REVIEW

Biogeography of diseases: a framework for analysis

A. Townsend Peterson

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Abstract A growing body of literature offers a framework for understanding geographic and ecological distributions of species; a few applications of this framework have treated disease transmission systems and their geography. The general framework focuses on interactions among abiotic requirements, biotic constraints, and dispersal abilities of species as determinants of distributional areas. Disease transmission systems have key differences from other sorts of biological phenomena: Interactions among species are particularly important, interactions may be stable or unstable, abiotic conditions may be relatively less important in shaping disease distributions, and dispersal abilities may be quite variable. The ways in which these differences may influence disease transmission geography are complex; I illustrate their effects by means of worked examples regarding West Nile Virus, plague, filoviruses, and yellow fever.

Keywords Disease transmission · Ecological niche · Geographic distribution · Dispersal · Reservoir · Vector

Introduction

The past decade or so has seen considerable progress toward a general framework for understanding species' geographic distributions, essentially addressing the question, why is a species where it is and why is it not where it

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A. T. Peterson (⊠) Natural History Museum and Biodiversity Research Center, University of Kansas, Lawrence, KS 66045, USA e-mail: town@ku.edu is not? Key steps toward this understanding have included (1) mathematical formulation and clarification of ecological niches of species and how they affect species' geographic distributions (Pulliam 2000; Hirzel et al. 2002; Soberón 2007), (2) exploration of the role of historical events in shaping species' geographic distributions (Wiens and Graham 2005; Martínez-Meyer and Peterson 2006; Peterson and Nyári 2008; Waltari et al. 2007), and (3) analysis of the time scale on which change in ecological niche characteristics is likely to occur (Holt 1996; Holt and Gomulkiewicz 1996; Peterson et al. 1999). Altogether, this body of work provides a basis for understanding the complexities of distributions of species in both geographic space and ecological dimensions.

Diseases are caused by pathogens—generally viruses, bacteria, fungi, and protozoa—that invade an individual's body and cause ill effects (for the purposes of this review, I distinguish between pathogens and parasites simply on the basis of size—pathogens being microorganisms and parasites being of larger size—and focus on pathogens). Because disease processes are dynamic, taking place on extremely diverse scales of space (microscopic to continental) and time (minutes to centuries), and are the products of interactions among species (pathogens, reservoirs, vectors, etc.), their ecological and distributional dynamics may differ from those of more "normal" species. These differences are the focus of this review, which I will illustrate via a series of examples.

Factors shaping species' distributions

A recent concept paper presented a simple heuristic for understanding questions regarding species' distributions (Soberón and Peterson 2005), which aims to provide a framework for thinking about species' distributional patterns, even if not a comprehensive summary of the phenomenon. This framework (Fig. 1) centered on a Venn diagram showing interactions among three factors: abiotic conditions, biotic conditions, and accessibility considerations. The former two factors correspond roughly to the fundamental and realized niches as outlined by Hutchinson (1978); although Hutchinson focused more specifically on the role of competition among abiotic conditions, a more modern view would include other biotic interactions, such as predation, parasitism, and mutualism. Accessibility considerations, including both current dispersal ability and opportunity for dispersal and colonization in the past (Waltari et al. 2007), were not integrated into Hutchinson's (1978) framework.

The heuristic diagram centers on requirements of particular abiotic conditions (I refer to this circle as "A" for abiotic conditions) and how they relate to biotic interactions (referred to as "B" for biotic conditions) and how they modify the abiotic requirements. For example, a species may have fairly broad tolerances of abiotic conditions such as temperature, solar radiation, rainfall, and soil chemistry. The interactions of this species with other species, however, may restrict it to only a subsector of the abiotically suitable areas—that is, some areas may not be suitable owing to the presence of a particular predator or pathogen or owing to the absence of a key mutualist or symbiont. As such, the potential distribution of the species can be seen as $A \cap B$ —or to put it into words, in these areas, both abiotic and biotic conditions are appropriate for the species to maintain populations.

Finally, the species may be limited from occupying the entirety of its distributional potential by accessibility considerations (referred to as "M" for mobility). That is, a hypothesis of the actual distribution of the species would be $A \cap B \cap M$, which are those areas that are simultaneously

Fig. 1 Diagrammatic representation of three important factors in determining species' geographic distributions. Circle A summarizes abiotic variables that circumscribe species' geographic potential, circle B adds biotic considerations, and circle M indicates limitations on dispersal or movement ability $A \cap$ B is the potential geographic distribution of the species, and $A \cap B \cap M$ is a hypothesis of the actual distribution of the species. Three example disease systems are illustrated: West Nile Virus, the filoviruses (Ebola and Marburg viruses), and plague: Changes are illustrated as the difference between broken (original) and entire (present) outlines of circles; particular geographic occurrences of the disease are labeled to illustrate points discussed in the text



appropriate from both abiotic and biotic perspectives and that are accessible to the species in terms of dispersal. Although this area cannot simply be assumed to hold populations of the species (consider, e.g., metapopulation dynamics, which may lead to absences in suitable, accessible areas), this area does provide an index to the likely distribution of the species. Considerable exploration and testing has demonstrated both that $A \cap B \cap M$ is an appropriate prediction of the actual distributions of species (López-Cárdenas et al. 2005; Elith et al. 2006) and that $A \cap B$ offers highly accurate predictions of species' distributional potential when dispersal constraints are relaxed (Peterson 2003; Benedict et al. 2007).

This framework (the "BAM" diagram), although simple, offers considerable inferential power regarding distributions of species (Soberón and Peterson 2005). I emphasize that this framework is only heuristic and cannot cover the full diversity and complexity of disease phenomena—a more complete and mathematical treatment has been developed elsewhere (Soberón 2007). A wide variety of biodiversity phenomena (including diseases) can be placed in this framework and understood with greater clarity—for example, species' invasions are simply the broadening of the M circle to include more of the potential distribution of the species (Peterson 2003). Particular factors (e.g., abiotic conditions) can be visualized as more or less constraining by increasing or decreasing the size and overlap of the circle relative to the other circles.

Why diseases are different

The BAM framework encounters some intriguing challenges in applications to disease biogeography. For example, in coarse-scale biodiversity applications, the focus is generally on the role of abiotic conditions in constraining species' distributions (Soberón 2007)—in contrast, disease applications can emphasize biotic interactions as dominating the process. The following paragraphs highlight these differences.

Interactions rule A basic characteristic of a disease is its transmission cycle. Generally, these cycles involve several species—one or more reservoirs, vector(s), incidental hosts, and the pathogen itself. In an autecological world, then, a disease transmission system could be seen as a suite of species, each distributed according to its own ecological needs (i.e., its own particular A). In this sense, we could consider each element in a disease transmission system (species 1, 2, ... *i* ... up to *n*) to have its own particular version of A (which we can denote A_i)—disease transmission would then occur only where $A_1 \cap A_2 \cap ... \cap A_i \cap ... \cap A_n$. From the perspective of any single species in the system, the combined

intersections of the A's for all other species in the system compact into that species' B (Peterson 2007).

Put more simply, however, in disease transmission systems, interactions rule. Much of the dynamics of these systems will be determined not simply by abiotic considerations but by the interactions among many species. Examples are more than common across the world of diseases: Malaria transmission occurs only when appropriate mosquito vector species are present (Gu et al. 2006); plague transmission is most efficient when certain flea species are present (Krasnov et al. 2006); much more complex examples can be found in any introductory parasitology text. This interactions-dominated landscape appears to stand in sharp contrast with the abiotic-dominated landscape in the biodiversity world (Peterson 2003).

Stable and unstable interactions Beyond simply being dominant, interactions in the disease world can be stable or unstable (i.e., enzootic or epizootic). Specifically, some disease systems show dramatic associations of pathogens and hosts (Dragoo et al. 2006; Field et al. 2007)—in these cases, pathogens and hosts coevolve over evolutionary time and may establish stable relationships that may even involve evolution of resistance on the part of the host and/or avirulence on the part of the pathogen (Lenski and May 1994). These stable relationships can also lead to the evolution of parallel phylogenetic patterns, indicating a long period of shared evolutionary change (Charleston and Page 2002).

On the other hand, species interactions in disease transmission systems can also be extraordinarily unstable, particularly when interactions are relatively new. Some disease systems have epizootic transmission phases in which hosts are 'burned through' at surprising rates: Excellent examples include plague (Yersinia pestis) transmission among prairie dogs (Cynomys spp.; Ubico et al. 1988; Cully et al. 1997), rabies (rabies virus, Rhabdoviridae) transmission among mammals other than vampire bats (Desmodus spp.; Wandeler 1993; Davis et al. 2006), and Ebola (Ebolavirus spp., Filoviridae) transmission among great apes (Leroy et al. 2004). In these (and other) cases, the host-pathogen interaction is so unstable that host mortality almost inevitably results, producing either unstable epizootic transmission systems or brief outbreaks that burn out after a few generations of transmission, although some instances of unstable transmission maintenance of diseases are known (e.g., Bingham 2005).

A can be of minor importance Another characteristic of disease systems that differs markedly from much of the remainder of biodiversity is the potential for A to be of relatively minor importance. That is, particular for microbial pathogens (viruses, bacteria) that may not have free-living stages, abiotic considerations may place few constraints on the distributional potential of species. Some examples are obvious-influenza transmission on the International Space Station or at an Antarctic research base-but more subtle examples should be considered more carefully, such as outbreaks of Afrotropical diseases such as Ebola virus in Virginia (Rollin et al. 1999) and Marburg virus (Marburgvirus spp., Filoviridae) in Germany (Murphy et al. 1990). More generally, some pathogens may not have free-living life stages or any other interface with the outside environmentas such, their "ecological niche" requirements may constitute simply that of having a live host. In other words, in many disease transmission systems, A may place only broad constraints on the potential geography of the system, and B and M may determine much more of the spatial dynamics of the system.

M can vary dramatically Pathogens and parasites are generally of small body size and often are not particularly well equipped for movement. As such, one might expect their dispersal abilities to be quite limited. However, because of the tight associations between pathogens and their much larger hosts, dispersal events of surprising magnitude become possible. That is, because pathogens can ride around with their much bigger and much more mobile hosts, dispersal events can yield surprising results, often termed disease "emergence" events, but really just constituting extreme dispersal events.

Disease dispersal ability can thus be surprising in its effects. For instance, bat-hosted coronaviruses related to that which causes severe acute respiratory syndrome (SARS) had certainly been present in Asia for what is effectively the entirety of human history in the region (Guan et al. 2003; Li et al. 2005). Although such viruses may, from time to time, have infected humans and caused disease events, the major recent SARS outbreak not only had serious public health consequences in Asia but also jumped rather dramatically to North America (McDonald et al. 2004). Similarly, monkeypox (monkeypox virus, Orthopoxviridae) is well known as a central African disease (Arita and Henderson 1969), but a seemingly innocent importation of African rodents for pets in the USA resulted in a small-scale monkeypox outbreak in North America (Hutson et al. 2007). These dramatic variations in dispersal ability must be borne in mind in considering disease biogeography.

Three case studies

In this section, I offer three brief case studies intended to illustrate the oddities of disease biogeography, as contrasted with the biogeography of most customary elements of biodiversity. The three studies are designed to illustrate different aspects of distributional biology and offer some intriguing insights into disease biogeography. These case studies also offer an illustration of the basics of tools for modeling species' ecological niches—I have used simple niche-modeling tools to develop some interesting coarse-scale results about disease biogeography. In niche modeling, known occurrences of species (or diseases, in some cases) are related to raster geographic information system (GIS) coverages summarizing relevant environmental parameters in an evolutionary computing environment; the result is a picture of the species' ecological distribution, which can be projected onto geography to identify a potential distribution for the species (Peterson 2007; Soberón 2007).

West Nile Virus West Nile Virus (WNV; west nile virus, *Flaviviridae*) was first described and characterized from a patient in Uganda in 1935 and came to be known as a cause of mildly serious encephalitis cases across eastern and southern Africa (Taylor et al. 1956; Nir et al. 1967; McIntosh et al. 1969); eventually, its sporadic occurrence, occasionally in the form of major outbreaks, in the southern tier of Europe was also documented (Hubálek and Halouzka 1999). In 1999, however, WNV appeared in New York, in the USA, and quickly spread west and south across essentially all of the Americas (Komar and Clark 2006). The disease is clearly now "endemic" to (i.e., established in) much of the Americas as a permanent component of the pathogen landscape of the Western Hemisphere.

Figure 2, however, paints a picture that contrasts actual and potential distributions of WNV. The area shaded brown in the figure in Africa, southern Europe, and Southwest Asia represents the virus' known distribution as of 1998; the blue-shaded area represents areas globally that fit the same precipitation–temperature profile as the brown-shaded area, showing that the same climate regime was present across a much broader portion of the world. In 1999, WNV managed to jump across the Atlantic Ocean and become established in New York City—in the ensuing 7–8 years, thanks probably to dispersal via migratory birds, the virus has spread west to the Pacific Ocean and south to Argentina (Komar and Clark 2006), fulfilling much of the spatial extent of its potential distribution in the Americas.

Ebola and Marburg viruses Discovered only in 1967 (Marburg) and 1976 (Ebola), the two virus genera that make up the *Filoviridae* family are exclusively African insofar as their known geographic distributions (Peters et al. 1993). What is more, however, Ebola virus is restricted to humid lowland evergreen tropical forests in Africa (Congo Basin and a small area along the Liberia–Ivory Coast border), and Marburg virus is restricted to less humid



Fig. 2 Actual and potential distributions of WNV as of 1998: *brown shading* indicates an approximate boundary around areas of known WNV occurrence prior to 1999; *blue shading* indicates areas matching the annual mean temperature×annual precipitation profile of the

brown areas using the BIOCLIM algorithm (Nix 1986). Note that the dispersal event of 1999 allowed the colonization of essentially the entire American portion shaded *blue* in the course of just 8 years

tropical forests in eastern and southern Africa. As such, these two related virus genera occupy a predictable ecological niche space across African landscapes (Peterson et al. 2004a; Peterson et al. 2006).

As an illustration of the volatility and unpredictability of disease geography given the association of pathogens with larger hosts, it is worthy of note the several times that Ebola or Marburg viruses have appeared outside the usual suite of conditions for filoviruses (Feldmann et al. 2004). Several filovirus-caused disease outbreaks have appeared under conditions distinct from the norm for these viruses and in areas well outside the usual distributional area of these viruses: (1) Marburg virus in Marburg, Germany, in 1967; (2) Marburg virus in Johannesburg, South Africa, in 1975; and (3) Ebola (Reston species) in Virginia, Texas, and the Philippines at various points in the 1990s (Peters et al. 1993; Miranda et al. 1999). As can be seen in both geographic and ecological dimensions (Fig. 3), these filovirus occurrences are outliers and clearly illustrate how pathogens can be carried around and into odd situations by their larger and more mobile vertebrate hosts. Clearly, this example illustrates the point that the "normal," endemic transmission mode may lend itself much better to predictive modeling than the irruptive, epizootic phases.

Plague Finally and the most complex, plague is a zoonotic disease that is held in a small mammal reservoir and vectored by several flea species (Gage 1998); the particular species involved depend on the region in question. Plague was originally—apparently—restricted to central Asia and interior China (Pavlovsky 1966); with the advent of large-scale movements (i.e., silk route trade, intercontinental shipping), however, a series of pandemics began, in which

plague became established in urban *Rattus* populations (Gage 1998). Particularly in the early twentieth century, however, these plague outbreaks could be extinguished by reducing the contact between humans and *Rattus*; however, in a number of cases around the world (e.g., western North America, Andean and northeastern South America, eastern and southern Africa, Madagascar), the plague "jumped" into native rodents and has established itself as an endemic zoonosis (Levy and Gage 1999; Enscore et al. 2002).

Viewed ecologically, the endemic (i.e., not epizootic) plague appears to occur under a consistent suite of environmental conditions (Enscore et al. 2002). As can be appreciated in Fig. 4, the environmental conditions across its original range in Asia are fairly restricted with respect to the extent to which it spread via global shipping (Echenberg 2007). Then, however, plague retreated somewhat as it moved from epizootic to enzootic, for example, not being maintained on Hawaii or in Australia (Fig. 4). Overall, then, in the history of the plague, two shifts have been observed: (1) broadened distributional potential thanks to improved dispersal abilities and (2) reduced distributional potential thanks to reduction in urban rat populations.

Disease geography

The above framework can be applied to understanding the geography of many biological phenomena, including diseases of various types (Soberón and Peterson 2004). The distribution of disease occurrences can be seen as the joint spatial distribution of suitable ecological conditions for all of the biological species involved in the transmission



Fig. 3 Summary of the geography and ecology of filovirus occurrences on global scales. *Top panel*: potential geographic distribution of Ebola virus (*green*) and Marburg virus (*blue*), in comparison with extralimital occurrences (*yellow stars*) resulting from exportation of infected primates. *Bottom panel*: plot of annual precipitation (mm) versus annual

mean temperature ($^{\circ}C \times 10$), showing conditions globally, conditions reconstructed as suitable for Ebola virus (*gray symbols*) and for Marburg virus (*red symbols*; Peterson et al. 2004a), and conditions at extralimital occurrence sites (*yellow squares*; Virginia and Texas, USA; Marburg, Germany; Johannesburg, South Africa)

cycle: pathogen, reservoir, vector, etc.—in essence, the geographic projection of the ecological distribution of the pathogen as limited by the ecological and geographic potential of each of its interacting species. This interplay among ecological and geographic spaces, among the

ecological niche characteristics of various species and as constrained by the spatial configuration of suitable habitats and dispersal abilities of the species involved, provides a framework for understanding disease distributions (Peterson 2007).



Fig. 4 Summary of the geographic distribution of plague (*Y. pestis*) over recent centuries: *red shading*, approximate original distribution of plague; *yellow squares*, a sampling of cities colonized in the last

The discussions above emphasize the role of humans in increasing the geographic scope of disease transmission, yet many diseases have yet to expand distributions broadly beyond their original distributional areas. For example, Chagas disease remains uniquely American, although it may be expanding northward (Beard et al. 2002). The filoviruses and monkeypox are endemic only to tropical Africa. Scrub typhus (*Orientia tsutsugamushi*) and Nipah Virus (nipah virus, *Paramyxoviridae*) are uniquely Asian. These diseases either have yet to disperse successfully to other regions, or perhaps they have dispersed but have failed to find appropriate interacting species (reservoirs, vectors, etc.) upon arrival. The distributions of such endemic diseases will be governed by the joint ecological requirements outlined above.

Some enigmas remain, however, in understanding disease geography. For example, yellow fever (yellow fever virus,

pandemic (1855–1959); gray shading, approximate present-day distribution (simplified from CDC 2005)

Flaviviridae) was originally distributed across humid tropical Africa; early shipping (probably via slave ships) transported it to South America, and it spread broadly across the humid tropical portions of that continent. Curiously, however, yellow fever has never colonized humid tropical portions of Asia, in spite of ample areas presenting appropriate climatic conditions (Fig. 5). Similarly, particular strains of malaria (e.g., falciparum malaria) have patchy and odd distributional patterns that would seem to challenge purely ecology-based explanations of distributions.

Frontiers and conclusions

This review may be seen as a nontraditional way of presenting disease geography. Instead of a broad review of



Fig. 5 Ecological potential for establishment of yellow fever in Asia. *Dark gray areas* correspond to countries with current yellow fever transmission (WHO 2000); *red areas* indicate portions of Asia that

match yellow fever-endemic regions in terms of annual mean temperature and annual precipitation

distributional patterns disease by disease (e.g., Ackerknecht 1965), I have instead attempted to present a general framework for understanding geographic distributions of species and to point out the ways in which disease transmission systems differ from other biological systems. My hope is that the generalities that emerge will assist in a new, more synthetic view of disease geography.

Many challenges remain to be addressed in this field. In particular, the way in which interactions among species act (and how these interactions can vary across geography) remains almost completely unexplored. That is, the $A \cap B$ framework described above is certainly an oversimplification—interactions among species may easily vary spatially and may even vary as a function of interactions with still other species. These considerations could produce complexity well beyond what has been appreciated in disease geography studies to date and indeed might explain some of the complexities and nonlinearities that confound the understanding of disease geography.

Another issue is the many diseases that have poorly known or unknown components to their transmission cycles. In such cases, niche modeling tools can only be applied to the overall cycle as a "black box"—human cases would be used as an integration over the entire $A_1 \cap A_2 \cap ... \cap A_i \cap ... \cap A_n$, even though the species involved are not identified—several such studies have already been developed (e.g., Peterson et al. 2006). Indeed, under some circumstances, niche-modeling tools can be used to identify the particular species participating in the transmission cycle (Peterson et al. 2002) or at least to identify likely candidate species (Peterson et al. 2004b, Peterson et al. 2007).

When participating species can be identified clearly, however, the power of these approaches increases considerably. Not only can the ecology and geography of the transmission cycle be reassembled in detail in a GIS environment, but exceptions to these predictions offer insights into the additional complexities in these systems. That is, where 1+1+1 does not equal 3 may indicate that some additional factors are acting. Such next-step insights will become available as more detailed, range-wide views of the ecology and biogeography of disease transmission systems are developed.

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References

Ackerknecht EH (1965) History and geography of the most important diseases. Hafner, New York

- Arita I, Henderson DA (1969) Smallpox and monkeypox in primates. Primates Med 3:122–123
- Beard CB, Pye G, Steurer FJ, Salinas Y, Campman R, Peterson AT, Ramsey JM, Wirtz RA, Robinson LE (2002) Chagas disease in a domestic transmission cycle in southern Texas, USA. Emerg Infect Dis 9:103–105
- Benedict MQ, Levine RS, Hawley WA, Lounibos LP (2007) Spread of the tiger: global risk of invasion by the mosquito *Aedes albopictus*. Vector-borne Zoonotic Dis 7:76–85
- Bingham J (2005) Canine rabies ecology in southern Africa. Emerg Infect Dis 11:1337–1342
- CDC (2005) World distribution of plague, 1998. Centers for Disease Control and Prevention, Ft. Collins, CO Available at: http://www. cdc.gov/ncidod/dvbid/plague/world98.htm
- Charleston M, Page RDM (2002) TreeMap2.0β: a Macintosh program for the analysis of how dependent phylogenies are related, by cophylogeny mapping. Available at: http://taxonomy.zoology.gla. ac.uk/%7emac/treemap/index.html
- Cully JF Jr, Barnes AM, Quan TJ, Maupin G (1997) Dynamics of plague in a Gunnison's prairie dog colony complex from New Mexico. J Wildl Dis 33:706–719
- Davis PL, Bourhy H, Holmes EC (2006) The evolutionary history and dynamics of bat rabies virus. Infection, Genetics and Evolution 6:464–473
- Dragoo JW, Lackey JA, Moore KE, Lessa EP, Cook JA, Yates TL (2006) Phylogeography of the deer mouse (*Peromyscus maniculatus*) provides a predictive framework for research on hantaviruses. J Gen Virol 87:1997–2003
- Echenberg M (2007) Plague ports: the global urban impact of bubonic plague, 1894–1901. New York University Press, New York
- Elith J, Graham CH, Anderson RP, Dudik M, Ferrier S, Guisan A, Hijmans RJ, Huettman F, Leathwick JR, Lehmann A, Li J, Lohmann LG, Loiselle BA, Manion G, Moritz C, Nakamura M, Nakazawa Y, Overton JM, Peterson AT, Phillips SJ, Richardson K, Scachetti-Pereira R, Schapire RE, Soberón J, Williams SE, Wisz MS, Zimmermann NE (2006) Novel methods improve prediction of species' distributions from occurrence data. Ecography 29: 129–151
- Enscore RE, Biggerstaff BJ, Brown TL, Fulgham RF, Reynolds PJ, Engelthaler DM, Levy CE, Parmenter RR, Montenieri JA, Cheek JE, Grinnell RK, Ettestad P, Gage KL (2002) Modeling relationships between climate and the frequency of human plague cases in the southwestern United States, 1960–1997. Am J Trop Med Hyg 66:186–196
- Feldmann H, Wahl-Jensen V, Jones SM, Stroher U (2004) Ebola virus ecology: a continuing mystery. Trends Microbiol 12:433–437
- Field HE, Mackenzie JS, Daszak P (2007) Henipaviruses: emerging paramyxoviruses associated with fruit bats. Curr Top Microbiol Immunol 315:133–159
- Gage KL (1998) Plague. In: Hausler WJ, Sussman M (eds) Bacterial Infections. Edward Arnold, London, pp 885–904
- Gu W, Regens JL, Beier JC, Novak RJ (2006) Source reduction of mosquito larval habitats has unexpected consequences on malaria transmission. Proc Natl Acad Sci USA 103:17560–17563
- Guan Y, Zheng BJ, He YQ, Liu XL, Zhuang ZX, Cheung CL (2003) Isolation and characterization of viruses related to the SARS coronavirus from animals in southern China. Science 302:276–278
- Hirzel AH, Hausser J, Chessel D, Perrin N (2002) Ecological-niche factor analysis: how to compute habitat-suitability maps without absence data? Ecology 83:2027–2036
- Holt RD (1996) Adaptive evolution in source-sink environments: direct and indirect effects of density-dependence on niche evolution. Oikos 75:182–192
- Holt RD, Gomulkiewicz R (1996) The evolution of species' niches: a population dynamic perspective. In: Othmer HG, Adler FR, Lewis MA, Dallon JC (eds) Case studies in mathematical

modeling: ecology, physiology and cell biology. Prentice-Hall, Saddle River, NJ, pp 25-50

- Hubálek Z, Halouzka J (1999) West Nile fever—a reemerging mosquito-borne viral disease in Europe. Emerg Infect Dis 5:643–650
- Hutchinson GE (1978) An introduction to population ecology. Yale University Press, New Haven
- Hutson CL, Lee KN, Abel J, Carroll DS, Montgomery JM, Olson VA, Li Y, Davidson W, Hughes C, Dillon M, Spurlock P, Kazmierczak JJ, Austin C, Miser L, Sorhage FE, Howell J, Davis JP, Reynolds MG, Braden Z, Karem KL, Damon IK, Regnery RL (2007) Monkeypox zoonotic associations: insights from laboratory evaluation of animals associated with the multi-state US outbreak. Am J Trop Med Hyg 76:757–768
- Komar N, Clark GG (2006) West Nile Virus activity in Latin America and the Caribbean. Rev Panameña Salud Publica 19:112–117
- Krasnov B, Shenbrot G, Mouillot D, Khokhlova I, Poulin R (2006) Ecological characteristics of flea species relate to their suitability as plague vectors. Oecologia 149:474–481
- Lenski RE, May RM (1994) The evolution of virulence in parasites and pathogens—reconciliation between 2 competing hypotheses. J Theor Biol 169:253–265
- Leroy EM, Rouquet P, Formenty P, Souquiere S, Kilbourne A, Froment JM, Bermejo M, Smit S, Karesh W, Swanepoel R, Zaki SR, Rollin PE (2004) Multiple Ebola virus transmission events and rapid decline of central African wildlife. Science 303:387–390
- Levy CE, Gage KL (1999) Plague in the United States 1995–1996, with a brief review of the disease and its prevention. Infect Med 16:54–64
- Li W, Shi Z, Yu M, Ren W, Smith C, Epstein JH, Wang H, Crameri G, Hu Z, Zhang H, Zhang J, McEachern J, Field H, Daszak P, Eaton BT, Zhang S, Wang LF (2005) Bats are natural reservoirs of SARS-like coronaviruses. Science 310:676–679
- López-Cárdenas J, González-Bravo FE, Salazar-Schettino PM, Gallaga-Solórzano JC, Ramírez-Barba E, Martínez-Méndez J, Sánchez-Cordero V, Peterson AT, Ramsey JM (2005) Fine-scale predictions of distributions of Chagas disease vectors in the state of Guanajuato, Mexico. J Med Entomol 42:1068–1081
- Martínez-Meyer E, Peterson AT (2006) Conservatism of ecological niche characteristics in North American plant species over the Pleistocene-to-recent transition. J Biogeogr 33:1779–1789
- McDonald LC, Simor AE, Su IJ, Maloney S, Ofner M, Chen KT, Lando JF, McGeer A, Lee ML, Jernigan DB (2004) SARS in healthcare facilities, Toronto and Taiwan. Emerg Infect Dis 10:777–781
- McIntosh BM, Dickinson DB, McGillivray GM, Sweetnam J (1969) Ecological studies on Sindbis and West Nile viruses in South Africa. V. The response of birds to inoculation of virus. S Afr J Med Sci 34:77–82
- Miranda ME, Ksiazek TG, Retuya TJ, Khan AS, Sanchez A, Fulhorst CF, Rollin PE, Calaor AB, Manalo DL, Roces MC, Dayrit MM, Peters CJ (1999) Epidemiology of Ebola (subtype Reston) virus in the Philippines, 1996. J Infect Dis 179:S115– S119
- Murphy FA, Kiley MP, Fisher-Hoch SP (1990) Filoviridae: Marburg and Ebola viruses. In: Fields BN, Knipe DM (eds) Virology. Raven, New York, pp 933–942
- Nir Y, Goldwasser R, Lasowski Y, Avivi A (1967) Isolation of arboviruses from wild birds in Israel. Am J Epidemiol 86:372–378
- Nix HA (1986) A biogeographic analysis of Australian elapid snakes. In: Longmore R (ed) Atlas of elapid snakes of Australia. Australian Government Publishing Service, Canberra, pp 4–15

- Pavlovsky EN (1966) Natural nidality of transmissible diseases. University of Illinois Press, Urbana
- Peters CJ, Johnson ED, Jahrling PB, Ksiazek TG, Rollin PE, White J, Hall W, Trotter R, Jaax N (1993) Filoviruses. In: Morse SS (ed) Emerging viruses. Oxford University Press, Oxford, pp 159–175
- Peterson AT (2003) Predicting the geography of species' invasions via ecological niche modeling. Q Rev Biol 78:419–433
- Peterson AT (2007) Ecological niche modelling and understanding the geography of disease transmission. Vet Ital 43:393–400
- Peterson AT, Nyári Á (2008) Ecological niche conservatism and Pleistocene refugia in the Thrush-like Mourner, *Schiffornis* sp., in the Neotropics. Evolution 62:173–183
- Peterson AT, Soberón J, Sánchez-Cordero V (1999) Conservatism of ecological niches in evolutionary time. Science 285:1265–1267
- Peterson AT, Sánchez-Cordero V, Beard CB, Ramsey JM (2002) Ecologic niche modeling and potential reservoirs for Chagas disease, Mexico. Emergi Infect Dis 8:662–667
- Peterson AT, Bauer JT, Mills JN (2004a) Ecological and geographic distribution of filovirus disease. Emerg Infect Dis 10:40-47
- Peterson AT, Carroll D, Mills JN (2004b) Potential mammalian filovirus reservoirs. Emerg Infect Dis 10:2073–2081
- Peterson AT, Lash RR, Carroll DS, Johnson KM (2006) Geographic potential for outbreaks of Marburg hemorrhagic fever. Am J Trop Med Hyg 75:9–15
- Peterson AT, Papeş M, Carroll DS, Leirs H, Johnson KM (2007) Mammal taxa constituting potential coevolved reservoirs of filoviruses. J Mammal 88:1544–1554
- Pulliam HR (2000) On the relationship between niche and distribution. Ecol Lett 3:349–361
- Rollin PE, Williams RJ, Bressler DS, Pearson A, Trappier SG, Peters RL, Greer PW, Zaki SR, Demarcus T, Hendricks K, Kelley M, Simpson D, Geisbert TW, Jahrling PB, Peters CJ, Ksiazek TG (1999) Ebola (subtype Reston) virus among quarantined nonhuman primates recently imported from the Philippines to the United States. J Infect Dis 179:S108–S114
- Soberón J (2007) Grinnellian and Eltonian niches and geographic distributions of species. Ecol Lett 10:1115–1123
- Soberón J, Peterson AT (2004) Biodiversity informatics: managing and applying primary biodiversity data. Philos Trans R Soc Lond B 359:689–698
- Soberón J, Peterson AT (2005) Interpretation of models of fundamental ecological niches and species' distributional areas. Biodivers Inform 2:1–10
- Taylor RM, Work TH, Hurlbut HS, Rizk F (1956) A study of the ecology of West Nile virus in Egypt. Am J Trop Med Hyg 5:579–620
- Ubico SR, Maupin GO, Fagerstone KA, McLean RG (1988) A plague epizootic in the white-tailed prairie dogs (*Cynomys leucurus*) of Meeteetse, Wyoming. J Wildl Dis 24:399–406
- Waltari E, Perkins S, Hijmans R, Peterson AT, Nyári Á, Guralnick R (2007) Consilience testing to determine location of Pleistocene refugia: Comparing phylogeographic, fossil and ecological niche model predictions. PLoS ONE 2:e563
- Wandeler AI (1993) Wildlife rabies in perspective. Onderstepoort J Vet Res 60:347–350
- WHO (2000) WHO report on global surveillance of epidemic-prone infectious diseases—yellow fever. World Health Organization, Geneva Available at: http://www.who.int/csr/resources/publications/ yellowfev/CSR ISR 2000 1/en/index.html
- Wiens JJ, Graham CH (2005) Niche conservatism: Integrating evolution, ecology, and conservation biology. Ann Rev Ecolog Evol Syst 36:519–539