

Reconnecting habitat will have consequences, in many forms and on many scales. The trick is to recognize these consequences, the negative ones as well as the positive, and understand them ahead of time, and in doing so to insure that human-engineered reconnectedness does the least harm and the most good.

# INFORMED DECISIONS

## Conservation Corridors and the



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**T**HE IDEA THAT HUMAN travel and migration play a crucial role in spreading infectious diseases has, finally and permanently, set up shop in our modern consciousness. Numerous books—*The Coming Plague*, *Secret Agents: the Menace of Emerging Infectious Disease*, *The Hot Zone*, and even Hollywood movies (*Outbreak*, based on the latter volume)—have driven home the message that we

to staunch the flood of global species extinctions, questions of how travel—the reintroduction, migration, and translocation of animals, both inadvertent and deliberate—and non-human animal diseases interact remain largely unanswered.

Why these questions remain unanswered is complex. The problem is due in part to the limited pool of money available to fund research on wildlife diseases, in part to insufficient attention being paid to wildlife disease ecology, and in part to internecine disagreements over

# Spread of Infectious Disease

are all, like Pigpen with his cloud of dust, trailing behind us a miasma of bacteria and viruses.

We may have first been awoken to the relation between travel and disease as far back as the early 1980s when AIDS epidemiologists searching to uncover a “patient zero” discovered that the Typhoid Mary of his day was an airline steward. Thus, through the steady accretion of many such events have travel and the spread of human disease come to be inexorably linked in our minds.

Then again, maybe the ideas of travel and infection were always somehow intertwined. In the fifteenth century, long before the fundamentals of germ theory and disease transmission wafted into the realm of science, European towns often preemptively closed their gates to wayfarers in hopes that the town could be spared an outbreak of bubonic plague. Maybe the idea just took a while to really sink in.

There are those, however, who would argue that when it comes to diseases of non-human animals, the idea has yet to sink in, or at least not sufficiently. For example, despite the popularity of wildlife corridors as a strategy

how, or even whether, disease transmission might affect corridor success. Another factor may be fear in the conservation community of openly discussing and examining the potential negative effects of reconnecting habitat. The admission that there could be any downsides to connectivity might be used by those who benefit financially as an excuse to continue prioritizing human land use and development without considering conservation concerns.

Few conservationists, however, would argue with the statement that unforeseen human-mediated pathogen contaminations—what Peter Daszak, Director of the Consortium for Conservation Medicine terms “pathogen pollution”—have been disastrously affecting biodiversity and species conservation since the late 1800s, and probably long before that. One of the most notorious instances of pathogen pollution was first recognized in 1889, when cattle rinderpest devastated native ungulates in East and Southern Africa. Although rinderpest spread via a so-called “natural” corridor, there is no reason to think that human-designed wildlife corridors could not function in this capacity.

**By  
Leslie Bienen**  
a freelance writer,  
veterinarian, and wildlife  
researcher living in  
Missoula, Montana.

Today, no one would disagree, at least in principle, that moving animals around blindly, or moving *anything*, sentient or insentient, around blindly—be it ballast water, rubber tires with mosquito larvae, or cattle carrying rinderpest—is a bad idea. Clearly, the concept that we ought to know just what it is we are letting in or out before we throw open the town gates is,

Wildlife Trust, Harvard Medical School's Center for Health & the Global Environment, and the USGS National Wildlife Health Center (NWHC).

The database was originally the brainchild of Gary Tabor, one of the pioneers of conservation medicine who was the founding director of Tufts CCM. Tabor had always seen one of

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like most things under the sun, no longer new.

**W**hat, then, is to be done? Why is it that although the effect of rinderpest on wild ungulates was well understood more than one hundred years ago, as late as 1986 24 percent of terrestrial vertebrate translocations “occurred without any disease screening, and fewer than 25 percent involved investigations into causes of death of translocated animals”? (1) Whether or not those numbers have improved appreciably in the intervening 15 years is anybody's guess.

The above questions and statistics underscore a two-fold problem: first, we lack comprehensive data on wildlife diseases; and second, existing data are not currently compiled and analyzed in a way that is conducive to making informed conservation decisions. Both of these phenomena are driving forces behind Dr. Colin Gillin's research.

For the past five years, Gillin, a veterinarian with a Master's degree in zoology and physiology from the University of Wyoming, has been developing a large-scale, multispecies database of occurrences of wildlife diseases. The database is a multi-institutional collaborative effort; Gillin is funded by the National Fish and Wildlife Foundation, the Fanwood Foundation, the Wilburforce Foundation, and the V. Kann Rasmussen Foundation, and his research is housed at Tufts University Veterinary School's Center for Conservation Medicine (CCM), one of four institutions that form the Consortium for Conservation Medicine. The other three are

the center's missions as expanding the role of disease ecology in conservation decisions. He and Gillin refined the basic concept, adding a GIS mapping component, and the database came into being.

## The Consequences of Connectivity

The big-picture ideological scaffold of the project runs as follows: reversing the trend of habitat fragmentation as much as possible is necessary to conserve biodiversity, but disease can spread through corridors designed for this purpose. Therefore, as Tabor put it, “The design and management of conservation reserves need to be open to the consequences of disease threats—not as a marginal issue of concern, which is the case at this moment in time, but as one more central in achieving long-term conservation success.” (2)

Gillin and Tabor's concept is new in degree, if not in kind, because its underlying philosophy is that by failing to integrate disease ecology into *all* of our thinking about preserving biodiversity, particularly in regard to wildlife corridors, conservation policy has gotten behind the eight ball, potentially with disastrous consequences.

It is undoubtedly true, as many scientists have pointed out, that wildlife diseases have not been forefronted as a conservation problem because we have not sufficiently elevated them as

a priority. There is a circularity to this logic, however, which fails to take into account the underlying reasons that contribute to the so-far minimal role of disease ecology in corridor design. One such factor may be that diseases are, at least initially, mostly invisible to the naked eye. Research attempting to assess corridor success primarily has focused on examining and analyzing aspects of habitat and habitat use that are easily visible—roads, traffic, habitat type, habitat loss, land use, animal use, human density, and numerous others.

It is possible that pathogen pollution ultimately may have as great or greater an effect on the success of corridors as do their more immediately obvious features. In a worst-case scenario, reconnecting fragmented habitat could cause pathogen invasions that would result in a net negative effect for species conservation. George Hess's models of animal movement and disease spread show that under certain circumstances not only could corridors fail to help the situation but that species extinctions could increase as a result. Hess wrote in *Ecology* "The essential message of my work is that increasing the movement of individuals among populations is a

conservation strategy that may carry a greater risk than is recognized commonly." (3)

Beier and Noss, in an article in *Conservation Biology*, responded to those they term "corridor skeptics" by charging that "those who would destroy the last remnants of connectivity should bear the burden of proving that corridor destruction will not harm target populations" and concluded that "empirical evidence of negative impacts from corridors designed or preserved for conservation purposes has not yet emerged." (4)

So far, it appears that corridors specifically designed to enhance connectivity have yet to cause a catastrophic spread of disease. It may also be true that corridors are causing disease transmission at as yet undetected levels. Since we do not know the baseline disease levels present in most populations of wildlife, currently researchers would be unable to recognize transmission of diseases in forms other than large-scale and simultaneous mortality. Therefore, it is virtually impossible to prove or disprove whether reconnecting fragmented habitat has caused or will cause negative effects due to disease transmission.

## Rinderpest

# A Global Pandemic

**R**inderpest, a disease related to measles, was once the most dreaded livestock scourge on earth and the most fatal disease of wild artiodactyls. In the first well-documented panzootic (lasting from 1889-1898), the virus spread far and fast, from East Africa to the Cape in Southern Africa—a distance of 3,000 miles—by 1896. The virus also reached the west African coast and Europe, where the latter continent's first veterinary schools were established to deal with the epidemic.

In Africa, the losses in cattle and wildlife were enormous, though difficult to estimate because of the vast distances involved. Buffalo, warthog, eland, kudu, giraffe, bongo, bushpigs, and wildebeest were among the worst hit, with mortalities in some species of up to 90 percent. In certain areas, bongo and kudu may have never recovered. Pastoralists, who rely heavily on cattle, starved in great numbers. Mortality was so high the disease burned itself out at the turn of the century, only to reappear repeatedly and with a vengeance around WWI. Many of East and southern Africa's game parks were established on the basis that segregation of wildlife would help stop

the exchange of the virus between cattle and wildlife. Draconian and expensive measures were taken in the 1930s and 40s, including attempted elimination of game on the Rhodesian border and, in 1941, construction of a game-proof 167-mile-long fence between lakes Tanganyika and Nyasa, with a 25-mile "game-free" strip on either side of the fence. Within this zone, any game could be killed without license, and estimates made at the time put the number of animals destroyed at about 10,000. Soon, there were few animals within the fenced area, and the shooting patrols became economically unrewarding in the late 1940s. However, none of these measures worked completely and—for reasons that remain unclear—the virus was not controlled until the 1960s.

**Sources:** 1. Plowright, W. 1982. The effects of rinderpest and rinderpest control on wildlife in Africa. In Edwards, M.A and U. McDonnell (eds.) *Animal Disease in Relation to Animal Conservation: The Proceedings of a Symposium held at the Zoological Society of London*. Academic Press Inc., London; 2. Vaughan-Jones, T. 1953. Notes on the rinderpest fence and cordon. *Bulletin Epizootic. Diseases of Africa* 1:286-290.

## Making the Invisible Visible

The problem, then, in looking at corridors and disease (and disease monitoring in general) becomes how to make pathogens and their consequences visible to us before we notice them as die-offs, when it's too late. In a paper in the *Journal of Zoo and Wildlife Medicine*, Anna Lyles and Andrew Dobson criticized the propensity of conservation biologists to “ignore disease concerns or, at most, treat outbreaks of disease as catastrophes that occur at random.”<sup>(5)</sup> They try to dismantle the road block that this type of thinking sets up by stating, “Yet, both empirical data and theoretical models have established that the dynamics of pathogens and their hosts can be understood and managed using deterministic mathematical models.” This latter state-

diseases had to meet at least one, and preferably more than one, of three criteria. Each disease had to be: 1) detrimental to wildlife, 2) a zoonosis (a disease transmissible from animals to humans, or 3) important to livestock. On that basis, Gillin selected the following diseases and hosts as his starting point: bovine tuberculosis in wild and ranched ungulates, sylvatic plague in rodents, brucellosis in elk and bison, Sin Nombre virus in deer mice (known as hantavirus pulmonary syndrome when it occurs in humans), and chronic wasting disease in elk and deer.

The database was set up to draw from three sources: data on wildlife diseases gathered by U.S. state and federal agencies and the Canadian government, data being generated by university research, and data meant to fill in gaps not addressed by the first two, which Gillin and

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ment, particularly the words “can be understood and managed,” points to the heart of the matter and indicates where Gillin’s database becomes crucial.

There are undoubtedly individuals scattered around the ranks of conservation science who have a good understanding of the dynamics of pathogens and their hosts. But at this stage in the evolution of conservation policy, for the reasons discussed above and many others, relationships between hosts and pathogens are not usually considered in the planning and implementation of wildlife corridors *before* those corridors are chosen and opened. Gillin hopes his database, and eventually the resulting models based on actual disease data, will speed up this transition from theory to application so that both disease data and concepts of disease ecology will truly become part of our everyday conservation decisions.

To make the job of developing this database slightly less Herculean, Gillin and Tabor began by limiting the data to five diseases. The

veterinary students working under his supervision are collecting. Even casting as broad a net as this to gather data cannot dispense with the problem that wildlife disease surveillance is inadequate in scope and in geographic coverage. However, the existence of a central clearinghouse that merges many discrete data bundles should help overcome some of the challenges that small data sets engender. Hopefully at some future point, an increased interest in and understanding of the importance of wildlife disease ecology will translate into more comprehensive and geographically widespread disease surveillance.

Gillin is uniquely qualified to take on the sometimes forbidding task of getting data from agencies because for the decade prior to enrolling in veterinary school, he worked as a biologist for Wyoming Game and Fish Department. He is therefore well apprised of the fact that whereas state and federal agencies often hold the most thorough and up-to-date data, and sometimes the only data, that information

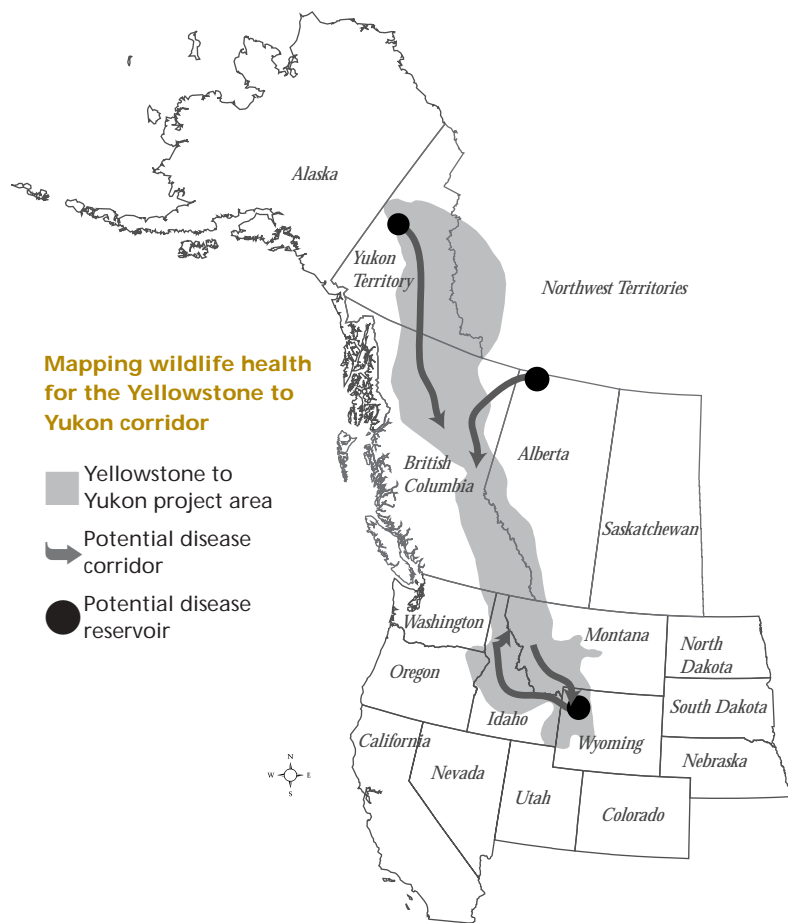
can be time-consuming and cumbersome to track down.

The database also tracks and maps human occurrences of plague, tuberculosis, hantavirus, and brucellosis using data from the Centers for Disease Control and other human public health agencies. Human health data are included so that the relationships between wildlife diseases and human health can be taken into consideration. All the information in the database will eventually be given back to agencies in a common format that crosses jurisdictional boundaries. Information that is not publication-sensitive or in violation of laws governing the privacy of human health data will be made available in a public domain such as the Internet and will be linked back to the providing sources.

## Brucellosis: A Case in Point

Brucellosis is one of the diseases in Gillin's database that receives a great deal of attention. In a sense, the disease sets up a crucible of competing interests among conservationists, agriculturalists, and the general public. For various reasons, the disease falls under the purview of many different agencies: the U.S. Department of Agriculture, several state wildlife agencies, state departments of livestock, the National Park Service, and the U.S. Fish and Wildlife Service. Brucellosis is a zoonosis and in humans the disease is called undulant fever. Add to this that two of the important hosts of the disease, bison and elk, are widely viewed by the public as magnificent symbols of America's wildlands and freedom, and you have a situation whose contentiousness (and at times, litigiousness) has clouded relations between the public and government land managers as well as between state and federal agencies and has helped make sound policy very difficult to formulate or implement.

Brucellosis is caused by a bacterium that currently is divided into seven species, which in turn have multiple biovars, or strains. In wildlife it is found commonly in bison, elk, and caribou. Brucellosis affects many species of livestock, as well as dogs. Cattle are the primary concern, but the disease can infect pigs, goats, and sheep.



In most species, the primary effect is abortion of fetuses, but in cattle brucellosis can also cause decreased fertility and lowered milk production.

Elk winter feeding grounds are thought to promote the spread of brucellosis, with some feed ground herds testing as high as 30-45 percent positive. (6,7) Outside of feeding grounds, brucellosis prevalence in elk is thought to be about 1-2 percent. To complicate the matter further, brucellosis vaccines have different efficacies in different species, and even the statistics concerning these efficacies are a matter of disagreement. In cattle, the vaccine is very effective and in combination with "test and slaughter" programs, brucellosis in bovines has been well controlled in the US. In wildlife, the efficacy of various vaccines is still being tested—and contested.

A great deal of the attention brucellosis receives arises from the fact that states must be certified brucellosis-free by the U.S. Department of Agriculture for meat or other cattle products to leave the state. In states that have both large cattle ranching concerns and large

concentrations of elk and bison, such as Wyoming, Montana, and Idaho, fear of losing brucellosis-free status has dictated a great deal of the management policy for wildlife species that host the disease.

This is true despite the fact that transmission of brucellosis from elk or bison to cattle has not been documented under non-experimental conditions. However, the Montana Department of Livestock's (DOL) policy to kill bison that leave Yellowstone National Park is

corridor, and Gillin anticipates using the database to examine and compare the risks and costs of spreading brucellosis via each route. This would be done by collaborating with statisticians and biomathematicians to generate models that could predict the effects of the spread of disease based on differing biotic and abiotic variables in each particular corridor. Many factors within a corridor will interact with disease transmission: for example, numbers and density of cattle, human density, quality of habitat,

hunting activities, amount of public versus private land, and others too numerous to list. Such models could also be used to help policy makers, ranchers, NGOs, and

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based on the assumption that such transmission is possible. In trying to respond to ranchers' financial concerns, the DOL has received harsh criticism from conservationists and animal rights activists. Dan Brister, the outreach coordinator for the Buffalo Field Campaign (BFC), a group of activists who station themselves at the boundary of the park every winter to protest the killing and harassing of bison, said in a recent newsletter: "These agencies are wasting 2.8 million taxpayer dollars this winter to unnecessarily slaughter America's last wild buffalo." This year Crosby, Stills, Nash and Young dedicated a concert to benefit the BFC.

Whether the brouhaha over brucellosis is based on groundless fears or sound science, at the very least it means that a wildlife corridor capable of spreading the disease could come under heavy fire. One of the priorities in reconnecting fragmented habitat in North America is to link up the Greater Yellowstone Area to the Northern Continental Divide. Sociopolitically, this corridor is a difficult one to navigate because comparatively it contains a high percentage of private land, particularly ranch land. So far, serological sampling of wildlife in the Northern Continental Divide has not revealed the presence of brucellosis, and keeping the disease from spreading farther north than Yellowstone Park will be a major consideration in reconnecting these habitats.

Several alternate routes could form this

the public—anyone with a stake or an interest in wildlife management—understand and foresee the potential costs of introducing disease, costs related to vaccinating, loss of livestock productivity, and increased management budgets for agencies.

## Living With the Consequences

Reconnecting habitat *will* have consequences, in many forms and on many scales. The trick is to recognize these consequences, the negative ones as well as the positive, and understand them ahead of time, and in doing so to insure that human-engineered reconnectedness does the least harm and the most good. Instead of our being forced to presuppose that "all else being equal, and in the absence of complete information, it is safe to assume that a connected landscape is preferable to a fragmented landscape," (1) Gillin's database and the models that arise from it will offer an alternative by helping planners understand the long- and short-term ramifications, and the costs, of disease transmission due to the use of a particular corridor. In this way, Gillin's work will also help bridge the ideological gap between corridor boosters and corridor skeptics, at least when it comes to disease transmission. The first step, however, is collecting

and mapping the data, which Gillin is planning to do in the next several years.

Though Gillin's efforts have so far focused on disease in the context of corridors, his work has many other implications. The number of captive animals released into the wild every year seems to be growing, though no one knows exactly what these numbers are. (8) Reintroductions of captive-bred endangered species, as well as accidental releases of exotic pets or animals from game farms, have posed many questions about the spread of disease in all directions, from introduced animals to surrounding species and from the surroundings to introduced animals. Many releases have failed or have been compromised by insufficient understanding of disease transmission and ecology. For example, reintroductions of the whooping crane in Florida and Idaho were adversely affected by the cranes' infection with avian tuberculosis. (8)

Joshua Dein and Kathryn Converse, at the National Wildlife Health Center, are calling for "a national database and monitoring program for propagation, introduction and translocation programs" (8); presumably, models similar to those described above would be of great use in deciding where and how to reintroduce captive animals. The U.S. Fish and Wildlife Service has undertaken disease ecology studies to identify productive habitat to set aside as preserves for Hawaii's endangered birds. And, hopefully, the increasing use of molecular-level work using gene flow to assess corridors will help shift the focus from primarily tangible factors to a broader understanding of corridor success that more often includes disease studies. Mary Poss's work at the University of Montana manages to combine issues of assessing connectivity and assessing disease by looking at the evolution of viruses as a way to measure connectivity.

**A**s far as Gillin knows, no one has yet devised a model quite like what he is proposing, using real disease data to examine and tease out the interactions between infectious diseases of wildlife and elements of connectivity and land use. In the context of human health, some models looking at interactions between environmental factors and the spread of HIV and tuberculosis have been generated and they will be helpful as reference

points. When the models Gillin has in mind are a reality, debates over corridors shouldn't have to take place on the level of "should we or shouldn't we?" and policy decisions shouldn't have to be made on the basis of fears and assumptions rather than on epidemiological data.

A decade ago, then-director of the National Wildlife Health Center, Milton Friend, said in an article discussing wildlife diseases as a conservation problem, "if we don't change our way of looking at wildlife and habitat, we will end up spending millions of [conservation dollars] for naught." (9) This is more true today than ever, in the United States and in every other country that is attempting to manage its biodiversity, when conservation dollars are facing fierce competition from a multitude of global problems. If all goes according to plan and Gillin's database is used the way he envisions it, his work will help those changes take place, changes that have been a long time coming and are long overdue. ♡

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