hundreds of partners and multiple government agencies and nonprofits, and costing hundreds of millions of dollars. (In the United States, such regional-scale projects include the Everglades, the California Bay delta, Lake Tahoe, and the Mississippi delta.)

Community involvement and volunteerism are hallmarks of ecological restoration. These efforts often include opportunities for assessing and informing public perceptions of natives, exotics, landscape, and "wilderness." Community participation in restoration activities can offer occasion for hands-on environmental education, as well as instill a sense of local land stewardship.

A substantial proportion of restoration projects are the result of legally mandated mitigation for development that has as its (largely unsubstantiated) underlying assumption that losses of species or habitats at one site can be recouped through restoration of another site.

INVASIVE SPECIES AND RESTORATION

In many sites, invasive species are one of the major sources of ecosystem degradation, and their control is often one of the primary goals of (and challenges to) restoration efforts. Invasive species can sometimes completely preempt successional regeneration, leading to the formation of a highly invaded stable community state. Invasive plant (and animal) species can be a degradative force in their own right, whose initial control is a prerequisite for restoration, and whose long-term control often requires the restoration of a native plant community that is resistant to further invasion (Fig. 1). In highly invaded sites like island ecosystems or the western grasslands of the United Sates, it has been said that there can be no successful restoration without effective weed control, and there can be no effective weed control without successful restoration. Conversely, in some ecosystems, nonnative species can be used to assist in restoration.

SEE ALSO THE FOLLOWING ARTICLES

Endangered and Threatened Species / Fire Regimes / Grasses and Forbs / Herbicides / Hydrology / Land Use / Mechanical Control / Succession

FURTHER READING

Baker, J. P. 2005. Vegetation conservation, management and restoration (309–331). In E. van der Maarel, ed. *Vegetation Ecology*. Malden, MA: Blackwell.

Ewel, J. J., and F. E. Putz. 2004. A place for alien species in ecosystem restoration. *Frontiers in Ecology and the Environment* 2: 354–360.

Falk, D. A., M. A. Palmer, and J. B. Zedler. 2006. *Foundations of Restoration Ecology*. Washington, DC: Island Press.

Hobbs, R. J. 2007. Setting effective and realistic restoration goals: Key directions for research. *Restoration Ecology* 15: 354–357.

Society for Ecological Restoration International. www.ser.org/

Van Andel, J., and J. Aronson. 2006. *Restoration Ecology*. Oxford: Blackwell.

Young, T. P., D. A. Petersen, and J. J. Clary. 2005. The ecology of restoration: Historical links, emerging issues, and unexplored realms. *Ecology Letters* 8: 662–673.

RINDERPEST

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Rinderpest is a virus in the Morbillivirus family that causes disease in cattle; it created the largest pandemic ever recorded when it was introduced into East Africa in the early 1890s. Its invasion and spread caused the deaths of around 50 to 90 percent of cattle and wild artiodactyl species (wildebeest, buffalo, giraffe) in sub-Saharan Africa. The loss of hosts for tsetse flies created a human epidemic of sleeping sickness throughout sub-Saharan Africa. Rinderpest is closely related to human measles virus and canine distemper virus; control was only achieved once methods used to develop a measles vaccine were applied to rinderpest in the early 1960s.

INTRODUCTION HISTORY IN AFRICA

Africa has been afflicted by many disasters in the relatively short period for which we have historical records: droughts, dictators, deforestation, swarms of locusts, and of course disease. Although over 20 million Africans are currently infected with the HIV virus that causes AIDS, and several thousand children die each day from malaria, arguably the worst disaster ever to have hit the African continent was the great rinderpest pandemic that started in 1889. In the ten years of its initial spread from the coast of Ethiopia in the Horn of the Africa until its arrival in Cape Town, it is estimated to have killed 80 to 90 percent of the cattle population and similar proportions of many artiodactyl species (wildebeest, giraffe, and particularly buffalo). These losses essentially devastated the protein supply for most of the human population of sub-Saharan Africa while simultaneously triggering an epidemic of sleeping sickness when starved tsetse flies switched to human hosts in the absence of their natural hosts.

Rinderpest provides detailed and important insights into the impacts that pathogens can have when they

invade host populations in locations that have no prior experience of them. While the population dynamics of rinderpest are, at first sight, deceptively simple, there is ultimately a significant level of subtle complexity that sharply echoes that seen for measles. For instance, it was quickly realized that early measles models that divided the host population into susceptible, infected, and recovered classes required more details about how transmission was driven, for example by mixing rates among age classes and by spatial heterogeneity across the landscape in the host population. These pathogens thus provide an important set of insights into studies of invasive species and conservation of endangered species as pathogen dynamics are inherently those of other species distributed as metapopulations, because each host is, in effect, a habitat patch that can be colonized (causing infection) or can experience local extinction (disease clearance or host death). Ultimately, the invasion success of any pathogen and the efficacy of its control require on an understanding of how it manages to persist in the patches of habitat that are its hosts.

THE MORBILLIVIRUS FAMILY

Rinderpest (RPV) is likely the basal species in a Morbillivirus family. It is a single-stranded RNA paramyxovirus and is the most likely ancestral species for canine distemper in dogs (CDV), peste des petites ruminants in goats and gazelles (PPRV), and measles in humans (MV). The trifurcation in the evolutionary split between RPV, CDV, and MV is too poorly resolved to accurately determine the order with which these pathogens spread into their major classes of hosts. It seems likely that either rinderpest's association with cattle predates the domestication of aurochs or that CDV's association with dogs predates dog domestication. Whichever is the case, the domestication of cattle and dogs allowed the ancestral morbillivirus to jump between domestic species and humans and gave rise to three viruses that have had major impacts on the welfare and abundance of humans, canids, cattle, and goats. There is no other closely related group of pathogens with a comparative impact on humans and their domestic livestock. The symptoms of infection are initially subtle and are often missed by farmers and herders, thus allowing the pathogen to be transmitted before intervention can occur. After several days, infected animals stop feeding, become dehydrated, and have difficulty breathing. Soon thereafter, open sores appear around the mouth and nasal passages. These manifestations are rapidly followed by diarrhea, further dehydration, and eventual death. Mortality rates within herds range from 30 to 90 percent, causing the pathogen to have devastating effects whenever outbreaks occur.

DYNAMICS OF RINDERPEST

The population dynamics of rinderpest are inherently similar to those of measles, arguably the pathogen for which we have developed the deepest quantitative understanding, so it is worth briefly considering the population dynamics of measles for insights it provides into the dynamics of rinderpest. In the text that follows, one could readily replace the word "measles" with "rinderpest," as broadly similar things will happen at both the individual and population levels when rinderpest infects cattle and measles infects humans. Indeed, were measles or rinderpest to resurge and create an epidemic, it would be perfectly possible to use the human measles vaccine to protect susceptible cattle, and vice versa! There is almost perfect cross-immunity between the vaccines, as the pathogens are essentially indistinguishable to the immune system.

Early work on the dynamics of measles identified a key epidemiological principle: measles was able to persist only in a population of greater than half a million people. This pattern was consistent in relatively isolated human populations living on oceanic islands, as well as those living in cities that were connected to other cities by emerging rail and road networks. In these larger cities, the birth rate was high enough to sustain an input of susceptible hosts into the population, thereby maintaining a constant chain of infection for the currently infected individuals in the population. Constant chains of infection are required by all morbilliviruses if they are to persist in the host population, and this will occur only if the population is larger than a "critical community size." As individuals who have recovered from measles are immunologically resistant for the rest of their lives, new susceptible hosts can enter the population only by birth or by immigration. Newborn individuals are said to have maternal immunity if their mothers had previously been infected and were placed in the resistant class of hosts that maintain antibodies protecting them from further infection. However, maternal immunity is lost after a period of around six months, so all infants will from then on become susceptible to infection. When these individuals contact infected hosts, they are likely to become infected themselves and enter a period a period of infectiousness, during which they either die from the infection or, more usually and if well nourished, recover and enter the immunologically resistant class.

The dynamics of measles, rinderpest, and all the other morbilliviruses can thus be well described by dividing the host population into four classes of hosts: susceptible,

infected, resistant, and with transient maternal immunity; we designate their abundances as S, I, R, and M, respectively. This leads to a set of four coupled differential equations that describe the dynamics of a morbillivirus infection in a well-mixed host population.

$$dM/dt = bR - (\delta + d)M$$
$$dS/dt = bS + \delta M - dS - \beta SI$$
$$dI/dt = \beta SI - (\alpha + d + \sigma)I$$
$$dR/dt = \sigma I - dR$$

Here, $S + I + R = N$, the total size of the adult population, and maternal immunity is lost at a rate δ (whence newborn hosts are, on average, protected for a period of $1/\delta$ years). The population gives birth at a rate b and dies at a rate d, and it is assumed that infected individuals are too ill to give birth. Transmission occurs at a rate β between infectious and susceptible hosts, and individuals are infectious for a period of $1/\sigma$ years. To regulate the host, we could assume direct density dependence in, say, births (details are not explicitly shown in the above equations). The properties of this model have been exhaustively explored in the mathematical epidemiology literature; these properties provide a number of important insights into the dynamics of many viral pathogens. As mentioned above, a central property of pathogens whose dynamics can be described by this model structure is that the host population size needs to be large enough to allow the pathogen to persist. A slightly more subtle consequence is that the average age of infection will be coupled to the size of the population. Thus, in large and aggregated host populations, transmission rates will be high, and average age of infection will be skewed toward the younger individuals who have recently lost their maternal immunity. In contrast, in host populations that are below the critical community size, the average age of infection will be higher and will depend more on contact rates between the local small population and the external populations that are themselves large enough to maintain a constant chain of infection.

The structure of the equations also reveals a second key feature of most morbillivirus infections: the relative proportions of the host population in the different classes of infection will be roughly proportional to the time each individual host spends in each class of infection: $1/\delta$, $1/(\alpha+\sigma)$, and $1/d$. As hosts are susceptible for only a couple of years in endemic areas, and infective for, at best, a couple of weeks and then immune for life, the largest proportion of the host population will be in the recovered class. There will be significantly fewer susceptible hosts, and infected hosts will be comparatively rare, often constituting less than 0.01 percent of the population. This creates an important paradox that has confounded ecologists' perspective on the importance of pathogens until recently; although infectious individuals are comparatively rare, the presence of a pathogen can significantly reduce the abundance of a host population. However, once the pathogen has established, it holds the population at a significantly lower abundance (around the local critical community size), and although the chain of transmission is maintained in the reduced host population, infected individuals will be comparatively rare. We tend only to see large numbers of infected individuals when the pathogen first invades a host population and all hosts are susceptible. Rinderpest provides one of the best examples of this; when first introduced into sub-Saharan Africa, it produced a dramatic pandemic, as all host populations were composed only of susceptible individuals, so everyone became infected at almost the same time, causing widespread disease and mortality. It is likely that some of the plagues of Egypt recorded in the Old Testament were of similar form when measles first crossed over into human populations from cattle; similar devastating epidemics were recorded when Central American Indians were introduced to measles and smallpox following the Columbian conquest.

The equations described above can be rearranged to provide an expression for the basic reproductive number of the pathogen, commonly termed R_0. This can be defined formally as the number of secondary cases produced by the first infected individual introduced into the population. A moment's thought suggests we are highly unlikely ever to witness, let alone sample and quantify, this event, so other methods are used to measure R_0 formally. Nonetheless, deriving and inspecting an algebraic expression for R_0 provides important insight into the demographic components of the host–pathogen interaction that determine the magnitude of a disease outbreak. It has also been argued that deriving expressions for R_0 for other types of invasive species might provide insights into how best to control them. The expressions for R_0 for rinderpest, measles, or distemper are essentially identical:

$$R_0 = \frac{\beta N}{(\alpha + d + \sigma)}$$

Inspection of this expression provides a key biological insight; R_0 is the rate at which an infected host contacts and successfully transmits to susceptible hosts, times the duration of time over which it is infectious. This is

arguably analogous to one measure of the fitness of a free-living organism: the number of viable offspring it produces during its reproductive lifespan (often called "lifetime reproductive success"). Conditions that create large values of R_0 will produce dramatic, large epidemics that quickly exhaust the supply of susceptible individuals and then die out. In subtle contrast, conditions that produce R_0 values that are slightly larger than unity will produce small epidemics that may persist for quite some time. One can use R_0 as a measure of pathogen fitness. However, there are tradeoffs for a virulent pathogen between optimizing its epidemic potential (a traditional perspective on "fitness," i.e., growth rate) and optimizing persistence; the latter may favor strains that are less competitive in the short run, particularly in spatially structured host and pathogen populations, permitting local extinctions and recolonizations. These processes may even interact; when a pathogen is repeatedly spilling over from a reservoir host, the initial outbreaks may be dramatic and then die out. Subsequent epidemics are less dramatic, as R_0 is reduced, owing to the presence of immunologically resistant hosts who recovered from earlier outbreaks. Persistence will now increase, and the pathogen may be able to establish a longer-term chain of transmission in the host population.

Ultimately, the principal goal of vaccination is to increase the proportion of resistant hosts in the population to levels at which the chain of transmission is broken and the pathogen dies out. Estimating the magnitude of R_0 provides an important quantitative guideline for the fraction of hosts that need to be vaccinated, quarantined, or culled in order for chains of transmission to break and cause the pathogen to die out in the host population. Vaccination and quarantine reduce the quantity β, whereas culling boosts d. The level of vaccination needs to reduce the current reproductive number for the pathogen below unity for control to be complete (i.e., the infection tends toward extirpation); these conditions are usually met when the proportion vaccinated, p_v, meets the following inequality:

$$p_v > 1 - \frac{1}{R_0}$$

Pathogens with high estimated values of R_0 require much higher levels of vaccination than do those with lower R_0 values. Two important caveats apply here: (1) this initial calculation assumes that the vaccine is administered once and lasts for the rest of the host's lifespan; this seems to be the case for measles, rinderpest, and canine distemper (although two doses of vaccine are increasingly used to boost individual levels of protection). It is not often the case for other pathogens. These either mutate rapidly and create new variants for which new vaccines must be developed (e.g., influenza), or the vaccine produces only short-term immunity, in which case hosts must be repeatedly vaccinated if eradication is the goal, and (2) while individual shots of vaccine protect the host to whom they have been administered, increasing the numbers of immunologically resistant hosts in the population creates a secondary important effect termed "herd immunity," which entails a reduced risk of infection for hosts who have been neither vaccinated nor naturally infected. In the case of measles, cultural beliefs and misgivings about possible vaccine side-effects act to reduce levels of vaccination coverage to below levels where eradication can occur. Similar problems beset rinderpest vaccination, as herders are reluctant to have cattle vaccinated at times of stress (e.g., during droughts), or when emergency vaccination schemes in response to outbreaks lead to rumors about whether the vaccine or the pathogen led to cattle deaths.

A key potential difference between rinderpest and measles is that rinderpest may be present as a pathogen in a community of different host species; this introduces a number of significant complications in the population dynamics of outbreaks. These can be reduced to a manageable scale by recognizing that rates of between-species transmission are likely to be considerably lower than rates of within-species transmission. Furthermore, rates of between-species transmission are likely to be significantly determined by levels of spatial niche overlap between potential host species. Thus, species with very different feeding niches are unlikely to transmit aerosol pathogens between each other. Similarly, if species use similar niches sequentially, as occurs in some "grazing successions," then rates of between-species transmission will be asymmetrical and will be more likely to occur from the earlier species in the succession to the successive ones, rather than vice versa. More subtly, it is likely that some group of host species will act as a reservoir for the pathogen, while others will act as spillover hosts. A key point here is that spillover hosts may play a key role in indicating the presence of background transmission in the reservoir hosts that would go undetected owing to less pronounced pathology and etiology in the reservoir hosts. This occurs with Nipah and Hendra virus, emerging pathogens that are fairly closely related to rinderpest and other morbilliviruses. Their reservoir hosts are pteropid fruit bats, which show essentially no symptoms of infection with the virus. However, when it is transmitted from

bats to horses or pigs, significant pathology and mortality are very apparent.

Rinderpest tends to show similar pathologies in most of its host species, although different strains of the virus may show significant differences in pathology that range from mild to severe. Similarly, with morbilliviruses, older individuals may exhibit much higher levels of pathology and mortality than do younger ones, who are characteristically infected in a population large enough to support the continuous presence of the pathogen. The classic studies on measles in the Faeroe Islands are instructive here; measles had been absent from this isolated group of islands for over 50 years, and when it was introduced by a passing fisherman, it created an epidemic that infected most individuals under the age of fifty; those older than this still had immunity from exposure during the previous outbreak. The mortality rates of those under fifty increased exponentially, from relatively low mortality when under the age of five to very severe mortality in those in those in their thirties and forties. The dramatic *grand mal* seizures and mass mortalities of adult humans exposed to measles observed in the Faeroes, Hawaii, and ancient Athens echo those seen when rinderpest devastated the cattle, wildebeest, and buffalo populations of sub-Saharan Africa in the 1890s. However, once the pathogen became endemic, younger animals that had survived infection created a significant level of herd immunity that slowed the rate of spread of the pathogen and lowered its average prevalence. New cases were largely restricted to young animals born in the previous 12 months; this feature led to rinderpest being known as "yearling disease" to African veterinarians and farmers. This is comparable to human populations with endemic measles, which mainly infects susceptible youngsters.

CONTROL OF RINDERPEST

Once a vaccine had been developed for measles in the 1950s, it was relatively straightforward to develop one for rinderpest, and subsequently for canine distemper. The principal motivation for developing the vaccine was to protect the cattle herds of British East Africa from recurrent outbreaks of rinderpest, which conventional wisdom dictated were the result of frequent spillovers from wildlife reservoirs in wildebeest and other wild game species. The British government even debated the seriousness of the problem, worried in part about the national cattle herd's ability to keep beef on the British Sunday dinner menu and in part about an increasing reliance on Argentinian beef, which might be cut off in a political pique if relations with Argentina were to become sufficiently strained over ownership of the Malvinas/Falkland Islands. Indeed, it might be argued that the rise of the Argentinian beef-based economy in the early twentieth century was significantly aided by the presence of rinderpest throughout the rest of the world. The absence of the pathogen and the advent of fast-traveling steamships allowed Argentina to supply much of Europe with beef at a time when rinderpest hampered production in most other cattle-producing countries.

Once the rinderpest vaccine had been developed, it was quickly deployed to protect cattle in East Africa and other parts of the world (Fig. 1). The results were quite startling; certainly the vaccine provided excellent protection and allowed vibrant and viable cattle herds to reestablish in areas of sub-Saharan Africa from which they had been largely excluded for over 60 years. Simultaneously, the disease began to disappear from wildlife—although not a single wild animal was vaccinated. It is hard to think of a better experimental way to identify the true reservoir of a pathogen!

The effects of rinderpest removal on wild host species such as wildebeest and buffalo were spectacular. The Serengeti National Park had been established in 1959 to protect the herd of around a quarter of a million wildebeest; the eradication of "yearling disease" allowed the population to increase by a factor of around six to over 1.5 million animals. Buffalo were felt to be rare or absent from the region because of their popularity as game for "sportsmen" and poachers. Rinderpest control, however, allowed buffalo numbers to increase massively, suggesting that disease, not overhunting, may have been the culprit in keeping buffalo scarce. In like manner, buffalo had been rare in Ngorongoro Crater; by the early 1970s, there

FIGURE 1 Widespread vaccination of cattle has eradicated rinderpest in the wild. In this picture Maasai cattle are vaccinated against rinderpest during an annual control campaign in the Ngorongoro highlands of Tanzania in 1991. (Photograph courtesy of Andy Dobson.)

were over 5,000 in Ngorongoro Crater and over 20,000 by the late 1990s.

IMPACT IN THE SERENGETI

The full impact of rinderpest at the ecosystem level has only recently been fully appreciated and quantified. This can be done largely as a consequence of long-term studies in and around Serengeti National Park. Removal of rinderpest by vaccination of cattle can be seen as a form of experiment that has been replicated in other areas of East Africa where roughly similar phenomena are recorded. The differences between Serengeti and other areas most likely reflect differences in the ability to prevent poaching of wild game as their numbers recover and agricultural expansion into areas in and around the national parks. The Mara region of Kenya that forms the northern extension of the Serengeti provides the ultimate "control." The migratory wildebeest use this area as grazing habitat in the summer when the southern part of the ecosystem (in Tanzania) has dried from lack of rain. While both the resident and migratory populations of wildebeest in the Mara initially increased following the eradication of rinderpest, the resident populations have now declined by as much as 95 percent following agricultural expansion around the park and the removal of river water for agriculture. Ultimately, the mantra for all who work in Serengeti is "We need to prevent the same things from happening in Tanzania that have happened around the Kenyan Mara region!"

Removal of rinderpest from the Serengeti created cascading impacts that affected the abundance and diversity of nearly all other species in the ecosystem (Fig. 2). It is again hard to think of another invasive alien for which this has been so extensively documented. This is testimony not only to the importance of long-term ecosystem studies, but also, more ominously, to the power of viral pathogens to produce dramatic impacts on their host's abundance, and thus to significantly alter the ecosystem processes that they are involved in. The removal of rinderpest provided a blunt illustration that the large-bodied herding herbivores of the Serengeti were not limited in abundance by their predators. The six- to eightfold increase in wildebeest and the huge increase in buffalo following rinderpest release led to an increase in both lions and hyenas, the principal predators of these species. This strongly suggests that, in the absence of disease, food availability is more important than predation for regulating herbivore populations in East African savannas, at least among free-living herding species. It is noteworthy that not all grazers increased in abundance. Indeed, some

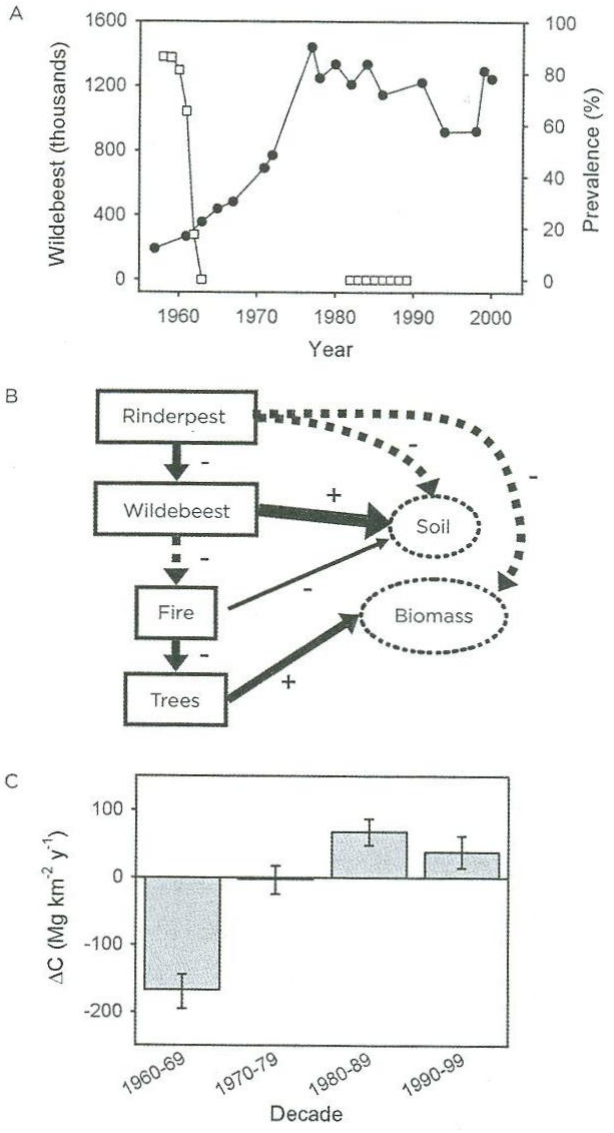

FIGURE 2 (A) Serengeti wildebeest population (•) reported for the period 1958–2000, and rinderpest seroprevalence (□) reported for the periods 1958–1963 and 1982–1989. (B) Pathways linking rinderpest with ecosystem C pools (in soils and biomass) as a result of a trophic cascade (solid and dashed lines signify direct and indirect effects, respectively). (C) Simulated annualized decadal net changes in ecosystem C balance (mean and 95% credible intervals) as a result of rinderpest eradication in the 1960s. (From Holdo et al. 2009, *PLoS Biology*.)

of the smaller grazers, particularly Thompson's gazelles, declined by around 50 percent, most likely because of increased hyena predation. There thus may be an emergent indirect interaction between rinderpest and gazelles: relaxation of rinderpest boosted wildebeest and buffalo, boosting hyena numbers, which then inflicted heavier predation upon the smaller-bodied gazelles. Curiously, the huge herds of plains zebra that comigrate with the wildebeest and exhibit considerable dietary overlap have remained at approximately the same level of abundance over at least the last 50 years. The zebra are completely

resistant to rinderpest and associate with the wildebeest, as lions and hyenas significantly prefer wildebeest to zebra as prey. Increasing wildebeest numbers permitted by the decline in rinderpest may have buffered any impact of increased predator numbers on zebra mortality, and this effect might even outweigh competition for forage with the burgeoning wildebeest population.

The changes in herbivore abundance have precipitated a trophic cascade that has significantly altered the fire regime of the Serengeti. In the early 1960s, prior to rinderpest vaccination, around 80 to 90 percent of the Serengeti burned each year, following lightning strikes during the dry season in areas with large amounts of unconsumed grass. As wildebeest numbers increased in the 1970s and 1980s, fire frequency declined to less than 25 percent, and acacia-dominated woodland and bush began to colonize the central and northern regions of the park. This recovery was also partly aided by low numbers of elephants following the ivory-poaching epidemics of the 1970s. Long-term studies of tree recruitment suggest that the Serengeti ecosystem has changed from a source of atmospheric carbon when rinderpest was present to a major carbon sink, now that rinderpest has been removed. Carbon accumulated in the trees is more than matched by carbon build-up in the soil of both the wooded and grassland areas of the park; in fact, as much as 80 percent of the carbon is stored in the soils, and carbon continues to accumulate there at a significant annual rate. It is suspected that similar processes apply in other East African national parks. This ecosystem effect should permit them to be promoted as areas where airlines and businesses could offset their carbon budgets by investing in schemes that control cattle and wildlife diseases, minimize poaching, and provide health and education facilities for people living around the parks.

A SUCCESSFUL EXTINCTION?

There are increasing reports that rinderpest has been eradicated in the wild, which would place it alongside smallpox as one of only two pathogens to be eradicated in the wild; in both cases, this was achieved by a sequential combination of mass, and then targeted, vaccinations. Celebrations are still muted, as it was previously thought that rinderpest was eradicated following the JP15 (Joint Project) African project initiated in 1962. This project focused on vaccinating all cattle for each of three years and then only calves in subsequent years. In Tanzania and Kenya, its success gave rise to the eruption of wildebeest and buffalo described above. The project seemed a great success, and by the mid-1970s, rinderpest seemed to have disappeared from each of the 22 countries involved. Unfortunately, initial signs of success led to apathy and a reduction in vaccination coverage levels, which allowed the pathogen to resurge from troubled areas along the

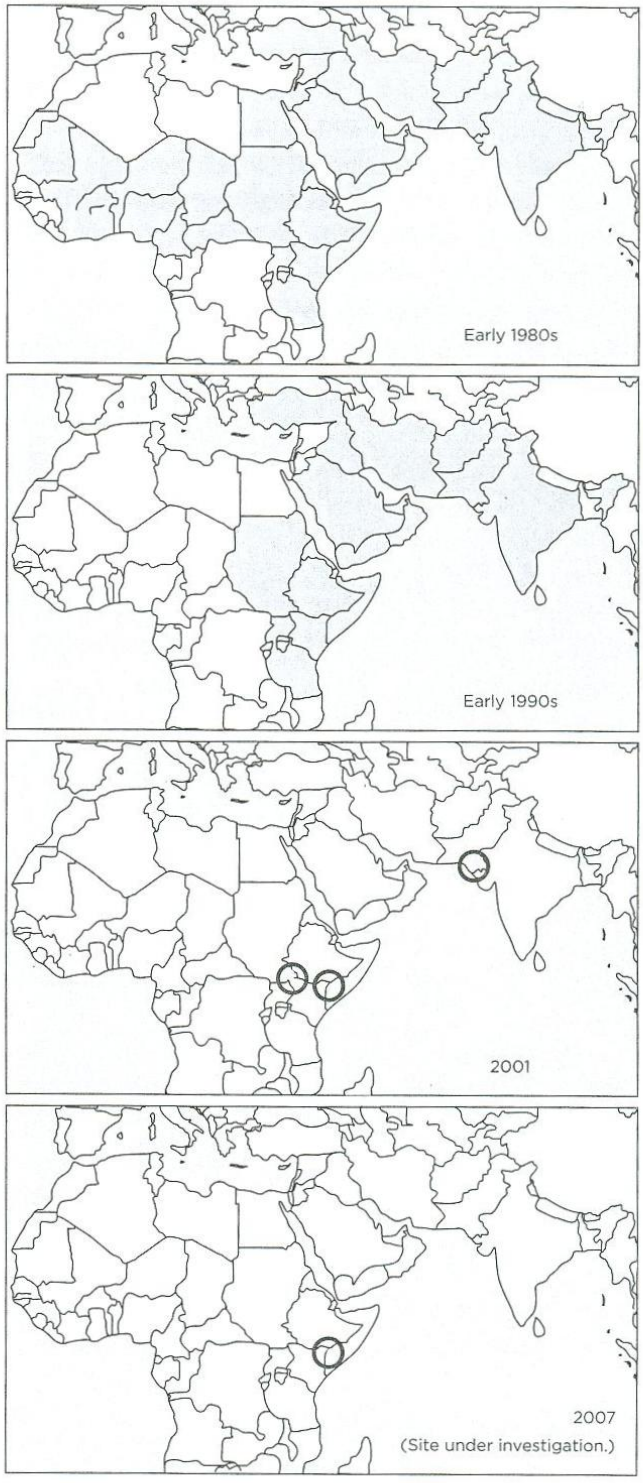

FIGURE 3 Four maps illustrating the declining geographical distribution of rinderpest in the face of mass cattle vaccination. (From Normile, 2008, *Science*.)

Mali–Mauritania border in the west and from southern Sudan in the east. Losses from this new pandemic in the early 1980s were thought to have matched those in the original pandemic, with an estimated 100 million cattle deaths and an economic cost to Nigeria alone of over $2 billion. Similar problems prevailed in areas from Turkey to Bangladesh, where persistence in metapopulations of host patches allowed rinderpest to resurge. A second eradication campaign was launched in 1987 (the Pan-African Rinderpest Campaign); it was then fairly swiftly realized that attempts to eradicate rinderpest in Africa were being undermined by a constant trickle of infected cattle from the Indian subcontinent. This realization led the Food and Agriculture Organization to patch together the Global Rinderpest Eradication Campaign in 1993, which set the goal of global rinderpest eradication by 2004, followed by a period of surveillance to ensure that the pathogen was extinct. At present, it seems to have accomplished these goals (Fig. 3).

RINDERPEST AS AN INVASIVE SPECIES

Rinderpest has run the full gamut of possibilities as an invasive species: it has caused massive changes in ecosystems on several continents, has had major effects on human health, and has caused major economic disruption, yet ultimately it provides one of the very few examples of global control and eradication. Its impact on cattle and wild artiodactyls was largely driven by its ability to use domestic livestock as a reservoir and then repeatedly spill over into wildlife populations that were insufficiently large to maintain continuous chains of infection. This case study of rinderpest as an invasive species on one hand illustrates the importance of community interactions in determining the spread and control of invasions and, on the other, exemplifies the potential amplification of impacts of invasive species due to interspecific interactions and ecosystem processes.

SEE ALSO THE FOLLOWING ARTICLES

Epidemiology and Dispersal / Eradication / Flaviviruses / Influenza / Pathogens, Animal / Pathogens, Human

FURTHER READING

Anderson, R.M., and R.M. May. 1982. Directly transmitted infectious diseases: Control by vaccination. *Science* 215: 1053–1060.

Bolker, B., and B. Grenfell. 1995. Space, persistence and dynamics of measles epidemics. *Philosophical Transactions of the Royal Society of London Series B–Biological Sciences* 348: 309–320.

Ferrari, M.J., R.F. Grais, N. Bharti, A.J.K. Conlan, O.N. Bjornstad, L.J. Wolfson, P.J. Guerin, A. Djibo, and B.T. Grenfell. 2008. The dynamics of measles in sub-Saharan Africa. *Nature* 451: 679–684.

Fine, P.E.M. 1993. Herd immunity: History, theory, practice. *Epidemiologic Reviews* 15: 265–302.

Holdo, R.M., A.R.E. Sinclair, A.P. Dobson, K.L. Metzger, B. Bolker, and R.D. Holt. 2009. A disease-mediated trophic cascade in the Serengeti and its implications for ecosystem C. *PLoS Biology* 7.

Mariner, J.C., J. McDermott, J.A.P. Heesterbeek, A. Catley, and P. Roeder. 2005. A model of lineage-1 and lineage-2 rinderpest virus transmission in pastoral areas of East Africa. *Preventive Veterinary Medicine* 69: 245–263.

Normile, D. 2008. Rinderpest: Driven to extinction. *Science* 319: 1606–1609.

Plowright, W. 1982 The effects of rinderpest and rinderpest control on wildlife in Africa. *Symposia of the Zoological Society of London* 50: 1–28.

Runyoro, V.A., H. Hofer, E.B. Chausi, and P.D. Moehlman. 1995. Long-term trends in the herbivore populations of the Ngorongoro Crater, Tanzania (146–168). In A.R.E. Sinclair and P. Arcese, eds. *Serengeti II: Dynamics, Management, and Conservation of an Ecosystem*. Chicago: University of Chicago Press.

Sinclair, A.R.E. 1979. The eruption of the ruminants (82–103). In A.R.E. Sinclair and P. Arcese, eds. *Serengeti II: Dynamics, Management, and Conservation of an Ecosystem*. Chicago: University of Chicago Press.

RISK ASSESSMENT AND PRIORITIZATION

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Risk assessment is a set of analytical techniques for estimating the frequency of undesired outcomes and their consequences. As applied to invasions, risk assessment typically concerns analyzing the likelihood that a particular species, if allowed into a region, will harm the environment, agriculture, or human health. Prioritization for managing invasions generally concerns the risk-based ranking of different invasive species already within a region. Important recent developments in invasion risk assessment have explored the risks posed by particular pathways (e.g., ballast water, the pet trade, shipping containers) for entry of species. The field is in its early stages, with one strand of science focusing on creating increasingly elaborate models for assessing invasion risk, and another taking a more skeptical approach, arguing that attempts to predict invasion risk are unlikely to succeed.

RISK ASSESSMENT CONTEXT

Three drivers have stimulated demand for invasion risk assessment and prioritization. The first is the trend toward risk-weighted resource allocation in government, a trend that has increasingly permeated natural resource policymaking. The second has been the policy need to minimize