# Disease ecology

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**The interaction of the behavior and ecology of hosts with the biology of pathogens as relating to the impact of diseases on populations.** Although human diseases have been studied from an ecological perspective for centuries, diseases in plant and animal populations have only recently received much attention. The reason that ecologists have generally overlooked diseases is that diseases tend to be "invisible" compared to predators or competitors. The toll that diseases can take on all organisms (even bacteria are attacked by diseases) is enormous, and the ecology of diseases is one of the most vigorous areas of research in modern biology. *See also:* DISEASE; ECOLOGY.

## Threshold theorem

For a disease to spread, on average it must be successfully transmitted to a new host before its current host dies or recovers. This simple observation lies at the core of the most important idea in epidemiology: the threshold theorem. The threshold theorem states that if the density of susceptible hosts is below some critical value, then on average the transmission of a disease will not occur rapidly enough to cause the number of infected individuals to increase. In other words, the reproductive rate of a disease must be greater than 1 for there to be an epidemic, with the reproductive rate being defined as the average number of new infections created per infected individual.

Human immunization programs are based on applying the threshold theorem of epidemiology to public health; specifically, if enough individuals in a population can be vaccinated, then the density of susceptible individuals will be sufficiently lowered that epidemics are prevented. Diseases such as smallpox have been eradicated because vaccination rates were high enough worldwide (70–80% immunization rates) to preclude the possibility of a smallpox outbreak. More generally, because diseases spread rapidly when densities of hosts are high, and less effectively (or not at all) when host densities are low, diseases can profoundly influence the population dynamics of their hosts—either by causing fluctuations in host density or by regulating host densities to low levels in conjunction with other factors. Even human populations seem to have been dramatically influenced by diseases. For example, between the fifth and eleventh centuries, European populations were ravaged by cycles of the plague. The initial plague epidemics killed 25–40% of the urban populations in Europe. Later cycles of plague were less deadly because the populations had evolved resistance and individuals had acquired immunity. *See also:* EPIDEMIOLOGY; VACCINATION.

Diseases have profoundly altered the course of human history. For example, the conquest of the Aztecs by a few hundred Spanish soldiers was due primarily to the fact that the Spaniards brought to the Western Hemisphere

new diseases that decimated the Aztec populations. In particular, the population of Mexico fell from about 20 million to 3 million between 1518 and 1568. The deaths were from successive epidemics of smallpox, measles, and typhus—diseases that had never before been seen in the New World, but to which the Europeans had built up a resistance (after centuries of exposure). Even today, new diseases can have a major impact on humans. The HIV epidemic in Africa is depleting the population of healthy workers so severely in cities that the economies of many nations are hampered. Almost 20 million people are infected with HIV in sub-Sahara Africa, with many of them being babies.

In general, the rate of reproduction for diseases is proportional to their transmissibility and to the length of time that an individual is infectious. For this reason, extremely deadly diseases that kill their hosts too rapidly may require extremely high densities of hosts before they can spread. All diseases do not behave as simply as hypothesized by the threshold theorem, the most notable exceptions being sexually transmitted diseases. Because organisms actively seek reproduction, the rate at which a sexually transmitted disease is passed among hosts is generally much less dependent on host density. For this reason, sexually transmitted diseases are especially hard to control.

## Population effects

Cycles in many animal populations are thought to be driven by diseases. For example, the fluctuations of larch bud moths in Europe are hypothesized to be driven by a virus that infects and kills the caterpillars of this species. Cycles of red grouse in northern England are also thought to be driven by disease, in this case by parasitic nematodes. The interaction between the red grouse and parasitic worms is subtle; the presence of a worm infection does not necessarily mean that a grouse is harmed. Instead, it is only when grouse are laden with heavy worm burdens that effects are seen, and those effects take the form of reduced breeding success or higher mortality during the winter. This example highlights a common feature of diseases: their effects may be obvious only when their hosts are assaulted by other stresses as well (such as harsh winters and starvation). The most severe human plagues in the Middle Ages were associated with times of famine, presumably because malnourished victims were less able to withstand the plague.

*Animals.* The introduction of novel diseases to wild populations has created massive disruptions of natural ecosystems. For example, the introduction of rinderpest virus into African buffalo and wildebeest populations decimated them in the Serengeti. African wild ungulates have recovered in recent years only because a massive vaccination program eliminated rinderpest from the primary reservoir for the disease, domestic cattle. But the consequences of the rinderpest epidemic among wild ungulates extended well beyond the ungulate populations. For example, human sleeping sickness increased following the rinderpest epidemic because the tsetse flies that transmit sleeping sickness suffered a shortage of game animals (the normal hosts for tsetse flies) and increasingly switched to humans to obtain meals.

*Plants.* It is widely appreciated that crop plants are attacked by a tremendous diversity of diseases, some of which may ruin an entire year's production. The emigration of Irish people to the United States was prompted by a famine caused by a potato disease. A corn leaf blight destroyed approximately one-third of the United States corn crop in 1970 at a cost of almost $1 billion. Diseases are equally prevalent among wild populations of plants, but their toll seems to be reduced because natural plant populations are so genetically variable that it is unlikely that any given pathogen strain can sweep through and kill all of the plants—there are always some resistant genotypes. But when agronomists have bred plants for uniformity, they have often depleted genetic diversity and created a situation in which a plant pathogen that evolves to attack the crop encounters plants with no resistance (all the plants are the same) and sweeps through the fields with drastic consequences. For example, when leaf blight devastated the United States corn crop, 70% of that crop shared genetically identical cytoplasm, and the genetic uniformity of the host exacerbated the severity of the epidemic. In fact, hundreds of agricultural experiments have been performed in which the severity of plant diseases is contrasted between fields planted with a mixture of varieties and fields planted with only one variety. The results reveal that, on average, genetic variety reduces disease incidence by 50% compared to genetically homogeneous (only one variety) plantings. This is one reason why biodiversity and genetic diversity are viewed as a natural resource; such diversity is likely to harbor resistance traits, which can be bred (or transferred by genetic engineering) into food crops. *See also:* PLANT PATHOLOGY.

## Disease emergence

Humans are dramatically altering habitats and ecosystems. Sometimes these changes can influence disease interactions in surprising ways. Lyme disease in the eastern United States provides a good example of the interplay of human habitat modifications and diseases. Lyme disease involves a spirochete bacterium transmitted to humans by ticks. However, humans are not the normal hosts for this disease; instead, both the ticks and the bacterium are maintained primarily on deer and mouse populations. Human activities influence both deer and mice populations, and in turn tick populations, affecting potential exposure of humans to the disease. The pathways for this influence are numerous and hard to predict. But one well-documented human effect has arisen from the control of natural predators of deer and the clearing of forest habitat; both changes have favored the buildup of large deer populations. As deer populations increase, so do populations of deer ticks and the opportunities for transfer of Lyme disease to humans. Lyme disease could never be maintained in human populations, but excessive deer populations and proximity of deer to human outdoor activities have created a significant health hazard in many northeastern states.

Much less certain are the impacts of anticipated global warming (caused by the burning of fossil fuels) on diseases. There is clearly some cause for concern about the expansion of tropical diseases into what are now temperate regions in those cases where temperature sets limits to the activity or distribution of major disease vectors (such as the species of mosquito that vector malaria).

The role of natural ecosystems in diseases, and the possibility that human activities can profoundly alter the ecology of diseases, are very significant. *See also:* LYME DISEASE; POPULATION ECOLOGY.

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## Bibliography

J. Burdon, *Diseases and Plant Population Biology*, Cambridge University Press, Cambridge, 1987

J. Diamond, *Guns, Germs and Steel*, W. W. Norton, New York, 1999

B.T. Grenfell and A. Dobson (eds.), *Ecology of Infectious Diseases in Natural Populations*, Cambridge University Press, Cambridge, 1995

W. H. McNeill, *Plagues and People*, Anchor Press/Doubleday, New York, 1976

R. S. Ostfeld, The ecology of lyme-disease risk, *Amer. Scientist*, 85:338–346, 1997

## Additional Readings

V. J. D. Casino, *Social Geography: A Critical Introduction*, Wiley-Blackwell, Chichester, West Sussex, UK, 2009

P. Daszak et al., Interdisciplinary approaches to understanding disease emergence: The past, present, and future drivers of Nipah virus emergence, *Proc. Natl. Acad. Sci. USA*, 110(s1):3681–3688, 2013
DOI: http://doi.org/10.1073/pnas.1201243109

M. S. Meade and M. Emch, *Medical Geography*, 3d ed., Guilford Press, New York, 2010

J. H. Vandermeer, *The Ecology of Agroecosystems*, Jones & Bartlett Learning, Sudbury, MA, 2011