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Analysis of the Disease Transmission

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ABSTRACT

Zoonotic diseases also known as zoonosis are illnesses that can spread from vertebrate animals to people, and examples include rabies, salmonella, Lyme disease, bubonic plague, and Lyme disease. Humans can become ill from disease-carrying animals, or reservoirs, in a number of ways, including when they consume them, when they bite them, or when arthropods like mosquitoes or ticks that have previously fed on them later feed on a human host. According to estimates, zoonosis account for more than 60% of infectious diseases that affect humans and are on the rise globally, making up more than 75% of new diseases.

KEYWORDS:

Disease Ecology, Disease Transmission, Host Populations, Infectious Diseases, Macro Parasites.

I. INTRODUCTION

The World Health Organization (WHO), the Food and Agriculture Organization of the United Nations (FAO), and the Office International des Epizooties (OIE) jointly convened a study group on veterinary public health (VPH) in 1999. The group, which was made up of 28 experts from 18 different countries, defined veterinary public health as the contribution to the complete physical, mental, and social well-being of humans through an understanding and application of veterinary science. The focus was on the human ecology, where veterinary science has made a significant and long-lasting contribution to human health. Most people do not realize that this contribution extends beyond animal husbandry and food production to include animal transportation and transportation, which created the foundation for the majority of urban societies worldwide. A much of what we know about the dynamics and management of infectious human diseases as well as the improvement of environmental quality has its roots in the study and management of animal diseases.

Zoonotic diseases also known as zoonosis are illnesses that can spread from vertebrate animals to people, and examples include rabies, salmonella, Lyme disease, bubonic plague, and Lyme disease. Humans can become ill from disease-carrying animals, or reservoirs, in a number of ways, including when they consume them, when they bite them, or when arthropods like mosquitoes or ticks that have previously fed on them later feed on a human host. According to estimates, zoonosis account for more than 60% of infectious diseases that affect humans and are on the rise globally, making up more than 75% of new diseases. Since many significant human diseases have their origins in animals, changes in the habitats of animals that serve as disease reservoirs or vectors may have both beneficial and negative effects on human health. For instance, it is thought that the Napa virus first appeared after forest clearing fires in Indonesia drove bat carriers to neighboring Malaysia, where the virus affected intensively farmed pigs before spreading to people. While improving nutrition is good for health, intensive animal production has also helped to create conditions that encourage the spread of illness. Increased human interaction with wild animals, bush meat derived from forest encroachment and dietary changes also increase the risk of disease transmission [1], [2].

Certain populations of mosquitoes, ticks, and midges appear to have been impacted by trends ranging from forest clearing to habitat changes brought on by climate change, changing the patterns of how diseases like malaria and Lyme disease are transmitted. Zoonotic illnesses have not previously been considered to be a component of ecological systems. Zoonosis are common, thus a multidisciplinary subject called disease ecology has arisen in response. Any ecological system with diseases and complicated multiple interactions must be studied in this context. The research field examines the fundamental mechanisms that underlie the interactions between ecosystems, climate, and infectious disease, focusing in particular on the various ways that climate might affect the emergence and spread of infectious disease pathogens. For instance, to better comprehend the myriad of variables that controlled the occurrence and spread of malaria in Oaxaca, Mexican researchers used an ecosystem approach. This study comprises statistical studies, a geographic information system-based surveillance system, community views of malaria, and the molecular biology of the parasite and the vector.

The majority of zoonotic diseases are spread by mammals, with rodents leading the pack. Rodents are responsible for the spread of several diseases, including the plague (*Yersinia pasties*), Lyme disease (*Borelli burgdorferi*), Hantavirus pulmonary syndrome, and Rocky Mountain spotted fever (*Rickettsia rickettsia*). Rodents are considered to be in the center of the food chain ecologically speaking. They provide a source of food for vertebrate predators like foxes and owls because they are primarily herbivores with diets high implant material. The transmission of human parasites by aquatic animals also contributes to sickness and mortality. The cost to humans is high: In the world, according to the World Health Organization (WHO 2004, 2), 1.8 million people die each year from diarrheal diseases, 200 million people are infected with schistosomiasis, and more than 1 billion people suffer from soil-transmitted helminths infections. Sadly, irrigated agriculture systems in particular have a long history of being linked to severe human ill health caused by water-related illnesses. The main reason is that the sector of water resources, which has generally concentrated on possible economic benefits of water bodies in terms of food production and power generation, has not been concerned with public health and disease control programmes.

This applied ecology theme seeks to advance understanding of the connection between water, human health, and ecosystems as well as to provide useful strategies to mitigate harmful effects on environmental health managing agricultural use of contaminated water sources including urban and industrial waste to maximize food production and livelihood benefits and minimize negative health and environmental impacts; investigating the trade-offs necessary to meet environmental water requirements in river basins; and making wise use of wetland ecosystems are all examples of ways to reduce the negative effects of malaria and other water-related parasitic diseases. The risky integration of situations that arise when animals and consumers from several ecosystems interact is another significant concern. Humans and other animals serve as replicating reservoirs for viruses and bacteria that can quickly adapt and evolve due to the lack of resistance to novel diseases. The tremendous number of animals and people in contact also transforms the one-in-a-million chance of a disease transfer into a nearly everyday probability [3], [4].

This reservoir of bacteria, viruses, and other pathogens provides the ideal environment for illnesses to spread quickly and leap between species to take advantage of new potential hosts, something the most successful diseases do all too well. In this case, there are two issues that arise. The first is the significant chance that emerging illnesses will infect people. Second, this may instill a fear factor in people, leading them to try and eliminate the threat by killing the wildlife if they believe it to be unhealthy. Although the link between the disease and flying foxes has not been conclusively established and the sickness is rarely seen in flying foxes, shooting flying foxes was suggested in Southeast Asia when they were supposed to be carrying the nape virus. In addition to spreading disease among humans, livestock, international trade, rural livelihoods, native wildlife populations, and ecosystem health are all at risk due to the worldwide traffic in wildlife. Globally, outbreaks brought on by the wildlife trade have cost the economy hundreds of billions of dollars.

II. DISCUSSION

Although infectious diseases have long been recognized to cause severe illnesses in people, cattle, and crops, it was previously believed that infections had little effect on populations of wild plants and animals outside of the occasional, occasionally dramatic die-offs. In the past two decades, it has become increasingly clear that parasitic organisms are not only a typical and essential component of ecosystems but also have an impact on wild population abundance, have the potential to wipe out their hosts, and act as evolutionary engines. This understanding of the widespread involvement of pathogens in ecosystems has given rise to the subject of disease ecology, which is defined as the ecological study of host-pathogen interactions within the context of their environment and evolution. The study of pathogen dissemination over time and geography as well as its effects on host populations forms the basis of disease ecology. These objectives are distinct from those of adjacent disciplines like epidemiology, which attempts to pinpoint risk factors for infectious and non-infectious diseases, and parasitology, which concentrates on the taxonomy and life cycles of parasites. Here, we go through the theoretical underpinnings and mathematical framework of disease ecology and discuss how ecological and evolutionary studies might be applied to better disease management.

Introduction to Host-Parasite Ecology

In disease ecology, the phrases parasite, pathogen, and infectious disease are sometimes used synonymously to refer to organisms that live in or on a host and draw their resources from it, generally to the host's harm. However, strictly speaking, a disease is a pathogenic condition of a host that is occasionally brought on by a pathogen or parasite; hence, while pathogens and parasites that because disease can be passed from one host to another, actual diseases cannot. To reflect distinctions in their population biology, parasites are typically divided

as micro parasites and macro parasites. In their hosts, micro parasites which include viruses, bacteria, fungus, and the majority of protozoa, including malaria proliferate quickly and on timelines that are far shorter than those of their hosts. This frequently, but not always, results in transient infections that can cause host mortality or immune development. Disease ecologists classify hosts for micro parasites into susceptible (S), exposed, infectious, and, for some infections, recovered (R; or immune) classes that reflect their stage of exposure. Compartment models are described as, for instance, SIR or SI models depending on the host categories that best match the biology of the pathogen [5], [6].

These models are used to track changes in the numbers of hosts in each of these classes but not the number of parasites in each host's body. To remove classes that aren't necessary to reflect gearbox dynamics, models can and should be made simpler. For instance, if the latent phase is extremely brief in comparison to other stages or processes the exposed class may be deleted, and the recovered class may be eliminated if few hosts recover from infection. In addition, if the disease is spread via water, soil, air, etc., models can be modified to include a free-living stage of the pathogen. In contrast, macro parasites are larger, live longer, and seldom complete their life cycle within a single host. These include parasitic worms called helminths and parasitic arthropods like lice. As opposed to this, adult macro parasites typically release infectious stages into the environment, such as eggs or larvae, which may or may not infect the same host that the adult macro parasites inhabit. The host's immune response to these pathogens is frequently insufficient or transient, leading to persistent infections and ongoing reinfections. Disease ecologists keep track of the number of macro parasites in each host and mathematically quantify their distribution across hosts because the effects of macro parasites on their hosts and frequently parasite survival and fecundity depend strongly on the number of adult parasites in each host. Most often, there is evidence of aggregation or clumping in the parasite distribution among hosts, which means that most hosts have few or no parasites whereas a small number of host individuals have numerous.

Due to the fact that parasite effects accelerate for hosts with the highest parasite populations, this clumping tendency can both predict the macro parasite impacts on populations at the population level as well as the level of parasitic competition inside a host. In addition to direct contact between hosts, pathogens can also spread through the air, water, soil, or other surfaces, or by biting arthropods, which can serve as hosts for the pathogen to proliferate. Pathogen survival outside of a host can be determined by environmental conditions including temperature and humidity, with considerable effects on transmission. Environmental factors can also influence how an arthropod pathogen replicates within the vector, which in turn affects whether the vector will subsequently transmit the pathogen. In order to create and evaluate ideas, integrate data, and direct disease management efforts, disease ecologists typically employ mathematical models. This quantitative approach was derived from population ecology. The pathogen's basic reproductive ratio (R0), which characterizes the pathogen's beginning proliferation in a population of previously uninfected hosts, is a cornerstone of this strategy. R0 defines whether the virus may penetrate and spread in a deterministic environment (for example, if R0 surpasses 1).

For values of $R0 > 1$, the number of transmission chains is longer and disease invasion is more probable (but not guaranteed) in the real stochastic environment. R0 is frequently derived intuitively by dividing the rate of infectious individual loss (the sum of the three loss terms: natural death, disease-caused death, and recovery by the ratio of new infections (the SI) caused by one infectious individual ($I = 1$) when the population is fully susceptible ($S = N$). The effective reproductive ratio (Re) in a host population where some individuals may be immune to infection due to prior exposure, vaccination, or other factors for example, inherited maternal antibodies is a more generic quantity. Re is frequently just R0 times the proportion of the host population that is susceptible, or $Re = R0S/N$. Re for macro parasites is the number of adult offspring that a single adult parasite gives rise to over its lifespan, whereas Re for micro parasites is the number of hosts that a single person infects over its infectious lifespan. The question of whether and how host population density affects pathogen transmission is crucial.

Density-dependent transmission is when the spread of a pathogen increases with the number of infected hosts this is shown in Figure 1a, where the probability of infection (I) for each susceptible is proportional to the density of infected hosts and the rate of infection (SI) for all susceptible as a whole per unit of time. As a result, there will be a threshold density (NT) of hosts below which transmission is ineffective and the disease cannot persist (R0 1) in that host population for these density-dependent infections. As will be explored shortly, this has significant consequences for control. Alternately, host density might not have a significant impact on a pathogen's ability to spread. The force of infection, or the rate per capita at which a susceptible person contracts the pathogen (I/N for frequency dependent transmission or I for density dependent transmission), increases with the proportion of the host population that is infectious but does not increase with overall host density (Figure 1). As a result, if transmission is frequency dependent, there is no threshold density for disease invasion. Such diseases have the

potential to survive at incredibly low host concentrations. The majority of infections are likely transmitted somewhere between these two extremes, according to empirical research on mice, voles, ladybird beetles, frogs, and plants [7], [8].

Whether or not a disease transmits via frequency or density-dependent means depends critically on the mechanism of transmission. Direct contact kissing can spread herpes viruses, aerosol sneezing can spread influenza viruses, indirect contact drinking water contaminated with faces can result in cholera infection, or vectors ticks and mosquitoes can spread viruses and bacteria between hosts are all ways that pathogens can be transmitted. Pathogen dissemination via water and aerosol frequently increases with host density. Conversely, it is believed that some vector-borne diseases and sexually transmitted viruses spread more often assuming that rates of sexual interaction do not necessarily rise with host density.

Impacts of Pathogens on Communities and Populations

The size of the host population can be significantly impacted by parasitic organisms. Pathogen virulence, or the pathogen's ability to reduce host fitness, affects how a pathogen affects host populations. The pathogen's ability to limit host survival parameter in the models in Figures 1, reproduction, or both (as depicted in Figure 1) will also affect how much of an influence it has. Pathogens with moderate virulence typically have the most detrimental effects on host populations when they diminish host survival. This is due to the fact that hosts infected with extremely virulent infections typically pass away fast, shortening the infectious period during which parasites can spread. Similar logic dictates that sterilizing infections those that decrease fertility, can lead to larger losses in host population size than those that decrease survival. When there are no other density-dependent factors affecting host abundance, pathogens with density-dependent transmission (Figure 1) can control host populations by reducing and maintaining them at a lower density than they would be without the disease. This necessitates that the pathogen's mortality rate exceeds a figure that is dependent on the host's ability to survive, reproduce, recuperate, and lose immunity.

Numerous research systems, ranging from conjunctivitis in house finches to parasitic nematodes in red grouse and feral Soya sheep, provide increasing empirical evidence for parasite-mediated host regulation. Chytridiomycosis in amphibians, chestnut blight in American chestnuts, avian malaria in Hawaiian birds, devil facial tumor disease in Tasmanian devils, and sudden oak death in Californian trees are just a few examples of diseases brought on by novel pathogens that have resulted in dramatic host population declines. According to theory, specialized diseases that are dependent on density those that infect a single host will rarely be able to eradicate their hosts on their own, however host populations might be so low as to trigger stochastic extinctions. However, diseases that demonstrate frequency-dependent transmission, have long-lived infectious phases, or are multi-host pathogens that can spread between common reservoir hosts and more vulnerable target species are more likely to result in host extinctions. When a host is scarce, these three features enable prolonged transmission and reduced host fitness.

Host species that can escape infection by a pathogen by moving to a new place may have much better population growth rates and invasion potential, which is one effect of the reduced fitness brought on by infections. For instance, European green crabs are larger and more fertile in North America than they are in Europe because they only had a small subset of the diseases that infect them there when they arrived there, probably hitchhiking on ships. Introduced or exotic species are ones that humans have introduced to new areas, such as green crabs. As demonstrated by the par poxvirus of grey squirrels and its detrimental effects on native red squirrels throughout the United Kingdom, introduced host species can also bring and introduce viruses to new places with them. These pathogens can have sometimes disastrous effects on native host species. In addition to preventing competitive exclusion and modifying predation pressure, pathogens can also have an effect on host species interactions in other ways that improve host community diversity.

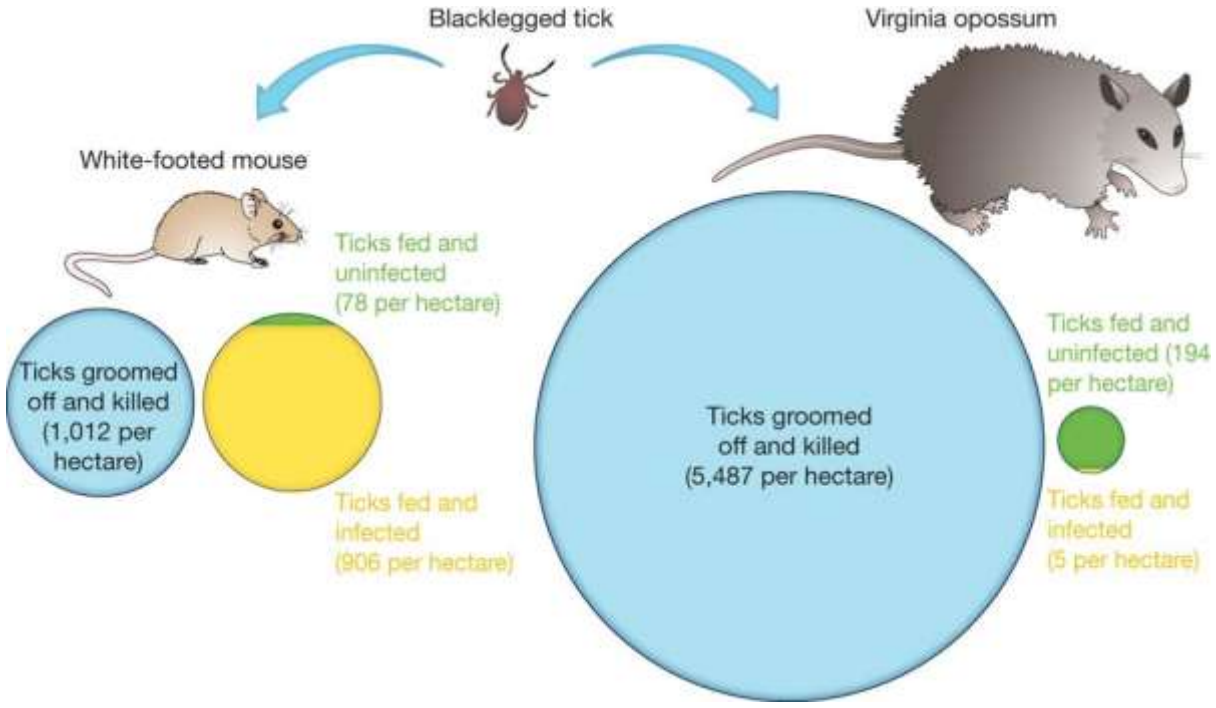

Figure 1: Represting the impacts of Pathogens on Communities [Nature].

Information for the Prevention and Control of Disease

Significant attempts have been made to stop the spread of infections or completely eradicate them from populations in humans, domestic plants, and animals. The most frequently used tactics are vaccination, behavioral changes including confinement and social seclusion, and culling for animals, plants, and disease vectors. When transmission is thought to depend on density and efforts are made to get densities below the threshold density, NT, culling is performed. For human infections like SARS and HIV, quarantines and social segregation have been used successfully to reduce the contact rates between infectious and susceptible people. Vaccination, the third key technique, aims to raise herd immunity, or the proportion of the population that is immune to infection due to either past exposure or vaccination. A common aim of vaccination is to inoculate a sizable enough portion of the host population to stop the pathogen's spread or locally eradicate it. Because it lowers Re (= R0S/N) below 1, the crucial vaccination threshold in a homogenous population is 1-1/R0. Unvaccinated vulnerable hosts are less likely to contract an infection since they are surrounded by immune people, which is one of the main advantages of vaccination. Additionally, new research on rabies virus protection for Ethiopian wolves has demonstrated that even relatively low vaccination coverage can inhibit disease transmission and possibly stop the extinction of local hosts.

A Heterogeneous Host

Despite the fact that the majority of basic models assume that each person is the same, there is variation in host traits that are connected to pathogen transmission. These include spatial and temporal variability in host features or the environment, as well as variations in susceptibility, contact rates, and infectiousness between hosts. This heterogeneity has significant effects on the spread of pathogens and, consequently, on efforts to control disease. Studies on a variety of parasites have revealed that at least 80% of subsequent transmission is caused by 20% of the host population. Supers' readers are people who, in the extreme, spread more secondary infections than would be predicted e.g., people who fall in the 95th or 99th percentile of a Poisson distribution. SARS, HIV/AIDS, and measles are just a few of the human diseases for which supers' readers have been discovered. As demonstrated in recent research on yellow-necked mice, the effectiveness of control methods can be significantly increased if individuals that play dominating roles in transmission can be identified and targeted.

West Nile virus, a vector-borne disease, exhibits host heterogeneity. Pathogens that can spread amongst a variety of different host species are a significant subcategory of heterogeneity. These include zoonotic pathogens, which are spread from non-human animals to humans. Numerous of the most significant infectious diseases that affect people, such as HIV, influenza, and SARS, were transmitted from one or more animal species to humans and continue to do so. Some host species for these multi-host infections act as amplifiers, increasing the pathogen's transmission, whilst others may dampen transmission, decreasing it. It has been demonstrated that the identity and abundance of different host species play a significant role in the transmission of both animal and plant

illnesses, such as brucellosis, rinderpest, and chytridiomycosis. Examples of plant diseases include sudden oak mortality, Jar rah dieback, and root rot in Australian trees. The dilution effect, which postulates that illness risk would decrease as the species diversity of hosts grows, is one concept that is actively being explored. It may operate through a variety of different pathways. Since vectors frequently only consume a small number of blood meals, the dampening effect of host variety can be particularly significant for pathogens that are transmitted through vectors (Figure 1).

The West Nile virus and the Lyme disease bacterium Borelli burgdorferi, two infections that are spread by ticks and mosquitoes, respectively, between dozens of animal species, are the most researched examples. In ecosystems with fewer host species, the dilution effect may be present if vectors feed more heavily on the most capable hosts those with a high ci. For instance, it is hypothesized that in cases of Lyme disease, a diversified mammal and avian community lowers the proportion of ticks that feed on highly skilled and obliging white-footed mice. Disease ecologists have shown how spatial structure, dispersal patterns, and landscape-level variation can affect the spatial transmission of infections over the past 20 years. When it comes to pathogens like rabies in raccoons, measles and influenza in humans, and baculoviruses in insects, researchers have looked at how spatially localized motions might result in moving waves of infection an infection peak followed by a trough. In these circumstances, the dispersal kernel's distribution that is, the range and distribution of distances over which infected people or pathogen particles spread strongly influences the pace of spatial spread. In the context of met populations, where hosts and diseases can travel among interconnected patches, other studies have looked at host-parasite dynamics. Numerous studies have demonstrated how even a small amount of host movement within patches can boost the long-term persistence of both hosts and parasites, in part by enabling animals to flee from areas that are highly infested with parasites.

Virulence, Resistance, and Coevolution in Host-Pathogen Evolution

Both hosts and parasites frequently change in reaction to their environments and one another. Why do parasites injure their hosts (parameters and in models in Figure 1) when they depend on their hosts for their own transmission is a crucial question in disease ecology. According to conventional opinion, parasites should develop into benign species and extend the lifespan of the animals they infect. However, a lot of parasites seriously injure their hosts, in part because reproduction invariably wears down host tissues and uses up host resources. One theory for the preservation of virulence is that while parasites that replicate too quickly will kill their host before they can spread, parasites that replicate too slowly will not create enough transmission stages which would increase. As a result, selection may favor parasites with intermediate degrees of within-host replication and thus, virulence that balance the advantages of increased replication for transmission with the drawbacks of quicker host mortality. In other words, it is assumed that there is a tradeoff between raising, which would increase transmission, and raising, which would decrease transmission [9], [10].

Even though this trade-off theory is well-liked, there are other explanations for pathogen virulence, and only a few studies such as experimental research on rodent malaria, bacterial pathogens of Daphnia, and a protozoan parasite of monarch butterflies have so far offered conclusive evidence in favor of the trade-off theory. In response to infection, hosts can also change in ways that affect virulence. The studies of Australian rabbits and the maxima virus, crickets and parasitic flies, and bacteria and phages are some of the best instances of host adaptation in response to disease that have been documented. According to Boots et al. host techniques for warding off infection can be divided into two categories host tolerance and host resistance. The ability of a host to tolerate infection with a disease by minimizing the harm caused while preventing the pathogen's multiplication or transmission is known as host tolerance. In contrast, host resistance tactics lessen the likelihood that a host will contract an infection, slow down pathogen multiplication inside the host, and/or hasten pathogen clearance.

A compelling question is, why aren't hosts more resistant to pathogens? Given that hosts would benefit from thwarting infection, possible answers include: a trade-off between resistance traits and other fitness-related traits, pathogen evolution to thwart or counter host resistance traits, and a trade-off among defenses targeted at various parasite types or strains. Contrarily, it is generally predicted that features that promote tolerance will eventually lead to fixation if their advantages outweigh their drawbacks. Thus, host-parasite interactions can result in co-evolutionary processes that, through co-speciation events and genetic arms races, can increase the genetic diversity of both hosts and diseases. Negative frequency-dependent selection, where numerous host and parasite genotypes exist and only some host-parasite combinations result in infection, is one approach that might result in co-evolutionary change. Local parasite genotype dynamics have the potential to influence and feedback on the frequency of resistance genotypes in a population over time. Long-term investigations of treaded parasites infecting freshwater snails in New Zealand have demonstrated this. This research also demonstrated how unique

host genotypes may be produced that are resistant to infection by widespread parasite clones by promoting host sexual reproduction in the long run.

Upcoming Directions

At least four crucial areas make up the expanding field of disease ecology interactions between pathogen species and strains including the dynamics of infection within the host and interactions with the immune system, explicit consideration of the spatial context of transmission, dynamics and drivers of multi-host pathogen transmission, and host and parasite evolution in the context of environmental change. The previous section dealt on the final three of these topics. Functionally diverse parasites can interact with each other through both immune-mediated and resource-competition-based mechanisms, according to research on interactions between various pathogen species at both the population level and inside individual hosts. The results of infection by microbial pathogens for hosts already infected by intestinal helminths have been demonstrated to be influenced by immunological tradeoffs, for instance, when energy spent to one branch of the immune system that is utilised to fight off one sort of parasite cannot be used for another. The trade-off between tuberculosis and HIV infections in humans and bovine tuberculosis in African buffalo has been researched. Other times, even in the absence of co-infection or cross-immunity, diseases can dynamically interfere with each other's predominance at the population-level, as was shown for measles and whooping cough in various European cities.

III. CONCLUSION

For diseases with many hosts, disease transmission can take both within and between species. It is necessary to assess the relative importance of each type of transmission if these diseases are unwanted for either health or economic reasons. One such dreadful illness is Mycobacterium bovid-caused bovine TB (Tb). Feral ferret Mustafa furor populations in some areas of New Zealand have high rates of M. bovid infection, raising concerns that they might serve as a reservoir for the infection of domestic livestock, much like brush tail possums *Trichosurus vulpecula* do. To test for and quantify the transmission of M. bovid from brush tail possums to feral ferrets and intraspecific transmission of M. bovid within feral ferret populations, we conducted a manipulative large-scale field experiment.

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