Infectious Diseases of Wildlife, in Theory and in Practice

Robert D. Holt

THE ROLE OF INFECTIOUS DISEASE has in recent years been an increasingly prominent theme in ecology and evolution. A 1982 Dahlem Conference organized by Roy Anderson and Robert May[1], which evenhandedly treated human and nonhuman infectious diseases, signally catalysed interest in this field. Progress made since the Dahlem Conference in elucidating the population dynamics of human infectious disease was recently summarized by Anderson and May[2]. A significant step toward a comparable synthesis of our current understanding of nonhuman disease dynamics was taken this past spring in the elegant setting of the new Isaac Newton Institute of Mathematical Sciences at Cambridge University, UK, where an eclectic yet congenial group of theoreticians, empiricists, ecologists, wildlife biologists and veterinarians assembled to discuss the topic of 'Infectious Diseases in Wildlife Populations'.

The conveners, Bryan Grenfell (Cambridge University, UK) and Andrew Dobson (Princeton University, NJ, USA) fostered lively discussions and an ecumenical spirit, not least by generously including infectious diseases of invertebrates and plants under the rubric of 'wildlife disease'. The workshop, deliberately modeled after a Dahlem Conference, opened with a batch of formal presentations, followed by group discussions and a final round of reports synthesizing the groups' conclusions. The proceedings will appear in a Cambridge University Press volume.

The main themes of the 1982 Dahlem Conference – the impact of infectious diseases on host populations, transmission dynamics, control and evolution – were also central issues in the Isaac Newton meeting. One area that received more explicit attention in Cambridge was the interweaving of classical ecological concerns (e.g. population regulation, community structure) and disease epidemiology. In contrast to human infectious diseases, knowledge of infectious diseases of wild populations is often quite scattered; in few cases are all the relevant bits understood in satisfactory detail. These pragmatic problems heighten the need for a smoother interweaving of theoretical and empirical studies, so as to maximize the value of scant but often hard-won data. A particularly useful feature of the meeting was that presenters and discussants attempted to articulate not only what is known, but also what is not known about wildlife infectious diseases, both empirically and theoretically.

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The first five talks provided synoptic overviews of infectious diseases in wildlife populations. Frances Gulland (Institute of Zoology, London, UK) kicked off the meeting by reviewing the impact of infectious diseases on wildlife populations, particularly vertebrates. Dramatic cases of population limitation by infectious disease do exist, but clear demonstrations of diseases acting as classical, density-dependent regulatory factors are still rare. Studies of wildlife disease typically rely on pathological examination of carcasses or lists of parasites from host samples; these procedures underestimate mortality, particularly if predators are present, and moreover miss sublethal effects on host reproduction. Gulland also emphasized the need for analysing how species differences in life history and social structure influence disease transmission.

Dobson provided an excellent overview of observed patterns in microparasites. He amplified the point that we often know a lot about the pathology of a wildlife disease, but little about how to translate this into individual fitness. In order to connect models with data, there is a crying need for statistical estimators to estimate transmission rates (e.g. from age–incidence data). Yet most epidemiological models and inference techniques assume a single host species, whereas many wildlife diseases in fact have a multispecies distribution, and many hosts harbor multiple infectious diseases: analyses of host–disease interactions need to be cast in a broad, community context. Dobson further suggested that the net transmission rate parameter might be usefully decomposed into components corresponding to direct physiological responses, basic ecology (e.g. diet) and an inter-individual spacing parameter. He also emphasized the potential of molecular techniques of phylogenetic reconstruction for elucidating cross-species relationships.

Nigel Barlow (Canterbury Agriculture and Science Centre, Lincoln, New Zealand) collated all models he could find of microparasitic infections in wildlife. He concluded that few are reasonably 'complete' with respect to either crucial biological details or spatial effects. For instance, the 'βIS' (= transmission rate × infected hosts × susceptible hosts) term of classical epidemiology assumes homogeneous mixing, yet in almost all field data disease incidence is spatially aggregated. Barlow argued that we need a better characterization of how transmission varies with population size and spatial dispersion patterns, that is, to provide concrete ecological underpinnings for abstract epidemiological parameters.

Peter Hudson (Game Conservancy, Inverness-shire, UK) complemented Dobson's review with a summary of observed patterns in macroparasites. There are impressive success stories in macroparasite population ecology, such as the work by Hudson and Dobson themselves on the role of a nematode (*Trichostongyle tenuis*) in red grouse (*Lagopus lagopus scoticus*) population dynamics. Yet many significant problems remain. Some of these center on understanding the magnitude of aggregation in numbers of parasites per host (a key determinant of population regulation in macroparasite models). Though aggregated distributions are generally observed, biases in data collection tend to overemphasize the degree of aggregation, so it is difficult to parameterize models accurately. Moreover, there is growing evidence from age–prevalence relations and other sources that acquired immunity to macroparasites exists, the magnitude of which varies among hosts. Such immunity could have important dynamic consequences. Documenting acquired immunity empirically and analysing its effects are challenging problems. Hudson amplified for macroparasites several topics also sketched by Dobson: the relatively poor evidence for host regulation (versus limitation) by macroparasites; the importance of effects on fecundity and vulnerability to predators, as well as direct effects of parasitism on host survival; and, the need to consider basic ecology (e.g. diet, social organization) in sculpting models of disease transmission and host vulnerability.

Mick Roberts (Wallaceville Animal Research Centre, Upper Hutt, New

Zealand) reviewed state-of-the-art models for helminth parasites and their animal hosts. He argued that among those aspects requiring further work, particular attention needs to be paid to the effects of acquired immunity, seasonal dynamics and interactions between multiple parasite species in the same host.

The next five talks concentrated on particular aspects of the host–parasite interaction. Chris Dye (London School of Hygiene and Tropical Medicine, London, UK) described systems in which parasite transmission occurs indirectly via vectors. He pointed out that vector dynamics introduces a range of distinct nonlinear processes (e.g. saturation in vector prevalence) and that many vectors attack multiple hosts. The implications of these complexities are not well-characterized in any natural system.

Jonathan Swinton (Imperial College, London, UK) emphasized the potential of plant host–pathogen systems for addressing many issues in epidemiology. Because plants move more slowly than do their pathogens, a consideration of spatial dynamics is paramount in plant epidemiology. Moreover, the role of genetic variation in plant disease processes, if not fundamentally more important than in animal systems, at least seems easier to document. Swinton reviewed different approaches to spatial heterogeneity (e.g. mean field theory, cellular automata) and concluded that the existence of robust scaling rules in spatially patchy systems remains an important open question. Dennis Mollison (Heriot-Watt University, Edinburgh, UK) provided a complementary overview of spatial models in epidemiology.

Sheelagh Lloyd (Cambridge University, UK) concentrated on how the environment influences the magnitude of immune responses. Developing a quantitative theory incorporating acquired immunity is hampered by the immensely complex natural history of such responses. Ecology influences the magnitude of the immune response; abiotic stress or resource limitation often suppress the immune system, and concurrent infections by different disease organisms may prevent an effective immune response. Bryan Grenfell also examined acquired immunity, but in the context of incorporating this biological factor into host–macroparasite models. He stressed that integrating acquired immunity into population models forces one to consider age effects, typically leading to very complex models that must be studied by simulations. He illustrated this approach with a model for a directly transmitted gastrointestinal nematode of sheep. Preliminary intriguing results from this system suggest that observed reductions of parasite aggregation with host age may require intrinsic differences between hosts. This modeling exercise crisply illustrates how theoretical work on infectious diseases can suggest new directions for empirical studies.

The final two speakers concentrated explicitly on the importance of genetic heterogeneity and multispecies interactions. Michael Begon and Roger Bowers (both of Liverpool University, UK) observed that one must move beyond single host–single pathogen dynamics to characterize fully many empirical systems. They reviewed in detail theoretical studies of host–host–pathogen and host–pathogen–pathogen systems, which to date emphasize the traditional community ecology problem of species coexistence. For instance, cross-species infection opens up an avenue for the indirect competitive exclusion of one host by another. Whether this occurs, rather than, say, coexistence, depends on both the detailed pattern of transmission and the magnitude of other, nondisease regulatory factors. The coexistence of multiple pathogens or parasites within a single host population requires either direct interference among pathogen strains, or independently aggregated parasite attacks. Begon and Bowers noted that these clear theoretical expectations have not yet been scrutinized in any but the most general way in empirical systems, and that the analysis of multiple-pathogen systems will require a better mechanistic understanding of within-host dynamics.

A final insightful talk by Curt Lively (Indiana University, Bloomington, USA) and Victor Apanius (Leiden University, The Netherlands) examined the genetic counterpart of the species coexistence problem, namely the maintenance of genetic variation. The traditional view since the time of Haldane has been that parasitism generates a form of frequency dependence that promotes the maintenance of variation. Lively and Apanius cautioned that a number of factors lead toward allelic fixation; these include long time lags, strong fitness effects, strongly oscillatory dynamics and density effects in transmission. Thus, the very factor that is believed to make parasites important in the evolution of sex – strong negative fitness effects on hosts – also makes it more difficult for genetic diversity to be maintained. This effect tends to vitiate the selective value of outcrossing. Despite this cautionary note, their review of the available evidence strongly suggests that parasites do play a significant role in maintaining sex, recombination and genetic diversity; Lively and Apanius argued that migration and truncation selection might be involved and need to be considered in future empirical and theoretical studies of host–parasite coevolution.

Following these stimulating formal lectures, the meeting participants divided into working groups. One group focused on microparasite dynamics. Pathogen persistence emerged as a central theme. Current models have difficulty in explaining persistence of disease between epizootics without invoking unrealistically low levels of infection (e.g. a fraction 10^{-18} of a fox population in a rabies model). There was general agreement that, although many (if not all) of the likely mechanisms for parasite persistence (e.g. spatial structuring, carrier states, vertical transmission and alternative hosts) were known, there were few if any empirical cases where parasite persistence was well-understood. A consensus emerged that it would be valuable to mount a determined attack on the problem of parasite persistence, starting with thoroughly studied diseases such as rabies. This group also emphasized that the role of parasites in regulating their hosts is still an open question, and that the issue of population limitation and regulation needs to be examined in a more inclusive community context (e.g. alternative hosts).

A second group was concerned with macroparasites and concentrated more on characterizing current deficiencies in modelling approaches, and in particular on the development of tractable methods for generating parasite distributions as an integral part of the model (rather than superimposing aggregated distributions, *ex cathedra*). This group also emphasized the problem of dealing with the complexities of environmentally modulated acquired immune responses without drowning in a sea of detail, and further concurred with the first group in noting the need for a systematic exploration of multispecies effects.

The third group dwelt on spatial dynamics. It was agreed that spatial models usefully focus attention on critical questions about mixing and local density that are swept under the rug in nonspatial disease models.

A number of questions were raised. How does one gauge the spatial scales in both models and field studies that best permit one to understand particular systems? Given that multiple scales matter (e.g. one for dispersal, another for transmission), how can a hierarchical structure be sensibly integrated into models? When can spatially explicit models be replaced by appropriately parameterized nonspatial models? Can spatial disease models be used to help design field sampling programmes? These issues are likely to be central in future efforts to understand disease dynamics in natural populations.

The final group faced a twofold mission. What is the relevance of genetics for epidemiology? Conversely, what are the evolutionary consequences of host–parasite interactions? Because these two short questions blithely span a daunting swathe of the biological sciences, this group made no pretence at being comprehensive, but instead focused on a set of general questions worthy of concerted attention. A sample of these is as follows. (1) Are there any novel epidemiological patterns that result from the existence of genetic variation? (2) Introducing genetics into ecological models, or ecology into evolutionary models, greatly increases the dimensionality of both; are there ways to ward off this proliferation of parameters, without losing the essence of the connection? (3) How can complexities of the immune system be incorporated into models of host–parasite coevolution, and are there useful parallels to be drawn with models in behavioral ecology (e.g. learning models in foraging theory)? (4) How is the evolution of virulence influenced by dynamical instability? Are there systemati differences between epidemics and endemic infections as evolutionary forces?

Judging from this meeting, the field of wildlife disease epidemiology is in an exciting growth phase. It is likely that in coming years host–parasite interactions will increasingly emerge as playing a role in practically all areas of wildlife ecology and evolutionary biology, ranging from diet choice and reproductive behavior to population regulation, geographical range limitation and the determination of community structure. Some important directions of research in infectious disease epidemiology discussed in the meeting (e.g. the role of spatial dynamics in parasite persistence, the elucidation of indirect interactions in multispecies host–parasite assemblages) exemplify conceptual developments within the ecological sciences as a whole. The Newton meeting provided an invaluable function by crystallizing attention on many of the crucial empirical and theoretical questions which now face us.

References

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Coral Reefs: Health, Hazards and History

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TROPICAL CORAL REEFS cover barely 0.18% of the world's oceans[1]. The great scientific interest they elicit might thus seem totally to outweigh their importance. Historically, much of this interest can be attributed to their striking biological richness. For example, the diversity of fishes on reefs is some two orders of magnitude higher than the oceans' average[2]; at the level of Orders and Phyla, coral reefs are probably the most diverse ecosystem on the planet.

Recently, the world's attention has been focused on reefs for more pragmatic reasons: their role in a changing climate. The global phenomenon of coral bleaching (loss of symbiotic algae from tissues, often followed by colony death), recorded with increasing frequency since the 1960s, has been suggested as a harbinger of global warming[3]. Additionally, the fates of entire island nations may depend on the ability of reefs to keep pace with sea-level rise by growing upwards. Recent widespread reports of coral reef deterioration and death have therefore sounded alarms in scientific circles and beyond.

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Proclamations of the impending demise of coral reefs are not new. In the 1960s massive outbreaks of the coral-eating crown-of-thorns starfish on the Australian Great Barrier Reef caused widespread prophecies of doom. Thirty years the later, the Great Barrier Reef and the crown-of-thorns are still there. To cut through the rhetoric and decide just how coral reefs really are faring in the 1990s, 125 reef scientists and managers gathered in Miami in June to examine the evidence at a conference entitled 'Global Aspects of Coral Reefs: Health, Hazards and History'. In a welcome break from scientific norms, the organizers resurrected the original meaning of the word 'colloquium' and most of the meeting was centred around discussion of 62 preprinted case histories documenting the condition of the world's reefs. To sharpen the minds of the participants, the final two days of the five-day meeting were thrown open to press and public. The meeting sought answers to three critical questions: (1) are reefs worldwide in decline? (2) what are the major hazards to reefs? (3) how will global warming affect reefs?

The geological record shows that reefs have survived through many changes of climate. Arthur Bloom (Cornell University, Ithaca, NY, USA) showed how, in the Holocene, reefs had experienced rates of sea-level rise an order of magnitude faster than those predicted for the coming 50 years. Ann Budd (University of Iowa, USA) noted that the two major periods of extinction in Caribbean reef corals since the closure of the Isthmus of Panama, 3.5 million years ago, did not coincide with periods of rapid climate change. Clive Wilkinson (Australian Institute of Marine Science) summed up the consensus among participants saying that 'coral reefs will probably enjoy a little bit of climate change'! However,