species interaction in the network improves or harms population fitness. Evolutionary studies involving more complex network dynamics including individual knowledge of fitness, kin ties in social networks, and "rule-breaking" individuals in a population have only recently been incorporated into disease dynamic models to better understand this story [29, 35, 38]. The divergence in epidemiological patterns between social scenarios that differ in seemingly minor ways demonstrates the need for further study in this area. This work can extend to understanding evolutionary constraints on social populations that may be critical for persistence under any number of threats, including deaths due to disease epidemics.

5 Extensions and Questions for Further Study

The studies discussed in the previous sections of this paper focus almost exclusively on dyadic interactions. For example, these models describe social contacts between pairs of individuals, and then use a union of pairs to capture simultaneous interactions between more than two individuals. This representation may fail to capture the full diversity of types of social interactions, thereby hampering our ability to fully characterize the range of achievable outcomes. While some tools have begun to be developed to investigate higher-order interactions in social behavior [30, 71], a more complete methodology needs to be developed.

Even within the scope of risks from transmissible illness, large gaps remain in our understanding of the impacts of infection on social behavior. While we have reviewed here many of the existing investigations into the impacts of risks from pathogens that evolve according to traditional epidemiological models (e.g., SIS or SIR models), these models cannot describe all infections that may significantly impact the evolutionary fitness of individuals in a population. In particular, large

fitness consequences from parasitic infections have been demonstrated in many social species. Parasites differ in their dynamics in critical ways from microbial pathogens and may, therefore, select for very different types of social behaviors to confer population-level resilience or robustness to their circulation [32, 50]. Self-grooming and allogrooming behaviors directly impact the severity of health outcomes to individuals suffering from parasite infection, and the frequency/patterns of allogrooming can be directly determined by social organizational patterns [66]. Furthermore, in addition to reducing the parasite load of the groomee (the target of allogrooming), the groomer is potentially exposed to risk of parasite infection from undertaking this social hygienic act [70]. Perhaps most importantly, the evolutionary impacts of parasitic infections act directly on both individual health and also on mating opportunities in ways that infections from pathogens may not, especially for chronic infections [85]. Given the ubiquity of parasitic infections across the animal kingdom, dynamic network models need to be developed and analyzed in order to understand the long-term dynamics and evolutionary consequences of these diseases. Some studies on parasitic infections are described below, leading to specific questions that mathematical models could address.

A recent study, using the known social network of a group of wild lemurs as a basis for social organization, considered the effects of a hypothetical parasitic outbreak on the population of lemurs [68]. In this case, the network's modularity—amount of clustering in the social organization—correlated negatively with disease spread. The idea of the social group clustering into subgroups had been previously addressed in [55], and arrived at the same conclusion that the more individuals that stay within their subgroup the less likely a pathogen will spread to the entire social group. In [68], the outbreak was simulated on both dynamic and static networks and it was discovered that the average parasitic outbreak size was greater with dynamic networks vs static networks. Thus, supportive evidence exists that the results from dynamic networks differ from static networks and that dynamic networks are needed to model social interactions. However, the work in [68] did not include information about the relationship between the size of the network and the parasite type or parasite load of the individuals in the network. The results from this study demonstrate the theoretical difference in outcomes between static and dynamic networks, though it will be an important next challenge for the field to determine which model best matches empirical systems.

In [60], several studies regarding parasitic infections are combined to show that the spreading of parasites depended on parasite mobility and group size. As group size increases, parasite intensity, defined as the number of parasites per individual, increases for direct parasites or indirect parasites where direct parasites are parasites with one host, e.g., most ectoparasites, while indirect parasites are parasites with more than one host, e.g., endoparasites. However, with mobile parasites, e.g., mosquitoes and biting flies, the number of parasites decreases as group size increases. This is hypothesized to be because of the "dilution effect" whereby the parasites are spread among more individuals in the group, reducing

the number of parasites per individual. However, the social structures of the group or if the group had possible defensive strategies against particular parasites is not discussed [60].

We know that a cost of living in social groups may be the possible increase in parasite load, depending on group size and the mobility of the parasite; however, this is not always the case. For example with rodents, a study found sociality reduced the number of ectoparasites, speculating that one factor may be that social species evolved defensive strategies like allogrooming to reduce parasitic load [6]. Although allogrooming may reduce the number of ectoparasites in a social group, it also comes at a price to the animal [6]. For example, if the animal relies on the herd to signal when predators approach, slowing the recognition of danger may lead to one's death. In impalas, being engaged in the act of allogrooming has been shown to increase the time it takes to notice an approaching predator [51]. Further, the delay is even worse for the impala performing the act of grooming than it is for the impala being groomed. In bats, more parasites led to more frequent grooming and less rest, which meant that the bats needed more energy and time devoted to dealing with parasites [26].

In previous sections, we have discussed trade-offs between the circulation of infectious pathogens and efficient social organizations. However, these are not the only evolutionarily relevant effects of patterns of contact that emerge from individual social behaviors in a population. Many different features of social organization have been shown to be important to the fitness of individuals in populations of social species beyond the simple centrality, stability, and resilience we have explored. As the most naive extension, it is clear that social populations may need to balance many different types of organizational success to achieve sufficient evolutionary fitness to persist. For example, the same population may need to achieve both the ability to assimilate parallel information from scouts to make communal foraging or nest selection decisions efficiently while also enabling new individuals to assume appropriate positions in the social hierarchy without having to invest the time and social capital to establish actual dominance [37]. Structures that enable efficient information gathering and assimilation for centralized decision-making in some cases may be compromised by the need to also support distributed, consensus, or even individual decision-making in other contexts [14].

The evolutionary consequences of relative success at achieving social structures that meet the full diversity of simultaneous needs faced by a population will necessarily depend both on the frequency of the situations in which the structures are required and the severity of fitness impact suffered by failing to meet these needs well. We can, unfortunately, be certain that the complexity of selective pressures acting on social structures of groups will be at least as challenging as teasing apart the variety of selective pressures faced by individuals—an endeavor which has occupied the field of evolutionary biology since Darwin.

Each group's social organization may or may not contribute to the spread of a disease or parasite. Theoretical studies mentioned using three measures of societal organization—degree, betweenness, and closeness—showed that the type of organization contributed to the lowering or raising of the risk for pathogenic

infection [34, 36]. Observations from these studies emphasized that not all groups choose one type of organization and follow it closely. Thus, the goal is to investigate if a social group structure affects parasitic risk in the group's population. As stated above, the group's organization, including network modularity, also contributes to the risk of parasitic infection. Mathematical models involving social network structure and disease dynamics can be effective in understanding these ideas further, just as they have been useful in understanding the spread of infectious diseases.

6 Concluding Remarks

Classical evolutionary theory considers the fitness of an individual expressing particular traits. More recently, evolutionary theory has expanded to include concepts of "group" and "multilevel" selection because when individuals live in groups, the traits one individual expresses may impact the fitness of others. While an abundance of literature has considered how social dynamics impact the spread of disease, only recently have researchers considered how social behavior has evolved in response to disease. Because susceptibility to disease has historically served as a strong driver of selection, it is likely that the social behaviors observed in modern populations would be those that were favored to minimize the spread of disease.

In this work, we reviewed the literature on the evolution of social organization and social behavior, such as grooming, through its ability to improve the capability of populations to cope with disease. While close social connections may facilitate the spread of pathogens and parasites, social behavior such as grooming may act to lessen the disease burden of individuals. While the majority of studies have considered only static social networks, when social networks are allowed to vary dynamically, population-level disease burdens can be substantially different.

Though progress has been made, many questions remain and we have outlined areas of further study. First, social organization and disease are not the only forces influencing population-level selection and other population-level characteristics such as the ability to work in subgroups or forage warrant further study. Second, dynamic networks have thus far only considered centrality of an individual as an attractive trait, but it is possible that social behavior such as allogrooming or parasitic burden may itself influence an individuals' social standing. Finally, we note that while mathematical modeling has proven an important tool for studying sociality and disease spread, there is not a single mathematical approach capable of fully characterizing the behavior of social organization and disease. Differential equations will have difficulties capturing the stochastic effects from dynamic networks, and computational simulations do not easily allow us to probe system stability of sensitivity to parameters. Tackling the open questions in the evolution of sociality will continue to require an interdisciplinary approach between mathematical and computational modeling, network theory, and empirical ecological studies.

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