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INTRODUCTION

Sociality and disease: behavioral perspectives in ecological and evolutionary immunology

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The coronavirus pandemic has made us all too familiar with the evolutionary ecology of disease and the role of social behavior in the transmission of infection. Although the human behavioral dimensions of the pandemic are well-documented (e.g., Seitz et al. 2020; van Bavel et al. 2020), few likely recognize that we are not unique in increasing inter-individual distance ("social distancing") to mitigate virus spread (Lopes 2020) and that we share other behavioral mechanisms of infection control with non-human social species. The past two years provide a timely illustration of the importance of basic and applied socioecoimmunological research from a broad phylogenetic perspective (Schmid-Hempel 1998, 2021a; Cremer et al. 2007, 2018; Rosengaus et al. 2011).

The study of disease ecology, pathogen transmission, and group living began approximately 70 years ago (Collias and Southwick 1952; Traniello and Bakker 2020). Since then, research in the discipline has expanded and diversified through integrative evolutionary, ecological, behavioral, genetic, physiological, and comparative immunology (Anderson and May 1979, 1982; Hamilton 1980, 1982, 1993; May and Anderson 1990; Schmid-Hempel 1998, 2001, 2021a; Rosengaus et al. 2011; Malagoli 2016). As the new hybrid discipline developed, an important paradox concerning sociality and infection risk was recognized: while social evolution may be favored by high genetic relatedness among group members (Hamilton 1964a, b), kin groups are more susceptible to disease due to decreased genetic variation (Anderson et al. 1986; Hamilton 1987; Schmid-Hempel 1998; Sherman et al. 1998). Therefore, the costs of increased pathogen loads and/or disease transmission could potentially outweigh the benefits of group living. Eusocial insects then emerged as significant and tractable model systems to advance our understanding of the significance of genetic diversity (decreased due to haplodiploid sex determination) in disease susceptibility (Shykoff and Schmid-Hempel 1991; Schmid-Hempel 1998; Tarpy 2003; Seeley and Tarpy 2007; Reber et al. 2008; Wilson-Rich et al. 2009; Simone-Finstrom et al. 2016).

An interdisciplinary approach that embraces the role of behavior within a broad definition of immunocompetence (Owens and Wilson 1999) expanded our understanding of comparative and evolutionary immunology (Sheldon and Verhulst 1996; Schmid-Hempel 1998, 2021a; Siva-Jothy et al. 2005; Rolff and Reynolds 2005; Sadd and Schmid-Hempel 2009). Additionally, life history theory and concepts of energy allocation have been applied to quantify relative investment in immunocompetence and identify potential trade-offs between fitness parameters and the induction of an immune response (Hamilton 1978; Hamilton and Zuk 1982; Sheldon and Verhulst 1996; Schmid-Hempel 1998; Owens and Wilson 1999; Sadd and Schmid-Hempel 2009). This perspective emphasizes the high energetic cost of immunity and its significance as a life history trait (Zuk and Stoehr 2002; reviewed by Tieleman 2018).

The dynamics of behavior and disease transmission are in large part influenced by the degree of sophistication of host immune systems. Although invertebrates generate immune responses at cellular and humoral levels with some degree of immunological memory (i.e., priming effects) and specificity (reviewed in Cooper and Eleftherianos 2017; Prakash and Khan 2022), they lack important aspects of the antibody-based adaptive immunity characteristic of vertebrates. Yet, despite their presumed "less robust" immunity, throughout evolutionary time the immune function of invertebrates appears to have been extremely effective in reducing rates of infection because their physiological immune responses

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are deployed in conjunction with behavioral and biochemical adaptations.

The recognition that social behavior is a mechanism by which invertebrates can control disease and thus improve upon the benefits derived from a less-sophisticated immune system stimulated research on eusocial insect pathobiology. Eusocial phenotypes — potentially enormous and complex colonies composed of millions of individuals — are highly diverse, and in the case of soil-dwelling ants and termites may exploit microbial-rich and pathogenic environments. Honeybees as well are susceptible to diverse and potentially virulent pathogens and parasites (Tarpy 2003; Evans and Spivak 2010; Hristov et al. 2020). Unlike the haplodiploid eusocial Hymenoptera, termites are diploid and enable comparisons of strategies and mechanisms of immunocompetence, some of which are convergent. The behavioral, biochemical, and immunological responses of termites against both bacterial and fungal pathogens (Rosengaus and Traniello 1993, 1997; Rosengaus et al. 1998, 1999, 2007; reviewed in Rosengaus et al. 2011), together with the role of the gut microbiome in disease resistance (Rosengaus et al. 2014), suggest that pathogens colonizing the prototermite nesting, feeding, and/or foraging resources posed significant selection pressures during their social evolution (Rosengaus et al. 2011). Termites were the first eusocial insect clade in which social facilitation of disease resistance and social immunity were demonstrated (Traniello et al. 2002). Eusocial insects, as superorganisms, exhibit significant parallels in immune function with those of multicellular organisms (Cremer et al. 2007, 2018; Cremer and Sixt 2009; Cremer 2019; Pull and McMahon 2020). These studies led to extensive empirical and theoretical research on the collective mechanisms by which group-living organisms defend against pathogens. Today, the concept of social immunity has expanded beyond eusocial insects and has been applied to clades as diverse as social microbes and primates (Nunn and Altizer 2006; Cotter and Kilner 2010; Walke et al. 2011; Meunier 2015; Van Meyel et al. 2018; Spivak et al. 2019).

Invertebrates have also provided excellent models to examine mechanisms underlying the vertical transmission of immune protection. Transgenerational immune priming (TGIP) appears to be widespread (Tidbury et al. 2010; Roth et al. 2018) across solitary (Little et al. 2003; Moret 2006; Freitak et al. 2014; Salmela et al. 2015) and eusocial species (Moret and Schmid-Hempel 2001; Saad et al. 2005; Saad and Schmid-Hempel 2007, 2009; Barribeau et al. 2016; Cole et al. 2020). Several mechanisms underlying TGIP have been identified, including the transfer of immune elicitors, immune proteins, and/or epigenetic markers that render progeny less susceptible to disease.

The dynamic integration of vertebrate pathobiology and sociality is likely influenced by their more sophisticated immune function, characterized by immunological memory and high degrees of specificity (Schmid-Hempel 2021a). Vertebrates also appear to rely on behaviors like self-medication (de Roode et al. 2013), avoidance of infectious conspecifics (Stockmaier et al. 2021), and (allo)grooming (Mooring et al. 2004) to reduce pathogenic/parasitic loads and control infections. Most vertebrates have both innate and acquired immune systems, each with different costs and benefits (McDade et al. 2016). There will be a balance of investment in both immune systems that is contingent upon level of nutrition, degree of pathogen exposure, and signals of extrinsic mortality risk during sensitive periods of immune development (McDade et al. 2016), and on the etiology of disease (Boots and Bowers 2004).

The field of socioecoimmunology addresses fundamental questions of how social groups may achieve reduced pathogen transmission and infection through the evolution of diverse anatomical, behavioral, biochemical, and physiological adaptations at the level of the individual and the society (Rosengaus et al. 2011). Through this multidisciplinary and synergistic perspective, the significant infection-control advantages of social living across diverse taxonomical groups have been recognized. Today, previously unidentified subtle factors such as seasonality, resource distribution, community dynamics, within-group demography, rates of information transmission in respect to the presence of pathogens, and degree of hierarchical behavior within a group have been incorporated (Romano et al 2020). These factors, in turn, appear to influence the magnitude, strength, and permanence of social bonds of group members. The existence of possible feedback loops can further reinforce these social interactions (i.e., social connectivity) and, ultimately, affect within-group social cohesion, structures, and networks (Romano et al. 2020).

The *Sociality and Disease* article collection provides a timely focus on the bidirectional dynamics between social hosts and their pathogens in a diverse array of invertebrate and vertebrate clades. Contributions to the collection range from model group-living systems with relatively simple organization to complex eusocial societies, and pathogens and parasites as diverse as viruses, bacteria, fungi, nematodes, and helminths. The contributions consider empirical tests of hypotheses and theoretical perspectives and reflect cutting-edge multidisciplinary research on the importance of host densities, spatial/temporal distribution of hosts and pathogens, pathogen loads, modes of infection, host and parasite life histories, immune defenses by hosts and immune evasion by parasites, the frequency with which social interactions among group members occur, and how environmental pressures pose differential selection pressures on immune-related genes.

Several broad themes emerge from the contributions to this topical collection.

(a) Social host/pathogen/parasite coevolution

Parasites and pathogens pose important selection pressures on their hosts, and it is recognized that hosts living at high densities — as in social groups — have an increased risk of infection. The positive correlation between high host density and high infection rates is exemplified by Ritchie et al. (2021) involving manipulated social group size in captive California slender salamanders: skin-to-skin contact increased an individual's risk of infection with a fungal pathogen. In this case, significant costs (increased infection) are incurred after physical contact with infected group members. Interestingly, eusocial insects may exhibit an opposite pattern provided that pathogen load is not excessive. At intermediate pathogen loads, grouped ants and termites, for example, are less susceptible to disease than isolated individuals (Rosengaus et al. 1998), indicating that social defenses against pathogens are not equivalent among taxonomic groups. That pathogens pose significant selection pressures on their hosts is also supported by Bulmer and Stefano (2022), whoaddressed the question of whether molecular evolution of two β-1,3-glucanases (GNBP1,2) intensified during the transitionfrom nesting and feeding within the same piece of decayed wood to foraging outside the nest in termites. The evolutionary transition in feeding and foraging strategies of termites presumably resulted in increased encounter/exposure to soil entomopathogenic fungi. Bulmer and Stefano (2022) accentuate that not all immune-related genes are under the same selection pressures. The communal adaptations of termites to resist infection (including mutual grooming, sharing and depositing of antimicrobial secretions in the nest, social immunization and/or variolation; i.e., social immunity (reviewed in Rosengaus et al. 2011)) appear to have resulted in the relaxation of selective pressures on several components of the termite physiological innate immune system.

Host sociality can drive pathogen/parasite evolution. Janecka et al. (2021), using guppies as a model, stress the importance of recognizing the role that host social behavior has on generating fine-scale changes in the spatial distribution of parasite genotypes, their influence on parasite non-random mating, gene flow, genetic drift, changes in population effective size, bottlenecks, genetic diversity, and, ultimately, parasite virulence and host/parasite coevolutionary history. Interestingly, Moore et al. (2021) add yet another level of complexity to host/pathogen coevolution. By focusing on cliff swallows and their alphavirus interactions, they report that the introduction of an alternative host (house sparrows) alters the group-size consequences for the cliff swallows (the principal host). Their results show that virus loads carried by bloodsucking bugs increased with cliff swallow group size when the house sparrows were absent. However, when house sparrows were present, the loads decreased. The virus appears to have diverged into two lineages, one carried in high numbers when the colony size in cliff swallows is large and another lineage well adapted to infect house sparrows without requiring large numbers of bugs and cliff swallows for effective transmission and persistence. This work highlights the significance that alternative invasive hosts have in influencing host/pathogen dynamics.

Schmid-Hempel (2021b) suggests that, in the face of pathogenic/parasitic risks, the major difference between solitary and social hosts is not in the deployment of defenses by individual hosts and the within-host parasite success, but rather the within-group transmission pathways. He argues that within-group transmission is the most important selective episode for the evolution of social hosts and their parasites. He coined the concept of "generalized transmission distance," which captures significant elements of parasite transmission success such as the temporal, spatial, genetic, and ecological proximities associated with social life and social organization. The combination of all these elements determines parasite fitness: short-distance transmission among genetically similar hosts within the social group, for example, is the most frequent process by which parasites attain high fitness. A logical conclusion is that the "generalized transmission distance" also helps us predict that the most effective defenses a host can evolve are those that diminish within-group spread of infection, especially when members of the group are genetically related.

Ruiz-González et al. (2022) compare susceptibility between bumblebee gynes (the reproductive caste) and workers (which are sterile), concluding that a trypanosome infection does not equally affect the two different castes. Gynes were not only less susceptible to infection than workers, but the two castes differ in their immune profiles. Therefore, it appears that even against a similar genetic background of the host, the trypanosome exerts differential selection pressures, likely driven by reproductive potential. These results point to the existence of multiple phenotypes from a single genotype that maximize fitness in a social group context.

(b) Disease and social structure/network

Several articles in this collection leverage social structure and network analyses to provide insights into the role of behavioral interactions on disease transmissibility. Deere et al. (2021), for example, found that wild chimpanzees that spent more time with more individuals in the same area had higher helminth parasite richness, concluding that same shared space, rather than grooming contact, is responsible for higher parasitic risk. However, Lynsdale et al. (2022) failed to find an association between gastrointestinal nematode load and sociality (measured by individual solitary behavior, work group size, and work group sex ratio) in semi-captive Asian elephants employed in timber logging.

Social networks influence not only disease transmissibility in primates; parasites may in turn influence host social networks, as shown by Poulin and Filion (2021). In this case, correlative evidence across primate species suggests bidirectional effects between host social structure and their pathogens.

Silk and Fefferman (2021) examine the role of social structure and social dynamics in maintaining endemic disease. Through their synthetic theoretical and empirical work, they highlight the importance of both social structure and dynamics in maintaining reservoirs of agricultural and zoonotic diseases. Distinguishing between local and global persistence of infection, they provide a framework by which sociality contributes to the long-term maintenance of infectious disease in wildlife hosts. Evans et al. (2021) use theoretical simulation models to ask whether trade-offs between the spread of information within a group and the spread of infection exist. They conclude that modular networks can promote the spread of information relative to the spread of infection, but only when the network is fragmented and group sizes are small. Therefore, it appears that highly fragmented networks and multilevel societies can be effective in modulating the infection-information trade-offs within a group.

Roitberg and Rosengaus (2022) generate two dynamic state variable models incorporating the roles that termite social structure (demography and caste composition), collective immune responses, parental contributions to progeny, and varying pathogenic loads have on the expression of transgenerational immune priming (TGIP). Their synthetic and empirically informed termite-specific model as well as a more simplified version applicable across the sociality spectrum should have heuristic value to generate future research that focuses on this widespread phenomenon. One of their main conclusions is that trade-offs among competing demands and the cost/benefit analyses of engaging in TGIP are not constant throughout a colony's life cycle. Given that the demographic and social structure of a eusocial insect colony changes dramatically from an incipient to a mature colony (and, consequently, the colony's collective immunocompetence), the adaptive value of TGIP may be context-dependent.

(iii) Disease and behavioral change

Pathogens influence the behavior of social hosts. In the face of disease risks, group cohesion can become weaker or stronger. In other words, diseased individuals can exhibit higher social tendencies or, on the contrary, demonstrate increased antisocial behavior. Kramer and Bressan (2021) review the literature and argue that humans were selected to detect "sickness" traits. The perception of such cues can influence mate choice and sexual activity, and help explain why social ties strengthen within groups but turn hostile toward others who look, smell, or behave in an unusual fashion, in turn permeating the foundation of moral and political views. Blersch et al. (2021) focus on sickness behavior in wild vervet monkeys in a semi-arid region of South Africa. After quantifying monkey activity budgets and behavioral predictability as a function of infection with two gastrointestinal parasites, they conclude that sickness behaviors in monkeys was context-dependent, contingent on the type of parasite and food availability, and that ecological stress likely overrides the ability to express sickness behavior in an adaptive fashion. Lemanski et al. (2021) generate an agent-based model focused on how territorial behavior in eusocial insects (a type of "antisocial behavior" in the authors' opinion) prevents the introduction of infected foreign workers into a colony and concluded that territoriality can flatten the curve of an epidemic, delaying the introduction of an infectious disease and reducing its maximum prevalence. However, this was again context-dependent; the benefits of territoriality against risks and prevalence of infections were only observed when pathogens ranged between low to moderate transmissibility. Demandt et al. (2021) further support the idea that some social species exhibit antisocial tendencies when infected by investigating changes in shoaling behavior in three-spined sticklebacks infected with a cestode parasite. After manipulating group size as well as the number of infected fish within a group and subsequently exposing those fish to simulated bird predation, they found infection reduced shoal cohesion and speed. They conclude that the significant impact of infection on the groups' anti-predator behavior has important implications for collective decision-making.

Declarations

Conflict of interest The authors declare no competing interests.

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