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Cover art by Kalyani Ganapathy

For a lot of us, disease conjures up images of human suffering. But what about animals and plants? Those of us lucky enough to own pets and unlucky enough to lose them to illness have a certain dread for pet disease. Most of us have also heard about historical plant infections that have wiped out food crops and plunged entire countries and civilisations into famine; however, botanical outbreaks on that scale are seemingly on the decline. By virtue of being living organisms, animals and plants get diseases and infections caused by the same agents that tend to infect humans: bacteria, viruses, fungi, and protozoa. This issue is an attempt to highlight the ways in which these organisms impact wildlife, and the implications of the resulting infections.

When we address the pressing issues of wildlife and biodiversity conservation today, disease is something that needs to factor into the equation. Thierry Work elaborates on this issue, giving examples from intricately connected ecological systems. Many times, animal disease can jump to humans in a process known as "spillover"– the topic of David Quammen's bestselling book, reviewed here by Caitlin Kight. As discussed by Aswathy Vijayakumar and Andrea Phillot, spillovers are particularly common in tropical areas and other habitats where pathogens and the agents that spread them have been expanding their ranges as a result of climate change.

Another critical factor in wildlife disease is the dynamics between hosts and reservoirs. This is exemplified by the case of India's stray dogs and Hawaii's mosquitoes, as described by Aniruddha Belsare and Farah Ishtiaq, respectively. However, Caitlin Kight's primer on plant disease serves as a reminder that animals are not the only organisms afflicted by infection.

By definition, wildlife diseases impact free-living species—but this doesn't mean that these illnesses don't also affect humans in some way. This is the message from Pramod Patil and Milind Watve, who write about how research into wildlife disease can actually improve our ability to study and understand human health.

Although most people would probably deem it preferable that there were no such thing as disease at all, the examples highlighted in this issue indicate that susceptibility to sickness is yet another thing that unites living organisms.

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Diseases of free-ranging dogs: Implications for wildlife conservation in India

Dogs are almost everywhere on this planet. With the exception of Antarctica, this domesticated subspecies of the gray wolf occurs on every continent (Though dogs played an important role in the exploration of Antarctica prior to 1991, they were subsequently banned from the continent due to the potential threat of canine distemper, a dog disease, spreading to Antarctica's seals). Widely distributed across the rest of the continents, dogs have a staggeringly large global population—estimated at one billion individuals—and have therefore achieved the distinction of being the world's most abundant carnivore.

Some 58 million of these dogs (~6% of the global dog population) roam the Indian landscape. Dogs are very much a part of the backdrop everywhere in India, so commonplace that they barely warrant a second glance. Dogs occur in cities, towns and villages; around markets, garbage dumps, slaughterhouses and meat shops; outside restaurants and dhabas; on highways and farmlands; and even within protected areas. These dogs are mostly unowned or community owned ('loosely owned'), and therefore unsupervised, unvaccinated, and free-ranging. Such dog populations typically have a high population turnover, mainly due to high rates of both birth and mortality. Further, dog populations in any given landscape form a metapopulation, or a series of interconnected populations between which individuals move freely. These two factors—interconnected populations and high population turnover—allow the persistence and transmission of disease-causing pathogens. Most dog pathogens have a wide host range, meaning they can infect other species as well. For instance, dogs are the known reservoirs of rabies virus which causes fatal disease in all mammals, including humans. More than 50,000 humans die every year due to dog-transmitted rabies.

THREATS TO WILDLIFE CONSERVATION

Dog-transmitted rabies also poses a conservation threat. Introduction of canine rabies resulted in the local extinction of African wild dog populations in the Serengeti-Mara system (Tanzania/Kenya) in 1989; similar spillover events have resulted in dramatic population declines of the Ethiopian wolf population in the 1990s. Several other multi-host pathogens can also persist in large dog populations. Dogs have been implicated as a source of canine parvovirus (CPV), contributing to gray wolf mortality on Isle Royale, and as a potential source of canine adenovirus (CAV) transmitted to maned wolves in Bolivia.

Canine distemper virus (CDV) has also caused several well-documented epidemics in wild carnivores. The most infamous CDV epidemic occurred in the Serengeti in 1994, wiping out a third of all lions (>1,000 individuals) and many hyenas, leopards and bat-eared foxes. Several other species, including African wild dogs, Caspian sea seals and Lake Baikal seals have also experienced high mortality rates as a result of CDV introduced from dogs. Domestic dogs may be the source of CDV infections that have recently been reported to impact endangered Amur tigers living in the Russian far east. These examples indicate that multi-host pathogens can pose a serious conservation threat when reservoir dog populations occur alongside susceptible populations of wild carnivores.

MUCH ADO ABOUT NOTHING?

In India, several wild carnivore species, including leopards, snow leopards, lions, tigers, wolves, hyenas, jackals and foxes, occur in human-dominated landscapes where dogs are omnipresent. Further, dogs are known to interact with wildlife in myriad ways; for instance, several studies in India have found that dogs are an important component of the diets of leopards. Dogs are also known to attack wild carnivores. Interactions such as chasing, fighting, or feeding events at carcasses are all potential opportunities for pathogen transmission between species. But so far, there has been no dramatic disease outbreak or disease-related population declines in wild carnivores in India—or, at least, none has been documented.

The lack of evidence for disease-related population decline in wild carnivores may simply result from the fact that we have not yet looked for it. Unless there are extreme circumstances such as mass die-offs, diseases in free-ranging populations can go completely undetected because of the inherent difficulties of monitoring wild populations. Also, wild animals instinctively mask the symptoms of illness or disease; so a diseased wild animal will appear 'normal' to an observer. Further, it is difficult to find carcasses of wild animals quickly enough to assess the cause of their death. In India, there has not been much research on infectious diseases of wildlife, nor do we have a system of obtaining and analysing surveillance data. This is also true for diseases of dogs: measures of the prevalence of important pathogens in dog populations are virtually lacking. Despite the serious public health threat of rabies in India (one person dies every 30 minutes due to dog-transmitted rabies), the epidemiology of rabies in dog populations has not been properly investigated. Neither do we have any reliable demographic data for dog populations in India.

DISEASE ECOLOGY STUDY IN INDIA

The lack of epidemiological and demographic data on the free-ranging dog populations in India is a major impediment to understanding the real public health and conservation threats posed by dog pathogens, in achieving effective disease control, and even in managing dog populations. To address this data void, a pilot study was undertaken around the Great Indian Bustard Wildlife Sanctuary (GIB WLS) at Nannaj (near Solapur) in 2005. The protected grassland patches at Nannaj are surrounded by villages, agricultural fields, communal grazing lands, and forestry plantations. Dogs are ubiquitous in this region, and are free-ranging irrespective of ownership status. Abi Vanak, as a part of his dissertation research, was studying the ecology of Indian foxes in and around GIB WLS. Using radiotelemetry, he also studied the activity, movements and interactions of dogs and foxes in this region. Abi and I decided to use this opportunity to obtain baseline disease prevalence data from blood samples (called serologic data) for Indian foxes and free-ranging dogs in the region.

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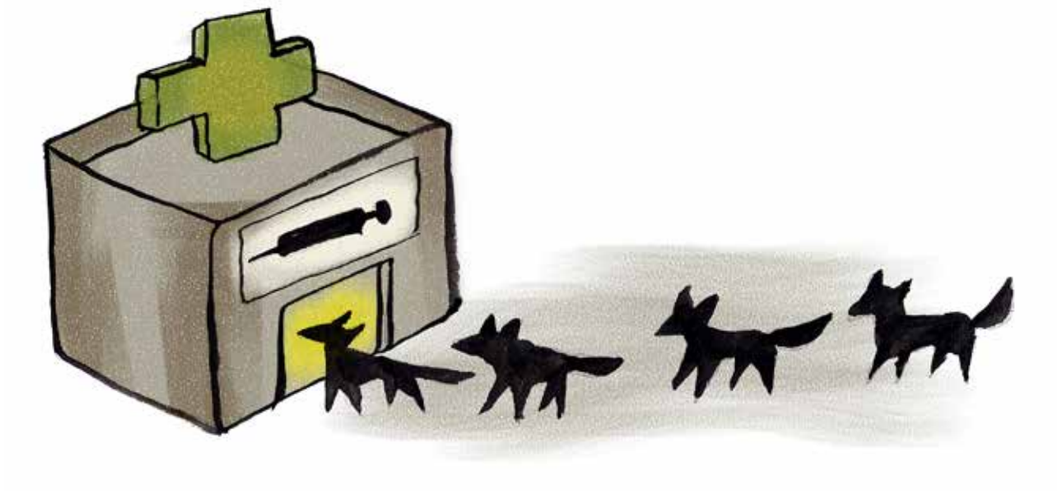
Serologic surveys indicated exposure to viral pathogens in both dog and fox populations. Most of the dogs we tested were exposed to CPV, CDV and CAV, indicating that the pathogens were enzootic (meaning 'constantly present in animals of a specific region'—the animal version of 'endemic') in the dog populations around GIB WLS. The exposure rates in foxes were low compared to those in dogs, indicating the susceptibility of foxes to dog pathogens. The fox serological data also revealed an ongoing CDV epizootic in the fox popu-



lation (meaning that new cases of CDV in foxes were being recorded). Further, foxes infected with CDV had a high rate of mortality. We tentatively hypothesised that high dog-fox contact rates facilitated the transmission of CDV from dogs to foxes, as the latter species would not have maintained CDV in isolation given its relatively low population density and the apparently high pathogenicity of CDV in foxes. These observations prompted the Maharashtra Forest Department to undertake mass vaccination of dogs in the villages surrounding the GIB WLS as an approach to protecting wild carnivores inhabiting the protected area.

The mass vaccination programs provided an excellent opportunity to further study the disease ecology of free-ranging dogs. I planned my dissertation research around the vaccination campaigns in six villages near the GIB WLS. Long-term serologic data was obtained for the dog populations, along with baseline demographic data. As virtually all the dogs in this region are free-ranging and not habituated to restraint of any sort, a non-invasive method using photographic mark-recapture approach was optimised for estimating dog abundance. Dogs occurred at high densities in the villages around GIB WLS (> 526 dogs per square kilometre); the dog populations were male-biased and comprised mostly adult dogs. The serology data confirmed the previous findings of high exposure rates to the three viral pathogens of interest. An in-depth analysis of the epidemiological data indicated that adult dogs had consistently high exposure rates to these pathogens, indicating that the viruses actively circulate in the dog populations. The high exposure rates of adult dogs also indicated survival following early natural exposure to these pathogens; lifelong immunity results from natural infection with CPV, CDV or CAV. Collectively, the findings suggest that most adult dogs in the study populations are immune to pathogens such as CPV, CDV and CAV, and play no current or future role in the maintenance or transmission of these pathogens.

THE VACCINATION EXPERIMENT

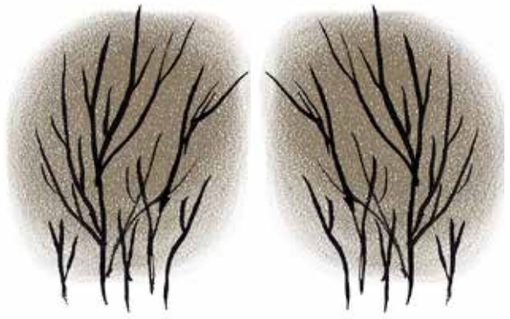
Vaccination of dog reservoir populations has been recommended as a potential measure that can be used to protect wild carnivores from dog diseases. By vaccinating dogs against pathogens of conservation concern, it is expected that the number of susceptible dogs in the population will be reduced, thereby reducing the occurrence of clinical cases and the likelihood of transmission events between infected dogs and wild carnivores. To determine the extent to which such mass vaccination programs are applicable and practicable for large, free-ranging dog populations, I undertook a village-level vaccination experiment. Dogs from three villages were vaccinated against rabies virus, CAV, CPV and CDV (treatment dogs), while those from three other villages were only vaccinated against rabies virus (control dogs). All dogs in the control and treatment villages were vaccinated against rabies because of the public health risks posed by dog-transmitted rabies in the region. For both the groups, we determined the proportion of dogs with protective antibodies on four occasions over a period of one year.

Young dogs (pups and juveniles) appear to play a critical role in the transmission of these pathogens, and targeted vaccination of this age class could be considered as a disease management intervention.

Vaccination failed to increase the proportion of dogs with protective antibodies against CAV, CPV or CDV in the treatment group compared to the control group, as much of the effort was put into vaccinating dogs that were already antibody-positive by virtue of prior natural exposure to these pathogens. Furthermore, several unvaccinated adult dogs acquired protection against these pathogens during the study. In such situations, vaccination of adult dogs against enzootic viral pathogens seems unnecessary, and would escalate the cost-benefit ratio of dog disease control programs. Young dogs (pups and juveniles) appear to play a critical role in the transmission of these pathogens, and targeted vaccination of this age class could be considered as a disease management intervention. However, further research on such approaches is necessary. It should be noted, though, that mass vaccination programs will readily work for a pathogen like rabies, as exposure to rabies virus is always fatal and therefore an unvaccinated population consists entirely of susceptible dogs that will benefit from vaccination.

MODEL EXPLORATIONS

Another important outcome of this work was the realisation that effective management of diseases in free-ranging populations requires a better understanding of pathogen dynamics—specifically, the conditions favouring the persistence and transmission of the pathogens in the system under study. Experiments can be designed to study and unravel these mechanisms, but such an approach would necessitate repeated interventions requiring the capture and handling of a large number of animals. Unfortunately, legal, ethical, and logistic constraints make such interventions impossible when free-living or wildlife species are involved. In such situations, computational models can be formulated to simulate disease transmission based on the current best understanding of the system. The model can then be used to play out various scenarios under different assumptions, and thereby explore potential disease control strategies. Using data from the ecological and epidemiological studies, I formulated a model of CDV transmission between dogs and foxes in and around the GIB WLS. The model simulated movements of dogs and foxes in the landscape, and the transmission of CDV between infected and susceptible individuals. There were two model outputs: the average number of new CDV cases in the dog population every year, and the number of times CDV was transmitted between dogs and foxes (number of 'spillover' events). I then investigated which potential disease control interventions would best mitigate the disease spillover threat in the model fox population.



My results indicated that spillover could be significantly reduced by a reduction in the size of dog populations, as well as by a limitation on the free-roaming tendencies of dogs. Vaccination of dogs against CDV in such settings, however, was ineffective.

IMPLICATIONS FOR MANAGEMENT

Collectively, these findings have important implications for dog disease control programs, especially in settings where dog populations are large and free-ranging, and where pathogens like CPV, CDV and CAV are enzootic. This work underscores the importance of investigating the population pattern of pathogen exposure before considering mass vaccination programs for free-ranging dog populations. Vaccination of local dog populations will be an ineffective disease control strategy in settings where viruses are enzootic in large, free-ranging dog populations. This is an important point to emphasise, given that the National Tiger Conservation Authority has recently recommended vaccination of dogs around protected areas to prevent CDV transmission in tigers (http://projecttiger.nic.in/whtsnew/CVD.pdf). Based on the predictions of my model, disease control programs should also have a strong component of public outreach, emphasizing responsible dog ownership. For example, dog birth control programs implemented in and around areas of conservation concern, in combination with restrictions to the movements of dogs in habitats occupied by species of conservation concern, would reduce the disease threat to susceptible wildlife. It is likely that similar efforts in other domestic animal taxa would also be beneficial.

Overall, management of diseases in free-ranging populations is challenging, given the complex ecological and epidemiological interactions of multiple hosts and pathogens. Strategies for investigating and mitigating disease risks to wildlife should be based on scientific evidence obtained using a combination of ecological, epidemiological, and computational studies.

References:

Belsare AV & ME Gompper. 2013. Assessing Demographic and Epidemiologic Parameters of Rural Dog Populations in India During Mass Vaccination Campaigns. *Preventive Veterinary Medicine* 111, no. 1-2: 139-46.

Gompper ME (Ed.). 2014. *Free-Ranging Dogs and Wildlife Conservation*. Oxford University Press. New York, USA.

Vanak AT, AV Belsare & ME Gompper. 2007. Survey of disease prevalence in free-ranging domestic dogs and possible spill-over risk for wildlife: A case study from the Great Indian Bustard Sanctuary, Maharashtra,India. The Rufford Small Grants Foundation, UK, 1-13.

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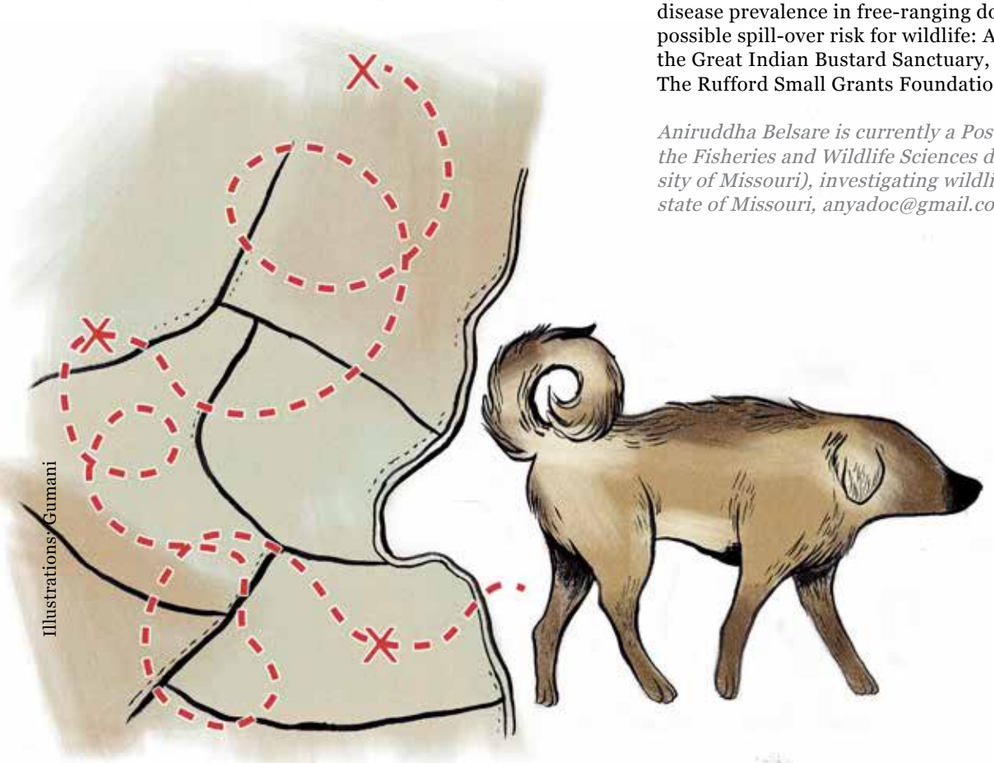


Illustrations: Gumani

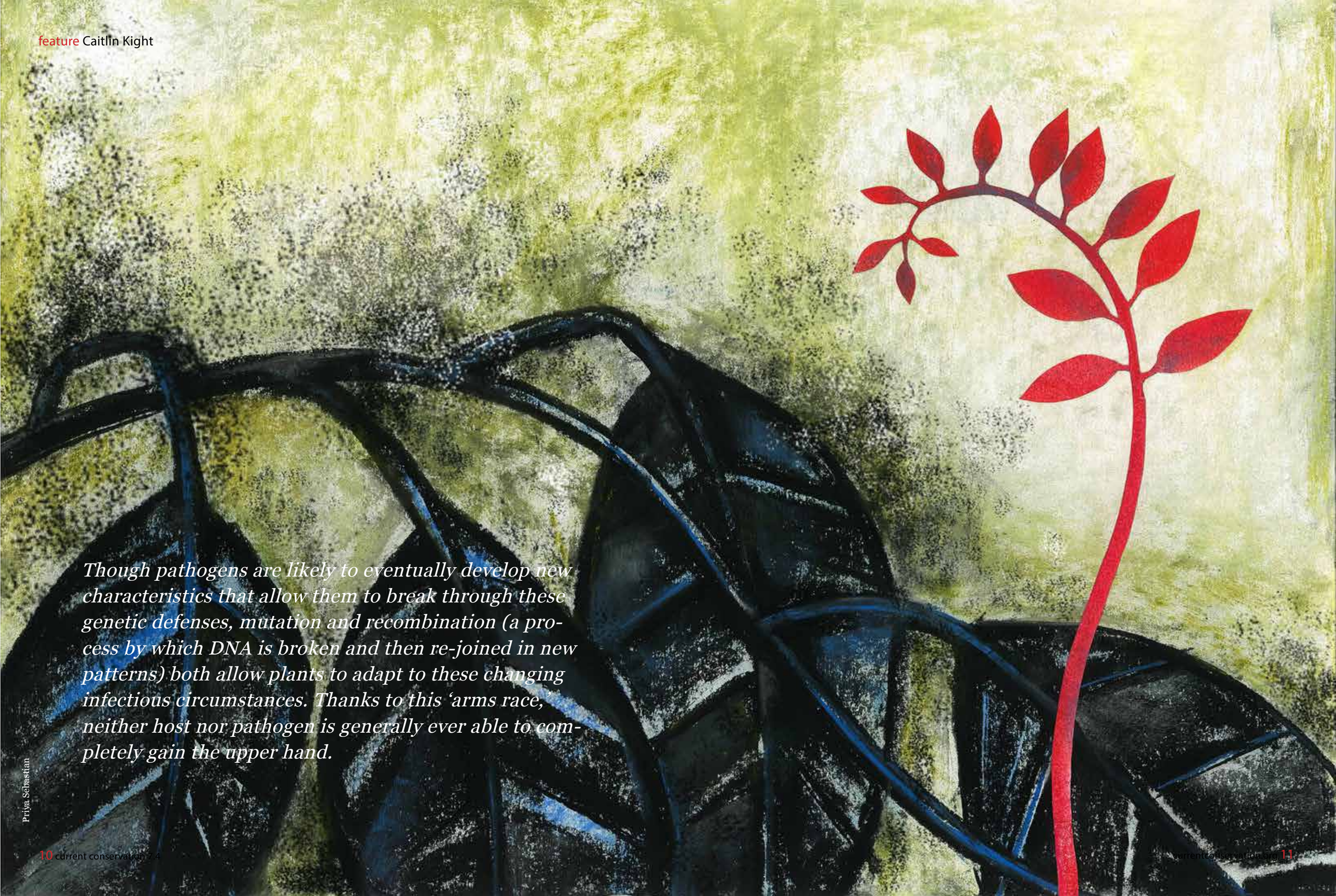
Plant Pathogens: A Primer

If you were asked to tally up all the diseases you've ever heard of, chances are that you would think of animal rather than plant infections. Except when they have created widespread problems—think of the potato blight that affected British and Irish farmers in the mid-19th century—plant pathogens have typically been interesting only to the farmers whose livelihoods have depended on quickly recognising, reacting to, and preventing the spread of these diseases. More recently, agriculturalists have been assisted in their efforts by plant geneticists and phytopathologists (researchers who specialise in the field of plant disease) tasked with developing disease-resistant crops and reducing the spread of infection between various plants and fields. However, despite the efforts of these individuals—not to mention the millions of dollars that the agriculture and horticulture industries have poured into phytopathology research—we still lack a fundamental understanding of plant disease. In some cases we haven't even identified the agents responsible for certain sicknesses; where these have been found, we don't always know why they have suddenly become problematic, or how widespread the infection might ultimately become.

One way of predicting where, when and under what conditions plant infections will appear and spread is to examine the characteristics of emerging infectious diseases (EIDs)—those that have recently become more visible to us, either be-

International Maize and Wheat Improvement Center (CIMMYT)

Corn kernels impacted by partial bunt, caused by smut fungus



Though pathogens are likely to eventually develop new characteristics that allow them to break through these genetic defenses, mutation and recombination (a process by which DNA is broken and then re-joined in new patterns) both allow plants to adapt to these changing infectious circumstances. Thanks to this 'arms race,' neither host nor pathogen is generally ever able to completely gain the upper hand.

Priya Sebastian

cause they are newly evolved or discovered, have changed their method of infection, or are found in higher numbers or over a broader geographical area. An improved understanding of the mechanisms that allowed these pathogens to become so successful could not only have huge agricultural (and therefore economic) implications, but also inform conservation and management decisions relevant to sensitive or endangered wild species.

EMERGING INFECTIOUS DISEASES IN PLANTS

Plants, like animals, can be infected by a variety of microorganisms, such as viruses (the most common type of EID, causing nearly half of infections), fungi, bacteria and nematodes (the least common form of EID, responsible for ~1% of infections). Perhaps counterintuitively, a discussion of the factors responsible for the emergence of phytopathogens is best initiated with a summary of the mechanisms that prevent pathogens from running rampant in the first place. First and foremost among these is the evolution of tolerance or even complete resistance in potential host species. Though pathogens are likely to eventually develop new characteristics that allow them to break through these genetic defenses, mutation and recombination (a process by which DNA is broken and then re-joined in new patterns) both allow plants to adapt to these changing infectious circumstances. Thanks to this 'arms race,' neither host nor pathogen is generally ever able to completely gain the upper hand.

Another important characteristic of natural ecosystems is their diversity. Even though a habitat may offer an abundance of a particularly good host, it may also be home to plenty of terrible hosts that represent dead-ends for even the most infectious (transmissible) and virulent (harmful) of pathogens. Likewise, high biodiversity can negatively impact pathogens that rely on 'vectors', or organisms that shuttle infectious agents from a reservoir (an organism in which the pathogen is 'stored') to a host; where there are many alternative vectors, infectious agents may not end up at their intended destination. An abundance of natural predators and parasites may also waylay pathogens before they are able to infect any hosts.

A number of abiotic factors can also keep pathogens in check. Physical and geographical barriers—even something as simple as a few feet between an infectious host and its uninfected neighbour—can prevent or reduce the spread of disease. Further, changing weather conditions can kill off pathogens that are highly sensitive to temperature and moisture, while a lack of wind or hard rain can prove problematic for microorganisms that rely on these events to create injuries through which they can invade their hosts.

Given these natural checks on pathogen spread, it is not surprising that both habitat disturbances (including those caused by humans) and changes in vector populations have been identified as major players in the emergence of phytopathogens. Recombination can also play a role, since it can—temporarily, at least—allow either plants or pathogens to pull ahead in their evolutionary arms race. Fluctuating weather patterns are thought to be responsible for approximately one-quarter of emergences; many of these variations are likely tied to climate change facilitated by anthropogenic activities.

By far the most important factor, however, is the introduction of non-native species—of both hosts and pathogens. On average, introductions are responsible for just over one-half of all emerging diseases. However, the exact figure varies from one type of pathogen to the next; viruses seem particularly skilled at capitalising on interactions with organisms they have not previously encountered. Whether introductions are accidental or deliberate, things tend to go very badly for plants exposed to pathogens to which they have no innate defense.

The emergence of novel pathogens may also be facilitated by farming methodologies. Particularly problematic is the fact that we tend to grow monocultures comprising plants with a similar genetic makeup. This technique creates huge tracts of land covered by crops frequently lacking the resistance genes needed to combat that next big infection—whatever it might be. Modern farmers also tend to sow seeds fairly close together, thereby increasing the likelihood that infections will spread between grown plants. Recently, there has also been a move towards leaving plant remnants in the field once the harvest has been completed. While this helps enrich the soil, it also facilitates the spread of last year's diseases to next year's crops.

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Compounding all of these problems is the fact that agricultural efforts have been intensifying in order to satisfy the demands of a growing human population. Given the flexibility of gene transfer amongst many pathogens, there is an almost endless supply of infectious agents available in the environment. Sooner or later, chances are good that we will experience the emergence of an infection that will be the next potato blight or cassava mosaic disease—two epidemics with extreme financial, social, and health repercussions.

EMERGING INFECTIOUS DISEASES IN WILD PLANTS

Although the bulk of phytopathogen research to date has focused mainly on domestic species, epidemics can, of course, also occur in wild plants. Detailed studies of native organisms have generally been pursued only in species acting as a reservoir for infections impacting crops or ornamentals. There are, however, two notable exceptions: the American chestnut blight (caused by the fungus *Cryphonectria parasitica*) and the spread of Dutch elm disease (caused by fungi in the genus *Ophiostoma*), each of which has been fairly well documented. In both cases, the infectious agents appear to have been introduced into new habitats after hitchhiking on exotic products; *C parasitica* was hiding in Japanese chestnut trees bound for an American nursery, while the original *Ophiostoma* species (from which two others eventually evolved) was stowed away in a shipment of lumber.

Anthropogenic activity also appears to be responsible for infections threatening eucalyptus species in Australia and dogwood trees in the United States. In both cases, the pathogens (both fungi) were introduced and spread by humans. Origins of other known plant diseases are more mysterious. The *Florida torreya*, a conifer native to the northern portion of the US state of Florida, has suffered an extreme population decline likely resulting from one or more fungal infections; it is now considered critically endangered. Researchers are still searching for the pathogen responsible for the severe decline of pondberry, a rare and endangered North American species that had already been hit hard by habitat loss when it also began to succumb to an unknown—probably fungal—infection.

Other recent notable EIDs include ash dieback (caused by the fungus *Chalara fraxinea*) and sudden oak death (caused by *Phytophthora ramorum*). The latter of these is only the most recent disease resulting from the activity of a species in the *Phytophthora* genus; previous victims have been the New Zealand kauri (collar-rot), a variety of ornamental rhododendrons (root rot), beeches (stem and leave rot), and several domesticated species, including strawberries, soybeans, coconuts, and cocoa.

EFFECTS OF CLIMATE CHANGE ON PHYTOPATHOLOGY

One of the biggest questions in contemporary plant phytology is how the spread and emergence of disease are impacted by climate change. Analyses of ancient sediment and ice samples have revealed evidence that both the distribution and prevalence of pathogens have been affected by historical fluctuations in weather and climate patterns. Thus, there is every reason to believe that the high rates at which infectious plant diseases have been emerging over the past several decades may be related, both directly and indirectly, to climate change.

One factor that affects emergence is moisture: fungi and bacteria tend to benefit from increases in humidity and precipitation, while viruses and insect-borne diseases thrive under drier conditions. As certain habitats have become wetter or drier (patterns affected by numerous factors, including topography and proximity to the Equator), local conditions have likely shifted to favor new pathogens. Such shifts are expected to continue—and perhaps become even more common—over the next several decades.

Thus, there is every reason to believe that the high rates at which infectious plant diseases have been emerging over the past several decades may be related, both directly and indirectly, to climate change.

Variations in temperatures and broader weather patterns may also benefit pathogens by altering the distributions and abundances of both vectors and hosts, allowing disease agents to move into new habitats. Some pathogens are free-living, capable of surviving in water or soil for long periods before encountering a suitable host. Even slight variations in environmental conditions can extend the length of time over which these organisms can lie in wait, thus increasing the likelihood that hosts will eventually become infected. Researchers have even suggested that thawing glaciers might release frozen pathogens that could either infect hosts directly or contribute pathogenic genes to other infectious agents in the environment.

Climate also influences host physiology, affecting fundamental processes such as respiration and metabolism. These are characteristics that can impact how easy it is for a pathogen to infect a

Ronnie Nijboer, Wikimedia

Beetle feeding galleries on an elm tree; beetle feeding can spread the fungi responsible for the disease

host, as well as the speed and strength with which an infected individual can mount an immune response to an infection. Animal pathologists have recently identified several systems in which physiological processes likely played an integral role in linking climate change with disease emergence; in all likelihood, an analysis of plant data would yield similar results.

WHAT WILL HAPPEN IN THE FUTURE?

Paolo Bacigalupi's 2009 award-winning novel *The Windup Girl* envisions a future where the world has been re-shaped by the activity of phytopathogens; agriculture companies race to develop disease-resistant genetically modified crops, while 'natural' plant resources are all but depleted. It is a chilling scene made all the more frightening by the fact that we have already experienced outbreaks and destruction similar to those the author describes. Luckily, there are many ways for us to escape such a future.

Given the influence of introductions on disease emergence, one of the most important tactics will be improving our monitoring systems—especially those associated with the import and export of plants and plant-based products. Many regions and countries have already initiated more stringent procedures in response to outbreaks. For example, flour export is often banned from regions affected by the smut fungus *Tilletia indica* (the cause of Karnal, or partial bunt), which is so destructive that it is considered a biological weapon.

There are also a number of ways in which farmers could adjust their agricultural practices to reduce the likelihood of disease spread. A greater emphasis might be placed on native, rather than exotic, crop species. Alternatively, crops could be rotated more frequently in order to reduce infection rates (though this tactic would not work as well against pathogens that can infect multiple domestic species). The development of new pesticides and genetically modified crops could also be helpful; however, these techniques must be used carefully, as they could have negative implications for both humans and wildlife, and are currently heavily regulated in many parts of the world.

Seedbanks—stores of seeds from both cultivated and wild species—may also prove useful in preserving biodiversity in the face of pathogenic activity. Seedbanks have two main benefits. First, they can be used to reintroduce plants that can no longer be found growing in the wild. Second, if we preserve a variety of seeds from each species, the bank stocks can be used to reintroduce specific genes—for example, those conferring resistance to a particular infection—that may have vanished from extant populations.

One of the most important weapons against plant epidemics is, of course, knowledge. Further scientific research will be critical in helping us identify, predict, and respond to the activity of phytopathogens. There is growing interest in improving our understanding of plant disease not just in agricultural crops, but also in wild plants. Because mild infections in native species may become widespread outbreaks in agricultural crops—and vice versa—it is vital that we study infections in a broad range of organisms and habitats. It will be equally important to consider what might happen as various disease strains hybridise and produce novel infectious agents.

Finally, we need to elucidate the ways in which specific human activities alter disease dynamics—and how these anthropogenic effects might act in conjunction with each other and other stressors to impact the spread and emergence of phytopathogens. It will be particularly important to model the possible effects of climate change in a variety of scenarios that differ according to level of mitigation; it is unclear how soon or how intensely we will adjust our behaviour to prevent further climatic variation, and so we need to be prepared for a range of potential pathogenic responses.

Unlike other agents of widespread ecological change, we are in the unique position of being cognizant of our own effects on the natural world. We can significantly reduce the likelihood of plant diseases emergence and spread by making informed decisions about farming and gardening practices, shipment of plant materials, travel practices, and land management (among other things). Perhaps the most important goal, however, is education. By raising awareness and fostering an improved understanding of disease dynamics, we can initiate a broader discussion about what we need to do to prevent both domestic and wild plant species from being eradicated by phytopathogens.

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Health, disease, and wildlife conservation in the Pacific Islands

Kalyani Ganapathy

Mention “disease” or “death” in a conversation, and the listener will typically cringe, wince, and turn away in disgust. Yet disease and death are part and parcel of life as we know it and play an important role in wildlife conservation, particularly for threatened and endangered species. Like humans, wildlife die from various causes, including infectious disease and non-communicable causes of death such as predation or poisonings.

Take the Laysan duck, for example. It is one of the most critically endangered water birds of the world, found only on the small island of Laysan in the northwestern corner of the Hawaiian archipelago. These ducks forage around a shallow hypersaline lake at the center of the island and raise their ducklings near freshwater seeps around the lake. In 1993-1994, massive duck mortality occurred in conjunction with a drought. The ducks had been killed by a parasitic worm, the quick and widespread transmission of which had been aggravated by the ducks’ dense congregations around scarce sources of fresh water.

Because Laysan was the only location where the ducks existed, this die-off introduced the possibility that a future disease outbreak, in conjunction with some other environmental fluctuation (e.g., storm surges) could wipe out the population completely. To prevent this, 42 ducks were translocated in 2004 from Laysan to Midway Island, a wildlife refuge managed mainly for seabirds, sea turtles and monk seals. In anticipation of the ducks’ arrival, numerous small wetlands were created on Midway. The translocation was a success, and the duck population increased six-fold over the next three years. Unfortunately, about half of the Midway ducks were lost over a two-month period in 2008 because of botulism, a natural toxin produced by bacteria in the newly created wetlands.

Botulism mortality in waterfowl can be effectively reduced with a combination of carcass removal and water management that alters conditions sufficiently to stop toxin production, and Midway has now implemented some of these practices to stem losses from botulism. As a result, in addition to monk seals, sea turtles and seabirds, Midway must now also be managed as a waterfowl refuge.

THE FORENSICS OF ANIMAL DEATHS

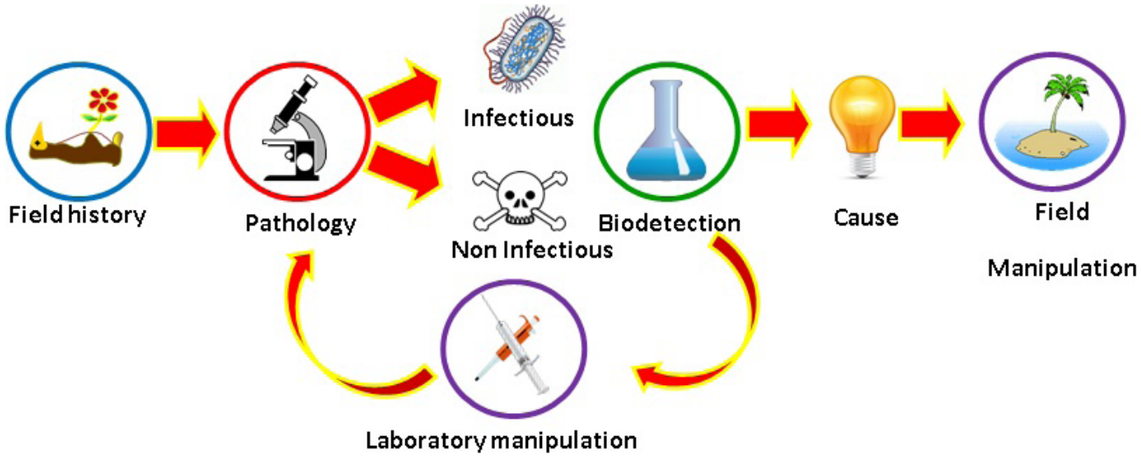
Investigating wildlife mortality is a deductive process. Wildlife health experts and biologists must consider a range of possible causes and, using data from environmental and laboratory observations, systematically and efficiently eliminate these possibilities to arrive at the most likely cause of death. Valuable clues include which species are affected, the extent and timing of the mortality, and whether there have been any changes in behaviour or land use patterns. For example, the deaths of multiple species over a short time period in a localised area might suggest a non-selective agent, such as a toxic spill, rather than an infectious disease that usually targets a given species or group of animals.

Wildlife mortality is driven by three factors: the agent or cause of mortality, the host (animal affected) and the environment in which the agent and host interact. Understanding the interactions between agent, host, and environment is what makes the study of wildlife health so fascinating. To understand how mortality impacts wildlife, you need to know about things like physiology, anatomy, molecular biology, pathology (the study of disease), veterinary medicine, animal behaviour, and animal ecology just to name a few. Clearly, no one person has a grasp of such varying disciplines, so to address such complex issues, wildlife mortality investigations are best done by an interdisciplinary team comprising animal health experts with an appreciation for ecology, and biologists with an appreciation for animal health.

Ask a biologist how his or her wildlife populations are doing, and most will answer in terms of population levels or recruitment rates: If these are stable or increasing, then things are going well. Mortality, a major driver of animal populations, is often explained away as a natural phenomenon that we can do nothing about. Furthermore, many biologists do not have the necessary expertise to investigate animal mortalities or understand why animals die. Cultural norms also play a role –disease and death are not exactly pleasant topics of conversation. As a result, many animal populations are managed in absence of knowledge of the major drivers of mortality. This is unfortunate, because if we know what kills wildlife, we can, in some instances, do things to alleviate or reduce those causes of mortality, thereby aiding recovery of animal populations. Furthermore, an understanding of wildlife health ultimately facilitates an understanding of ecosystem health. Hawaii, for example, has one of the highest numbers of threatened and endangered animals per unit land area in the world; in places like this, it is critical to understand why species are declining. Wildlife health expertise can sometimes aid biologists in identifying the cause of such declines and aid management to stem such declines.

Laboratory investigations typically begin with a necropsy of dead animals and examination of tissues under the microscope; this guides additional laboratory investigations. For instance, the presence of bacteria in tissues associated with cell death would prompt investigators to order laboratory cultures to identify the bacterium and potential cause of death. Once a cause of death is determined, then the really hard work begins: determining how to mitigate or eliminate this cause to aid recovery of the population. Achieving this requires one to know about how the agent, host, and environment interact and to identify appropriate critical points where an intervention can interrupt the cycle. For example, the discovery that mosquitoes transmitted malaria led to mosquito abatement and use of bed nets to reduce the contact between malaria-infected mosquitoes and humans. In rare cases, vaccines can be used to reduce wildlife diseases; the use of orally delivered rabies vaccines has successfully pushed the virus out of large geographic areas.

Islands are, of course, surrounded by water, and what happens on land will often impact the surrounding ocean. Ecosystem degradation often manifests as the presence of sick or dying wildlife. One example in Hawaii is the turtle tumour disease fibropapillomatosis that has been linked to polluted or degraded marine ecosystems. Researchers suspect that a virus is associated with the tumours, which grow on the eyes, mouth, and skin, and cause turtles to waste away and die. Unfortunately, this disease is difficult to manage at this time because researchers have not yet established an unequivocal link between the virus and



Investigating diseases of wildlife is a deductive process that follows a series of logical steps to systematically eliminate possible cause of death, and these are summarised as follows: (1) Field history– Where and when dead animals are found, location, time of year, species, age or sexes affected can all provide preliminary clues as to potential causes of death. (2) Pathology– Dissecting dead animals and describing morphologic changes of disease at the gross and cellular is critical because it provides both clues as to whether mortality is associated with infectious causes like bacteria or parasites or non-infectious causes like poisons. (3) Biodetection– Laboratory assays are used to identify and characterise suspect causative agents. (4) Laboratory trials– In case a new pathogen is found, laboratory experiments are done on animals to confirm the cause of disease; both biodetection and laboratory trials are guided by pathology. Eventually, a cause of death is confirmed, and field studies are done to understand the ecology of the disease in animal populations with a view to developing ways to manage or reduce impacts of diseases in wild animals.

tumour development. For this to happen, it will be necessary to culture the virus in a laboratory and then experimentally reproduce the disease in turtles. Unfortunately, growing the virus in the lab has proved elusive. This is one of many examples of the challenges faced by wildlife health professionals who often lack the necessary laboratory tools to investigate disease in the myriad animals they encounter, and who therefore must develop them de novo.

Loss of coral reefs is analogous to loss of rainforests, and in some regions of the world, such as the Caribbean, diseases have decimated coral reefs over the past 30 years. Unfortunately, because biologists and wildlife health experts did not team together to address the problem, we know little about the cause of coral diseases and their declines.

If we do not have the laboratory tools for charismatic megafauna such as turtles, imagine how difficult it is when investigating diseases in less loved life forms, such as molluscs and other invertebrates. For many of these species, we know little about physiology, anatomy, or behaviour. Yet the health of these organisms can often drive the health of entire ecosystems. For example, tropical marine ecosystems are underpinned by tiny animals called corals that form massive reef structures and provide habitat for myriad fish and other invertebrates. By buffeting the effects of storm surges, corals also protect human communities living along the coasts. Corals are an extreme in wildlife disease ecology, in that the animal is, in essence, the environment. Loss of coral reefs is analogous to loss of rainforests, and in some regions of the world, such as the Caribbean, diseases have decimated coral reefs over the past 30 years. Unfortunately, because biologists and wildlife health experts did not team together to address the problem, we know little about the cause of coral diseases and their declines. In the Pacific Ocean, home to the highest diversity of corals, we are trying to avoid a repetition of this mistake by applying some of the standard biomedical tools we use for other wildlife species to figure out what is killing corals—with the hope that this knowledge will allow us to manage and mitigate the effects of disease on these populations.

As these examples show, the developing field of wildlife health is intellectually exciting, and promises to yield much new information about infectious and non-infectious causes of mortality in terrestrial and marine ecosystems. In this day and age of specialisation, there are few fields that afford the opportunities to work with such a variety of organisms, disciplines, and ecosystems in a truly integrative fashion. Biologists and wildlife health experts working in concert have much to learn and discover as they strive to maintain healthy ecosystems and conserve natural resources and species in an ever-changing world.

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What's in the Genes: The molecular age of avian malaria

When we think about malaria, we immediately think of human beings—people in the tropics being bitten by mosquitoes, and getting consumed by malaria afterward. We rarely make a mental connection between malaria and birds, yet malaria parasites influence virtually all aspects of their avian hosts' lives. I have been studying the effects malarial infection can have on bird populations, in susceptible ecosystems like the Hawaii archipelago.

Although the word 'malaria' has previously been used to refer to any type of infection caused by a blood parasite, we now use the term commonly to indicate infections originating from the genus *Plasmodium*. Parasites from both this genus and *Haemoproteus* are cosmopolitan (except for Antarctica), and have been reported from a broad range of bird species. These parasites are transmitted between hosts through vectors; *Plasmodium* species use mosquito vectors, whereas *Haemoproteus* species prefer biting midges from the genus *Culicoides* (Diptera: Ceratopogonidae) and louse flies (Diptera: Hippoboscidae).

Avian malaria is closely related to human malaria, but unlike the human form, it is not strictly a tropical disease, and is found in many temperate birds. With recent advances in molecular genetics techniques, an astonishing diversity among blood parasites has been revealed–much of which was not evident from morphology alone. In addition, the majority of distinct lineages identified from mitochondrial DNA sequences have been shown to represent reproductively isolated entities–effectively, biological species. It has been suggested that there might be as many lineages of parasites as there are species of birds!

The severity of malarial infections in bird populations depends on whether the birds have encountered the parasite previously during the course of evolutionary history. In island systems like Hawaii, which have been isolated for millions of years, birds have evolved in blissful oblivion, without any exposure to the malarial parasite. However, in areas where malaria is found, like in Asia, some birds have been co-evolving with the parasite.

WHEN MALARIA REACHES WHERE IT'S NEVER BEEN BEFORE

Islands in the Hawaiian archipelago are a classic example of how biological invasions can have a profound effect on endemic species (those that are found only in one area and nowhere else in the world). These islands have been isolated from the mainland for many millions of years. Geographical isolation and colonisation from the mainland facilitated the evolution of countless unique life-forms–including, for example, the Hawaiian honeycreepers. The absence of disease-causing agents has been suggested as one of the reasons why such a diversity of life forms is often found on isolated islands. Based on fossil evidence, the Hawaiian Islands were once home to more than 100 endemic species and subspecies of land and water birds.

However, things changed in Hawaii when the Polynesians, and later the Europeans, landed in the late 19th and early 20th centuries. Numerous birds from different continents were introduced to the islands. At least 17 bird species new to Hawaii became established in forest habitats after this initial introduction. The malarial pathogen (*Plasmodium relictum capistranoae*) was probably present in some of the introduced birds. For forest-dwelling native birds, the accidental introduction of mosquitoes (not to mention additional diseases such as avian pox and avian malaria), have had extremely negative consequences.

Insects are among the groups of organisms most likely to be affected by climate change, because climate has a particularly strong direct influence on their development, reproduction, and survival. Given that suitable vectors are present to transmit and maintain the infection, such migrants can form an effective bridge for parasites between wintering and breeding grounds.

Illustration: Priya Sebastian
Photoshop image: Gimani

Across the mountains of Hawaii, birds, mosquitoes and avian malaria seem to be playing out a predictable drama. At lower elevations, there are many introduced bird species and almost no native Hawaiian bird species. The introduced mosquito, *Culex quinquefasciatus*, abounds in these areas. The introduced birds, however, are resistant to malaria. Malaria in the low elevations is only found in native Hawaiian birds that have somehow survived the infection–malaria prevalence is very low. As you go higher into the mid-elevation forests, the prevalence of malaria increases in native Hawaiian birds. Even higher up the mountain, abundances of both the mosquito and the *Plasmodium* decline due to the lower temperatures, and native Hawaiian birds are free to thrive and reach their peak numbers. Some of the native birds also escape to higher elevations to escape from the parasites.

Introduced birds seem to be doing rather well, with no mortality. On the other hand, 50-90% of the endemic Hawaiian bird species are dying out. There could be many other factors causing extinctions of these native birds, but avian malaria is believed to be one of the key reasons behind the crash of bird populations in the otherwise very suitable lowland and mid-elevation habitats.

There is an exception to every rule, and this case is no different. In the Hawaii amakihi (*Hemignathus virens*), a species of Hawaiian honeycreeper, malarial infections affect the survival of adults. However, the disease does not affect reproductive success or prevent populations from growing. Most other native bird species are on the decline in the forests of Hawaii, but the amakihi appears to be evolving tolerance to infection: Lowland populations have rebounded dramatically in recent years.

WHEN MALARIA GETS INTO AN ARMS RACE

The effect of malaria is quite different in locations where avian malaria parasites have been living for millions of years. If Hawaii is considered to be one end of the disease spectrum, the Asian mainland—where there is a long history of hosts and parasites co-evolving and engaging in arms races—is an example of a counterpart at the opposite end.

Even in areas like the Western Himalaya, where resident species (non-migrant species) might have been exposed to malaria before, there can be scenarios where the parasite can result in the sudden emergence of disease epidemics. This can happen because a new host (the endemic birds) is exposed to a parasite (the *Plasmodium*) to which it has no protective immune response; this is similar to what happened in Hawaii. Alternatively, a parasite can gain increased virulence (the power to infect) by various means, like mutations. Or, a change in the environment can affect the equilibrium that was established between the host and parasite over millions of years. For example, a change in climate may result in a longer transmission season to which birds might not be able to adapt quickly enough. New climatic conditions can also result in the spread of the parasites into new habitats where they may encounter new, susceptible hosts.

HITCHING A RIDE WITH MIGRATING BIRDS

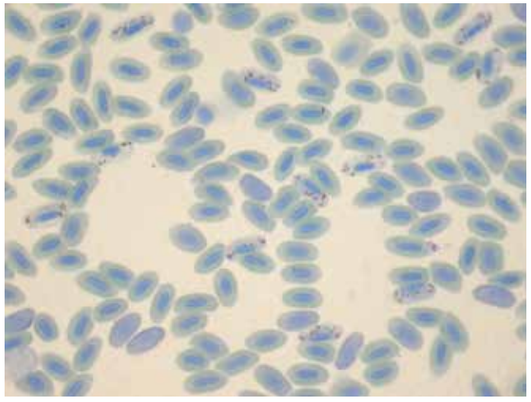
Some birds travel great distances on their migratory journeys, and carry their parasites with them when they head back from their warm wintering grounds. The role of migratory birds in the spread of diseases between regions has been widely documented. However, journeys between different altitudes in the same mountain range, and whether parasites also travel, have not been documented. Migrants move between altitudes or down to the plains, and thereby encounter more diverse faunas of parasites compared to their non-migratory counterparts.

In the plains, the resident birds may act as reservoirs for blood parasites, increasing the risk that migrants will become infected with new parasites on their wintering grounds. Insects are among the groups of organisms most likely to be affected by climate change, because climate has a particularly strong direct influence on their development, reproduction, and survival. Given that suitable vectors are present to transmit and maintain the infection, such migrants can form an effective bridge for parasites between wintering and breeding grounds. If the high altitude avian fauna has evolved in the absence of these blood parasites, their risk of infection is potentially increased.

Studies on human malaria have shown that climate change can alter both where *Plasmodium* is found, and how much of it is found in different areas. The period when the Plasmodium grows inside the mosquito is very sensitive to temperature; the development of the parasite is completely blocked below 15C. To make the situation even more complicated, the vectors that *Plasmodium* depends on for transmission are also affected by climate change. As the environment changes, some habitats that are currently too cool to sustain vector populations may become more favourable, while others that are drying may become less favourable for insect breeding. For instance, a small rise in in ambient temperature and rainfall can increase the breeding season of mosquitoes in a particular area. This can increase the time window of malaria transmission, resulting in a larger number of generations of parasites per year.

The Wellcome Trust-DBT India Alliance has funded a study of a possible altitudinal variation in the presence of avian blood parasites transmitted by vectors in the Western Himalayas. The main aim of the project is to study how changes in temperature, the community of insect vectors, the migration patterns of birds, and changes in habitat affect both rates of avian malaria infection, and its spread in high altitude areas that do not currently have malaria.

The project focuses on the distribution of both *Plasmodium* and *Haemoproteus* blood parasites. It also studies the extent to which these parasites are exchanged between migrating and resident bird populations of *Phylloscopus* warblers (Leaf warblers) and *Parus* species (Titmice species) distributed across an altitudinal gradient, and how the vectors that carry these parasites between hosts are expanding to new areas in the Himalayas. The study is challenging because although we know the Western Himalayas are species-rich, the area is also relatively understudied, and there has been no research on the dynamics of the avian diseases and their vectors to date.



Farah Ishtiaq

Blood smear from white-eye bird (*Zosterops palpebrosus*) showing malaria parasite (purple dots)

Across habitats as diverse as the tropical forests of Hawaii or the snow-capped peaks of the Himalayas, avian malaria parasites are important study systems for testing hypotheses about host-parasite evolution and ecology and the results of these studies have huge conservation implications for a variety of avian species. These findings can also be used to create models of the epidemiology of human malaria. Despite the similarity of disease susceptibility and transmission in humans and birds, birds may be easier to study because they are not subject to the complexities of human socio-economic and cultural patterns. Thus, they provide an excellent system in which to directly determine how ecological changes such as climate change, temperature, vector community, and habitat affect the prevalence and spread of diseases worldwide.

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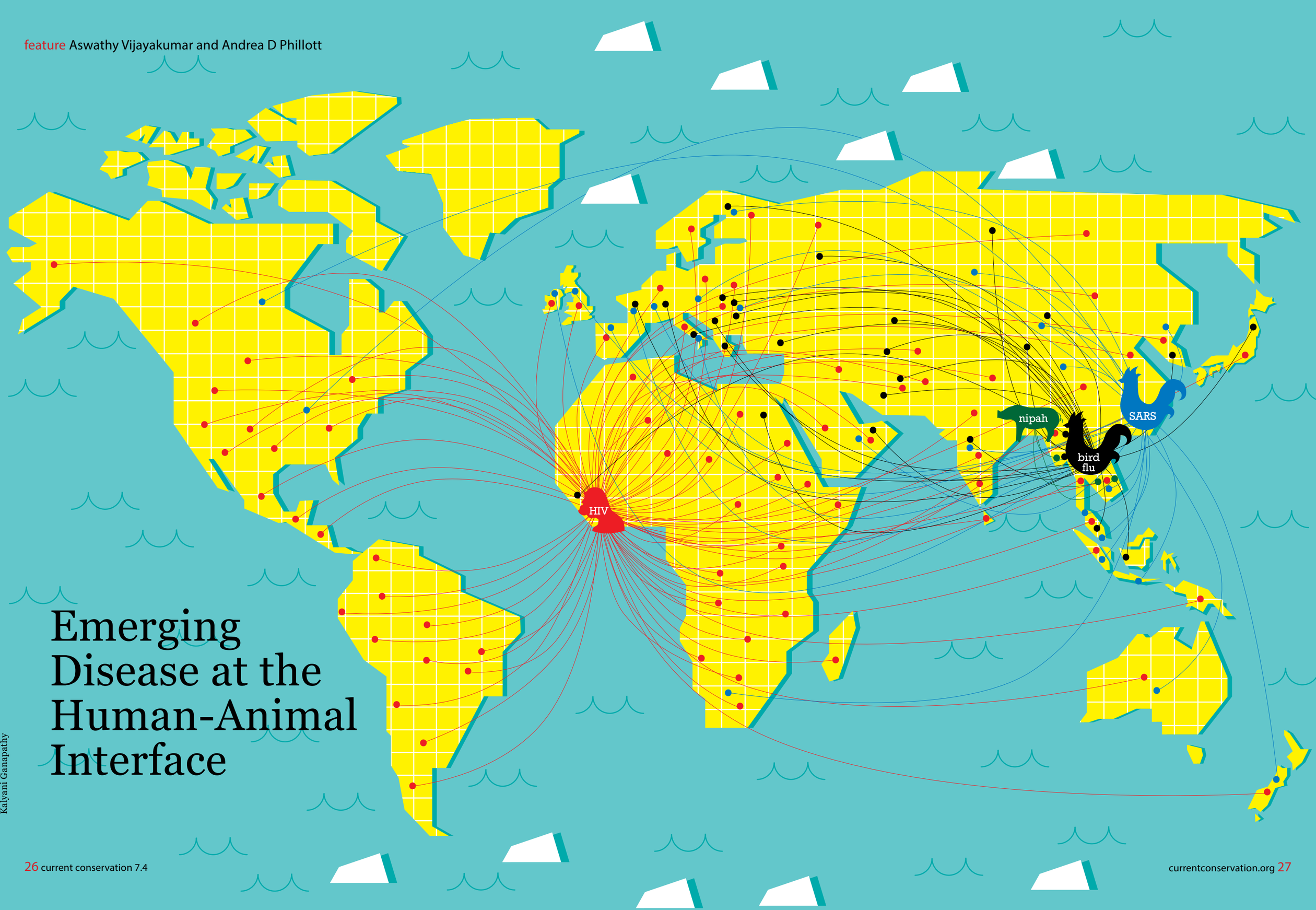
feature Aswathy Vijayakumar and Andrea D Phillott

Emerging Disease at the Human-Animal Interface



Kalyani Ganapathy

More than 60 percent of infectious diseases that affect humans are caused by pathogens that we share with wild and domestic animals. Such diseases, known as "zoonoses," are transmitted from other vertebrate animals to humans in a process known as "spillover," and pose a significant threat to human health. Spillover is more common in developing countries, which experience rapid environmental change as their human populations grow. Some zoonoses, such as rabies and anthrax, occur only when there is transmission directly from animals to humans. However, there is the potential for a shift from animal-to-human to human-to-human disease transmission. The result may be a localised outbreak of disease, such as the periodic emergences of Ebola virus in Zaire/Democratic Republic of the Congo, or a global spread of epidemic proportions such as the recent outbreak of Severe Acute Respiratory Syndrome (SARS).

In order to better anticipate, contain, and even prevent such outbreaks, it is first necessary to improve our understanding of the basic epidemiology of zoonoses, including the factors that cause new diseases to emerge, and those that cause re-emergence of known diseases. The first step in this process is considering the anthropogenic activities that have been linked with the dramatic increase in incidence and frequency of zoonoses over the past 30 years.

CHANGES IN HUMAN POPULATION SIZE AND DENSITY

Pathogens are more likely to occur in populations above a certain density; below this "threshold density," pathogens cannot survive. Before the Second World War, most human settlements in tropical developing countries were scattered, with few large cities. This pattern has changed over the past decades: areas that previously consisted of scattered settlements are now occupied by large mega-cities surrounded by semi-urban settings, with only small areas of undisturbed forest area remaining near croplands and degraded lands. In 2006, there were 18 mega-cities in the world, cumulatively containing more than 10 million inhabitants. By 2025, Asia alone is predicted to have 10 mega-cities; by 2030, these areas may be home to as many as 2 billion people. Both the size and high density of human populations and their associated domestic animals increases the risk of new disease emergence. These conditions also allow rapidly reproducing pathogens more chances to undergo mutation and develop new traits that will promote survival and infectiousness. Even if there are no immediate outbreaks or emergences of new diseases, pathogens will adapt to conditions within the infected host. Later, under favorable circumstances such as a weakened host immune system, the resident microbes can cause an infection.

LAND USE AND ENVIRONMENTAL CHANGES

Another consequence of human population growth is an increase in deforestation, the conversion of forested land to non-forested areas such as cropland, plantation and urban habitats. This allows humans to settle in formerly isolated areas rich in previously unknown pathogens. This is probably what led to the spread of human immunodeficiency virus (HIV), which originated as simian immunodeficiency virus (SIV) in chimpanzees.

Deforestation is frequently associated with forest fragmentation, which reduces the number of predators that can occupy the habitat. Without predators to control their population, rodents and biting insects thrive; because these animals can act as reservoirs for human disease, their increase allows zoonoses to flourish. As the movement of humans and wildlife species between remnant forest and human habitation increases, pathogen-host interactions also increase and result in the spread of infections.

GLOBAL BIOSPHERE AND CLIMATE CHANGE

Anthropogenic changes to the global biosphere include shifts in land and water use, biodiversity loss, and introduction of new chemicals. These processes alter environmental factors such as temperature, humidity levels, and water availability. Disease vectors such as ticks and mosquitoes, which carry disease from one organism to another, are sensitive to such changes in the environment. The resultant fluctuations in vector populations can facilitate the emergence of vector-borne zoonoses such as West Nile virus.

Climate change, a phenomenon associated with global biosphere change, is likely to increase populations of vector-borne pathogens in cold regions, which are expected to become warmer. At the same time, it may decrease the transmission of infections in areas that become so warm that vectors are no longer able to survive in high numbers. Climate change is also predicted to change patterns of human activity. For example, continued warming of the Earth's surface temperature could reduce the quality of pasture lands, causing a decline in livestock numbers and human-animal interactions.

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This changing context presents many uncertainties. For example, droughts may decrease mosquito populations and, therefore, the incidence of mosquito-borne diseases; at the same time, the accumulation of dead vegetation might act as a reservoir for other pathogens. The effect of climate change on the spread of zoonotic disease is likely to vary according to geographic location and local habitat.

HUMAN CONSUMPTION OF ANIMALS

Recent advances in the way food is produced, processed, and preserved can also play a role in the emergence of zoonotic disease. One especially influential practice is the use of antimicrobial feeds and drugs. The latter are particularly to blame for an increase in the number of antimicrobial-resistant pathogens that can be transmitted from animals to humans through the food chain. In human *Salmonella* isolates collected in the United States, for example, resistance to antimicrobial agents rose from 0.4% to 1.0% between 1996 and 2001.

Among animals reared for agricultural purposes, repeated breeding of genetically similar stocks leads to weakened immune systems that leave animals less resistant to infections that may then be transmitted to humans. Further, these animals are often housed and transported in crowded conditions that increase the chances of exposure to, and transmission of, infections.

Another risk factor for cross-species spread of infection is subsistence hunting. The bushmeat trade–the tracking, capture and butchering of animals in the wild, and transportation of meat—brings humans dangerously close to potential vectors and the microbes they carry. Risk of infection is especially high during interactions with non-human primates such as chimpanzees; these close relatives of humans may transmit pathogens such as Ebola virus. The amount of annual wild meat consumption has been calculated to be around one billion kilograms for central African countries alone. In Cameroon, Central, and West Africa, bushmeat hunters and other persons who handle vertebrate pets are at a higher risk of zoonotic transmissions as a result of bites, cuts and exposure to the bodily fluids and tissues of infected animals. Bushmeat hunters are commonly infected by simian foamy virus; luckily, human-to-human transmission of this pathogen has not yet been established.

ZOONOTIC DISEASES IN DEVELOPING COUNTRIES

Zoonoses are most likely to emerge where humans come into close contact with animals. Such encounters are most likely in tropical regions, which are characterised by increasing human population densities and rapid urbanisation. Because of the high number of low-income individuals in these countries, a significant proportion of the population is faced with poor sanitation, substandard housing, inadequate disease control and management, and rapid urbanisation. Globally, over 600 million people are dependent on livestock for their income, and up to 70% of these people live in marginalised developing regions. Two-thirds of the workforce in sub-Saharan Africa and South Asia is involved in agriculture; livestock are vital as a source of both income and food. Unfortunately, they may also be a source of pathogens and, to compound the problem, there are few incentives for farm-level management and control of infections.

Once infected, people in this socioeconomic group often lack access to proper medical care. Health centers are few and far between, and potential patients lack the time and money needed to visit them often. As a result, most zoonotic infections are chronically under-diagnosed. The burden of looking after a seriously ill family member may push the household further into poverty and illness.

Another major challenge associated with disease management in developing countries is the lack sufficient information to make decisions, both at an individual and a national level. In India, for example, around 68% of the national workforce depends on farming, yet most people are unable to tell whether they are working with infected or healthy animals. Because of ineffective data collection and poor disease management practices, there are no data on death rates associated with zoonotic infections across the country. Further, there seems to be a reluctance to recognise that the study and management of zoonoses requires the combined efforts of medical and veterinary professionals, who have, historically, worked in isolation from each other. The transmission of swine flu across the country can be used as an example: Even though many control measures were taken to prevent infections among humans, few or no measures were taken at the veterinary level to prevent infections among livestock. This lack of coordination and information is only exacerbated by widespread illiteracy, poverty and unsafe living conditions.

LEARNING FROM THE 'NIPAH OUTBREAK' IN BANGLADESH

The emergence of new infections and the re-emergence of known infectious diseases are both major global concerns, particularly in developing countries that lack adequate medical surveillance, disease management practices, and financial resources. Successful management of zoonoses requires collaborations between experts at the local, national, and global levels, as currently being demonstrated in Bangladesh in association with the emergence of the Nipah virus.

Once infected, people in this socioeconomic group often lack access to proper medical care. Health centers are few and far between, and potential patients lack the time and money needed to visit them often. As a result, most zoonotic infections are chronically under-diagnosed. The burden of looking after a seriously ill family member may push the household further into poverty and illness.

Nipah infections in Bangladesh are very different to the first emergence of this zoonosis in pig farms in Malaysia in 1998, when pigs were exposed to flying fox urine, faeces or saliva carrying Nipah virus. The disease quickly spread to humans throughout Malaysia and Singapore as infected pigs were transported to slaughterhouses. Since 2001, periodic outbreaks of Nipah virus, with approximately 200 human fatalities, have been reported from Bangladesh and northern regions of India; however, these occurrences of zoontoc disease resulted from increased habitat loss which placed humans in closer contact with flying foxes. In Bangladesh, the infections coincide with the date palm sap harvesting season from November to March because the most common transmission pathway for Nipah virus is human consumption of sap contaminated with flying fox saliva and urine.

Date palm sap is collected through a tap or funnel that drains into a clay pot, often left in place overnight and frequently visited by flying foxes that enjoy the sugar-rich sap. The harvester, their families and friends drink the raw sap the next morning. Shields to keep fruit bats away from the sap collection pots are known to local harvesters, but rarely used until scientists from United States Centers for Disease Control and Prevention, and International Centre for Diarrhoeal Disease Research, Bangladesh, determined that home-made skirts covering the sap-producing surface and mouth of the collecting pot prevented most flying fox and bats from contaminating the sap. Community intervention trials are underway to determine if changes in sap harvesting practices can reduce local spillover of Nipah virus from flying foxes to humans, minimising the human-to-human transmission which may occur during the care for an infected patient.

PREDICTION AND PREVENTION OF THE FUTURE ZOONOTIC DISEASE

As human populations grow, and our interactions with the environment change, there is a greater likelihood of emerging disease at the human-animal interface. However, greater awareness and collaboration among doctors, veterinarians, wildlife carers, biologists and communities can identify potential diseases at the source, before there is the potential for diseases to be carried to other regions and have a greater impact on human health and economies.

As shown by the case of Nipah virus in Bangladesh, minimising the risk of zoonoses is best accomplished with the involvement of local people. Global co-operation to support monitoring programs in tropical developing countries, hotspots for disease emergence, and new molecular methods of identification will help to quickly isolate potential new pathogens. Advanced communications technology allows outbreaks to be reported quickly so susceptible communities can be informed of transmission pathways and prevention strategies. Our best weapons against future zoonoses are understanding the origin and dynamics of pathogens in wildlife, reacting quickly to spillover events so that the disease has limited time to be transmitted among the human populations.

Further reading:

Khan SU, ES Gurley, MJ Hossain, N Nahar, MAY Sharker & SP Luby. 2012. A Randomized Controlled Trial of Interventions to Impede Date Palm Sap Contamination by Bats to Prevent Nipah Virus Transmission in Bangladesh. *PLoS ONE* 7: e42689. doi:10.1371/journal.pone.0042689.

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Human diseases: Insights obtained from wildlife research

Kalyani Ganapathy

Current wildlife research in India is mainly driven by conservation priorities. Often, studies that do not have a conservation implication are treated as a sinful luxury. What is less appreciated is that basic wildlife research can yield many important insights into the fundamental principles of life, behaviour, society and health. Health is an ecological phenomenon: when you consider infectious disease as an interaction between two or more species, it becomes intuitive to consider it an ecological process. However, current opinion suggests that even non-infectious diseases, such as type 2 diabetes, hypertension and cardiovascular disease can be better understood as ecological processes. Increasingly, many principles of evolutionary physiology and animal behavior are providing a radically different and insightful view of the disease process.

Our major understanding of human diseases comes from experiments on captive animals. However, certain intricate patterns can only be revealed through wildlife research. Wildlife data have contributed to our understanding of some of the modern lifestyle-related disorders, and we will illustrate this using the example of type 2 diabetes.

A GLOBAL BURDEN OF HAUNTING DISEASES

Diabetes, one of the oldest diseases described in humans, has had an unprecedented impact on modern human health. In 2011, 366 million cases of diabetes were reported worldwide; this number is expected to rise to 552 million by 2030. The number of people with Type 2 Diabetes Mellitus (T2DM) is increasing in every country. India, with its huge numbers of T2DM patients, is considered the diabetes capital of the world. Though diabetes has been intensely studied, the exact cause and mechanism of the disease remains shrouded in mystery, and no permanent cure is in sight. T2DM begins with insulin resistance—the inability of cells to respond to the hormone insulin like normal cells would. Insulin resistance is usually associated with high levels of glucose, though which of the two comes first is debated. The current belief is that insulin resistance is central to T2DM, with blood sugar rises resulting from an inadequate insulin response.

There are three main hypotheses as to why T2DM is incurable. First, it may be associated with some sort of irreversible physiological or morphological change. If this is the case, then diabetes would function like retinal damage, which is permanent because retinas cannot regenerate in humans. However, recent research has shown that this is unlikely. The two main components believed to cause diabetes, namely insulin resistance and degeneration of beta cells, are both reversible. The second possibility is that the pathological components of diabetes are not, by nature, irreversible, but we do not have the technology to reverse them. This is also unlikely since there is extensive work on insulin sensitisation and stimulation of insulin secretion, both of which are unable to cure diabetes once it emerges.

The final possibility is that our current understanding of type 2 diabetes is fundamentally wrong, and therefore we have not yet been able to determine the correct approach for curing it. The lack of support for the first two possibilities makes the third more likely. This might be true not just for diabetes, but also for a number of other modern lifestyle disorders, including hypertension, cardiovascular disease, osteopenia, polycystic ovary syndrome, and chronic fatigue syndrome—all of which are increasingly prevalent but not yet well understood.

Results from recent studies of wild and semi-wild primates are changing our perceptions of diabetes, rapidly generating an alternative understanding of the condition, and raising hopes of finding a cure in near future. In approximately 15% of bonnet macaques reared in groups under natural conditions, insulin resistance arose spontaneously—independent of age, physical activity, diet, or body weight. This is surprising, given that old age, a diet high in fat and/or carbohydrates, and obesity are often blamed for insulin resistance. If these are not the causes of insulin resistance in primates, then what are they? Similarly, in wild chimpanzees, individuals that are strong, dominant and aggressive consistently have lower insulin resistance. On the other hand, weaker, subordinate and submissive individuals are insulin resistant despite having a lower calorie intake. This suggests that we should rethink the true causes of insulin resistance.

BEHAVIOURAL SYNDROMES AND THEIR ACCOMPANYING PHYSIOLOGIES

The concept of ‘hawk’ and ‘dove’ behaviours was introduced by John Maynard Smith, a British evolutionary biologist and geneticist. In a conflict situation, a ‘hawk’ behavior is an aggressive one, with the individual willing to risk the negative outcomes of an attack or fight. ‘Dove’ behaviour, on the other hand, avoids physical aggression, often through the use of deception. Whenever a ‘hawk’ and a ‘dove’ enter a conflict, the dove always retreats and the hawk wins. This may suggest that it is always better to be a hawk. However, a meeting of hawks often results in huge costs—including injuries or even death—to one or both individuals. Doves, on the other hand, escape with merely a temporary defeat. As a result, hawk and dove strategies can coexist in stable equilibrium in a given society.

In real life, one can see this and similar behavioural dichotomies in a wide variety of animals, ranging from frogs, rats, primates and even humans. For example, large frogs are able to use their sizeable vocal sacs to attract mates, while smaller frogs cannot do this and prefer to linger around larger males and take a chance by engaging in ‘sneaky’ matings with females. Such behaviour is also seen in other species—less sexually attractive ‘dove’ males wait near more attractive ‘hawk’ males in the hopes of sneaking a copulation with females attracted by the ‘hawk’ males’ displays the ‘hawk-dove’ model explains how individuals can be successful despite being physically weaker: Individuals that are not strong can still be socially ‘smart’.

In primate societies, weaker individuals show tactical and deceptive behaviours toward stronger individuals, relying on strategy rather than brute strength. Insulin plays a role in strengthening cognitive abilities as well as altering behaviour; it has been shown to help thinking and problem solving on the one hand, and reduce aggression and risk-taking behaviour on the other. Therefore, it likely helps the weaker but smarter subordinate individuals. If this is true, then it is no surprise that the subordinate chimpanzees have high insulin levels and accompanying insulin resistance.

SOLDIER VERSUS DIPLOMAT

These results from animal research have inspired a completely new interpretation of the origins of T2DM in humans. According to this theory, the human analogs to hawks and doves are ‘soldiers’ and ‘diplomats’ (referring here to personalities, rather than actual professions). The soldier trait is characterised by physical strength and aggression, risk-taking, swiftness, injury proneness, and tolerance to physical pain and discomfort. A diplomat, on the other hand, is physically weaker but socially smarter; this condition is brain-dependent and involves social manipulation. In both animals and humans, behaviour has been strongly linked to insulin, cholesterol and cortisol levels. Levels of these molecules alter behavioural preferences, and, in turn, engaging in specific behaviours alters the levels of these molecules. Typically, strong, aggressive and dominant hawk/soldier behaviour is characterised by low levels of insulin, cholesterol and cortisol, as well as high insulin sensitivity. Conversely, dove/diplomat behaviour is associated with high levels of insulin, cholesterol, and cortisol, and low levels of sex hormones and growth factors.

THE BEHAVIOURAL TRANSITION: FROM HUNTING-GATHERING TO MODERN URBAN LIFE

We evolved and lived as hunter-gatherers for millennia. But even in ancient human societies, the soldier-diplomat dichotomy existed: There were shamans or magic-men who would have hunted less and lived by shaping people’s faith. Over the course of modernisation, we have been engaging more and more in the diplomat way of life, and a majority of the population has lost its the hunter-fighter instincts—a by product of the transition from a muscle-dependent lifestyle to a brain-dependent lifestyle. This transition is associated with certain physiological changes in the body. Australian Aborigines are the classic example of what happens to human physiology during such a transition: Aborigines in urban environments rapidly become diabetic and hypertensive, but no longer experience these conditions after returning to the wilderness and resuming a hunter-gatherer lifestyle.

Though researchers have argued for quite some time that the Paleolithic diet is the critical factor, it is increasingly clear that the Paleolithic environment and behaviour are equally critical. The modern lifestyle is sedentary, and deficient in physical aggression, agility, endurance activities and quick and complex nerve-muscle coordination. These deficiencies bring about many changes in body chemistry; these are increasingly recognised as causing not only diabetes, but also many other modern lifestyle disorders. These are not superficial explanations; there is now detailed knowledge about how different behavioural strategies are associated with variations in gene expression and changes in metabolism, hormones, and immunity. More than 70 signalling molecules are now known to link behaviour to hormones, metabolism and immunity.

VANISHING WILD AND SERENE PLACES

Modern humans are increasingly found living in crowded urban environments away from wilderness and serenity. High population densities create an anticipation of future food scarcity, since this is what happens in nature. Populations of wild animals periodically rise and fall; these ups and downs have caused animals to evolve the ability to anticipate starvation and store fat. Experiments in worms and fruit flies have shown that crowding induces fat storage. Perception of crowding also affects behavioural responses, favouring diplomat traits over soldier traits. It also affects many hormone levels in the body. The unprecedented overcrowding of cities and lack of serene natural places is likely to be an important contributor to changes in human health.

MINOR INJURIES: GOOD FOR HEALTH

Hunter-gatherer and agricultural lifestyles were associated with frequent minor injuries and behaviours that anticipate injuries. Minor injuries maintain a delicate balance between central and peripheral immunity. Normally, minor injuries result in the movement of macrophages, a type of immune cell, to the skin. In the absence of injuries, macrophages fail to undergo this migration, instead remaining in the blood vessels, where they cause inflammation of the vascular tissue. Many of the molecules—particularly growth factors—involved in would healing are also shown to be essential components for the normal growth of pancreatic beta-cells that secrete insulin.

Does this mean that being a diplomat is wrong, and therefore leads to diabetes and other modern health problems? Not necessarily. Rather, the source of the problem is less being a diplomat than exhibiting a deficiency of hunter/soldier behaviours. In a non-violent society, these latter traits can be experienced in the form of aggressive sport and outdoor adventure. Physical exercise has long been known to protect against a number of modern disorders. Now we know that exercises are not mainly important for burning calories, but for filling a behavioural gap in our lives. Therefore it is the type of exercise that matters, not the number of calories burned.

Much of this new view of disease has originated in wildlife research. This is an important realisation for wildlife lovers, researchers, and managers. Not only can wildlife be observed through binoculars; they can also make us think and learn about our own lives and health. Wildlife research should focus on more than saving species; studies of basic animal biology can also help us improve our own lives. Our new understanding of type 2 diabetes best illustrates this potential.

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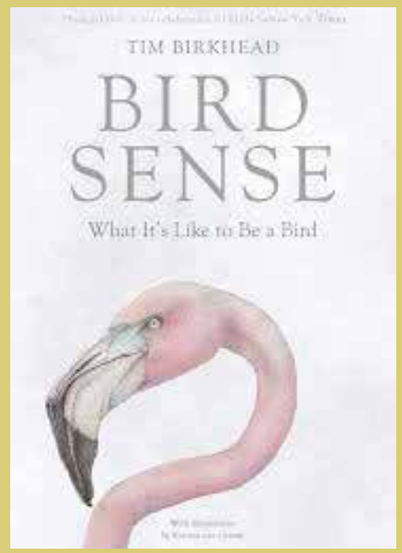
Being a bird



Bird Sense: What It's Like to Be a Bird
Tim Birkhead

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Bloomsbury USA; Reprint edition 2013

Written by an ornithologist, Bird Sense is a fascinating account of the senses that enable birds to carry out their day-to-day activities like feeding or avoiding predators. Author Tim Birkhead, who has studied zebra finches and common guillemots for most of his scientific career, has successfully hinted at what it's like to be a bird. Every chapter in the book deals with one sense—seeing, hearing, touch, taste, smell, magnetic sense and emotions—in birds as varied as owls and hummingbirds, making the science that goes into the discovery and understanding of the senses accessible to lay persons.

The book familiarises its readers with the amazing diversity of behavioural and anatomical adaptations that can be found in birds. A case in point is asymmetrical ears in some owl species that help owls locate the source of sound and find prey in the dark.

Bird Sense also informs its audience about the scientific process, suggesting how science builds on previous work. It talks about the debates and controversies some senses, such as those of smell and taste in birds, have sparked in the community of ornithologists. In author's words, 'For some inexplicable reason ornithologists have found it hard to accept that birds might have a sense of smell.' Whether birds could have a sense of taste was debated for long, too. And even now, the idea of consciousness in birds remains controversial.

Bird Sense gives its readers a good overview of most, albeit not all, bird senses. While the first five chapters of the book—'seeing', 'hearing', 'touch', 'taste' and 'smell'—are rich in science, those on magnetic sense and emotions—areas where a lot remains to be scientifically explored—are less detailed. One major downside is that the book is short of illustrations – there is only one at the beginning of each chapter. Colourful pictures of the lesser-known birds mentioned in the book would have helped readers relate to the text. These would also have improved the book's aesthetics. It is especially surprising that flamingos, one of which is featured on the book's cover, receive only passing mention for their mysterious ability to sense rain falling hundreds of kilometres away. More details on this intriguing talent are, sadly, missing.

Despite these drawbacks, the book is a compelling read. It includes interesting and unusual details, and is strewn with intriguing anecdotes. It vividly summarises what science has revealed about birds' senses so far and leaves clues to several potential research questions for future investigation. It is, therefore, no wonder that the book was shortlisted for the prestigious 'Royal Society Winton Prize for Science Books 2013'. Readers who enjoy Bird Sense may want to read Birkhead's latest book Ten Thousand Birds: Ornithology Since Darwin, or read his other works, including The Wisdom of Birds, Promiscuity, Great Auk Islands, Sperm Competition in Birds and The Red Canary, among others.

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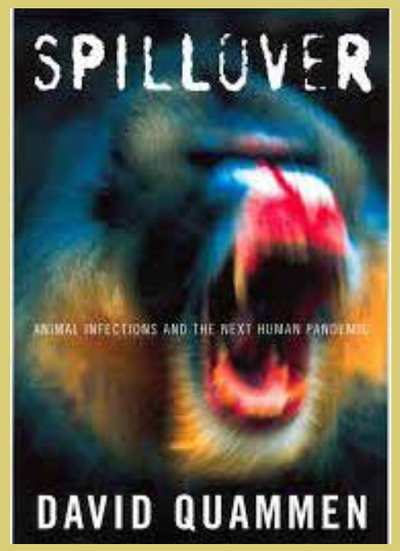
How and why animal diseases impact humans



Spillover: Animal Infections and the Next Human Pandemic
David Quammen

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W. W. Norton & Company, New York, 2013

Several years ago, I had the opportunity to interview award-winning science and nature writer David Quammen, author of (among other things), Monster of God, Flight of the Iguana, and The Song of the Dodo. At the time, he was celebrating the 200-year anniversary of Charles Darwin's birth by touring the United States to read from The Reluctant Mr. Darwin, Quammen's "intimate portrait" of the biologist who developed the theory of evolution.

Towards the end of our conversation, I asked Quammen if he was working on anything new. It turned out he was: something to do with animal diseases that could be transferred to humans. I was surprised; the majority of Quammen's previous books had focused predominantly on megafauna—particularly endangered species—and the increasingly threatened wildernesses in which they live. This new book sounded like quite a departure.

As it turns out, that is not exactly the case. The book, which was not finished until nearly three years after our conversation, was published in late 2012 under the title Spillover: Animal Infections and the Next Human Pandemic. A perusal of Spillover reveals that Quammen's favourite themes are still present, but are examined from a different perspective and at a different scale. The megafauna are, more often than not, humans, and the ecosystems are both those through which we move and those that are found within our own bodies.

As a result, Spillover is, in many ways, the most personal of Quammen's books to date. It should have particular resonance with readers who have nervously noticed the increasing frequency with which new disease outbreaks have been reported—diseases such as bird flu, SARS and Nipah, all of which have their origins in non-human animals. The book describes both why and how such pathogens are shared between humans and our animal neighbours; it also explores whether we can use information from previous and current zoonotic epidemics to predict those that might occur in the future.

Spillover has much to commend it, but two strengths in particular: its treatment of scientific research and the people who conduct it, and its conversational, easily accessible prose. Together, these give the book the intensity and excitement of an adventure novel, but also a breadth, depth, and accuracy that make the book a legitimate educational resource. Where many authors might have been content to merely summarise the major points of published papers, Quammen travelled the world to interview researchers on the front lines of epidemiology: scientists sampling bats in Australia and Asia, primates in Africa and monkeys in Bangladesh. As a result, the author provides not only detailed background information and accessible definitions to scientific terms, but also vivid first-hand descriptions of how immunological work is conducted in the field.

The plethora of case studies examined in Spillover—Hendra, ebola, SARS, Nipah and HIV, to name a few—act as variations on a theme, driving home the point that nearly all spillover events have certain characteristics in common, even if the associated infectious agents vary in location, virulence, transmissibility, and innumerable other traits. Indeed, Quammen describes disease outbreaks as a perfect storm of factors—"Maybe luck... Maybe circumstance. Maybe [population] density. Maybe genetics. Maybe behavior."

However, the author also takes pains to emphasise that humans do not have to passively accept their fate as pathogen hosts. By combining our brainpower with modern technology and the scientific method, we have a fighting chance of identifying "the next big one" before it becomes big—or, at the very least, of responding to it efficiently and effectively once it does make the leap from wildlife into humans. Thanks to this optimistic message, Spillover is an engrossing, informative, and ultimately rather hopeful book.

The following excerpt, from Chapter 74 of David Quammen's *Spillover*, describes the author's experiences volunteering as a field hand in Khulna (Bangladesh), helping a team of epidemiologists investigating whether local bats were carriers of the deadly Nipah virus.

The bats were all out for their nightly feeding. We would lurk here to catch them as they returned, sometime before daylight. Gofur and Pitu had already hoisted the net into place, an invisible wall of delicate mesh in the blackness somewhere above us, big as the screen for a drive-in movie. We hunkered down to wait. The night grew chilly--the first time in my limited Bangladesh experience I'd had occasion to get cold. I lay on my back upon the tarpaper, bundled as best I could be in a light jacket, and went to sleep. The first bat hit the net at 4:22 a.m.

Headlamps came alight, people jumped up, Gofur lowered the net on its pulleys while Epstein and Pitu converged on the animal and I staggered forward after them, safely blinded behind my safety glasses. Pitu untangled the bat and Epstein accepted it, using just the technique he had described: grabbing its head firmly, taking its legs and arms into his finger gaps—binga, binga, binga, binga—and then jouncing the bat into its bag. Close the bag's net, tie firmly with a piece of twine. Captured bats, like captured snakes, evidently relax better if you confine them in soft cloth. Reraise the net and repeat. I was impressed by the proficiency of Epstein's team.

Between the first bat and daylight, before call to prayer even sounded from the local mosques, they bagged five more. Six bats for a night's work was below par for Epstein—he liked to average about ten—but it was a good start for a new location. Adjustments to the net placement, to the height of the masts, would improve the yield here in coming days. For now, enough. As dawn filtered in, we

climbed down the ladder and repaired to the laboratory room. Here again, everybody had an assigned role. Mine was to stay the hell out of the way, and occasionally to assist with a swab.

Three hours later, blood samples drawn, swab samples taken, tubes in the freezer tank, it was time to release the bats. Each of them first received a drink of fruit juice to help restore bodily fluids lost in the blood draw. Then we all walked back to the grassy courtyard, beneath the karoi trees, where a small crowd of men, women, and children from the neighborhood had gathered. (The walls of the old depot compound were permeable to locals when something interesting was afoot.) Epstein, again now wearing welder's gloves, released the first five bats one by one from their bags, holding each animal high so it wouldn't crawl up his face, letting it free its legs and its wings, then relaxing his grip gently just as the wing beats began to find purchase on air, and watching—all of us watching—the animal catch itself short of the ground, rise slowly, circle languidly, and fly away. Eventually, after a circuit or two of the compound, a few minutes of befuddled relief, it would find its way back to the communal roost, sadder but wiser and no great harm done.

Before releasing the last bat, Epstein gave a brief talk to the assembled citizens, translated by Arif, congratulating them on their good fortune as a village at harboring so many wonderful bats, which are helpful to fruit trees and other plants, and assuring them that he and his colleagues had taken great care not to harm the animals while studying their health. Then he let the final bat drop. It climbed through the air, from knee level, and flew away.

Later he said to me: "Any one of those six bats could have been infected. That's what it looks like. They look totally healthy. There's no way to distinguish Nipah virus. That's why we take all these precautions." He dipped his boots again in the sterile footbath, as we left the lab, and washed up at the village pump. A little girl brought soap.

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