

1 Disease and epizootiology—basic principles

“Typically, diseases of wildlife have been investigated by performing pathological examinations on carcasses that are found incidentally, or producing lists of parasites identified in small samples of host species. There have been few attempts to assess the impact of a disease on the host population rather than the individual, or to describe the distribution of the disease agent in a manner sufficient to understand its epidemiology.”

(Gulland 1995)

1.1 Disease and diseases

The concept of disease is surprisingly difficult to define in terms that are sufficiently broad for application to the wide range of conditions that occur in free-ranging wild animals, and that are still sufficiently narrow to separate disease from other factors, such as predation and food supply, that affect wildlife negatively. Disease might be defined as any departure from health, but this leads to a circular discussion of the meaning of health and normality. Disease in wild animals is often considered only in terms of death or obvious physical disability, probably because these are readily identified parameters. However, the effect of disease on wild populations may be much greater than is evident by simply counting the dead or maimed, even if it were possible to do so accurately. The impact of DDT and certain other chlorinated hydrocarbon insecticides on some raptorial and piscivorous birds provides an excellent illustration of this fact. These compounds have low direct toxicity and rarely result in the death of birds or in obvious clinical signs of intoxication, yet they had profound population effects through decreased recruitment as a result of increased egg breakage.

Disease conditions should not be dismissed as inconsequential simply because they occur commonly, nor should one assume that a disease condition or parasite has a major effect on the host simply because it is conspicuous. Infection with parasites of various types is ubiquitous in wild animals and reference is often made to *normal* parasite burdens, the inference being that parasites at this level have little or no impact on the animal. However, the actual effect of these parasites on the host is largely unknown. *“Although most infectious agents do not result in obvious disease, the host must pay a ‘price’ for harboring parasites that live, grow, and reproduce at the expense of the host”* (Yuill 1987). In domestic livestock, this price can be measured in

terms of decreased efficiency of production and, even in livestock, the true extent of the cost is often not recognized until the parasite or disease has been eliminated. This type of assessment is seldom possible in free-ranging animals but observations from semi-free-ranging animals, of the same species, such as those on game-farms, provide some indication of the cost of parasitism. For example, Szokolay and Rehbinder (1984) reported a 20% increase in the growth rate of fallow deer after gastro-intestinal parasites, of the type common in wild deer, were partially controlled through the use of anthelmintics. Perhaps even more pertinent is the finding that treatment with anthelmintics resulted in an “almost 100% increase in body weight in the fawns” and increased antler growth in males among free-ranging roe deer (Duwel 1987). Special techniques may be required to assess the cost of a disease. For example infection with avian malaria (*Plasmodium peditocetii*), while not causing obvious illness in male sage grouse, had a significant effect on breeding that was only detected in detailed behavioral studies. Infected males attended the lek less regularly, copulated less frequently, and bred later in the season, with less “fit” females, than did non-infected males (Johnson and Boyce 1991). Female sage grouse selected against mating with males that had hemorrhagic spots on their air sacs of the type produced by lice (Spurrier et al. 1991). We currently do not have sufficient techniques for measuring effects such as subtle alterations in behavior or diminished intelligence that have been documented to occur in parasitized humans (Levav et al. 1995; Flegr et al. 2003).

The effect of disease may only be evident under certain conditions. For instance, infection with blow fly larvae (*Protocalliphora* sp.) had no effect on the weight, size, or age at fledging of young sage thrashers; however, parasitized birds had a higher mortality rate than unparasitized fledglings during a period of wet, cold weather, suggesting an interaction between parasitism and other stressors (Howe 1992). Similarly, Murray et al. (1997) found a synergy between intestinal parasites and nutrition in snowshoe hares when food was limited. It also is important to examine the correct portion of the population in evaluating the effect of disease. Iason and Boag (1988) failed to find any correlation between intensity of infection with an intestinal parasite and body condition or fecundity of adult mountain hares but suggested that it would be very important to determine the effect of the parasite on growth and survival of young hares before concluding that it was harmless. The members of any population are not homogenous and a small proportion of the population may bear the brunt of a disease. This is most clearly established for infections by larger parasites in which “most hosts have very low parasite burdens and a few hosts have very high burdens” (Shaw et al. 1998) but the same principle likely applies to many other diseases in which underprivileged individuals in the population are affected disproportionately. It may be very difficult to detect or measure the impact of disease in these situations because severe effects on a small number of animals may have relatively little effect on measures of central tendency such as the average rate of growth or the median body condition.

1.2 A definition of disease

The definition of disease that will be used in this book is that the term includes “*any impairment that interferes with or modifies the performance of normal functions, including responses to environmental factors such as nutrition, toxicants, and climate; infectious agents; inherent or congenital defects, or combinations of these factors*” (Wobeser 1997). No distinction will be made between infectious and non-infectious causes of disease because the basic principles of investigation and control are similar for both. However, the term **risk factor**, rather than **causative agent**, will be used when referring to some non-infectious diseases.

Within this broad definition of disease, groups of affected animals with similar features are classified into categories or are said to be affected by a particular *disease* that is identified by a specific name. There is no consistent pattern or rationale for naming diseases, so the current situation represents a hodge-podge of styles:

Name of disease	Derivation of name
Tyzzler's disease	Discoverer (E.E. Tyzzler)
Tularemia	Locale of first description (Tulare County, California)
Bluetongue	Clinical feature
Enzootic ataxia	Clinical and epizootiological features
Avian vacuolar myelinopathy	Pathological feature
Aspergillosis	Causative agent (<i>Aspergillus</i> spp.)

In many cases, the name applied to a disease reflects the current understanding of its nature and this name is open to change as the cause or nature of the disease is elucidated. Categories or diseases may be subdivided when it is discovered that several causes produce similar features. For instance, the disease tularemia is now known to be caused by four closely related bacterial species in the genus *Francisella* and types A and B tularemia are recognized. In general, the tendency with time is to define the characteristics of a disease more precisely, and to indicate the causation in the name. For example, a common disease of domestic cattle has gone through a progression of names from red nose, to infectious bovine rhinotracheitis, to bovine herpesvirus I infection. Unfortunately, several names may be applied to a single disease simultaneously, resulting in unnecessary confusion. Thus, a single disease of waterfowl caused by one virus is referred to as duck plague, duck virus enteritis, duck viral enteritis, and anadid herpesvirus infection.

1.3 Disease causation

The study and understanding of the cause and nature of disease have undergone a gradual evolution. Prior to discovery of the identity of specific infectious agents, there was considerable controversy between the alternate hypotheses of the *contagium vivum* (or living contagion theory) and the *miasma* or (bad air theory). The discovery of microbial pathogens silenced this controversy for a period and, at the turn of the past century, both human and veterinary medicine were concerned primarily with identification of specific agents responsible for acute infectious diseases. A set of rules (Koch's postulates) developed for establishing cause-and-effect relationships between infectious agents and disease were generally accepted and widely applied. These stated that the agent:

- must be shown to be present in every case of disease through its isolation in pure culture;
- must not be found in cases of other diseases;
- must be capable of reproducing the disease in experimental animals;
- must be recovered from the host in which experimental disease was produced.

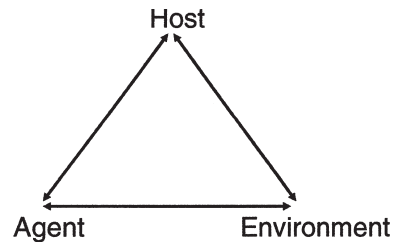
These laboratory-based criteria for judging causal relationships to disease were valuable in defining many diseases of simple etiology, and are still useful in the study of certain diseases such as rabies. However, this one agent-one disease model is not adequate for either the study of many diseases or for the explanation of how most diseases behave in nature. Koch's postulates are particularly inappropriate for many non-infectious diseases, for diseases caused by mixed infections, for diseases in which the predisposing factors are important, for diseases with a carrier state, for diseases caused by opportunistic agents that may or may not cause disease when present, and for many chronic diseases in which the inciting cause has disappeared before the clinical disease becomes evident. Hanson (1969) eloquently outlined the deficiencies of these postulates for the study of wildlife diseases in a presentation entitled "*Koch is dead*" to the Wildlife Disease Association annual meeting.

A more holistic view is necessary for the understanding of most diseases. Jekel et al (1996) proposed three categories that are useful for considering potential agents or factors as the cause of disease:

- If a **sufficient cause** is present the disease will always occur;
- If a **necessary cause** is absent the disease cannot occur;
- A **risk factor** is a characteristic that, if present and active, increases the probability of the disease occurring.

Multiple features of each of the **agent**, the **host** and the **environment** in which the disease is occurring must be considered in evaluating disease causation

Fig. 1.1 This schematic often is used to symbolize the interactions among agent, host, and other environmental factors that govern the occurrence of disease



(Fig. 1.1). (Consideration of environmental factors recalls the miasma theory of prior times). Even when dealing with a disease caused by a single agent, each of the three major components has a variety of determinants, any of which may influence whether or not overt disease will occur. This simple table presents only a few such variables:

AGENT	HOST	ENVIRONMENT
Strain	Species	Climate
Dose	Genotype	Weather
Route of exposure	Age	Altitude
Duration of exposure	Sex	Other species
	Nutritional status	Population density
	Reproductive status	Air and water quality
	Past exposure	Soil
	Concurrent disease	Human activity
	Immunocompetence	
	Food habits	
	Behavior	

For many diseases, even this agent-host-environment approach may be inadequate and it is more appropriate to consider disease in terms of a **web of causation** in which many factors interact to result in disease. It often is difficult in these situations to classify a factor as being distinctly a feature of the agent, the host, or the environment. Any single factor may be a necessary component but may not be sufficient, in and of itself, to produce disease without the presence of co-factors. The 'lungworm-pneumonia complex' of bighorn sheep (Forrester 1971) provides an excellent example of this type of situation. A wide variety of infectious agents including parasitic nematodes (*Protostrongylus* spp., and less commonly *Muellerius* sp. lungworms), bacteria (*Pasteurella* spp., *Mannheimia haemolytica*, *Arcanobacterium pyogenes*, *Streptococcus* sp., *Staphylococcus* sp., *Neisseria* sp., *Chlamydophila psittaci*, *Mycoplasma* sp.) and viruses (parainfluenza 3 virus, respiratory syncytial virus, bluetongue virus) have been recovered



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