CHAPTER 1

Ecology of wildlife diseases

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For real progress, the modeller as well as the epidemiologist must have mud on their boots. (David Bradley 1982)

1.1 Historical background to this book

The majority of living organisms are parasitic in one form or another, be they a virus invading a sea coral, a tapeworm within the guts of your dog, a cuckoo chick in a reed warbler's nest or a lion stealing a hyaena's kill on the African plain. The parasites are intimately linked with their host in a trophic interaction that has fascinating implications for both parasite and host. Most of the more traditional parasite species interact with their hosts at a range of organizational scales that stretches from the miniscule—the molecular battle between the host's immune response and the evading action of the parasite—through to the extensive—the structuring of communities and a driving force in the evolution of biodiversity and sex. These interactions also range over many orders of temporal scale, ranging from short time-scales of the history of an individual infection and an epidemic outbreak to time-scales of endemic co-exisitence and co-evolution. Few disciplines cover such breadth and few disciplines have developed so rapidly over the past 30 years.

Research areas evolve at different rates, at times needing periods of intense empirical work to set a foundation of understanding so that at other times, novel approaches and new theories can be formulated, and the discipline can expand rapidly. Modern epidemiology is such a discipline. For many years parasitologists focused on unravelling the complex and intriguing life cycles of parasites, describing in detail the stages of the life cycle, the parasitic mode of life, and banking the data in journals and texts, some of which were rather dry and repetitive descriptive lists. Then a series of workers started to realize that parasitology was not just a descriptive science but there were parallels with other disciplines, and the time had come to start synthesizing such disciplines. In our minds, an essential step was taken when Anderson and May (1978) synthesized our understanding of parasitology with the quantitative discipline of population biology. They pointed out that the parasite–host relationship was not simply the impact a parasite had on an individual, but an integral of these interactions at the population level, and at the same time a dynamic process where parasites were flowing from one host to the next; the rate at which this took place was determined by host behaviour and abundance. Where the hosts were suffering from the parasite insult with reduced survival and fecundity, they were also fighting back in terms of innate resistance and acquired immunity, leading to an evolutionary arms race where both were fighting for their lives and their fitness. They followed in the footsteps of major theoretical figures such as Ross, Macdonald, and Bartlett—but Anderson and May ignited the discipline at the right time. This was consolidated and extended in a meeting held in Dahlem in 1981 (Anderson and May 1982a). In many respects the Dahlem meeting was inspirational—the group of workers produced detailed group reports on what they felt needed to be done

and which direction the discipline should take. While the original impetus was derived from population ecology and parasitology in natural host populations, much of the subsequent work over the next decade focused on applying population biology to human infections—notably in response to the AIDS epidemic (Anderson and May 1991).

At the same time, an increasing number of workers applied these ideas to explore the impact of diseases in naturally fluctuating wildlife populations, particularly in the applied context of conservation biology. This work was synthesized in a meeting organized in 1993 at the Isaac Newton Institute in Cambridge. The resulting volume (Grenfell and Dobson 1995) summarized the state of play of wildlife-disease modelling. There has been a large body of subsequent theoretical and empirical work on the ecology and evolution of infectious disease in natural populations. The volume and quality of this work now demands another synthesis—hence the meeting upon which this book is based.

We also now need a wider unification—wildlife disease ecology should bring evolutionary biology and parasite population dynamics closer together but also reach out to immunology, genetics, and molecular biology. In this respect there has been inspirational work by evolutionary biologists like the late Bill Hamilton, who has addressed questions of how hosts signal their disease status to prospective mates and how parasites have been important in the evolution of sex.

Following the excellent tradition laid down by the meetings in Dahlem and Cambridge we organized a meeting in a location that was both beautiful and inspiring. The Grenfell and Dobson (1995) volume was set in a mathematical institute and consequently started to look at the disease issues from a theoretical, mathematical viewpoint. In contrast we thought it would be timely to base the next volume in an empirical research station and take a more empirical and ecological view. We chose the *Centro di Ecologia Alpina*, a young and dynamic research centre, based close to the top of Monte Bondone, Trento in the Dolomitic Alps. The scenery was breath-taking, the facilities excellent and the hospitality of the president, director, and staff beyond belief. Ideal for quiet thought but a location that brought people close together and one that would result in synergy. We obtained funding and support, and invited 50 research workers, selected to cover the breadth of the discipline, and also for their ability to think, to write, and to communicate. We know we did not get everybody we should have but the mixture was good and the discussions buzzed. This volume is the result of that meeting. We organized the day into some formal talks, where we asked two people from each discipline to provide contrasting views on specific questions before we all retired to group discussions. During these discussions the chair and rapporteur called on others to give 5-minute presentations about models, data, or thoughts, and guided the discussion into stimulating areas of disease research. We then got a lead author for each chapter and asked them to produce a chapter for the volume, which we could edit into one coherent text. A great idea but not a simple task. We hope this text will provide another step in our understanding of wildlife disease ecology.

1.2 Subject area and content

This book summarizes recent developments and thoughts in wildlife disease ecology. We asked 50 of the leading workers to identify the interesting and important issues and to look at these from a range of perspectives, to try and mould the veterinarians view with that of the ecologists, and to stimulate empiricists and theoreticians to work closely together. We wanted to provide an understanding and an insight into disease dynamics that spans ecology and evolutionary questions and uses techniques from the molecular sciences through to mathematics. We do not believe that any one individual, or even a small group of workers, could achieve this. Moreover, we believe that this insight will not only provide intellectual satisfaction to ourselves but also provide guidance in the applied fields of disease control, conservation of animals threatened by disease, and help set future research agendas.

This volume addresses a series of what the editors thought were the most intriguing and stimulating areas, where we could bring together workers with disparate visions. First, we wanted to build on the volume by Grenfell and Dobson (1995) to see

how the discipline was progressing but at the same time we did not want to fall into the trap of asking smaller and smaller questions that eventually run the risk of becoming insignificant. One key area for discussion was the role of spatial dynamics in disease transmission and the impact that the structuring of host populations has in influencing disease spread and persistence (Chapter 6). Second, we wanted to complement the earlier volumes and address areas of work that had not been fairly covered. One of these was tick ecology and research; here, improved molecular techniques and the use of satellite imagery have made remarkable progress and can make important predictions about how global climate change can affect disease prevalence (Chapters 7 and 6). Originally, workers thought that transmission rates were simply dependent on host density and that such diseases were unlikely to have an important effect on small, threatened populations. This is not the case—there are several species of canid that are currently under threat from diseases shared with reservoir hosts (Chapter 8). As we start to investigate multiple-host systems, parasite communities (Chapter 4), the role of reservoir hosts, and strain competition, we need to keep evaluating the role that parasites have in influencing host population dynamics and shaping community structure through apparent competition (Chapter 3). At the same time we need to develop new and tractable mathematical techniques to evaluate these questions and apply stochastic modelling techniques to problems of small populations and probabilities of disease persistence (Chapter 9). One area where there has been progress in recent years is in our understanding of the relative role of deterministic and stochastic forces in determining the onset of epidemics and their subsequent fate (Chapter 5).

Much of our understanding of disease is based on studies of individuals, particularly the very detailed studies that have been undertaken by veterinary workers. A major challenge is to link our understanding of individual level infections to how disease flows through host populations and so influences host dynamics. Such variation in parasitism between individual hosts are probably best understood for macroparasitic worms. We therefore start with a careful look at variations between individual hosts (Chapter 2), then examine how these influence the likelihood of regulating a host population (Chapter 3). Indeed, a central issue in the original models of Anderson and May (1978) was the impact of parasite aggregation, as captured by the negative binomial distribution. Understanding the origins and impact of aggregation are still a major focus for research. As ever, the relative role in determining aggregation of ecological and genetic heterogeneities, and the stochastic process of transmission, are key questions (Chapter 7).

We have tried in these first few pages to show you what excites us about our discipline and we hope encourage you to delve further into the book. Our publisher hopes you will get your money out and buy it.

1.3 How to use this book

This book is aimed specifically at those with biological interests—if you are interested in biology then this book will help to reveal the exciting new developments in wildlife disease ecology. We have a clear and simple objective: to unify studies of infectious disease biology and stimulate further growth in this field. So, if you are a medical or veterinary clinician, a molecular biologist, or a biology student just starting off on a career, this book is for you. The book is not a compendium of taxonomical life histories—there are already a number of excellent books on the market that cover these. One of our favourites, which focuses just on nematodes, is the outstanding volume by Anderson (2000).

The trick with any good non-fiction book is to learn how to find your way round it so that you can glean the details you need efficiently and effectively and enjoy doing it. We have tried to help you in this respect by structuring the chapters along the same lines and presenting the information in a way that we hope is easily accessible. Each chapter starts with a 'motivation statement' that identifies the key underlying questions that are addressed in the chapter and captures in a phrase the essence of the chapter. The background sets the scene and then we launch into the theory, the questions, what is known, and specific study cases to illustrate the issues. We have used boxes throughout the book for two main purposes. First, to provide background on a specific parasite–host system, for

example phocine distemper virus in seals or helminths of Soay sheep. Second, to provide some of the more detailed technical details, of mathematical models or statistical procedures. We wanted to include both the biology and the theory without cluttering up the text and yet it is these very details that we know readers and students wish to find quickly, so they can use the book as a source of information. Finally, we have been brave and provided a vision about where the discipline is going and what the questions are that need to be tackled—Chapter 9. Actually, this is not quite true, we the editors were not brave—we asked our colleagues from parallel disciplines to look at our discipline and to identify what needed doing. Try it—we would like to think of this book sitting next to the computer of the next generation of bright young epidemiologists or sitting next to the armchair of our own retired professors—as they cogitate on where their students took their discipline.

1.4 Who are the players in the parasite game?

Traditional parasitologists may well ask themselves if this book is about helminths or viruses? The answer is both. We follow the useful division of parasites into two groups: the macroparasites and the microparasites. We define these and then introduce a most important player in the world of epidemiology: R_0.

Macroparasites are the parasitic species where reproduction usually occurs via the transmission of free-living infective stages that passes from one host to the next. Direct reproduction rarely occurs within the definitive host, although asexual reproduction can occur in the intermediate hosts, e.g. Digenean trematodes often multiply within snails. It is true that compared to the microparasites they are relatively large, have long generation times, and are characterized by a great diversity of antigens so that immunity is transient and is a function of the history of infection. Infections tend to be chronic leading to morbidity rather than mortality. Our understanding of the system is based on the number of parasites per host, and models must capture the details of the intensity of these infections. They include the helminths (worms) and arthropods.

Microparasites in contrast tend to have rapid reproduction within a host and do not have a special infective stage. The generation time is short, such that populations rise rapidly within their host leading to a crisis that leads either to host death or the development of immunity. Antigens are usually simple and immunity is often life-long. The state of the host is the basis of our understanding, such that hosts can be classified as susceptible, latent (infected but not infectious), infectious, or recovered (immune). From this classification we can make compartmental models of disease dynamics—very different from the macroparasite models that explicitly consider the intensity of infection. The microparasites tend to include the bacteria, viruses, protozoa, and fungi.

R_0 (read as 'R nought'). One of the fascinations of disease dynamics is that many aspects of the biology of the disease can be captured by this parameter and it would be rather unfair to progress much further into this book without introducing you to it. R_0 is fundamentally a measure of parasite fitness, since it is the number of new infections arising from an infected individual (microparasites) or number of female worms that are established from a female worm (macroparasite) in a population of fully susceptible hosts, where there are no density dependent constraints operating (macroparasites). R_0 influences many features of the epidemiology of an infection and wholly or partly determines whether a parasite can invade a susceptible host population, the subsequent pattern of disease dynamics, which strain or competing species dominates, the persistence pattern of an infection, and a range of other conditions. We use R_0 for all types of parasites. For an introduction see Anderson and May (1991) and Diekmann and Heeseterbeek (2000), which includes a detailed explanation of its calculation.

The introduction of R_0 encourages us to point out a further fascination of parasites—a duality—parasites act at a range of scales and actions that can be looked at in two ways. R_0 determines both the epidemiological patterns of the infection in the host population and the fitness of the parasite. Another duality: parasites are a threat to biodiversity, wiping out some host species through apparent competition (Hudson and Greenman 1998) but

at the same time are considered a major selective force behind the evolution of biodiversity both within a host population and by selecting for hosts that are spatially distributed to avoid parasitism. Highly virulent parasites lead to rapid mortality of their host but by causing wounds and making a host cough and splutter they also increase transmission rate between hosts leading to the question: Where should individual parasite genotypes sit in the trade-off between virulence and transmission? Parasites inhabit hosts that can be considered suitable habitat patches, within a host population structured like a metapopulation, and yet the host population itself is structured as a metapopulation. How can we use these spatial structures to reduce spread of infections within and between host populations?

There is one final, if not obvious then somewhat ironic, duality that needs to be pointed out—the relative importance placed on parasitological studies in biology reflects the importance of disease issues to humankind. There is no question that disease has been a major force influencing the development of human society: countries like Italy were devastated by the plague and it took the human population more than 400 years to recover. We need only look back a generation to observe the devastating impacts that influenza and other infectious diseases had on human mortality and morbidity. However, during our lifetime there have been remarkable developments, the effects of penicillin application, pasteurization of milk, and the development of vaccines against the major childhood diseases have changed the face of the world. Indeed when we were young, smallpox was eradicated (or was it?) and a number of infectious diseases brought close to extinction. As such, when we went to study at university, we were told that parasites had little impact on human and wildlife host populations—parasites were dismissed by many ecologists as something the parasitologists had to teach so that we understood taxonomy. Now we have seen the emergence of infectious diseases such as AIDS that have made population growth rates negative in some parts of the world and diseases such as Ebola that wipe out whole communities of people in parts of Africa. We are concerned about the evolution of drug resistance against diseases such as tuberculosis and as the book goes to print, Europe has no idea about the true threat from nv-Creutzfeld Jacob Disease and Britain is recovering from a serious Foot and Mouth epidemic. These increasing threats have brought disease issues out of the closet and at the same time sparked off new and innovative studies of disease epidemiology and evolution in Biology.

That is where this book comes in.