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"Detecting disease and parasite threats to endangered species and ecosystems"

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These are preliminary lecture notes, intended only for distribution to participants.

Suitable data for such analysis are rarely collected[24]. The obvious limitation of these approaches is that correlation does not imply causation, and a third factor such as nutritional stress may be responsible both for high infection levels and decreased survival or fecundity. In addition, methods based on life tables detect impacts on survival more readily than effects on fecundity.

Suggestions on how to proceed

To establish that a disease or parasite affects a host population, there is no substitute for manipulative experiments but (1) they must be acceptable for use on a threatened species, and (2) they are too slow and expensive for use on every parasite or pathogen that an endangered species might harbour. Thus, we need indicative tests that suggest that a parasite might be a problem, and definitive tests that can be applied to candidates suggested by the indicative tests.

Indicative tests

If a parasite is causing substantial mortality, parasite levels (prevalence for microparasites and mean burden for macroparasites) will be much higher in morbid hosts than in the general population (see Fig. 2).

If there is an independent estimate of the death rate of infected hosts, and estimates of both r and the prevalence (microparasites) or mean parasite burden (macroparasites), these can be used to estimate disease impact (Box 2). This is a formalization of the conventional veterinary approach.

Definitive tests

These must involve manipulation. The only entirely satisfactory approach is manipulation of infection at the population level, but the degree of replication necessary for valid statistical analysis (a minimum of five treatment and five control populations[25]) will rarely be practical. We know of no examples of population-level manipulations of infection using entirely natural host populations. However, Scott[26] demonstrated the potential of *Nematospiroides dubius* to reduce host population density considerably in laboratory colonies of mice. When these experiments were replicated using *Apodemus sylvaticus* in semi-natural conditions, similar but less-dramatic results were obtained[27].

A more realistic option is to treat some individuals only within the one population, and to compare survival or fecundity with a control group of animals treated with placebos. However, if these experiments are undertaken within populations, they provide data only on effects of the disease on individuals, and the problem of translating these into consequences for the population remains.

It is essential that such experiments should be replicated adequately. The problem of estimating survival is essentially one of estimating a proportion, and a 95% confidence interval for a proportion is approximately $\pm(1/\sqrt{n})$, where n is the sample size. Thus, at least 100 individuals must be marked to estimate survival to within 10%. When a subsection of the host population is treated, and statistically significant differences are required between the treatment and control animals, it may be necessary to handle 400–500 animals to detect a 10% difference in survival between the two groups with 95% confidence.

There are several examples of endangered species being vaccinated against disease (e.g. mountain gorilla, *Gorilla gorilla beringei*, against measles[28] and African hunting dogs, *Lycaon pictus*, against rabies[29]), but comparison of survival with untreated control animals has not been undertaken.

In non-endangered species, there have been several manipulative studies of parasite impact in natural conditions. Work on red grouse (*Lagopus lagopus scoticus*) and the parasitic nematode *Trichostrongylus tenuis* in the north of England and Scotland illustrates these techniques. The study involved catching birds and treating half of the sample with a drug to kill the parasites while the control birds were treated with a drink of water. Radio telemetry aerials were attached to the control and the treatment birds, and the birds were then monitored for the next three to six months[30]. These experiments illustrated that the treatment birds, those with lower parasite burdens, had a higher rate of survival but also significantly better fecundity than the control birds with natural parasite burdens. Similarly, the control birds were found more-readily located by the dogs used as surrogate predators in an experiment to determine whether or not parasites increased the susceptibility of hosts to predation.

Other recent manipulative experiments on parasite impact in the field include studies of botflies (*Cuterebra angustifrons*) on white-footed mice (*Peromysas leucopus*[31], ectoparasites on gerbils (*Gerbillus andersoni allenbyi*)[32] and nematodes in Soay sheep (*Ovis aries*)[33].

Many managers may feel compelled to treat as many animals in an endangered population as possible, if they even suspect a disease problem[34]. However, no treatment for disease is without risk, and the capture and handling of animals necessary to administer treatment may involve substantial hazards to the animals. This problem is exemplified by reports of increased mortality detected in vaccinated or handled African wild dogs in the Serengeti[35].

The importance of rinderpest in structuring the ungulate community in the Serengeti (Box 3) indicates that removal of a pathogen from a system may have major impacts on the structure of entire communities.

Accordingly, it is necessary to establish that treatment for a pathogen is required before it is used on a large scale, and the possible wider consequences of pathogen removal should be investigated. An experimental approach to handling disease threats to endangered species is not only good science, it is good management practice as well.

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Box 1. Did disease kill the thylacine?

The thylacine (*Thylacinus cynocephalus*) was the largest carnivorous marsupial to have survived into historical times. Formerly distributed throughout Australia, it was restricted to Tasmania by the time of European settlement. At that time, it was sufficiently common to be perceived as a serious threat to the sheep grazing industry. A bounty of £1 (several days' wages) was placed on scalps and a total of 2268 were known to have been killed[36]. The last known thylacine died in Hobart zoo, Tasmania, in 1933, and despite persistent reports of sightings, the species is almost certainly extinct[37].

Guiler[36] proposes that three lines of evidence suggest that the disappearance of the thylacine may have been because of disease. First, hunting returns remained approximately constant with a mean of 115 from 1888, when records were first kept, until a precipitous decline that began in 1906 led to no returns by 1910. A decline occurring through over-hunting could be expected to be more gradual. Second, the decline was simultaneous across Tasmania, rather than occurring first in heavily settled areas, as might be expected if habitat destruction were responsible. Finally, anecdotal reports exist of animals dying simultaneously in about 1910 in one area from a 'distemper-like' disease. As is typical of most recent extinctions, hard evidence of disease remains absent.

Box 2. Measuring the impact of single-host–single-pathogen infections

If it can be assumed that the host–pathogen system is at equilibrium, simple models can be used to estimate D, the extent to which the disease depresses the host population (D = equilibrium host population size with infection present divided by the disease-free carrying capacity). For a microparasite affecting both fecundity and survival:

$$D = \left(\frac{\alpha + a(1 - f_1)}{r} \right) \phi$$

where α is the death rate of infected animals, r is the intrinsic rate of increase of the population, f_1 is the impact of the disease on host fecundity (1, no impact; 0, infected animals sterile), a is the birth rate of uninfected hosts (females/female/unit of time), and ϕ is the equilibrium prevalence of the pathogen[12]. An analogous equation for macroparasites is:

$$D = \frac{\alpha}{r} M$$

where M is the mean parasite burden in the population, and α is the increase in host death rate per parasite.

Pathogenicity α can be approximately estimated as the inverse of the life-expectancy of an infected host for microparasites, and as the gradient of the relationship between parasite burden and the inverse of life-expectancy for macroparasites[38].

There are many ways to estimate the intrinsic rate of increase, r, most requiring detailed demographic data. A simple allometric approach[39] can be used to obtain an approximate estimate, given mean adult mass.

the colony of ferrets was too small to sustain the pathogen, once it was introduced into the colony from prairie dogs, or feral canids, it quickly spread, causing the deaths of around 70% of the ferrets. This example illustrates two key epidemiological features that are crucial for successful disease management in endangered species: first, because endangered species have small populations they are unlikely to sustain infections by virulent pathogens. Endangered species will therefore tend to acquire virulent infectious diseases only after exposure to infected hosts of another more common and widespread species. Secondly, because most individuals in the population are never exposed to a pathogen, there is very little acquired immunity to infection. Under these circumstances, when an epidemic outbreak does occur, it tends to infect a large proportion of the population and mortality levels may be high.

The rinderpest example described in Box 3 is another example of this phenomenon, in this case, the rinderpest (RPV) was relatively benign to its ancient cattle hosts, but highly virulent to the wildebeest (*Connochaetes taurinus*) and cape buffalos (*Syncercus caffer*) that had only recently been exposed to the virus. It is important to notice that quite subtle coevolutionary forces may be operating here. At first sight, it would seem that the ancient association between cattle and RPV would select for benign strains of the virus and resistant cattle hosts. This may only be part of the story, because once RPV has passed through several sequential wildebeest or buffalo hosts, its virulence to these species declines but its virulence to cattle increases[15,16]. This suggests that the production of more-benign strains of virus by a mixture of mutation and selection may occur on a timescale that is much faster (by several orders of magnitude) than any coevolutionary response in the hosts.

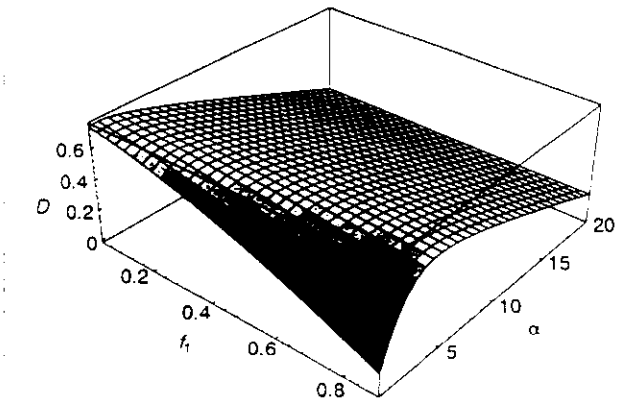

Fig. 1. The effect of disease pathogenicity, α, and the fecundity of infected hosts relative to susceptible hosts, f_1, on the extent that a host population is depressed below its disease-free carrying capacity, D. If pathogens primarily affect mortality, those with intermediate levels of pathogenicity have the greatest impact on their host population. If the major effect is on fecundity, the greater the decrease in fecundity, the larger the impact on the host. *From Ref. 12, using a modification of a model of Anderson*[11].

Finally, it is important to consider the potential of parasites as agents of competition that help one host species to exclude another from the part of their range where a pathogen can establish. Schmitz and Nudds[17] have examined a mathematical model for the meningeal helminth parasite, *Parelaphostrongylus tenuis*, that has been suggested as an agent that allows white-tailed deer (*Odocoileus virginianus*) to prevent moose (*Alces alces*) and caribou (*Rangifer tarandus*) from establishing in larger areas of the eastern United States[18]. A number of studies have suggested that high mortalities observed in moose and caribou, when introduced into a range occupied by white-tailed deer, were because of infection by *P. tenuis*. However, no experimental studies have been undertaken to falsify fully this hypothesis[18]. The models developed by Schmitz and Nudds[17] suggest that it is possible for the parasite to allow white-tailed deer to exclude other cervids from a potentially sympatric range, but that other hypotheses, such as direct competition, could not be excluded.

In a more general exploration of this phenomenon, Price, Westoby and Rice[19] have suggested that, where a parasite species utilizes more than one host species, the pathogen will be least pathogenic to the host with the larger range. The implications of this theoretical prediction for rare and potentially endangered species with highly localized distributions are obvious. However, the results of a comparative survey of pathogens that infect more than one host species are equivocal as the majority of examples they cite are for