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Science & Society

Stemming the Flow: Information, Infection, and Social Evolution

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Social information and socially transmitted pathogens are governed by social structure, and also shape social interactions. However, information and infection are rarely investigated as interactive factors driving social evolution. We propose exactly such an integrative framework, drawing attention to mechanisms of social phenotypic plasticity for information spread and pathogen control.

Social Decisions and Social Transmission

> It is certain that either wise bearing or ignorant carriage is caught, as men take diseases, one of another: therefore, let men take heed of their company (Shakespeare, *Henry IV*, part 2, 1600).

As William Shakespeare observed, the contacts that individuals make lead to exchange of information among them, for better or worse, as it does for the agents of disease. The current SARS-CoV-2 pandemic has brought this into sharp focus, but, like humans, other group-living animals constantly make decisions that affect their social lives, such as whether or not to interact with a particular group-mate. Individuals modify their social contact and social relationships based on environmental conditions. Such behavioral plasticity not only mediates an individual's own survival and reproduction but also alters its overarching social network, thereby influencing the dynamics of information and pathogen flow within the group. Maximizing information gain while minimizing pathogen spread is exactly what we humans try to optimize in the midst of a pandemic, but is there scope to use related animal behavior research in the search for solutions to promote global health while better understanding social evolution?

Social interactions influence population-level processes such as information and pathogen transmission [1,2]. Animals use social information acquired from conspecifics in a variety of contexts, such as in the identification of new foraging areas or predator threats. However, social contact among individuals, that is crucial for establishing and reinforcing social bonds, also risks contagion. Some pathogens, such as respiratory viruses and sexually transmitted diseases, are spread through sociosexual contact and/or spatial proximity. These costs and benefits of social transmission lead to an evolutionary trade-off – although social relationships favor the transmission of social information, they also favor the spread of socially acquired pathogens. It may be that transmission depends on the number and strength of social interactions (i.e., simple contagion) and/or on the occurrence of individuals in tight clusters, but the proportion of interacting partners expressing the behavior (i.e., complex contagion) is also relevant [3]. Nonetheless, both information and pathogen transmission depend on social connectivity.

The details of these social interactions, for example as revealed by social network analysis (SNA), help us to understand how individuals deal with the challenges of group-living. Pertinent to the present manuscript, through SNA, researchers have discovered that an individual's decisions about with whom to interact are sensitive to its status as informed or infected (e.g., [4]). Informed individuals might be more central within a network [4], whereas individuals avoiding contact with clinically ill group-mates might instead contribute to a more subdivided and less connected network [5]. Such behavioral and network plasticity ultimately feeds back into the structure of information and pathogen exchange, and is thus a crucial component of the resilience of populations facing socioenvironmental challenges. With this in mind, in Box 1 we propose an integrated framework for understanding emergent social structure given the competing pressures on individuals for information acquisition and pathogen avoidance.

Mechanisms of Social Avoidance

In its most simplistic form, the relationship between sociality and infectious disease is, on the surface, straightforward – animals living in closer proximity and with higher contact rates should experience higher rates of pathogen transmission. However, myriad defenses to prevent and/or respond to pathogen invasions have evolved. These antiparasite strategies have been collectively termed the 'ART of pathogen handling': avoidance, resistance, tolerance [6]. To complement immunological defenses, behaviors such as hygiene, self-medication, and social avoidance have evolved. Social avoidance may be among the most important mechanisms to prevent pathogen transmission [7]. Mechanisms of avoidance, however, may vary. Individuals may actively self-isolate or, owing to their own lethargy, may engage in fewer social interactions as part of a generalized sickness response. Uninfected individuals may also actively avoid infected conspecifics, especially those showing signs of sickness. Isolation of the infected may even be enforced by others. By whatever mechanism, restricted social interaction ultimately restructures the social network in ways that downregulate social transmission.

Examples of social avoidance in animals exist in taxa as divergent as arthropods and mammals (Box 2). One poignant example seen in black garden ants (*Lasius niger*)

Box 1. At the Interface of Social Transmission and Social Structure

Hinde's framework of animal society [10] posits that the quality and patterning of relationships are molded by social structure, while social structure itself is driven by socioecological pressures acting on individuals. Cantor and Whitehead [1] extended Hinde's framework by incorporating information transmission at the final level of the model (Figure I), highlighting bidirectional effects. An example might be the divergent vocal repertoires of cetaceans that cause clustering of individuals with similar vocal patterns [1]. Within clusters, individuals are then more likely to share information.

What remains missing from Hinde's framework is a role for deleterious forms of social transmission such as those that propagate infectious disease. Socially transmissible pathogens may negatively affect social cohesion, either directly or indirectly, by reducing social connectivity. Concurrently, the overarching social structure sets the conditions under which individuals are exposed to such pathogens, creating bidirectional feedback. We extend Hinde's framework to incorporate connection costs. Instead of considering transmission as an endpoint, or assuming that information flow is the only relevant variable, we suggest simultaneous examination of information and pathogen transmission as explicit and opposing entities that mediate social decisions and the emergent social structures they underpin. The result is a classical fitness trade-off in which the need to exploit social relationships while minimizing the associated costs dynamically drives the evolution of social behavior (Figure I).

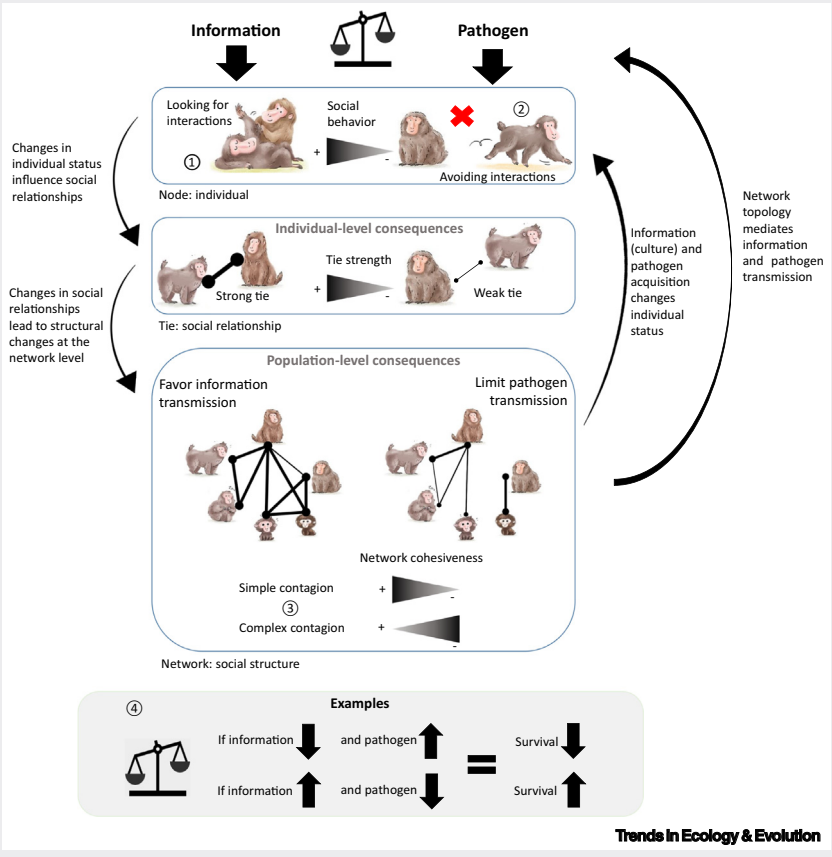

Figure I. A Framework Outlining the Feedback Loop between Individual Behavior, Social Structure, and Social Transmission, both Beneficial and Detrimental. Changes in individual status (informed and/or infected) lead to different patterns of social interactions. Informed individuals are seen as valuable interacting partners (1), whereas infected individuals are avoided by others or reduce their own interaction rates (2). These changes in social relationships lead to structural changes at the network level that affect social transmission. More or less cohesive networks affect the rate of transmission, which depends on the system. Simple contagion is linked to the number and strength of social relationships, whereas complex contagion depends on the proportion of social connections with informed or infected individuals (3). Network topology then mediates social transmission, which affects individual status. We propose a framework that integrates the two mechanisms, simultaneously examining information and pathogen flow as explicit and opposing entities, with emergent patterns of social behavior, and thus social connectivity, reflecting a trade-off between them. We exemplify this trade-off through a survival indicator: survival increases with increasing information gain and reduced pathogen exposure, and vice versa (4). Macaque images, credit: Delphine Vaufrey.

demonstrates that pathogen exposure induces behavioral changes that reinforce transmission-inhibitory characteristics in the contact network: namely increased modularity, clustering, and assortativity, and thus decreased transmission efficiency [5]. Not only do pathogen-exposed foragers isolate themselves, healthy foragers also decrease their time with the rest of the colony. Such pathogen-triggered behavioral responses reduce social connectivity and thereby limit pathogen transmission [5].

The aforementioned example involving ants highlights that social animals are capable of modulating their behavior dynamically in ways that increase the benefits and reduce the costs of social interactions, and this should lead to detectable changes in network structure (Figure 1). Nevertheless, how are individuals expected to behave when, despite the risks of infection, the need to acquire social information remains? Although there are few empirical studies, a recent theoretical model of roost selection in bats has demonstrated that fission-fusion behavior, whereby individuals fluctuate in their degree of sociality through time, resulting in more or less modular networks, is a strategy that can result from a collection of individual decisions aimed at maximizing information accuracy and minimizing infectious disease risk [8] (Figure 1).

The Evolutionary Mechanisms of Social Transmission

Individual decisions about with whom and how frequently to interact are flexible and responsive to the divergent pressures individuals face – and they have fitness consequences. Changes in social connections during and after information and/or pathogen transmission will affect how such entities propagate further throughout the network. Indeed, the potential for information or pathogen exchange is expected to shape social bonds [1,5], as we elaborate upon in our integrated theoretical framework (Box 1). Given some degree of flexibility, individuals that better adjust

Box 2. Examples of Social-Avoidance Behaviors across the Animal Kingdom

Evolved strategies other than the physiological immune system allow social animals to combat the spread of infection, such as conspecific avoidance. Examples of social avoidance and self-isolation abound in nature (Figure I). Caribbean spiny lobsters (*Panulirus argus*) avoid dens housing individuals infected with a lethal virus [11]. Immune-challenged mice (*Mus musculus domesticus*) reduce their own rates of social contact by avoiding encounters with group members [12]. Trinidadian guppies (*Poecilia reticulata*) avoid conspecifics infected with an ectoparasite in the late stages of infection [13]. Mandrills (*Mandrillus sphinx*) recognize parasitized conspecifics and avoid grooming contaminated body regions [14]. Social insects even go so far as to engage in collective defense against parasites through so-called 'social immunity' where they cooperate in different ways to mitigate colony spread [15]. These examples emphasize how social behavior can influence the dynamics of pathogen transmission via an added layer of defense.

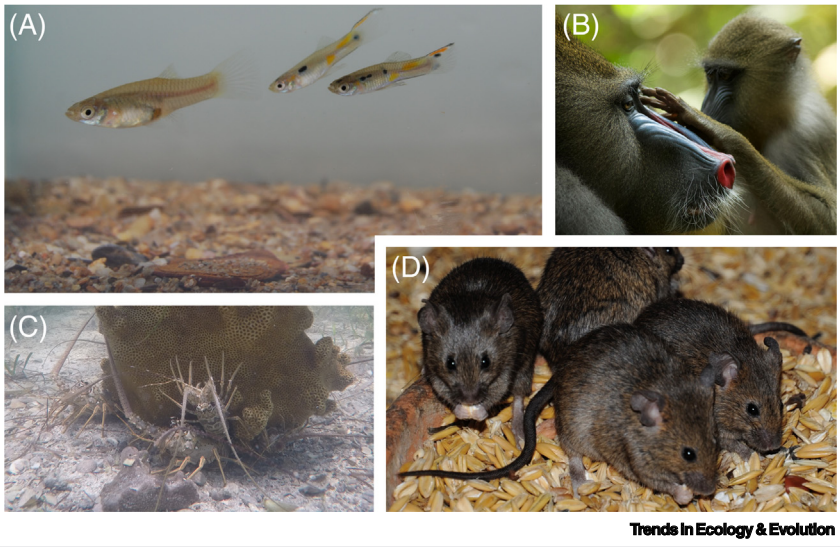

Figure I. Empirical Studies Demonstrating That Social Avoidance Is an Important Strategy for Controlling Pathogen Spread. (A) Trinidadian guppies (*Poecilia reticulata*) (credit: Darren Croft), (B) mandrills (*Mandrillus sphinx*) (credit: Paul Amblard-Rambert), (C) Caribbean spiny lobsters (*Panulirus argus*) (credit: Donald Behringer), and (D) house mice (*Mus musculus domesticus*) (credit: Barbara Koenig).

their behavior in response to the challenges both external to and inherent in social relationships, within the context of their own dynamic social networks, might better optimize their own fitness. Selection pressures driving information acquisition and pathogen avoidance across individuals – via their actions on the heritable components of the social phenotypes – can thus have far-reaching consequences. The interactions of individuals underpin the same networks that determine the efficiency with which information, or a pathogen, might spread. This creates a feedback loop between the selection pressures driving social decisions in individuals and the emergent properties of the social structure in which those individuals are embedded. Neither individual social decisions nor emergent network properties are expected to be static across time or stable across environments. Thus, exploration of this neglected trade-off has potential to deepen our understanding of how natural selection acts on social behavior and thus drives the evolution of social systems.

Prognosis: Going beyond Our Social World

Focusing on the role of individuals, as they navigate their social worlds and contribute to the various flows within their respective networks, highlights that effective epidemic control strategies depend on the collective sums of their behavior. We have shown that solutions do exist in social animals, and that, as in humans, other species can adapt their contact rates, their communication modalities, and their network structure to limit pathogen spread. Our modern understanding of pathogen spread and its mitigation has a history of less than two centuries, whereas evolution has used hundreds of millions of years to hone solutions to this crucial challenge in nature. More attention on this topic is needed – on a greater diversity of species and on the underlying mechanisms allowing for network plasticity. In this context, we call for more research into the information–pathogen trade-off in social evolution. Such research may reveal novel solutions to infectious disease outbreaks with relevance even to human societies. We propose that consideration not only of how infectious diseases emerge in animal populations but also of how they are regulated and even mitigated through processes such as those described in this essay is well within the purview of the 'one health' paradigm (i.e., recognition that human health is interconnected to animal and environmental health), and may thus contribute to fostering global health.

Perhaps one of the least controversial lessons we have learned during the current SARS-CoV-2 pandemic is that, for better and for worse, our own social networks are situated within a broader ecological context of interaction with nature. As the onrushing of human consumption invades deeper and deeper into the wild places of the world, we are increasingly exposed to all manner of novel infectious organisms that circulate undetected in wildlife. It is common knowledge that most emerging infectious diseases in human populations are zoonotic (i.e., of animal origin), and as many as 70% originate in wildlife [9]. Social distancing and digital communication can slow the spread of pathogens, but more responsible interaction with the natural

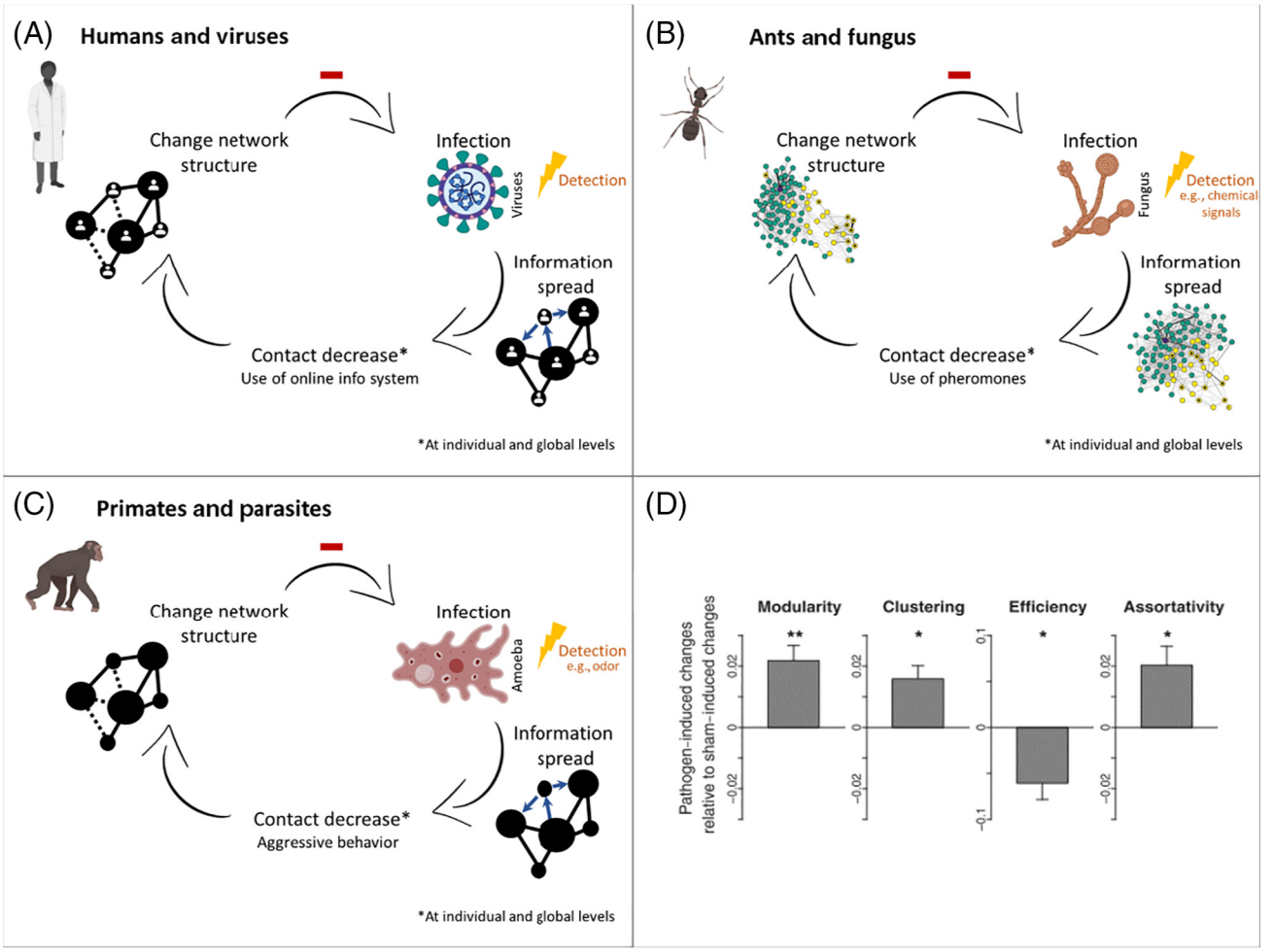

Figure 1. Evidence for Network Plasticity and Its Effects on Information and Pathogen Transmission. The behavioral mechanisms underlying human (A), ant (B), and non-human primate (C) social contact are shown. When a pathogen is introduced into a system and detected, infected and non-infected individuals usually change their contact rates. This leads to changes in network structure, such as increasing the number of subgroups, which affects the efficiency of pathogen transmission (D). Conceptually, the feedback is similar across the three systems depicted. The communication system can also change, as exemplified by both ants and humans, from a targeted and local contact-based modality to a global or more broadly disseminated modality (e.g., public information such as online communications or pheromone trails/clouds). Asterisks refer to the effect of pathogen-induced changes in network properties. *$P < 0.05$; **$P < 0.01$. The networks in (B) and graph in (D) were reproduced from [5] with permission from AAAS. The images of macro- and micro-organisms were created with BioRender.com.

word might have mitigated its emergence altogether.

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Deep-Sea Misconceptions Cause Underestimation of Seabed-Mining Impacts

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Scientific misconceptions are likely leading to miscalculations of the environmental impacts of deep-seabed mining. These result from underestimating mining footprints relative to habitats targeted and poor understanding of the sensitivity, biodiversity, and dynamics of deep-sea ecosystems. Addressing these misconceptions and knowledge gaps is needed for effective management of deep-seabed mining.

Deep-Sea Minerals and Mining Regulation

The deep sea, that is, ocean depths below 200 m, constitutes more than 90% of the biosphere, harbors the most remote and extreme ecosystems on the planet, and supports biodiversity and ecosystem services of global importance. Deep-sea minerals of commercial interest include: (i) potato-sized polymetallic nodules that precipitate on sharks teeth and other hard particles on some abyssal plains; (ii) polymetallic (massive) sulfides deposited at hydrothermal vents along seafloor spreading centers; and (iii) cobalt-rich (ferromanganese) crusts precipitating on rock surfaces on some seamounts and ridges [1]. The International Seabed Authority (ISA) regulates seabed mining in areas beyond national jurisdiction, with a responsibility to protect the marine environment from serious harm (https://www.isa.org.jm/). The ISA has issued 30 contracts covering ~1.5 million km^2 for lower-impact mining exploration, which includes: resource assessment, environmental baseline studies, and test mining. The ISA is currently drafting exploitation regulations for potentially high-impact, full-scale mining, with the regulations to include environmental impact assessment, monitoring, and habitat protection. The ISA's mandate pertains to international waters; however, its exploitation regulations will also be relevant within 'exclusive economic zones.' The United Nations Convention on the Law of the Sea (Part XII, Article 208), specifies that environmental protections for seabed mining within national jurisdictions should be 'no less effective' than those developed by the ISA.

Polymetallic nodules, massive sulfides, and cobalt-rich crusts all provide critical habitat for deep-sea biota. Polymetallic nodules in the Clarion Clipperton Zone (CCZ), an area in the equatorial Pacific Ocean with the richest nodule resources, harbor diverse megafauna (e.g., ~100 species within a 30 × 30 km area) [2] and microbes not found in surrounding waters or sediments [3]. The biotic communities of nodules and sediments vary with nodule abundance [2] as well as along and across the CCZ [4]. Polymetallic sulfides at active hydrothermal vents provide habitat for novel faunal assemblages that have altered our views of the primary energy sources and origins of life, and exhibit substantial local and regional variation in structure and connectivity [5]. Polymetallic sulfide mining is expected to target 'extinct' vents due to the extremely corrosive nature of hot venting fluids, but active vents are not yet protected and extinct vents also have characteristic, albeit poorly studied, biotas [6]. Ferromanganese-encrusted seamounts support productive hotspots of biodiversity that vary within and among seamount chains [7]. Where mining removes or buries any of these three mineral habitats, the associated fauna will be damaged or destroyed.

To manage deep-seabed mining effectively, regulators, such as the ISA (with 167 member states and the EU) and additional stakeholders (e.g., civil society, industry, scientists, and other concerned parties), should utilize the best scientific predictions of mining impacts. Here, we address several misconceptions in the recent peer-reviewed literature concerning deep-sea ecosystems and the potential impacts of seabed mining. We also highlight knowledge gaps and uncertainties in predicting