Ecological theory to enhance infectious disease control and public health policy

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Through the work of international public health organizations and advancements in the biological and technological sciences, substantial progress has been made in our ability to prevent, control, locally eliminate, and in one case eradicate infectious diseases. Yet each successful control or local elimination has been met with the emergence of new pathogens, the evolution of novel strains, or different epidemiological circumstances that have limited or reversed control methods. To respond to the increasing threat of emerging infectious diseases and bioterrorism it is vital that we design and implement efficient programs that prevent and control infectious pathogen transmission. The theoretical tools of ecology and epidemiology may be the cornerstone in constructing future programs aimed at preventing and controlling infectious diseases throughout the world.

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By the second half of the 20th century, improved hygiene, advances in diagnostic methods, and the development of new drugs and vaccines allowed industrialized nations to greatly reduce the prevalence of many deadly infectious diseases. Yet today, infectious diseases remain the leading cause of death in the world. At the advent of a century threatened by bioterrorism, rapid population growth, extraordinary levels of poverty, and an apparent increase in the emergence of infectious diseases, the control and eradication of pestilence is far from complete. Now, perhaps more than ever, there is a need to incorporate the tools of ecology into the design of programs aimed at preventing and controlling infectious diseases. To date, numerous achievements in theoretical ecology have influenced the development and implementation of such programs (Table 1). Here, we discuss these theoretical advances and describe a number of cases where theoretical ecology has influenced the construction of initiatives to prevent or control infectious diseases around the world. Four of these cases are detailed in the text, but many equally important examples exist (Table 2; and see web-only material). Public health institutions and NGOs charged with the design and maintenance of health programs should capitalize on the insights provided by ecological theory, and theoreticians should expand their research into infectious disease ecology.

In a nutshell:

- Infectious disease control initiatives for humans, livestock, and wildlife have benefited from theoretical insights from ecology
- As new infectious diseases emerge and old ones spread, the demand for surveillance, healthcare, vaccine development and distribution, and disease prevention will increase
- Although molecular biology and genomics are important in understanding the origins and pathologies of infectious agents, analysis of infectious disease ecology and parasite–host interactions, including pathogen transmission and disease manifestations, are central to successful control strategies

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Conceptual frameworks

Currently, three overlapping conceptual frameworks dominate the study of infectious disease emergence and spread. The medical community largely adheres to the individual, patient-based model which emanated from the revolutionary germ theory articulated by three eminent medical scientists, Pasteur, Koch, and Ehrlich. Within this framework, disease is viewed as the product of a particular pathogen infecting a particular patient. This view primarily focuses on the individual over the population, so the research strategy is driven by a desire to develop a drug that will cure the individual patient, or a vaccine that will prevent infection by the specific pathogen. This research community seeks to develop an intervention that targets the within-host pathogen population or mitigates the organism's pathogenic response to infection. This approach has had enormous success over the past century and is responsible for the eradication or management of many of the most ominous diseases in history.

Epidemiologists deviate from the individual–organis-

Table 1. Contributions of ecological theory to infectious disease control and prevention

Past

- Determining sensitivity in pathogen transmission cycles
- Determining rate-limiting steps in pathogen population growth
- Establishing effective and efficient vaccine strategies
- Understanding pharmacological treatment regimes
- Determining antimicrobial resistance among pathogens
- Establishing the spatial dynamics of pathogen spread
- Understanding host immune-system dynamics
- Determining the impact of host sub-population characteristics on pathogen spread
- Understanding critical community size and herd immunity

Present and future

- Using host demography and distribution to predict the emergence of infectious diseases
- Using ecological and evolutionary dynamics to predict pathogen–host shifts
- Using time-series analyses to predict temporal instability in disease epidemics
- Planning for biopreparedness
- Determining effective and efficient allocation of resources to disease surveillance
- Using "end-game theory" to determine the appropriate time to stop control programs

mic model, stressing the population-level pattern of infection within a system of interacting hosts and pathogens. Most frequently, epidemiologists attempt to uncover the factors that lead to statistical patterns of association between environmental, behavioral, and genetic influences and disease occurrence. The risk-factor approach is predominantly statistical and the research goal is most often the determination of an odds-ratio or relative risk statistic that predicts disease outcomes, given presumed causal factors. Risk factor analysis is important for management and decision making – if we know that a person is 17 times more likely to contract a sexually transmitted disease by one form of behavior over another, we can educate the population to behave in a manner that reduces that risk. This is important for policy, but we can only guess at the reasons individuals are less at risk from one behavior versus another.

Since the early 1980s, a complementary approach, articulated by ecologists, has focused on examining patterns of infectious disease occurrence not as a purely statistical pattern, but from a first-principles perspective of the natural ecological and evolutionary dynamics of host–pathogen interactions. Within this perspective, the pattern of case occurrence is the product of fundamental biological processes (eg mutation, gene flow and migration, contact and transmission rates) that, when modeled, can predict the time course of the infectious disease within a given population and how the pathogen will spread from source populations. The holy grail of this ecological–evolutionary approach is a mathematical model (usually a system of differential equations) used to capture the first-principle biological processes that generate disease patterns of occurrence in space and time. Since the system is based on underlying biological dynamics, we can not only predict but also generate a deeper understanding of the causal mechanisms underlying the disease processes.

These three approaches should not be viewed as sequential intellectual developments, but rather as coexistent, complementary frameworks, best used in concert. From an ecological perspective, risk factors are a kind of population heterogeneity. Thus, statistical analysis and odds-ratios can be used to identify probable causal forces that must be incorporated into first-principle models and can be used to help parameterize the biological processes embedded in an ecological dynamics model. Or, for instance, population genetics and dynamics models can be used to understand and predict the evolution of antimicrobial drug resistance that will then inform patient treatment. Bringing groups of practitioners together from each of these three perspectives is important.

Theoretical taxonomy of pathogens

The models developed by theoretical ecologists are based on quantifying underlying population biological processes. When faced with developing models to understand and explain the dynamics of new pathogens, ecologists are more interested in the number of infected individuals, the average age of infection, incubation time, contact rates between infected and susceptible hosts, and duration of immunity, than in the detailed mechanisms of pathogenesis. This focus has led to the development of a hierarchy of models that can be adapted to different pathogens and the complexities of host social behavior that drive transmission dynamics. Similarities in the basic ecology provide the basis for a "theoretical taxonomy" of pathogens that is mirrored by similarities in the mathematical models.

At the heart of the ecological taxonomy of pathogens is a split leading to two groups: microparasites and macroparasites. The microparasites, which include viruses, bacteria, protozoa, and prions, are typically studied using a series of compartmental models that characterize changes in the host population. First described by theoretical epidemiologists Kermack and McKendrick (1927, 1932, 1933), host populations are described by their infection status as the number (or proportion) that are *susceptible*, *infected*, or *removed* (or recovered and immune). The Kermack–McKendrick models, or SIR models, can be used to understand the dynamics of pathogen transmission and persistence. For macroparasites, the worms, ticks, and fleas, the modeling of pathogen dynamics is more complicated. For these pathogens, it is necessary to consider the numbers of parasites harbored by each host individual and the statistical distribution of these parasites across the host population.

This is primarily because most macroparasite populations (particularly the worms) are aggregated in a small proportion of the host population (see Web-only material on geohelminths). The complex mathematical framework required to describe this was first formulated by ecological parasitologists Crofton (1971a,b) and Tallis (Tallis and Leyton 1966; Tallis and Donald 1970), and then formalized and explored in detail by the broadly trained team of Anderson and May (1978; May and Anderson 1978).

When micro- and macroparasites (see examples below) have been modeled, the strategy tends to be one of elaboration, whereby important details are usually added to the basic models. Further complications arise where arthropod vectors transmit many human pathogens or have a non-human animal reservoir (Wilson 2001). All of these models display important similarities to those used to examine a variety of ecological problems where populations are fragmented into patches (Hanski 1997; Dobson 2003).

Measles

A great deal of our understanding of infectious disease dynamics and control comes from work on measles, beginning with that of Kermack and McKendrick (1927). This research produced a strong following in mathematics, but not until the 1950s did Bartlett apply these methods to develop an understanding of the dynamics of measles in urban and rural environments. The key concept was that of critical community size (CCS), the population threshold below which infections such as measles cannot persist. Bartlett demonstrated empirically that the CCS for measles was a community of approximately 500 000 people, and he argued that the stochastic nature of transmission made it impossible to estimate the CCS from deterministic models (Bartlett 1960, 1957). The concept of CCS was crucial in the formulation of vaccination policies, which became widespread following the successful development of vaccines for measles, mumps, chicken pox, and rinderpest (the cattle equivalent of measles) (Plowright 1967, 1982). Vaccination campaigns focused on reducing the size of the pool of susceptible hosts below the CCS. Critical to these campaigns was the concept of "herd immunity", the principle that transmission would not persist if a proportion of individuals became immune (either naturally or by vaccination). This herd immunity threshold is disease- and context-specific, but functions by reducing the potential for new infections for every infected host to less than one (see R_0 below) (Fine 1993).

The work on measles and other childhood diseases expanded and was again explored within a deterministic framework, but this time without the critical assumption that host population size remains constant (Anderson and May 1982; Anderson and Grenfell 1986). Initially, this work focused on deriving expressions for inter-epidemic periods and their dependence upon pathogen incubation period and duration of infectivity. This led to the development of more detailed age- and sex-structured models that could be used to examine the efficacy of concentrating vaccination programs on specific classes of hosts. These ideas guided the development of models for

Table 2. Additional infectious diseases for which theoretical ecology has improved control initiatives (see supplementary Web-only material for detailed descriptions)

Disease	*Insights from theoretical ecology*	*Associated public health initiatives*
Polio	Herd immunity and endgame planning.	Ongoing polio eradication program aims to halt the transmission of the poliovirus by the end of 2004.
Malaria	Ross and Macdonald models of pathogen transmission and mosquito survivorship. Garki model predicted variation in malaria prevalence in the human population	1955–69 WHO-coordinated, DDT-based global eradication campaign. Garki model was used in the analysis of control efforts that employed insecticide and drug intervention.
Schistosomiasis	Hairston and Cohen models of host–parasite population dynamics and patterns of infection determined the point in the transmission cycle when intervention strategies would be most successful. Anderson and May models of the effects of seasonality and the spatial clustering of infected snails on the success of transmission.	WHO molluscicide program minimized transmission in most African regions well into the 1980s. Recent application of mathematical modeling to various control methods has helped WHO adopt new intervention strategies, including biocontrol and education.
SARS	Models showed that the proportion of infective contacts that occur before symptoms appear (5–10% for SARS) are of major importance for determining the success of the control strategy.	When outbreaks occur the key step is isolating symptomatic individuals and tracing and quarantining their contacts.
Geohelminths	Crofton and Anderson and May models determined that the majority of the worm population is aggregated in a small proportion of the host population.	Control efforts focused on identifying why particular individuals were at risk and how control should be focused against these individuals or age classes.
River blindness	ONCHOSIM model simulated the effects of human and parasite population densities, the dynamics of vector populations, and interventions involving insecticide and chemotherapy on disease prevalence.	OCP managed the successful control of river blindness in 11 African countries. Today, this disease is no longer considered a public health problem throughout these areas and the parasite reservoir has been virtually eliminated.

Figure 1. *Burning cattle infected with foot and mouth disease.*

HIV/AIDS, where heterogeneities in the transmission of the pathogen are crucial to the spread of the disease. Work on the spatial and temporal dynamics of measles has continued to provide the benchmark by which to gauge our understanding of the dynamics of most other directly transmitted infectious diseases (Anderson *et al.* 1984; Bolker and Grenfell 1995; Keeling and Grenfell 1997; Grenfell and Bolker 1998; Grenfell *et al.* 2001; Bjørnstad *et al.* 2002).

Foot and mouth disease

A number of pathogens have emerged in the past few years that have had a major impact on human health and economic welfare. The foot and mouth disease (FMD) outbreak in the United Kingdom had a major effect on the agricultural community and an even greater impact on the rural tourist community (Ferguson *et al.* 2001; Keeling *et al.* 2003). An SIR model can again describe the dynamics of FMD among animals on a farm, but the relevant question in this case was farm-to-farm transmission. Detection of the pathogen on any individual farm led to the removal of all the stock on that farm, so the farm-to-farm dynamics were essentially SI.

There were two major priorities in understanding the UK foot and mouth epidemic. First, it was essential to develop ways of estimating R_0, the average number of secondary cases of infection from one primary case introduced into a defined population consisting solely of susceptible individuals. R_0 is a fundamental parameter in the study of infectious disease ecology as it governs the spread of infection and is related to the long-term behavior and level of vaccination necessary for local elimination or eradication. In its simplest form, R_0 serves as an index of how prone a population is to the risk of a given disease. Second, it was essential to develop a rapid understanding of the spatial spread of the disease. The epidemic was remarkable from an epidemiological public policy perspective, as it became apparent within the first 2 weeks of the outbreak that the policies then in place were inadequate to deal with the problem. This prompted Sir John Krebs, Chairman of the UK Food Standards Agency, to assemble a team of infectious disease ecologists to address the problem. These ecologists focused primarily on developing methods of estimating R_0 for farms; initial estimates suggested that each farm would infect about three to five others (Woolhouse 2001). The ecologists did a remarkably efficient job of educating the media and therefore the general public and policy makers about the importance of reducing R_0 to below one. It quickly became clear that the best way to accomplish this was to mount a more rigorous campaign to identify infected farms (and those nearby) as soon as the pathogen was detected. The herds on infected farms were quickly culled, producing the grim pictures which dominated the media (Figure 1) for some time.

Detailed quantitative work on the spatial dynamics of foot and mouth disease was considerably hampered by inadequacies in the database detailing the size and spatial distribution of British farms. The data required updating and confirming, all of which took valuable time (ironically, there are even less data on the size and spatial distribution of farms in the US). Nevertheless, it soon became possible to identify two forms of spatial transmission: local transmission by aerial plumes between neighbouring farms, and longer distance transmission typically resulting from the movement of farmers and veterinarians, and their contaminated vehicles. This led to the introduction of stringent movement restrictions and to ringed culling policies around infected farms (Figure 2; Ferguson *et al.* 2001a,b; Keeling *et al.* 2001, 2003). These policies were in place a month after the epidemic started and provide a remarkable testimony to the ability of the ecological epidemiology community to respond to a crisis. Once it was clear that the control policy was working and R_0 was declining, attempts were made to predict the end of the outbreak.

The UK foot and mouth outbreak provides some salutary lessons for public policy and future infectious disease outbreaks. Retrospective calculations of the time course of the epidemic suggest that the initial 2-week delay may have eventually led to a doubling of cases and of the number of cattle and sheep herds culled. All of this suggests that the ecologists should have been brought in sooner.

Rabies

The spillover of pathogens from domestic and wild animal hosts to human populations is responsible for many emerging infectious diseases worldwide, including SARS, Ebola, the US West Nile virus epidemic, and recent avian

influenza A (H5N1) outbreaks in Southeast Asia. Because these zoonotic diseases (infectious diseases transmissible from animals to humans) play such an important role in conservation biology, animal husbandry, and commerce, considerable attention has been paid to their prevention and control.

Mathematical models have influenced the development of global initiatives aimed at controlling rabies, including the ongoing fox rabies epidemic in Europe and the raccoon rabies epidemic in the eastern US. Although pet vaccination ensures that rabies cases in pets and humans are rare, strategies to control rabies in wildlife are limited. For many years, the only control program implemented was the reduction of wildlife densities below that required to sustain transmission (Anderson *et al.* 1981). More recently, an oral rabies vaccine (ORV) has been developed and used to control the disease in wildlife populations; this was widely distributed at the front of advancing waves of disease, to establish a zone where the density of susceptible individuals was low enough to prevent propagation. Mathematical models used to evaluate the cost effectiveness of ORV delivery versus culling revealed that the most economical strategy involves either culling or vaccination alone. The combination of culling and vaccination is cheaper than culling alone only when the per capita cost of vaccination is approximately one-fifth or less than the per capita cost of culling (Coyne *et al.* 1989). Although ORV distribution has become the tool of choice for wildlife rabies control in Europe, Canada, and the US (Brochier *et al.* 1991), ongoing research suggests that it has not been successful in the long term (Smith and Coyne pers comm).

In combination with extensive GIS mapping of rabies occurrence, mathematical models have also been effective in predicting the spread of rabies epidemics in wildlife and in helping to construct strategies that mitigate the extent of future epidemics (Murray *et al.* 1986; Russell *et al.* 2004; Smith *et al.* 2002). In particular, spatial models have been used to design *cordon sanitaires* that minimize both public health expenditure and negative impacts on wildlife populations, yet maintain effective barriers to rabies spread. Spatial models have also shown that the rate of disease spread is related to the density of wildlife populations. These models were combined with a map of fox density in the UK, to plan for a fox rabies epidemic (Murray *et al.* 1986). More sophisticated analysis showed that these strategies depended on restrictive and somewhat unrealistic assumptions about fox dispersal. For example, although long-distance translocation of foxes is rare, in the event that it does occur there is the potential for facilitated spread of rabies across the cordon sanitaire (Mollison 1986). The spatial network models used in the UK have been modified by the USDA to guide surveillance efforts and predict outbreaks of raccoon rabies in new geographic regions associated with the western expansion of the disease in the eastern US (Figure 2; Smith *et al.* 2002; Russell *et al.* 2004).

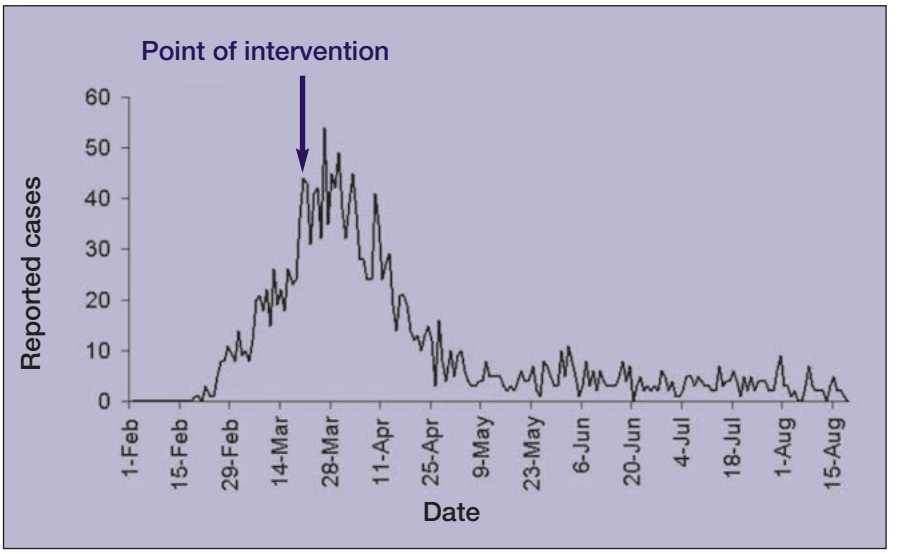

Figure 2. *Number of infected farms during the 2001 UK foot and mouth epidemic and the point at which the culling of infected herds and movement restrictions began. Adapted from Keeling* et al. *2001.*

HIV/AIDS

The slow but inexorable spread of the HIV/AIDS epidemic has created a global healthcare crisis (Anderson 1988; Anderson *et al.* 1988; Garnett and Anderson 1993; May and Anderson 1988). Ecologists have made important contributions to our understanding of how the virus spreads, the dynamics of infection, and the role of the host's immune system (Nowak *et al.* 1991). Theoretical ecology again provided two key public health insights in the early stages of the epidemic, the first of which emphasized the importance of quantifying the duration of the incubation and infectious periods (Anderson 1988; Medley *et al.* 1987; Blythe and Anderson 1988). Obtaining estimates of the infectious period is crucial for a sexually transmitted disease such as HIV, as the numbers of new cases created by any infectious individual will vary with duration of infectivity. However, estimating incubation period in the early years of the epidemic was a major statistical challenge, as data were only available from individuals who knew both when they were infected and when their symptoms developed (Medley *et al.* 1987). The second major determinant of the rate at which the disease spreads and the epidemic grows is the variance in the number of sexual contacts of an infected individual (May and Anderson 1988; Anderson *et al.* 1986). The key insight that people with many sexual partners contribute disproportionately to the rate of increase of the epidemic

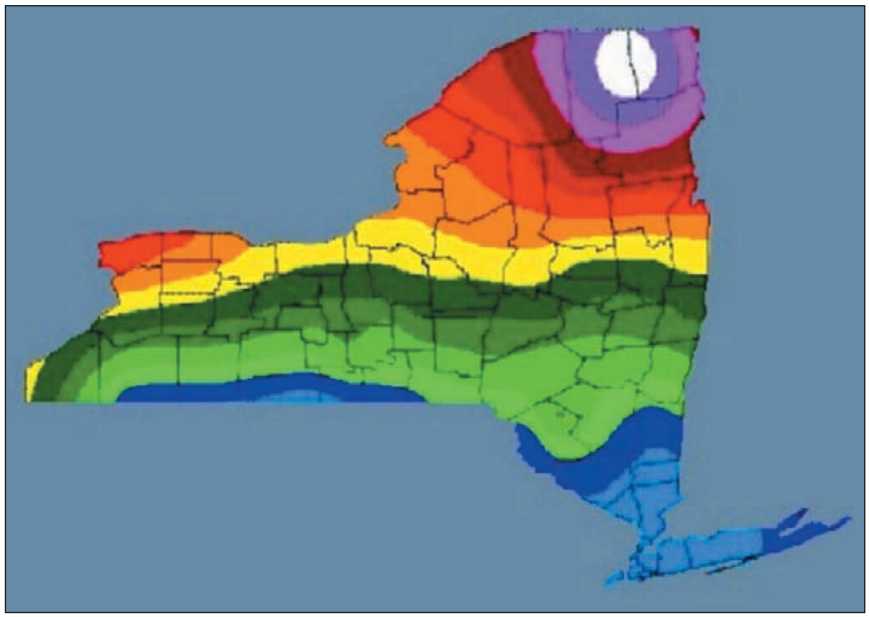

Figure 3. *Statistical characterization of the time to first appearance of raccoon rabies in the State of New York. Color bands depict the spread of rabies across the state, where blue represents the first occurrences of rabies and white denotes regions free from the disease. Band width represents the velocity of the wave front. Narrow bands represent slow spread and wide bands represent fast spread.*

strongly suggests that a reduction in the spread of the disease depends largely on a reduction in the number of new effective contacts of the most sexually active individuals. Understanding the variance in contact rates was crucial in developing models for the predictive spread of HIV in Africa and India, and indeed these models have proved to be extremely accurate (Anderson 1988; Garnett and Anderson 1993; May 2003).

Until the late 1980s, three strategies guided the treatment of AIDS in the US: medication of opportunistic infections, restoration or enhancement of immune activity, and anti-viral chemotherapy (Grmek 1990). By the 1990s, however, advances in theoretical epidemiology suggested two new strategies for HIV control: combination therapy and needle exchange programs. Theoretical research on viral multiplication in HIV patients revealed that the ability of the immune system to regulate viral replication declines as the diversity of viral strains increases with time since infection; thus, the interaction between HIV and the immune system is effectively a predator–prey system (Nowak *et al.* 1991). Extending this concept, Wodarz and Nowak (2002) developed mathematical models of HIV pathogenesis and treatment in order to understand the dynamics of HIV infection and therapy. Experimental results based on these models showed that early chemotherapy could substantially alter the dynamics between HIV and the immune system and that sustained virus control could be achieved (Wodarz and Nowak 2002). This research suggested that the treatment schedule could be modified to achieve long-term control in HIV patients that were chronic and in the asymptomatic phase of infection. In the mid 1990s, when it was discovered that combination chemotherapy could reduce mortality by inducing viral load suppression, the results of these models lead to the prescription of combination therapy at the onset of infection in HIV patients (Pomerantz and Horn 2003).

Needle exchange programs distribute clean needles and safely dispose of used ones for injections drug users (IUDs). Although widely controversial, needle exchange has been strongly advocated as an intervention to slow the spread of HIV among IUDs (Stimson 1989; Kaplan 1995). However, most US states have laws that make it a crime to possess or distribute drug paraphernalia, and 10 states, as well as the District of Columbia, have laws or regulations that require a prescription to buy a needle and syringe (Gostin *et al.* 1997). Consequently, the fear of police harassment or arrest has lead to increased rates of needle sharing among IDUs (Gostin *et al.* 1997). In the mid 1990s, two important probability models for needle exchange operations were developed, using data from the legal needle exchange program operated by the AIDS Division of the New Haven Connecticut Department of Health. The first model estimated the incidence of HIV infection among participating IDUs using the dates of client visits and the measured level of infection in needles (Kaplan and Heimer 1994; Kaplan 1995). The second model estimated the absolute reduction in new HIV infections as a result of needle exchange by extending earlier models of relative impact (Kaplan 1994; Kaplan 1995). These models revealed that needle exchange reduced HIV transmission among program participants from the onset to the conclusion of the program (Kaplan 1995). Furthermore, additional models based on the New Haven data predicted that needle exchange programs could prevent HIV infections among clients, their sex partners, and offspring at a cost of about $9400 per infection averted, a value far below the $195 188 lifetime cost of treating an HIV-infected person (Lurie and Reingold 1993; Holtgrave and Pinkerton 1997; Lurie and De Carlo 1998). These models and the results of the New Haven study were presented to public health decision makers in the US and abroad on numerous occasions (Kaplan 1995). Together with the results of other needle exchange studies (GAO 1993; Lurie and Reingold 1993), the New Haven models played an important role in shaping policy in major cities such as Baltimore, New York, and San Francisco (Kaplan 1995).

Conclusions

Ecologists first became interested in human infectious diseases because they provided rich datasets for testing

mathematical models developed to understand the population dynamics of host–pathogen interactions. It quickly became apparent that attempts to control infectious diseases could be perceived as experimental interventions, the impact of which could be used to parameterize and test the assumptions underlying such models. Perhaps most importantly, these models provided a novel way of examining the efficacy of public health interventions to control disease, and ultimately lead to numerous insights into the ways in which vaccination programs and other interventions may be carried out. As evidenced by the examples presented here, disease prevention and control has depended upon public health officials applying knowledge gained from the work of population ecologists. This has resulted in an increasing realization that understanding the dynamics of infectious diseases is as much an ecological as a medical problem and that future public policy initiatives cannot afford to ignore the results of research on disease ecology.

Due to the inherent heterogeneities present in all ecological systems, considerable benefits may accrue by identifying and focusing control efforts on the individuals (or locations) playing a disproportionate role in transmission. In many systems, less than 20% of individuals may be involved in more than 80% of transmission (the 20:80 rule) (Woolhouse *et al.* 1997). Identifying these individuals will be a critical component of future research in disease ecology and an important part of many intervention programs. New molecular genetic tools will be crucial in this arena, as will a more comprehensive blending of mathematical and ecological models with the new genetic tools derived from pathogen phylodynamics, an emerging discipline that combines the tools of immunodynamics, epidemiology, and evolutionary biology to understand the causes behind the wide variety of pathogen phylogenies observed in individual hosts and populations (Grenfell *et al.* 2004). The integration of these tools, together with the effective application of new knowledge, should enhance ongoing and future strategies for identifying and reducing the burden of infectious diseases.

As new infectious diseases emerge and old ones spread, there will be increased demands on surveillance informatics, vaccine development and distribution, and the healthcare industry. Similarly, the threat of bioterrorism requires both an understanding of the range of effects that outbreaks can cause and the identification and implementation of optimal control strategies to minimize those effects (Ferguson *et al.* 2003). The immediacy of these threats will add substantially to the pressure on national and international resources. Public health institutions charged with the development of control programs cannot afford to ignore the established and ongoing theoretical achievements in the field of infectious disease ecology and epidemiology. In addition to those described in the text and Table 2, there are numerous cases where theoretical ecology has facilitated the development and implementation of campaigns to fight infectious diseases around the world. These include bovine spongiform encephalopathy (Anderson *et al.* 1996; Ghani *et al.* 2003), anthrax (Webb and Blaser 2002), and smallpox (Kaplan *et al.* 2002), among others. We hope that these successes will stimulate greater collaboration between policy makers, public health workers, and theo-

Panel 1. Nelson Hairston, Sr: an ecologist at the forefront of schistosomiasis control

Nelson Hairston Jr

Nelson Hairston Sr was a pioneer in the application of ecological approaches to understanding and controlling human parasitic diseases. He came to explore the combination of these traditionally distinct sciences through a series of serendipitous personal experiences. Hairston majored in zoology at the University of North Carolina, obtained both Bachelors and Masters degrees and, at the urging of his mentor, RE Coker, moved to Northwestern University to do his PhD in ecology with Orlando Park. It was at Park's suggestion that Hairston began his seminal studies on salamander communities in the southern Appalachian Mountains.

After only one field season, Hairston was inducted into the army at the start of World War II. He was eventually commissioned as an officer, trained in parasitology at Walter Reed Army Medical Center, and was assigned to work on malaria prevention in the southwest Pacific, first in Australia and then in New Guinea. There he gained extensive experience with the life cycle of the parasite and the experimental therapies used to control symptoms in infected soldiers. He and his colleagues also studied transmission rates and parasite loads in native villages. After the US army invaded the Philippines, his unit moved there to study schistosomiasis, its life cycle and potential controls.

At the end of the war, Hairston returned to the US, completed his PhD on salamander ecology, and obtained a faculty position at the University of Michigan. Although appointed as an ecologist, one of his primary duties was teaching a course in parasitology. He had been at UM for 4 years when he accepted an invitation from the World Health Organization to spend 2 years in the Philippines working on the control of schistosomiasis. His assignment was to study the ecology of the parasite's intermediate snail host, and it was during this research that Hairston fully integrated his ecological and parasitological interests. It occurred to him that it ought to be possible to eradicate the disease if the snail population could be lowered sufficiently so that transmission rates were reduced below a level at which the schistosome fluke population would be self-sustaining. Discovering the critical minimum snail population size involved linking the parasite's life cycle with the host encounter rate as a function of population size in a simple mathematical model. In this way, he brought an ecological approach to the study of what had until then been treated solely as a medical problem.

Hairston's research set the stage for the seminal work of Joel Cohen, Roy Anderson, and Robert May, who collectively advanced the study of schistosome ecology and influenced the development and implementation of initiatives to control the transmission of schistosomiasis (see Web-only material).

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retical ecologists as we strive to improve and develop programs to control infectious diseases among humans, domestic livestock, and wildlife.

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