The Emergence of Disease Ecology

Shannon L. LADEAU * and Barbara A. HAN

Cary Institute of Ecosystem Studies, 2801 Sharon Turnpike, Millbrook, NY, U.S.A., 12545

[Received 15 February 2016; accepted 8 April 2016]

ABSTRACT

The number of newly recognized diseases affecting humans, domestic animals and wildlife has increased in recent decades and many of these diseases ‘emerge’ when environmental conditions change to alter contact rates between species. While traditional disease biology or epidemiological studies strive to understand the patterns of outbreak in a single species of interest, it is increasingly evident that management strategies demand more comprehensive understanding of the ecological interactions across wildlife, human, domestic animal and potential vector populations. This paper will introduce the ecological principles underlying pathogen dynamics in ecological systems and highlight current research frontiers in Disease Ecology. Pathogens and parasites are often considered as a disturbance rather than an inherent part of ecological systems, yet the fundamental principles of disease ecology derive from classical theory in population and community ecology.

Key words: community, dynamical models, ecosystem, pathogen, zoonotic

FUNDAMENTAL PRINCIPLES

The fundamental principles of disease ecology derive from classical theory in population and community ecology. Ecology may be summarized as the study of mechanisms controlling population growth and species coexistence. Likewise, disease ecology is the study of mechanisms controlling the growth of infected host populations, which relies on understanding how individuals, populations and species interact to support pathogen transmission. In Fig. 1, susceptible hosts (S) transition to the infected state (I) with transmission probability (β). This is the SIR model framework used as the conceptual backbone for much of the mechanistic modeling of infectious disease in humans [4]. Generating forecasts or inference in real-world systems may require integration of demography, as well as environmental variability and stochasticity into this conceptual model [5-10]. The oft-cited threshold parameter (R0), describing the likely number of newly infected individuals generated by the introduction of a single infected host into a
Shannon L. LADEAU and Barbara A. HAN

The pool of susceptibles, is derived from this conceptual model [11, 12].

SOME DEFINITIONS

Here, we refer to a pathogen as any causative agent of disease that may be transmitted from one host to another. If the pathogen may be transmitted from a non-human to a human host then it is considered zoonotic. Vector organisms, often blood-feeding arthropods, move a pathogen between hosts. Host species are those organisms that are susceptible to becoming infected with a pathogen. Hosts may either become infected but remain asymptomatic, or they may succumb to disease. In a multi-host system, a separate host species that maintains a pathogen long-term without high mortality is termed a reservoir host. An infected host may become infectious and able to transmit the pathogen to another individual directly or through a vector species. If a host becomes infected but does not transmit a pathogen, it is considered a dead-end host.

Pathogen transmission involves the interaction of at least two organisms, the host and the pathogen itself. A zoonotic pathogen may be transmitted to a human but predominantly persists in another species. West Nile virus, for example persists in avian reservoirs but can spill-over into humans when conditions permit, such as in urban habitat following permissive weather [13, 14]. Traditional epidemiological studies predominantly examine the patterns of pathogen infection in one host species of interest (often the human host). However, human infection is often the end result of a series of interactions (i.e., competition, predation) and environmental filters that influence the population growth of hosts, vectors, and pathogens. One example of environmental complexity supporting pathogen dynamics is seen in the tick-borne Lyme Disease system of the northeastern United States, where pathogen abundance can be linked to seasonal variability in food resources supporting reservoir host populations (Fig. 2). Fig. 2 doesn’t include the meso-predator species (e.g., Vulpes vulpes, red fox) or alternative hosts such as opossums (Didelphis virginiana) that help regulate mouse populations and tick survival [15-17]. There are a complex suite of mechanisms and interactions behind spatio-temporal patterns

![Fig. 1 Dynamical systems models (e.g., SIR) simplify epidemiological theory into a series of transition equations. For the past century, this structure has been the core of infectious disease models and management strategies [4, 29, 30]. Model estimates for transition from susceptible (S) to infected (I) states are described by a transmission term (β); contact rates are often assumed to be constant and random across individuals. Transmission is also a function of the duration of infectiousness, as quantified by the recovery rate (γ).](image1)

![Fig. 2 The causative agent of Lyme disease in the northeastern United States, *Borrelia burgdorferi*, is transmitted from an infected, often asymptomatic, reservoir species to blacklegged ticks, *Ixodes scapularis*. The predominant reservoir species is the white-footed mouse (*Peromyscus leucopus*), whose abundance is a function of food availability each season. Mice eat acorns and during a mast year when oak trees produce a superabundance of acorns, mouse populations grow. High mouse abundance allows more ticks to survive to pass *Borrelia* on to larger animals, including deer and humans. Additional complexity is evident when considering that high mouse populations may also be supported by gypsy moth outbreaks [31, 32], which could change oak tree populations and acorn availability. Graphic provided by Cary Institute of Ecosystem Studies.](image2)
in disease incidence. Ecology provides a natural framework for identifying and quantifying these dynamics.

**RESEARCH FRONTIERS**

Developing enough mechanistic understanding to identify strategies for management or epidemic forecasting is a critical research goal. We identify three current research objectives for the field of Disease Ecology - each of which acknowledges the very real need for inference to develop predictions and guide management.

Disease transmission among non-human animals is not easily studied in natural environments and the historical time series that have supported many advances in human infectious disease research do not exist for wildlife populations. Thus, in many cases of wildlife or zoonotic disease we are missing observations of infection status as well as observations of transmission events. This is a challenge for understanding wildlife disease systems and for predicting when or discovering how a zoonotic pathogen spills over into human populations.

Inferring an unobserved transmission mechanism from imperfect observations of infection status requires innovations in causal inference [18], and the development of modeling frameworks that can handle latent process estimation [19]. Integrating field data with transmission models to estimate critical infection, transmission and recovery rates in complex, multi-host or vectored systems is a critical goal for disease ecologists and public/wildlife health managers [20].

A second major focus for the field of Disease Ecology is the development of theoretical models that are generalizable across disease systems. This theoretical guidance is critical for identifying important research directions and for extending inference beyond the specific system, time and place of study. Perhaps the most important theoretical discussion specific to disease ecology is about the potential for species diversity to regulate pathogen abundance. The dilution effect hypothesis proposes that pathogen abundance can be limited in more diverse host communities, especially when host susceptibility is variable across multiple species [21]. A recent meta-analysis

---

**Fig. 3** Conceptual model depicting mechanisms by which diversity could reduce disease risk in a specialist host–pathogen system. The original Community X contains a single species with infected (filled circles) and uninfected, susceptible (open circles) individuals. The filled grey circle in Community X denotes recent transmission event in the single-species community. Individuals contact radius or home ranges are shown as dashed lines. The addition of a second species (black squares) could (a) reduce the probability of transmission (via reduced pathogen load) per encounter between susceptible and infected hosts (transmission reduction); (b) reduce the number of susceptible hosts (susceptible host regulation); (c) increase the rate of recovery of infected individuals (recovery augmentation); (d) increase the mortality rate of infected individuals (infected host mortality); or (e) reduce space used (contact radius) by the host species, reducing encounters between susceptible and infected individuals (encounter reduction). Redrawn from Fig. 1 in [21].
used results from studies on 47 parasites that infect wildlife exclusively and 14 zoonotic pathogens to demonstrate a net negative association between biodiversity and pathogen abundance, supporting the generality of the dilution effect across diverse systems [22]. A dilution effect may occur through several mechanisms (Fig. 3) [23-27]. Of course, it is important to note that there are exceptions to biodiversity’s benefits [22] and it is equally important to develop theory to understand when biodiversity conservation is unlikely to reduce infectious disease risk.

A third research frontier is in managing diverse and increasingly abundant data to identify the relevant reservoir and vector species in current and future disease epidemics. Until now, research to understand what precipitates infectious disease outbreaks has necessarily been a reactive endeavor, but as human activities increase the frequency of novel disease emergence, a preemptive strategy to disease mitigation is a global health imperative. For example, machine learning was recently used to predict novel reservoirs of human diseases[28], demonstrating the utility of these methods for revolutionizing disease mitigation by identifying a watch list of the most probable wildlife reservoirs. By analyzing comparative data on the intrinsic traits that distinguish reservoir from non-reservoir species, these approaches also reveal fundamental biological differences between organisms that underlie cross-species transmission and zoonotic spillover to humans. However, as with many interdisciplinary innovations, such advances require bridging historically disparate fields. Cross-training a new cadre of scientists in predictive analytics of infectious diseases will require combining computer science expertise with domain expertise in disease biology that zooms out from a traditional focus on single-host single-pathogen systems to adopt a macroecological view of what drives variation in infectious disease patterns across species.

ACKNOWLEDGEMENT

Shannon LaDeau is grateful to the Japanese Society of Zoo and Wildlife Medicine for the opportunity to present and discuss research at their Annual Meeting in July 2015.

REFERENCE

Disases Ecology（疾病生態学）の出現

Shannon L. LADEAU * and Barbara A. HAN

Cary Institute of Ecosystem Studies, 2801 Sharon Turnpike, Millbrook, NY, U.S.A., 12545

[2016年2月15日受領、2016年4月8日採択]

要約
ここ数十年間、ヒトや家畜、野生動物において新たに確認される疾病の数は増加している。これらの疾病の多くは、環境条件が変化した結果、生物種間の接触頻度が変わることで“emerge 現れる”のである。従来の疾病生物学あるいは疫学研究は、対象となる1つの生物種におけるアウトブレイクパターンを理解しようとするものである。しかしながら、疾病管理対策には、野生動物、ヒト、家畜、そして潜在的な媒介動物集団にまたがる生態学的相互作用について、より包括的な理解が必要であることが、ますます明らかになってきている。本論文では、生態系における病原体動態の根底にある生態学的原則について紹介し、“disease ecology”分野の最前線の研究について取り上げる。病原体や寄生虫は、生態系に固有のものであるというよりはむしろ生態系を乱すものと考えられがちであるが、“disease ecology”の基本原理は、個体群および群集生態学の古典論に由来するものである。

キーワード：エコシステム、群集、人獣共通感染症、動態モデル、病原体

—— 日本野生動物医学会誌 21（3）: 53-58, 2016

* 責任著者：
Shannon LaDeau（E-mail: LADEAUS@caryinstitute.org）