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Doorways in nature: Syndemics, zoonotics, and public health. A commentary on Rock, Buntain, Hatfield & Hallgrímsson

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New levels of biological understanding

Nature writer, Douglas Chadwick notes that because they are not a single organism but rather an interactive community of species, he thinks of lichens as kind of a *doorway* between organisms or individual species and ecosystems. "Look out one direction," he writes, "and you see individual things; look the other way, you see processes, relationships—things together. This is the new level in understanding biology" (Chadwick, 2003, p. 119). And it is this new understanding that the authors of *"Animal-Human Connections," 'One Health,' and the* Syndemic Approach to Prevention (Rock, Buntain, Hatfield, & Hallgrímsson, 2009) bring to their analysis of the significance of zoonosis—the movement of diseases between non-human animal species and humans—in the development of health threatening syndemics like bovine tuberculosis.

The concept of syndemics (Singer, 2009), defined as the concentration and deleterious interaction of two or more diseases or other health conditions in a population, especially as a consequence of social inequity and the unjust exercise of power, developed out recognition that the clustering of diseases in disparity populations was contributing in significant ways to the burden of disease. Research over the last decade (Freudenberg, Fahs, Galea, & Greenberg, 2006; Herring & Sattenspiel, 2007; Mavridis, 2008; Singer & Clair, 2003; Singer et al., 2006; Stall, Friedman, & Catania, 2007) has shown that syndemics, which have played a critical role in human disease history (and, as a consequence, in human history generally), are having a significant impact on diverse populations currently (e.g., both the food insecurity/HIV and tuberculosis/HIV syndemics in sub-Saharan Africa) and are likely to have a consequential influence on the emergent health profile of the 21st century.

The syndemic perspective, however, does not stop with a consideration of biological connections, myriad, complex, and fascinating as they may be, because human disease develops within and is greatly impacted by the conditions that comprise the built and interactive social worlds of disease sufferers. Human social relationships, including the operation of prevailing global and local structures of social inequality and injustice (as expressed in disparities in living conditions, exposure to social stressors and nutritional inequality, market distribution of toxic commodities, the privatization of needed resources, etc.) as well as through sociogenic environmental transformations (e.g., the spread of atmospheric brown clouds and other forms of air pollution; industrial pollution of freshwater sources; increases in rates of water- and vector-borne diseases, drought, and flooding because of global warming) contribute enormously to the biosocial chain of disease clustering, disease interaction, and the emergence of new syndemics. Without question, however, disease synergies are not limited to human populations and occur as well in the non-human animal world.

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Disease synergies in non-human species

Exemplary of interacting animal diseases, veterinary pathologists at the Indiana Animal Disease Diagnostic Laboratory at the Purdue School of Veterinary Medicine have identified consequential synergistic exchanges between a group of viruses called porcine circoviruses, first identified in Europe in 1974, and other pathogens, such as bovine viral diarrhea virus (a disease agent that may have spread to domestic pigs from wild deer populations). Interaction of this sort has been found to significantly increase the fatality rate of dually infected pigs. In recent years, porcine circoviruses have spread to pig populations around the world. In places where swine are infected with other viruses in addition to the newly introduced porcine circovirus, mortality rates have jumped by 35 percent to 50 percent. According to Roman Pogranichniy, a Purdue virologist involved in this research, "We think that the new co-factors, including bovine viral diarrhea virus-like pathogen and other swine viruses, work together with porcine circovirus to attack the animals' systems and become more virulent" (quoted in Steeves, 2008:1). Notably, circoviruses have also been detected in human body fluids, although, to date, they have not been found to be pathogenic to their human hosts (Biagini, 2004).

Similarly, various bacterial, fungal, and viral infections are more frequent and more intense in animals with tick-borne fever. Experimental research with sheep, for example, found that animals that are dually infected with tick-borne fever and louping-ill virus not only are more susceptible to louping-ill but almost all infected stock die of hemorrhagic syndrome involving a systemic fungal infection with *Rhizomucor pucillus*. By contrast, sheep given louping-ill virus alone do not develop this lethal syndrome (Brodie, Holmes, & Urquhart, 1987). In related research, Jolles and Ezenwa (2006) have examined the effects of interaction of gastrointestinal worms and tuberculosis in the African buffalo (*Syncerus caffer*). They found that mortality was heightened in co-infected individuals, a pattern that might be explained by adverse effects of helminth infection on individuals suffering from tuberculosis. These researchers concluded, however, that a simple disease dynamic model did not explain their mortality findings. Alternatively, they hypothesized that host defenses against one infection may block simultaneous immunity to the other, an explanation that accurately fit their findings when tested using computer modeling. In humans, Co, Hirsch, Toossi, Dietze, and Ribeiro-Rodrigues (2007) found that dual infection with gastrointestinal worms in patients with newly diagnosed tuberculosis skews the sufferer's cytokine profile toward a T helper 2 (Th2 immune) response, thereby creating an internal environment that supports a prolonged clinical course.

Animal syndemics?

In light of this body of research, the question must be asked: are disease synergies in non-human animal species syndemics, in that the structure of social relations and the issue of inequality and its health effects are not directly an issue in the spread, clustering and interaction of these animal diseases? In fact, of course, in some animal populations, such as non-human primates, social hierarchy is an important factor in predicting which animals get sick. Sapolsky (2005), for example, points out the ways in which social ranking behaviors among animals can have adverse adrenocortical, cardiovascular, reproductive, neurobiological, and immunological consequences, and, as a result, can produce syndemic-like effects. Among hierarchical animals, highly stressed individuals need not be subordinated members of a group. For example, while it is easy "to imagine that subordination can produce an excess of physical stressors," in that subordinate animals "may have to work harder for calories, or be calorically deprived" and be "the subjects of unprovoked displacement aggression" (Sapolsky, 2005, p. 397), among some species being at the top of a dominance hierarchy can also be quite stressful (because of aggressive challenges from-up-and-coming and would-be dominant animals, or even teams of genetically related individuals, as occurs in lion prides). Additionally, the health of animals, both domestic and wild, is significantly impacted by human activity and human social structures.

Domestic animals owned by the rich, for example, are likely to be fed better, housed better, and receive better veterinary care than animals belonging to the poor. Indeed, around the world, the animals of the rich tend to live far better than poor people. Thus, approximately three billion people in developing countries live on less than $2.50 a day, which the Human Society estimates is roughly the cost of maintaining a pet dog in the developed world. Additionally, people regularly move animals to new environments or change the environments they inhabit, exposing them to new diseases. Domestic animals, like their human handlers, live in built environments that are intended to serve human needs (e.g., increasing milk productivity among dairy stock) much more than the needs of animals. Similarly, anthropogenic changes in physical environments can significantly affect the quality of the environment (e.g., air quality, access to food and water) from the standpoint of the health of resident animals. In other words, whatever the natural social patterns of animal species, human social structures and economies are a factor in disease interactions that significantly affect animal health, even in wild populations.

In the end, it is probably not productive to draw too fine a conceptual line separating human from non-human animal species (or human and non-human animal diseases). The tendency historically (e.g., during the early period of animal domestication and resulting intensified cross-species interaction) has been for pathogens to jump across species boundaries. More recently, at a newly accelerated pace (as a result of environment change, human penetration of new ecozones, and the globalization of wild animals as commodities), pathogens have been successful in moving from animal to human hosts (and vice-versa). Diverse animal populations are known to serve as reservoirs of many current and (potential) future human infections, suggesting why it is ultimately impossible, as was once hoped, to eliminate all contagious human diseases. Even if a wild pathogen is eliminated from every human population (e.g., as occurred with *Variola major* and *V. minor*, the microbial causes of small pox), other pathogens will continue to leap from animals to humans, some of which will adapt to the cells and structures of their new human hosts, and trigger infections never seen before by the human immune system (Fauci, 2005). Thus while small pox may be gone (from everywhere but the laboratory) the monkeypox virus, a member of the same group of viruses (i.e., orthopoxviruses), appeared in the Midwestern U.S. in 2003 as an emergent source of human disease (i.e., the first detection of this ailment in a new geographic area, the Western Hemisphere). A number of people contracted monkeypox that year following contact with infected pet prairie dogs (probably as a result of the exposure of pet trade prairie dogs to an imported exotic animal). While causing self-limiting febrile rash in some patients, monkeypox produced severe neurologic infection in others (Sejvar et al., 2004).

In Central and West Africa, there are a large number of rodents, as well as some non-human primate species, that serve as animal reservoirs of monkeypox. Discovered in 1958 (in laboratory monkeys), the first human cases of infection with this virus were reported in the early 1970s. In 1996, 71 cases of monkeypox, including six deaths, occurred in 13 villages in the Kasai-oriental region of the Democratic Republic of the Congo. Since then, reports of monkeypox virus infections in humans have escalated, including

outbreaks involving direct human-to-human transmission (Parker, Nuara, Buller, & Schultz, 2007).

One health

This discussion lends strong support to the importance, stressed by the authors of *Animal-Human Connections*, of paying simultaneous and unified ("One Health") attention to the borderlines of animal and human diseases, including the close monitoring of outbreaks (and even the seemingly benign spread) of animal diseases as a means of protecting human populations at potential risk for emergent infectious diseases of animal origin (and vice-versa). Of 14,000 known infectious organisms in the world, 600 are shared between humans and various animal species.

In their discussion, the authors appropriately cite the case of HIV, a disease that in its various simian forms (SIV) is widespread among non-human primates but is most virulent when it makes the adaptive jump from a traditional to a new host species. As a consequence of the spread of this primate retrovirus to humans, at least 32 million people have died thus far (commonly as a consequence of syndemic interaction with other diseases and health conditions). Recent research (Worobey et al., 2008) suggests that the first HIV infection of humans occurred as early as the mid-1880s. Worobey et al. indicate that the city of Kinshasa was a critical site for the emergence of this new human disease, although the precise pathway taken from non-human primates to humans remains uncertain. Settled as a European trading post in 1881 by Henry Morton Stanley, Kinshasa (then called Léopoldville after King Léopold II of Belgium) became a vital transshipment center for the movement of diverse resources extracted from central Africa to colonial Belgium. As a result, the city from its origin was a mixing site of peoples, products, and pathogens. From this seeming backwater of world development, HIV/AIDS grew into the first modern global pandemic.

Significantly, what is sometimes referred to as the second global pandemic, SARS, also is of animal origin. Since the appearance of the first cases of SARS in 2003, there have been intensive efforts focused on identifying the animal source of the SARS-corona virsus (Vijaykrishna et al., 2007). Research in China linked this pathogen to palm civet cats (a relative of the mongoose), raccoon dogs, and ferret badgers sold in local food markets in the southern Chinese city of Guongdong. At the winding open-air Qingping market, for example, it had long been possible to find a wide variety of mammals, birds, and amphibians on sale and readily accessible to roaming shoppers. Subsequently, horseshoe bats of the genus *Rhinolophus* were identified as a natural reservoir of SARS-like coronaviruses. Vijaykrishna et al. (2007, p. 426) concluded that "bats are likely the natural hosts for all presently known coronavirus lineages and that all coronaviruses recognized in other species were derived from viruses residing in bats."

The role of human social behavior in the spread of animal diseases to human populations is seen as well in the case of another new pathogen call Nipah, an organism named after the region in Malaysia where it was first discovered (Chua et al., 2000). This occurred as the result of a significant yet puzzling outbreak of encephalitis and respiratory illness between September 1998 and April 1999. Epidemiologic investigation identified the cause as a previously unknown virus. During the initial disease wave, over 250 people were diagnosed, of which 40 percent died. Nipah virus was also found to cause relapse encephalitis. One Malaysian patient, for example, developed a renewed case of encephalitis over four years after his initial infection. He, like almost all of the early victims, was directly involved in the raising or butchering of pigs. Control of the infectious outbreak ultimately required the massive slaughter of pigs at considerable economic loss. Subsequently a small epidemic occurred in Singapore among individuals involved in handled pigs imported from Malaysia. Outbreaks have since occurred in Bangladesh and neighboring parts of India. Ultimately, it was determined that the Nipah virus was related to another pathogen responsible for several outbreaks in Australia, where it was at first called the equine morbillivirus and later the Hendra virus after the town where it first appeared (Chua, 2003). Among the victims of this virus was a famous horse trainer named Vic Rail. In fact, people who fell ill to Hendra infection all had close contact with horses that also sickened and later died. Both the Nipah and Hendra were eventually determined to be members of the *Paramyxoviridae* family of viruses.

It is now believed that certain species of "flying foxes" (pteropid fruit bats) are the natural hosts of both the Nipah and Hendra viruses. These bats are found across a wide area encompassing parts of Australia, Indonesia, Malaysia, the Philippines, and some of the Pacific Islands. While it is not yet fully understood how the virus is transmitted from bats to domestic animals or from animals to humans, research by Chua et al. (2002) found that reductions in the availability of flowering and fruiting forest trees used for foraging by fruit bats led to the encroachment of the bats into cultivated fruit orchards.

Noting various bio-sociocultural interactions that could account for the spread of this disease to humans, Chua (2003, p. 265) concluded that available evidence suggests "that climatic and anthropogenic driven ecological changes coupled with the location of piggeries in orchards allowed the spill-over of this novel paramyxovirus from its reservoir host to the domestic pigs and ultimately to humans and other animals."

The deadly dozen

The fundamental importance of zoonotic diseases in human health is reflected in the fact that the major human emerging infectious diseases in recent years had their origin in interspecies transmission from animals. Recently, at the International Union for the Conservation of Nature's World Conservation Congress (WCS) in Barcelona, the Wildlife Conservation Society (2008) identified twelve potentially lethal diseases of animal origin that could spread through human societies around the world because of global warming. Environmental changes tied to the build up of greenhouse gases blanketing the Earth's atmosphere are changing animal migration patterns and fostering their occupation of new areas. With mobile animal species go their diseases, including those already known to have adapted to the biochemical environments of human bodies.

Deemed "the deadly dozen," these ominous threats to human health include Lyme disease, yellow fever, plague, avian influenza, babesia, cholera, ebola, intestinal and external parasites, red tides, Rift Valley fever, sleeping sickness, and tuberculosis. In Barcelona, Wildlife Conservation Society researchers called for the establishment of a global wildlife health monitoring and education program to identify, track, and respond to early signs of disease outbreaks among animals before they spread to and infect nearby human populations.

Already, monitoring stations have been set up by the WCS in several locations with positive results in stemming animal-to-human disease transmission. For example, the WCS has created a network of hunters and other residents in local forested areas of the Democratic Republic of the Congo. These spotters report any sightings of gorillas or chimpanzees that appear to have died from outbreaks of Ebola. Vaccination efforts are then launched in areas where primate outbreaks are observed. Prior to the initiation of the program, hunters commonly brought dead animals back to their villages, a behavior that contributed to the spread of Ebola to

people. During the three year period since the initiation of the monitoring program, there have been no new outbreaks of the disease in the monitored areas. Similar projects have been established in Brazil, where researchers are gathering data on the spread of yellow fever as a consequence of global warming. At the same time, the Global Avian Influenza Network for Surveillance (GAINS) has developed a program to tap local ornithological knowledge and experience as a way of monitoring wild bird populations for signs of highly pathogenic avian influenza in over 20 countries in South America, Africa, Asia, and the Russian Federation. The aims of GAINS are to identify the location of avian influenza viral strains, analyze genetic changes in viral isolates, link information on the strains to wild bird distribution and migration, and provide an early warning system for global spread of avian influenza from wild birds to domestic poultry to humans. According to William Karesh (quoted in Wildlife Conservation Society 2008), Vice President and Director of WCS' Global Health Program:

> "The monitoring of wildlife health provides us with a sensitive and quantitative means of detecting changes in the environment. Without wildlife, we may not see what's coming until a crisis has occurred. Wildlife monitoring provides a new lens to see what is changing around us to help governments, world health agencies, and regional communities detect threats and mitigate them before they become health crises."

A new direction

In short, contemporary disciplinary and conceptual separation of human and animal diseases is ill-conceived; as the authors emphasize, "To redress health risks in relation to animal-human connections, new ways of thinking and working are required." The new direction called for by the authors would require multidisciplinary collaboration among biomedical, veterinary, public health, and social scientists, a turn fraught with challenges. Syndemics, however, involve both biological and social components, and, as the authors note, commonly involve animal diseases that are spreading to human populations because of anthropogenic impact on the environment. To borrow Kirmayer's (2003, p. 283) apt terminology, syndemics research "requires multiple languages of description" and analysis.

Yet the possibility of multidisciplinarity—the successful collaboration across not only disciplinary boundaries but conceptual worlds (and languages), and perhaps even more importantly, our ability to overcome the blinders imposed by disciplinary bias and discipline-centrism (as reflected in terms like "hard vs. soft sciences")—is far from assured. With reference to their respective disciplines, Richard Cone and Emily Martin (2003, p. 232), for example, ask: "Is collaboration between a biologist and a cultural anthropologist possible today? Would bringing insights from biological science and cultural studies together produce a synergy that scholars on both sides would find enlightening?" To the degree that the shared goal across disciplines is the promotion of health, the very nature of looming syndemics in a rapidly changing world, one in which complex threats to health grow with every passing day, is there any other road to success?

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