Part IV
Conclusions

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24 *Ways forward in the study of primate disease ecology*

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Introduction

The devastating impacts that diseases like Ebola have had on both human and wildlife communities (Walsh *et al*., 2003; Leroy *et al*., 2004) and the immense

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social and economic costs associated with viruses like HIV (Piot *et al*., 2004) underscore the practical and societal necessity of understanding infectious diseases in primates and the dynamics of their zoonotic transmission. Given that monkeys and apes often share parasites with humans, understanding the ecology of infectious diseases in non-human primates is of paramount importance to human health planning. This is well illustrated by the HIV viruses, the causative agents of human AIDS, which evolved recently from related SIV viruses of chimpanzees (*Pan troglodytes*) and sooty mangabeys (*Lophocebus atys*; Hahn *et al*., 2000), as well as by outbreaks of Ebola hemorrhagic fever in African communities, which trace their origins to zoonotic transmissions of Ebola virus from local apes (Formenty *et al*., 1999). Another example that has a deeper evolutionary origin is malaria. DiFiore *et al*. (Chapter 7 this volume) reviews the impacts of malaria on human health and reports that there were 396 million cases of human malaria and 1.1 million deaths in 2001 due to infection by *Plasmodium falciparum* alone.

Infectious disease also poses significant conservation risks to non-human primate populations, many of which are already threatened or endangered by habitat loss and/or hunting. For example, evidence indicates that Ebola virus outbreaks have contributed to the reduction of ape population densities by more than 50% over a broad geographic region between 1983 and 2000 (Walsh *et al*., 2003, 2005). Such examples clearly illustrate the importance of understanding disease dynamics from a conservation perspective. In addition, although humans have always shared habitats with non-human primates, the dynamics of human–primate interactions are changing radically (Chapman & Peres, 2001). Within the last several decades, humans have been responsible for irreversible changes to primate habitats. Most primates today live in anthropogenically disturbed habitat mosaics of farmland, human settlements, forest fragments, and isolated protected areas. As anthropogenic habitat change forces humans and primates into closer and more frequent contact, the risks of inter-specific disease transmission increase (Daszak *et al*., 2000, see also Wolfe and Switzer, Chapter 17, this volume).

When we (Huffman and Chapman) started work on this edited volume, we began with an appreciation that information about diseases from conservation and human-health planning perspectives had stimulated a considerable amount of recent research and the field of primate disease ecology was rapidly gaining momentum. To verify this impression we conducted a simple, but telling, analysis using the Web of Science. We simply put the terms "parasite" and "primate" into the topic search and quantified the number of articles published each year containing these terms. Figure 24.1 illustrates that our impression that the field was gaining momentum was correct. If one reads the titles of these articles, it is also clear that growth in the field has been dominated by

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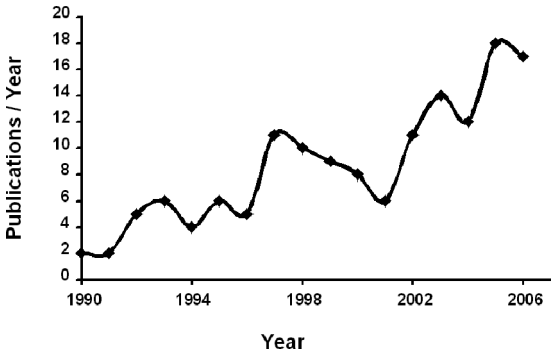

Figure 24.1. The number of publications per year revealed by a search using the Web of Science, with the search terms "parasite" and "primate."

studies of disease ecology and evolution, and that taxonomic research has continued to grow steadily, but at a much slower rate. The complexity of taxonomic identification, as illustrated by the chapters of Gasser *et al*. (Chapter 3) and Hasegawa (Chapter 2), indicates that taxonomic studies are as needed as ecological and evolutionary ones.

While authors of this contributed volume were not asked to identify the agenda of the next generation of research, their results clearly cannot help but do so. To build on or crystallize this agenda, rather than reviewing what has been presented in each chapter or presenting an overview, we would like to build on the impression that this area of study will soon see great advances. Thus, we will present some ideas/approaches that we hope will guide an insightful development of studies in this area. To do this, the authors of the edited volume (Huffman and Chapman) brought together three additional colleagues who we knew would bring yet more diversity to the perspectives and approaches and asked them to help to illustrate the ways forward for investigations into the field of primate disease ecology.

Methods and molecules

A number of chapters in this volume speak to a vital need to improve the "tool kit" that we use when asking questions in primate disease ecology. First, in the chapter by Greiner and McIntosh, it becomes clear that a variety of traditional methods can be used to characterize parasite infections, but that the selection of methods depends on the species of primate, the parasites in question, and logistical issues. This notion is amplified in Hasegawa's Chapters 2. It becomes clear in later chapters (Dupain *et al*. (14), Vitazkova (18), Weyker (20), and

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Chapman *et al*. (23)) that there is little consistency in the methods used and that the field in general needs to be able to answer some specific methodological questions concerning how to characterize infections in such a way as to make comparable cross-study comparisons, particularly when comparisons are made at the population level.

To illustrate the complexity of this issue, let us consider one question: "How many samples should be examined to contrast two groups or population?" We need to be able to answer this question for many of the inquiries being repeatedly raised, such as how does a specific type of anthropogenic habitat disturbance influence the parasite infections of a particular primate or what is the effect of seasonality across populations of any given parasite infection. If all animals are individually recognizable then this question becomes somewhat simpler and there is research suggesting how many samples of a single individual are needed to describe an infection (Muehlenbein *et al*., 2004). But when populations are being compared and individual identification is not possible, the way forward is more challenging. It is always important to avoid sample bias, using the same methodology over similar periods of the year for similar durations for each population being compared (Huffman *et al*., Chapter 16, this volume). Where individual identification is possible, the rules for sampling single individuals need to be applied, doing ones best to monitor all individuals of a population consistently over time. It is common for comparative studies to take a "slice of the pie approach," sampling group(s) for a short period. This runs into difficulties, especially because seasonality of infection can bring highly contrasting results from the same population depending upon when you sample it (Hernandez *et al*., Chapter 19, this volume; Huffman *et al*., 1997) or from different populations if the sampling period is too short, and or the periods sampled significantly differ with respect to season.

On the surface the question of how many samples to collect would appear to be a simple one and the answer would be that one would attempt to obtain as many samples as logistically possible. However, the situation is not so simple. There must be a compromise between increasing the sample size and doing assessments of infections using different techniques (e.g. sedimentation, flotation). In the Chapman lab at McGill University, performing both sedimentation (all of the sediment) and flotation takes 6 to 15 times longer than performing flotation alone. A means of increasing the chances of detecting differences among groups or populations would be to decrease variance in sampling unrelated to the question being asked. One might choose only to collect samples at a specific time of the day if diurnal variation in egg output were high, as is the case for many protozoan species (Ezenwa, 2003), or one might sample only from specific age/sex classes if variation were high along this dimension. Unfortunately, little information exists on the magnitude of diurnal variation or

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variation among age/sex groups for most primate or parasite species. Providing better guidelines as to what methods are best in what situations, will greatly increase our ability to compare studies and make generalizations.

A number of chapters in this volume also illustrate the improvement that comes from adding molecular approaches to our "tool kit." This is nicely illustrated by Gasser *et al*.'s Chapter 2 describing how molecular methods were needed to determine that *Oesophagostomum bifurcum* in humans is genetically distinct from that harbored by some non-human primates, that these genetic variants have distinct transmission patterns, and that non-human primates are not a source of human oesophagostomiasis. Thus, it is clear that molecular studies have contributed in substantial ways to our understanding of infectious disease in primates, and the past utility of molecular laboratory and analytical tools bodes well for their future in the field. Genetic data have, to date, been used in studies of primate disease ecology primarily for two related purposes: molecular subtyping and molecular epidemiology. Both uses have improved our understanding of primate infectious disease transmission dynamics.

The use of genetic data to subtype primate pathogens has not been merely an exercise in molecular systematics. Rather, studies examining the intra-specific molecular taxonomy of primate pathogens have shed light on key hypotheses relevant to the transmission of microbes between non-human primates and other species (including humans). For example, mountain gorillas in Bwindi Impenetrable National Park, Uganda, can be infected with the gastrointestinal protozoa *Cryptosporidium parvum* and *Giardia duodenalis* (Nizeyi *et al*., 1999, 2002). Both of these pathogens have genetically distinguishable subtypes (traditionally "genotypes" in the case of *C. parvum* and "assemblages" in the case of *G. duodenalis*) that are associated with transmission among ecologically distinct sets of hosts (Ey *et al*., 1997; Peng *et al*., 1997). Graczyk and colleagues (Graczyk *et al*., 2001, 2002) used molecular subtyping methods to demonstrate that Bwindi gorillas are infected with "genotype 2" *C. parvum* and "assemblage A" *G. duodenalis*, both of which are associated with zoonotic and anthroponotic transmission (as opposed to other subtypes that are considered to be more host restricted). Molecular subtyping of these protozoa in gorillas therefore strengthens the hypothesis that humans, and perhaps their livestock, represent a source of pathogen transmission for wild gorillas. Molecular data can also weaken hypotheses about the importance of interspecific transmission. Case-in-point are studies by Gasser and colleagues of the clinically relevant "nodule worm," *Oesophagostomum bifurcum*, in Togo and Ghana (Gasser *et al*., 2006, Gasser *et al*., Chapter 3, this volume).

Molecular epidemiology can be viewed as an extension of molecular subtyping, in that it attempts not only to classify pathogens, but also to infer patterns

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of transmission from degrees of genetic similarity among isolates and/or their phylogenetic history (see DiFiore *et al*., Chapter 7, this volume). In studies of primate pathogens, the molecular epidemiological approach has been applied most widely to viruses. Phylogenetic analyses based on nucleotide sequences, for example, led to the surprising discovery that both HIV-1 and HIV-2 originated from separate (and, in the case of HIV-2, multiple) cross-species transmissions of simian immunodeficiency virus from non-human primates to humans (Gao *et al*., 1992, 1999). Wolfe and colleagues examined other primate retroviruses (simian foamy viruses and simian T-cell lymphotropic viruses) and noted substantial "mixing" of human and non-human primate retroviral sequences on phylogenetic trees – a key observation supporting the concept of "viral chatter," or the continuous introduction of endemic primate retroviruses into "high risk" humans, such as hunters (Wolfe *et al*., 2005; Wolfe & Switzer, Chapter 17, this volume). In combination with careful analyses of geographic associations among outbreaks, molecular sequence data from Ebola virus isolates in Gabon and Republic of Congo demonstrated that localized epidemics in people probably originated through contact with different infected ape or duiker carcasses (Leroy *et al*., 2004).

Viruses with RNA genomes lend themselves well to molecular epidemiological studies of transmission, due to their high evolutionary rates (Domingo *et al*., 1996). However, molecular epidemiological methods for inferring transmission among pathogen isolates need not be restricted to such pathogens. Goldberg *et al*. (2007) used a PCR-based DNA fingerprinting approach in combination with population genetic and phylogenetic analyses to infer transmission of the common gastrointestinal bacterium *Escherichia coli* between humans and chimpanzees. The study inferred high rates of bacterial transmission between chimpanzees and people employed in chimpanzee research and tourism in Kibale National Park, Uganda, and showed that chimpanzees harbor bacteria that are resistant to multiple antibiotics used by local people. Novel laboratory and analytical methods may need to be developed for studies of bacteria and other microbes that evolve more slowly than viruses (e.g. Goldberg, 2003; Goldberg *et al*., 2006).

Molecular methods themselves are evolving at a remarkable rate. Techniques such as real-time quantitative PCR (Higuchi *et al*., 1993), for example, now significantly speed the diagnostic process and can indicate not only whether a specific pathogen subtype is present in a clinical sample, but also at what concentration. The rapid and cost-effective sequencing of entire eukaryotic genomes is now becoming a reality (Margulies *et al*., 2005). Refinement of molecular laboratory methods and associated analytical strategies should only enhance future studies of the transmission dynamics of a broad taxonomic range of primate pathogens.

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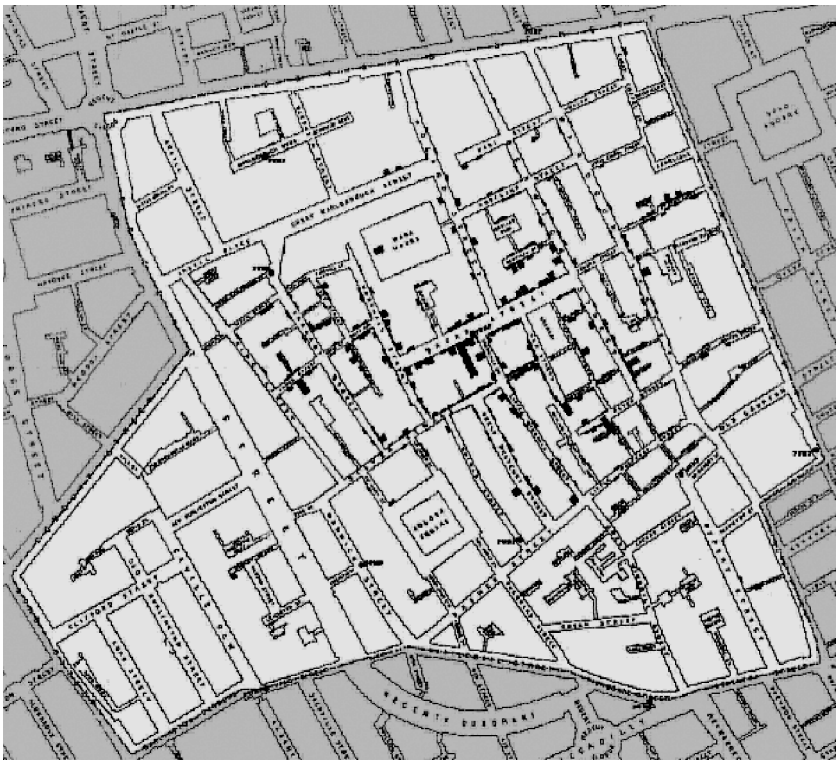

Figure 24.2. Modified map from John Snow's (1855) map of Cholera incidences (each case is shown as a bar) and location of hand pumps.

Combining spatial and temporal considerations

The collection of research chapters in this volume illustrated that while disease-transmission and agent-based modeling frameworks (Nunn, Chapter 5 this volume) are exciting trends in the study of infectious diseases, researchers often appear unintentionally to exclude the spatial dimension (Dietz & Hadeler, 1988; Blower *et al*., 1995; Oli *et al*., 2006). Perhaps because temporality is implicit in modeling, space is not (even typically within agent-based models). Yet, very early studies of infectious diseases recognized the importance of space. For example, John Snow was critical in tracing the origins of a cholera outbreak to a hand-pump and is considered a precursor of statistical mapping methods (Figure 24.2; Snow, 1855). Here we suggest that one way forward that will bring new insights into primate disease ecology will involve spatial epidemiology,

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with its emphasis on space and spatially explicit models of disease vectors and transmission (Elliot & Wartenburg, 2004).

Two parallel developments are likely to impact the growth of spatially explicit models in epidemiology significantly: (1) the ubiquitous availability of both spatial data and digital earth technologies (Gore, 1998; Masser, 1999; Fonseca *et al*., 2002), and (2) a growing awareness of basic spatial operations available within traditional GIS software (e.g. overlays and buffers; Xiang, 1993).

A rich repertoire of spatially explicit data is made available in the USA (and worldwide) through publicly funded data-gathering and dissemination initiatives. The 2000 Shuttle Radar Topography Mission (SRTM), for example, provided high-resolution topographic information (90 m $\times$ 90 m) for the terrestrial surface worldwide. Similarly, the Landsat series of satellites have obtained large amounts of multi-spectral information since 1972, having accumulated 1.7 million scenes and over 630 terabytes of data (http://www.landimaging.gov/about.html). These datasets are complemented with satellite data collected by other nations, such as Canada's RADARSAT, Europe's SPOT, and India's IRS series satellites. In addition, many countries have available large socio-economic and biophysical datasets and many are freely available for download. Digital Earth technologies are proving to be increasingly popular in the public domain, with technologies such as Mapquest, Yahoo Maps!, and Google Earth fast becoming part of daily life. The growth of such technologies, in combination with the existing repertoire of spatially explicit data, has the power to transform knowledge about the etiology of an infectious disease. For example, Butler (2007), a reporter for *Nature*, generated an animated sequence of H5N1 outbreaks using Google Earth, which helped illustrate the spatiotemporal dynamics of disease occurrence between 2003 and 2006 (Figure 24.3). Indeed, numerous blogs on the internet refer to this map, an indication of its power to influence both the scientific and non-scientific audience.

The next natural step is the use of analysis that spatially integrates environmental (e.g. elevation, landuse landcover) and socio-economic (e.g. income, population density) factors to understand causal mechanisms of infectious disease transmission. The two common GIS-based tools of "overlay" (that identifies relationships between two variables in the same spot) and "buffer" (that identifies relationships between a variable and another (or itself) as a function of distance) can play an important role in explaining disease etiology. Figure 24.4 is a reproduction from the journal *Emerging Infectious Diseases* that uses the "overlay" functionality of a standard GIS package (ArcGIS 9.2) to identify the predominance of certain eco-regions as hotspots for occurrence of bird flu (H5N1) (Sengupta *et al*., 2007). Only 25 ecoregions, representing 8.8% of the terrestrial surface area, accounted for 2407 (76.8%) of the reported H5N1 cases

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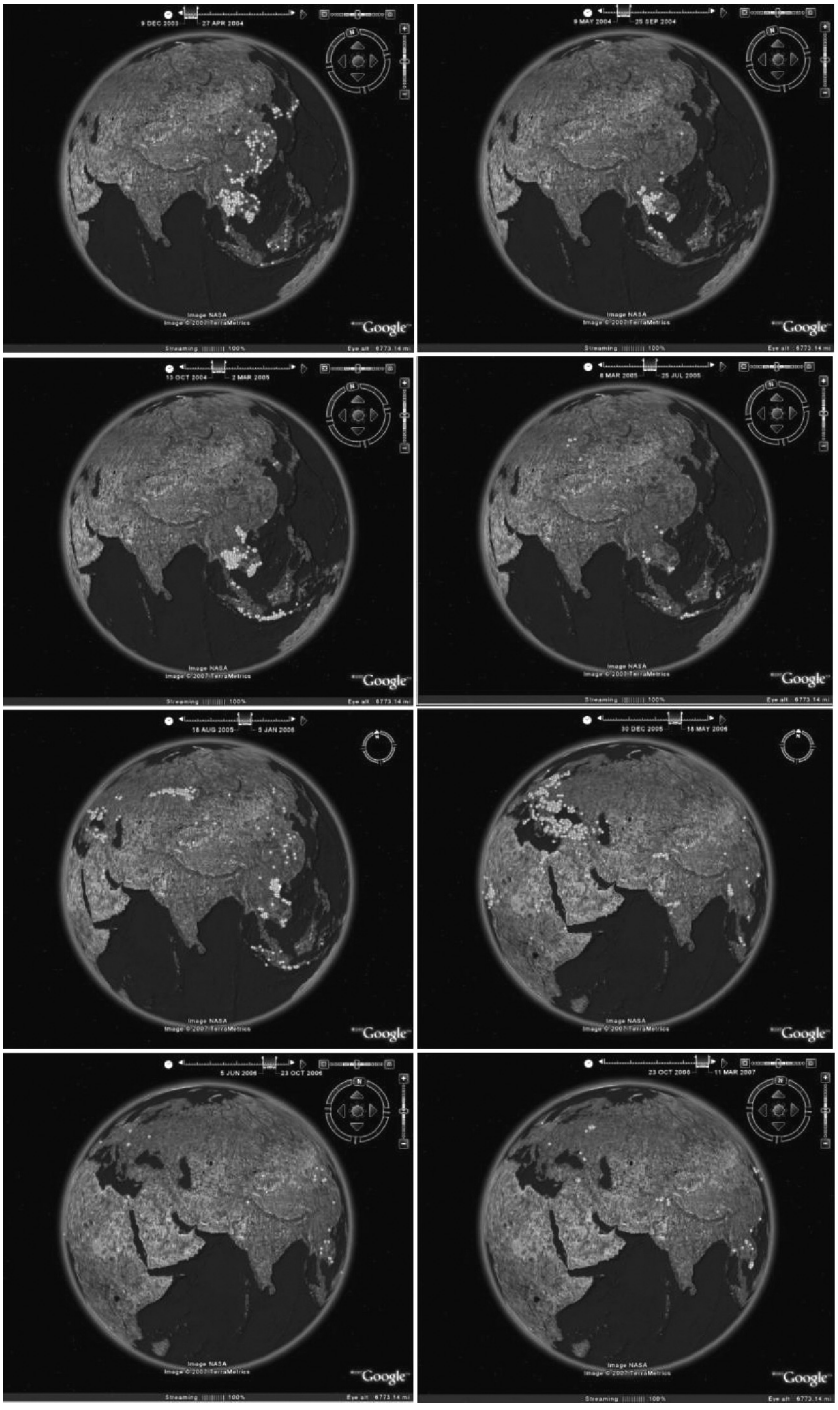

Figure 24.3. Snapshots of Declan Butlers' maps of Bird Flu occurrences between 2003–2006.

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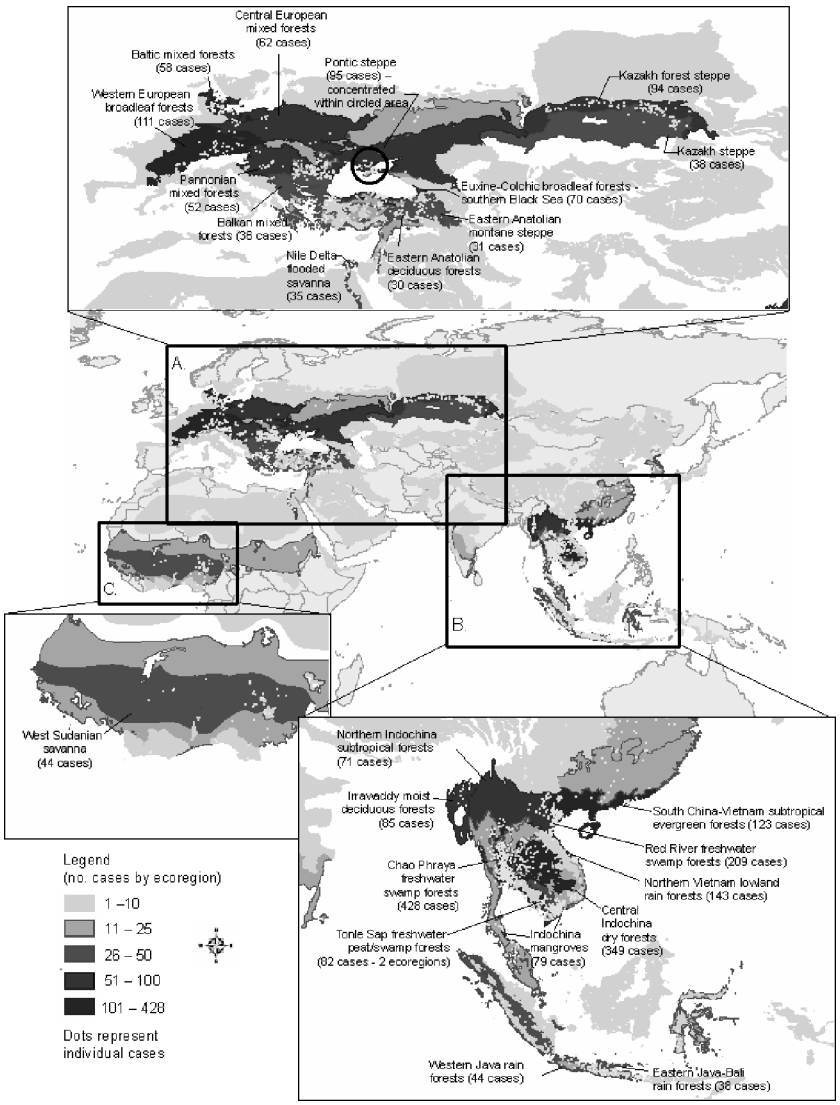

Figure 24.4. Map showing the 25 ecoregions with large numbers of avian influenza cases (November 2003–November 2006). (A) Eurasia; (B) Southeast Asia; (C) Africa. Dots represent individual cases (figure reprinted with permission © Centers for Disease Control and Prevention. *Emerging Infectious Diseases*, **13**, 1269–1271).

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between 2003 and 2006. Greater understanding of such techniques could prove useful for locating causes of disease transmissions that may have ecological underpinnings (Wolfe *et al*., 2005; Despommier *et al*., 2006). In effect, the consideration of multiple variables at the same location, or the values of the same variable across several locations, can be seen as the first step towards developing a true spatial process model to represent a phenomenon that varies through space and time.

Most infectious diseases do indeed vary in this manner. Infection between primate populations, for example, are a function of the presence of parasites within host populations, its prevalence, the interaction of infected individual(s) with other members of the population (or other populations), and a number of spatially explicit environmental variables (Thomson *et al*., 2000). An agent-based model, such as the one described by Nunn (Chapter 5 this volume) is able to capture the spatial and temporal dimensions of infectious disease transmission if and only if the spatial dimension has been explicitly specified within the model. Further, most models are univariate with respect to the environmental variables. This is likely not true for most infectious diseases. Research by Thomson *et al*. (2000) on eyeworm identified that variation in the prevalence of eyeworm among villages in west and central Africa can be explained by a number of environmental variables that included land cover, rainfall, temperature, topography, and soil (although one has to be careful here as some of these variables may co-vary).

Finally, one cannot underestimate the importance of integrating the temporal and spatial dimensions. Jacques (2000) correctly criticizes GIS for its static view and lack of process-based disease models. The development of temporal data storage and retrieval methodologies, and models that translate spatio-temporal data into meaningful health outcomes, are both necessary and legitimate demands to enable GIS to properly support spatial epidemiology (Kaur *et al*., 2004). However, just as temporality is a legitimate demand, so too is the need to incorporate the ability of process-based epidemiological models to access, retrieve, manipulate, and identify relationships between multiple environmental variables stored in spatially explicit datasets. This will truly put space alongside time in epidemiological models and help advance the field forward.

Scaling up: the need for models

To understand the role of disease in the persistence of primate populations, we believe that it will be extremely insightful to create appropriate and flexible

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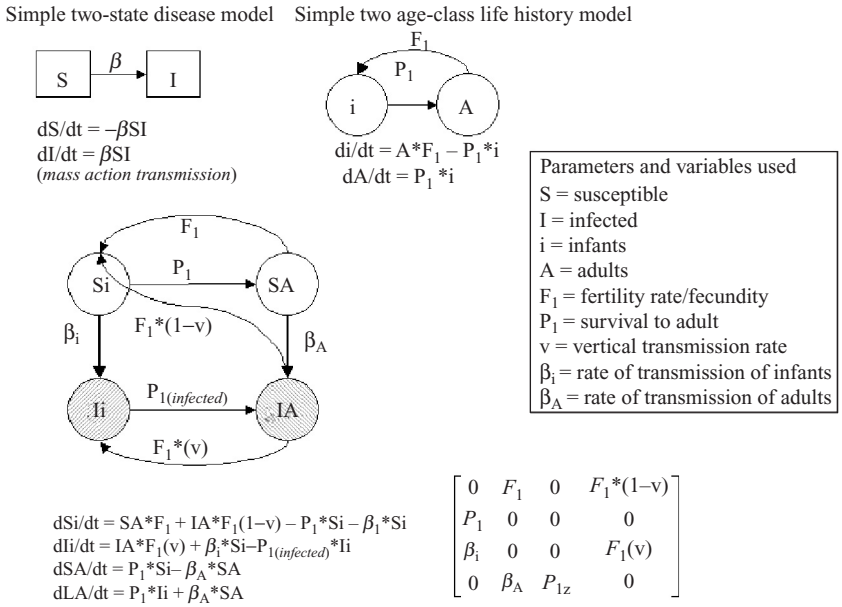

Figure 24.5. In this example, we demonstrate how an age-class model can be overlaid with a two-state transmission model to generate a framework for describing the transmission dynamics of a simple directly transmitted disease within a demographically explicit population model. The scaling up of this type of model to multiple age classes and multiple disease states is straightforward.

quantitative frameworks. Integrating population processes into models of disease impacts can help not only to guide data collection, but also lets the user explore how disease might interact with other forces, such as environmental fluctuations or climate change.

Simple process models of population increase and decrease (discrete or continuous equations) can be modified to include additional mortality due to disease impact, or adjusted to examine resource dependent responses. However, for a taxon in which social interactions, group living and population structure are often quite well known and definitely of interest, slightly more complex (but not complicated) models may be more appropriate. For example, a simple age-based demographic model of a primate population can be used to project the growth or decline of a population (Figure 24.5). Age-specific rates of survival and reproduction are the key components in traditional life tables, which for many primates are available in the literature (e.g. Kappeler & Pereira, 2003). These can be used in a deterministic matrix model for demographic projection

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to estimate whether the population is growing, declining, or remaining stable (Caswell, 1989; Ebert, 1999).

Matrix life-history models also allow the examination of which age classes are having the most impact on the trajectory of the population. This is at the core of many population viability analyses (PVAs) that inform conservation actions and policies. The sensitivity of the population trajectory to certain age classes can help reveal which parts of the population's structure needs protection to improve the recovery of the whole population. One highly cited example of the utility of a PVA is in the protection of loggerhead sea turtles (*Caretta caretta*), whose population was threatened both by beach use, endangering the eggs and hatchlings, and by adults drowning in fishnets. In their classic papers, Crouse and Crowder and colleagues (Crouse *et al*., 1987; Crowder *et al*., 1994) showed that adult survival was in fact more important to long-term population dynamics than protection of the younger life stages, and this led mandatory turtle excluder devices in fishing nets – which has been adopted globally for many turtle populations. In developing models for primates this will be important in the cases where diseases hit one component of a population and not others. In the development of population models for viability analyses it became apparent that stochasticity in both demographic and extrinsic inputs can drive a population dramatically in the short term, relative to longer-term effects in larger populations. This is important when considering primate populations, as many remnant populations are small or fragmented, meaning that population biology of small numbers is at play.

The impact of disease on a primate population can be accessed through models at many different levels. If the state of an infected individual is identifiable (observations of physical manifestations of infection, such as high parasite loads, lesions, lethargy) and can be associated with differential survival, it is easy to see how this can be incorporated into a sensitivity analysis in a PVA type of framework. This is particularly interesting if the disease impact changes with age and/or sex class of the individuals. For example, a disease causing high infant mortality will create a very different population signal than one that affects dispersing age animals (Oli *et al*., 2006). Even in the absence of empirical data, exploring potential theoretical impacts of disease by examining the sensitivities of different age classes can help inform the potential risks to that population.

It is also important to consider the potential impact of a disease due to its intrinsic traits. Some diseases, such as viral hemorrhagic fevers (e.g. Ebola and Marburg), cause mortality quickly, which may not allow rapid spread through a population, due to a short infectious period and a quick removal of the infected individuals from the population. In contrast to the "fast" dynamics of directly transmitted diseases with high mortality impacts, it is also important to

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consider the more prolonged role of chronic disease and of indirect transmission. Chronic diseases tend to lead to issues not of mortality, but of morbidity; these "slower" diseases may render individuals weaker, or less resilient in the face of other extrinsic shocks to the system. One characteristic of chronic weak states is the reduced ability to reproduce, or provide sufficient resources for offspring. Another factor is that weak or frail individuals are less likely to survive during low resource periods, and are less likely to rebound from short-term environmental shocks. The role of these life history and resilience impacts can be modeled quite simply, using state-dependent frameworks, or simple simulations in matrix frameworks.

Since primates characteristically live in groups, the structure of the population can act to "slow down" disease spread, if individuals get infected and die before emigrating from the group and thus there is no spread. If a disease has a long chronic phase or is infectious for a long time, the structure of a population becomes less relevant, as the between-group travel time becomes shorter than the duration of infectiousness and the disease can spread. The relationship between population structure and direct disease spread has been explored by Cross *et al*. (2005) within a theoretical framework, and the ramifications of different structure on disease due to social system differences in primates and other animals has been explored (Thrall *et al*., 2000; Altizer *et al*. 2003; Ryan *et al*., in prep). In particular, Thrall *et al*. (2000) demonstrate that the role of social structure and the sexual transmission mode for disease has varying effects on the prevalence and extent of an outbreak for different sexes in the population. Social structure and metapopulation structure can be modeled implicitly in deterministic frameworks, simply by continuing the 'compartmental' class structure into larger dimensions of a matrix. However, it will rapidly become apparent to the user that for primates, more realistic depictions of spatial structure may provide better information. Overlaying disease impacts on the individual and agent-based models as described by Charles Nunn in this book is another step, ranging in complexity from individual states (diseased or not) to explicit dynamics of the parasites themselves.

Explicit modeling of host–parasite dynamics wherein external phases of parasites are included is fairly well established (Hudson *et al*., 1992); however, the inclusion of multiple hosts and multiple parasites into models leads to quite complicated and data-hungry frameworks. The value of agent-based models with high levels of complexity is the potential to reveal novel properties of the system, commonly referred to as "emergent properties." These properties can demonstrate unexpected drivers in the system because of complex interactions that are not captured by more simple descriptions of the system. Importantly, the flexibility of parameters – wherein the user can simulate a range of values – provides a means of sensitivity analysis, and the ability to explore which

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components of the system are contributing to the drivers. This is a less precise means of examining sensitivity than the more analytically tractable nature of eigen value methods in matrix models (Mesterton-Gibbons, 2000; Yearsley, 2004); nonetheless, it is similarly useful.

Conclusions

Primate parasite ecology is in a period of rapid growth and development and it is our opinion, and we hope that the chapters in this edited volume provide a convincing case, that research in this field offers a great deal to enhance our understanding of this fascinating group of mammals and this diverse group of pathogens. We view that by employing the tools and approaches such as those outlined in this last chapter, unique insights will be derived moving forward this field dramatically.

Given the plight of primates in the tropics, we wish to end our volume by reminding the reviewer of the typical state of affairs of primate conservation and emphasizing the role parasitism can play in enhancing the effects of other factors. The tropical forests that most primate species occupy are undergoing rapid anthropogenic transformation. Cumulatively, countries with primate populations are losing approximately 125 000 km^2 of forest annually (Chapman & Peres, 2001). Other populations are being affected by forest degradation (logging and fire) and hunting (Oates, 1996). This is the typical means of considering conservation threats: in terms of readily apparent factors such as habitat loss/degradation and over harvesting. However, when situations are considered in more depth it often becomes apparent that population declines are associated with a complex set of interactions among the environment and biota, which in combination overwhelm the populations' ability to withstand change. It is clear that primate pathogens are important members of this complex set of interactions. For example, Ebola has clearly been related to the dramatic decline in gorilla populations (Walsh *et al*., 2003; Walsh *et al*., Chapter 8, this volume; Chapman *et al*., 2005), yet there is also evidence that Ebola outbreaks occur in habitats currently undergoing fragmentation in areas that are a mosaic of forests and open habitats (Morvana *et al*., 1999). Similarly, in forest fragments, nutritional stress is correlated with high gastrointestinal parasite infections and population declines (Chapman *et al*., 2006).

Conservation biologists and managers interested in protecting specific species or ecosystems benefit from understanding the interactions that characterize those ecosystems, because by doing so it becomes possible to predict population or ecosystem changes that may not be intuitively obvious (e.g. a population will decline if it is over-harvested). The field of disease ecology is

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an exciting area where by understanding how changes in the ecosystem, like climate change or habitat fragmentation, can alter host–disease interactions we can provide conservation biologists with such non-intuitive tools for species protection. Furthermore, by understanding how changes in host–disease interactions can cause population declines, we need not simply respond to change, but we can be proactive and manage the system to prevent declines.

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