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The ecology and emergence of diseases in fresh waters

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SUMMARY

1. Freshwater ecosystems, including ponds, lakes, streams and rivers, represent an interaction nexus between environmental change and a wide variety of infectious diseases, including human malaria, salmonid whirling disease, amphibian chytridiomycosis, crayfish plague and many others. However, few studies have explicitly examined patterns of disease in fresh waters and how they are changing over time.
2. Freshwater environments can function as transmission foci for pathogens because of (i) the importance of fresh water for organism survival, (ii) the aquatic life histories of many vectors and intermediate hosts, (iii) the concentrated aggregations of species – both freshwater and terrestrial – in and around freshwater habitats and (iv) the highly altered condition of freshwater ecosystems, which can affect species interactions and disease pathology.
3. To determine whether water-related diseases in wildlife are increasing, we used generalised additive models to quantitatively assess trends in the scientific literature (1970–2009) for major freshwater groups, including amphibians, molluscs, crayfishes, fishes, mammals, reptiles and birds. We further examined what types of pathogens were primarily responsible for observed patterns and whether recurrent groups or transmission modes could be identified.
4. After correcting for research effort and temporal autocorrelation, we find that reports of disease varied over time and across freshwater taxa, with significant increases in amphibians, fishes and crayfishes, a significant decrease in molluscs, and no significant change in freshwater reptiles, birds or mammals. The types and diversity of pathogens varied considerably among groups. Reports of infection in amphibians were dominated by helminths and a chytridiomycete, in crayfishes by viruses and fungi, in molluscs by digenetic trematodes, in birds, fishes and mammals by viruses, protists and helminths and in reptiles by helminths and bacteria.
5. These results provide some of the first quantitative evidence indicative of a long-term increase in disease-related research for freshwater taxa. Managing freshwater ecosystems to reduce or minimise human and wildlife disease risk – arguably one of the most significant ecosystem services – will require enhanced incorporation of ecological approaches alongside medical and veterinary tools.

Keywords: aquatic, biodiversity loss, emerging disease, global change, parasite

Introduction

Infectious disease emergence and re-emergence are a major concern for medical, veterinary and conservation-related disciplines (Lafferty & Gerber, 2002; WHO 2004; Ostfeld, Keesing & Eviner, 2008). In human populations, infectious diseases such as malaria, HIV/AIDS, tuberculosis, Lassa fever, Lyme disease and cholera collectively cause morbidity and mortality in millions of people every year. The list in wildlife is similarly diverse, including toxoplasmosis in sea otters, facial tumours in Tasmanian devils,

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canine distemper in African lions and white-nose syndrome in bats (Cleaveland *et al*., 2000; Miller *et al*., 2002; McCallum *et al*., 2007; Blehert *et al*., 2009). The parallel emergence of disease in humans and wildlife underscores two important and interrelated points. First, most emerging infections, regardless of host taxa, are linked to environmental changes such as habitat fragmentation, species translocations, altered food webs, climate shifts or pollution (Okamura *et al*., 2010; Daszak, Cunningham & Hyatt, 2000; Dobson & Foufopoulos, 2001; Patz *et al*., 2004; Poulin *et al*., 2010). Second, emerging diseases of humans and those of wildlife are intimately related; most emerging infections are zoonotic, such that they involve wildlife hosts, vectors or reservoirs, while human activity can also introduce novel pathogens to wildlife populations (Wallis & Lee, 1999; Taylor, Latham & Woolhouse, 2001; Woolhouse & Gowtage-Sequeria, 2005; Nunn & Altizer, 2006). In recognition of such relationships, an increasing number of studies have called for 'systems-level' approaches involving integration of epidemiological, epizootiological and ecological research (Lafferty & Gerber, 2002; American Veterinary Medical Association 2008; Riley *et al*., 2008).

The importance of disease emergence in freshwater ecosystems has received comparatively little attention relative to marine and terrestrial environments (Harvell *et al*., 1999; Harvell *et al*. 2002; Lafferty, Porter & Ford, 2004; Ostfeld *et al*., 2008). Outside of human diseases with water-borne (e.g. cholera) or vector-borne (e.g. malaria) transmission (Cotruvo *et al*., 2004; McNinch, Rose & Dreelin, 2009; Resh, 2009), few reviews have broadly explored disease patterns in fresh waters. We are aware of no reviews focused on wildlife diseases associated with fresh water specifically. Assessments of emerging threats to freshwater biota place little emphasis on infectious disease or omit it altogether (e.g. Dudgeon *et al*., 2006; Abell *et al*., 2009). This raises an important question: are patterns of disease emergence in freshwater habitats less pronounced than in marine or terrestrial ecosystems, for example owing to differences in ecology, evolution or anthropogenic influences, or does this trend result instead from a lack of information or synthesis (McCallum *et al*., 2004; Winfield, 2009)? Taxonomic specialisation and separation among researchers working on water-related diseases of wildlife (e.g. fishes, crayfishes and amphibians) and humans, for example, could obscure broad-scale patterns in freshwater epidemiology. Indeed, reports of severe disease and mass mortality in fresh water occur frequently in the epidemiological literature: viral haemorrhagic septicaemia (VHS) in freshwater fishes, chytridiomycosis in amphibians, Type E botulism in waterfowl and crayfish plague in European crayfish are but a few examples (Reynolds, 1988; USDA, 2006; Newman *et al*., 2007; Skerratt *et al*., 2007). Whether these examples underscore a wider pattern of emergence remains conjectural, however.

Here, we broadly explore the ecology of diseases in freshwater ecosystems. We begin with a discussion of disease ecology generally and the complexities inherent to host–pathogen-environment interactions, particularly in the face of rapid and pronounced environmental changes. Next, we explore the diverse roles of freshwater environments in host–parasite interactions, common types of water-related diseases and their modes of transmission, and the similarities and differences among diseases of freshwater, marine and terrestrial ecosystems. Although our focus is on wildlife diseases, we include selected medical examples associated with water to illustrate parallels between wildlife and human diseases (American Veterinary Medical Association 2008). To quantitatively explore the patterns of disease in fresh waters, including temporal trends, major pathogen groups and host taxa affected, we use ISI Web of Science to analyse the primary literature (1970–2009) for disease-related publications on amphibians, molluscs, crayfishes, fishes, birds, reptiles and mammals. Finally, we discuss ongoing threats and challenges confronting freshwater ecosystems and how they are likely to influence future patterns of infection and ecosystem services.

Ecology of disease

At its core, disease is an inherently ecological process involving interactions between at least one host and one pathogen. But such interactions are often subject to influences from a suite of co-occurring species, including other pathogens, other hosts and a community of non-host species (Cottingham & Butzler, 2006; Keesing, Holt & Ostfeld, 2006). Interactions among these species and with the abiotic environment can profoundly influence the likelihood of a disease outcome. Type E botulism, for example, which is a non-infectious disease of birds and sometimes

humans, is the result of ingestion of the bacterially produced toxin botulinum (Friend, 2002); however, while the bacterium (*Clostridium botulinum* Bergey *et al.*) that produces the toxin is relatively common, the levels of disease in vertebrate 'hosts' are influenced by both ecosystem conditions, such as environmental oxygen concentrations, and by community structure, such as the presence of species that bioaccumulate the toxin in the food web (Perez-Fuentetaja *et al.*, 2006). Disease is thus a specific state of pathology, and simple exposure to a pathogen may or may not induce disease depending on host condition, exposure dosage and factors such as local climate. Although the 'pathogen' is typically an infectious organism or particle (infectious disease) in such discussions, many diseases are caused by non-living agents such as pollutants, biotoxins or even trauma (non-infectious disease) (Johnson & Carpenter, 2008).

The central importance of host–pathogen-environment interactions is well recognised in epidemiology and often referred to as the disease 'triangle' or 'triad' (Jones, 1998; Scholthof, 2007). These connections create practical challenges for understanding disease causality and epidemiological patterns in natural environments. Environmental cofactors, multi-host parasite life cycles, state-dependent pathology (such as physiological stress or host age) or host–pathogen interactions that are difficult or unethical to manipulate in an experimental setting can all complicate the process of identifying the aetiological agents responsible for disease. For example, some coral diseases are the result not of a single pathogen, but of a 'consortium' of microbes that interactively cause pathology under certain environmental conditions (Voss *et al.*, 2007). Thus, many diseases cannot be understood by studying host and pathogen in isolation from the environment; an ecologically relevant understanding of disease patterns often requires a combination of modelling, laboratory and field experiments, broad-scale field surveys and diagnostic pathology. For example, Plowright *et al.* (2008) advocated a multifaceted approach to disease aetiology involving strong inference, causal diagrams and model selection applied iteratively, emphasising the complexities involved in studying disease emergence and highlighting the necessity of multidisciplinary collaborations.

Fundamental to this approach – and perhaps one of the most frequently misunderstood concepts of disease ecology – is the recognition that parasites and pathogens are an inherent and natural component of nearly all ecosystems. While researchers' interests are often centred on disease emergence, many forms of environmental change actually decrease or even eliminate parasites from a system, which is one foundation underlying the applied field of 'environmental parasitology' (Lafferty, 1997; Hudson, Dobson & Lafferty, 2006; Sures, 2008). Thus, the challenge confronting ecologists and epidemiologists alike centres around understanding and predicting which pathogens are likely to emerge (or decline) in response to specific forms of environmental change and how to manage such changes towards a desired outcome. Before examining these issues in freshwater ecosystems specifically, we begin with a discussion of the role of water in disease transmission and an overview of water-related diseases in humans and wildlife.

Fresh waters and disease

Freshwater environments serve multiple roles in disease relationships ranging from a transmission medium for water-borne diseases to a larval habitat for vectors that transmit infections. Warm, moist environments can enhance the persistence of infective stages and facilitate their movement among hosts in three-dimensional space, helping to reduce the challenges of desiccation and local dispersal facing many terrestrial disease agents. Because of the importance of fresh water to the survival of many organisms and its relative scarcity across the landscape (<0.08% of the world's surface), freshwater ecosystems often function as reliable points of species interaction and pathogen exchange between terrestrial and aquatic organisms. The small volume of many freshwater ecosystems relative to marine environments further increases the encounter probability between infectious stages and susceptible hosts. For example, animals frequently congregate at watering holes in the African savannah, which can facilitate disease transmission during dry seasons (Altizer *et al.*, 2006). Similarly, because people tend to establish developments and raise cattle close to local water sources, lakes and reservoirs in Africa function as reliable transmission foci for trematode diseases such as livestock liver flukes (fascioliasis) and human blood flukes (schistosomiasis), each of which is transmitted from

freshwater snails to mammals (Woolhouse *et al*., 1998). These conditions can be exacerbated during droughts when water availability is reduced (e.g. Lipp, Huq & Colwell, 2002).

Because of the dependability of interactions between freshwater habitats and a wide range of taxa, many pathogens have life cycles that include both aquatic and terrestrial stages. Digenetic trematodes, for example, use freshwater snails as intermediate hosts but often achieve sexual maturity in a terrestrial vertebrate, such as a mammal, bird or reptile (Combes, 2001). Trematodes responsible for medical and veterinary diseases, such as schistosomiasis, paragonomiasis and fascioliasis, all follow this pattern. Use of mobile definitive hosts allows these parasites to disperse between distant freshwater 'islands' separated by inhospitable areas. Other pathogens rely on vectors that use freshwater, including those transmitted by adult mosquitoes and black flies, or are transported as infected host propagules by waterfowl (e.g. Okamura *et al*., 2010). Perhaps as an evolutionary result of the scarcity of insects in marine habitats (Vermeij & Grosberg, 2010), terrestrial animals acquire relatively few infections from marine systems with the exception of some infections acquired through food. Collectively, pathogens with aquatic and terrestrial stages or hosts also represent significant trophic linkages between these ecosystems with potentially important effects on food web properties (Lafferty *et al*., 2008; Amundsen *et al*., 2009).

Finally, freshwater ecosystems are among the most altered environments on the globe. Because of their relatively small volume and low position in the landscape, freshwater habitats are subject to extensive runoff and disturbance from surrounding terrestrial areas, yet they lack the diluting potential of large marine systems (Dudgeon *et al*., 2006). Problems resulting from nutrient pollution, chemical contamination, dam construction, invasive species, climate change and food web shifts have all contributed to imperil biodiversity in fresh waters, which collectively have a higher proportion of threatened and endangered species relative to marine and terrestrial systems (Ricciardi & Rasmussen, 1999; Dudgeon *et al*., 2006; Abell *et al*., 2009). How such disturbances affect diseases is often uncertain, however. Many emerging infections are linked to environmental change, and environmentally induced increases in pathogens could further exacerbate declines among already threatened taxa (Daszak *et al*., 2000; Smith, Sax & Lafferty, 2006). On the other hand, parasites with specialised or complex life cycles are often sensitive to disturbance and the concomitant reductions in host density, such that some diseases will decrease in prevalence following particular forms of environmental change. Ward & Lafferty (2004), for example, suggested that disease reports in marine fishes have declined in recent decades owing to overfishing and reductions in host availability. These observations underscore the importance of evaluating temporal changes in both the frequency of disease-related reports in fresh water and the types of pathogens involved.

Overview of water-related diseases

Many pathogens of humans and wildlife depend on freshwater ecosystems. Infectious pathogens are typically classified as either microparasites or macroparasites. Microparasites are small, capable of multiplying within a host and include the viruses, bacteria, fungi and protists. Water-related examples include the apicomplexan *Plasmodium* (Marchiafava & Celli) (malaria in birds and mammals), the protist *Giardia lamblia* Stiles (giardiasis in mammals), VHS virus (VHS in fishes) and the oomycete *Aphanomyces astaci* (plague in crayfish). Macroparasites, in contrast, are typically larger in size and do not multiply directly within individual hosts; common groups include the flatworms (trematodes), the roundworms (nematodes), the tapeworms (cestodes), the spiny-headed worms (acanthocephalans) and ectoparasitic arthropods (ticks, copepods, mites and water lice). Macroparasites are responsible for diseases such as schistosomiasis in humans (trematode), yellow grub in fishes (trematode), heartworm in canids (nematode), eustrongylidosis in birds (a nematode) and certain limb deformities in amphibians (trematode). Diseases can also be caused by non-infectious agents, including chemical contaminants (e.g. lead poisoning), lack of oxygen (hypoxia) and toxin-producing cyanobacteria, bacteria or fungi. In the botulism example mentioned earlier, pathology is caused not directly by bacteria (*C. botulinum*), but instead by the botulinum toxin produced by the bacteria under certain environmental conditions (i.e. warm, anoxic waters) (Franson & Friend, 1999). Similarly, toxins produced during cyanobacterial blooms in fresh

water can cause serious pathology and mortality in both aquatic and terrestrial organisms, occasionally even in humans (Hallegraeff, 1993; Landsberg, 2002).

Transmission of freshwater-related pathogens is generally classified as either direct, in which infections move directly from an infected to a susceptible host, or indirect, requiring passage through another host species or life stage before returning to the original host species. Water-related parasites with direct transmission often produce water-borne infectious stages that either penetrate susceptible hosts or are consumed by them alongside food or water. Many parasites with indirect transmission that depend on water are considered 'water-based' rather than water-borne (see Bradley, 1977 for a more detailed classification). Some use intermediate hosts or vectors to complete transmission. Examples include the blood flukes (trematodes), which alternately infect freshwater snails and birds or mammals, and *Myxobolus cerebralis* (a myxozoan), which alternates between tubificid worms and salmonid fish and is the cause of whirling disease. Vector-borne pathogens can include parasites with complex life cycles involving obligate development in vectors. Freshwater examples include many viral and protist parasites transmitted by biting insects such as malaria, dengue fever, West Nile virus, Japanese encephalitis, yellow fever and river blindness (Resh, 2009).

Patterns of disease emergence in fresh waters

To explore patterns of disease emergence in freshwater ecosystems and how they have changed over time, we quantitatively analysed the primary scientific literature using an approach modified from Ward & Lafferty (2004). For a given freshwater host taxon, we examined the number of published papers in ISI Web of Science on disease relative to the total number of papers published for the group. This approach assumes that, over longer time horizons, research activity represents emergent patterns in nature. Ward & Lafferty (2004) validated the method in application to emerging diseases in marine ecosystems. Here, we applied this approach to diseases in freshwater ecosystems and specifically those affecting the following host groups: fishes, amphibians, molluscs, crayfishes, reptiles, birds and mammals. We selected these groups because their diseases are well studied, they encompass a wide range of vertebrate and invertebrate taxa, and because their diseases have relevance for conservation, human health or aquaculture. Our primary questions were: (i) Is there evidence of long-term trends in the number and frequency of disease-related publications (after normalising for total research effort)? (ii) How do patterns vary across the host taxa considered? (iii) Are there recurrent patterns with respect to the types of pathogens reported and their modes of transmission?

Approach

Using the literature search engine ISI Web of Science, we developed title-specific search strings composed of the major taxonomic families, genera and/or common names (e.g. muskrat, crayfish, anaconda) of each of our taxonomic groups so as to be specific to freshwater systems (see Appendix S1). Following Ward & Lafferty (2004), we did not search abstracts because ISI does not include abstracts for many journals prior to 1990. We included exclusion terms (e.g. NOT marine or experiment* or aquacult* or cell*) to reduce the likelihood of results focused on marine systems, laboratory experiments, aquacultural programmes or biochemical studies (Appendix S1). This process was iterative in that we refined our search terms to optimise their specificity. When search strings involved more than 50 terms, which is the limit for an individual search with Web of Science, we conducted multiple searches and subsequently combined them using the 'OR' command for advanced searches, which joins only unique records and excludes any duplicates. To further narrow the resulting records, we used the 'refine' function to exclude certain document types (e.g. reviews, editorials, corrections, etc.) and subject areas (e.g. biochemistry, psychology, pharmacology, oceanography, etc.). The lists of included document types and subject areas for each major taxon are presented in Appendix S2. We searched all Web of Science records between 1970 and 2009, as this is generally the time horizon considered relevant for emerging infectious diseases (Morse, 1995; Friend, 2002; Jones *et al*., 2008).

One challenge inherent to conducting habitat-specific searches is identifying which species can be reasonably classified as 'freshwater-related'. We identified freshwater taxa within each of the host groups using recent classifications developed by the Freshwater Animal Diversity Assessment and other

sources (Bailan *et al*., 2008; http://fada.biodiversity.be/) for amphibians (Vences & Köhler, 2008 and amphibiaweb.org), crayfishes (Crandall & Buhay, 2008), molluscs (Bogan, 2008; Strong *et al*., 2008), fishes (Leveque *et al*., 2008 and fishbase.org), birds (Dehorter & Guillemain, 2008; Clements *et al*., 2009), reptiles (Bauer & Jackman, 2008; Bour, 2008; Martin, 2008; Pauwels, Wallach & David, 2008) and mammals (Veron, Patterson & Reeves, 2008). We included genera or families known to be freshwater-dominated (>50% species freshwater-associated). For larger groups, we included major genera (with at least 25 species) and used common names or categories to increase specificity. Because some groups can move between freshwater and marine or terrestrial ecosystems, we endeavoured to make searches more restrictive (i.e. we tried to limit false-positive records) in accordance with our goal to examine broad trends in disease-related research, rather than to find all articles on the topic.

To evaluate the number of disease-related articles relative to the total number of articles, we combined the broad searches for each taxonomic group with a disease-specific search string (Appendix S1) using the 'AND' joining function, which yields only records appearing in both sets of searches within ISI. We recorded the year of publication for each study and extracted additional information on the host group, aquatic system, pathogen type and its transmission mode from articles published since 1990 to assess contemporary disease patterns. If more than 250 disease-related articles had been published since 2000 for a given taxon, we randomly selected >250 from which to extract information. To test whether the frequency of disease reports in each group changed over time (1970–2009), we calculated the annual proportion of records involving disease relative to the total number of papers. We then tested for a significant temporal trend using a generalised additive modelling approach, which allows for nonlinear relationships and uses a smoothing function to examine the relationship between a response variable and one or more predictors (in this case, time) (see Zuur *et al*., 2009). Cross validation was used to estimate the optimal amount of smoothing, if any. For each host group, analyses were performed for both the total number of disease publications per year as well as the proportion of disease-related publications per year. Because of the potential for temporal autocorrelation in time-series data, we used Autoregressive Moving Average Models to incorporate autoregressive and/or moving average terms at the appropriate lag (determined iteratively in conjunction with autocorrelation plots of model residuals). We used Akaike's Information Criterion (AIC; Burnham & Anderson, 2002), which penalises for the number of model parameters, to select among candidate models with different autocorrelation structures, including the model with no autocorrelation terms. We further verified that the residuals exhibited no obvious deviations from normality. For analyses involving count data (e.g. number of publications per year), we used the Poisson distribution. All analyses were conducted using the statistical programme *R* with the packages mcgv and nlme (R Development Core Team 2008).

Results

Based on our examination of all or a subset of articles from each search, our methods were generally effective in identifying records focused on diseases in the selected freshwater taxa. Both the total number of retrieved articles and the frequency related to disease varied substantially among the groups. After refinements for document type and subject area, we recovered 18 297 amphibian records with 5.5% related to disease, 6527 mollusc articles with 12.4% related to disease, 3396 crayfish articles with 3.8% related to disease, 42 145 freshwater fish articles with 6.7% on disease, 9495 bird articles with 8.1% related to disease, 5049 reptile articles with 3.8% related to disease and 3935 freshwater mammal articles with 11.9% on disease. Among birds and to a lesser extent fishes, however, searches returned a relatively high proportion of articles focused on molecular rather than epidemiological studies of disease which required the addition of further exclusion terms (Appendix S2).

The total number of disease-related articles per year increased significantly between 1970 and 2009 for amphibians ($R^2_{adj} = 0.89$, $P < 0.0001$), crayfishes ($R^2_{adj} = 0.50$, $P < 0.0001$), fishes ($R^2_{adj} = 0.95$, $P < 0.0001$), birds ($R^2_{adj} = 0.49$, $P < 0.01$), mammals ($R^2_{adj} = 0.32$, $P < 0.05$) and reptiles ($R^2_{adj} = 0.39$, $P < 0.0001$) (Fig. 1). Molluscs exhibited only a marginal increase ($P = 0.09$). Generalised additive models with smoothing terms rather than linear models provided a

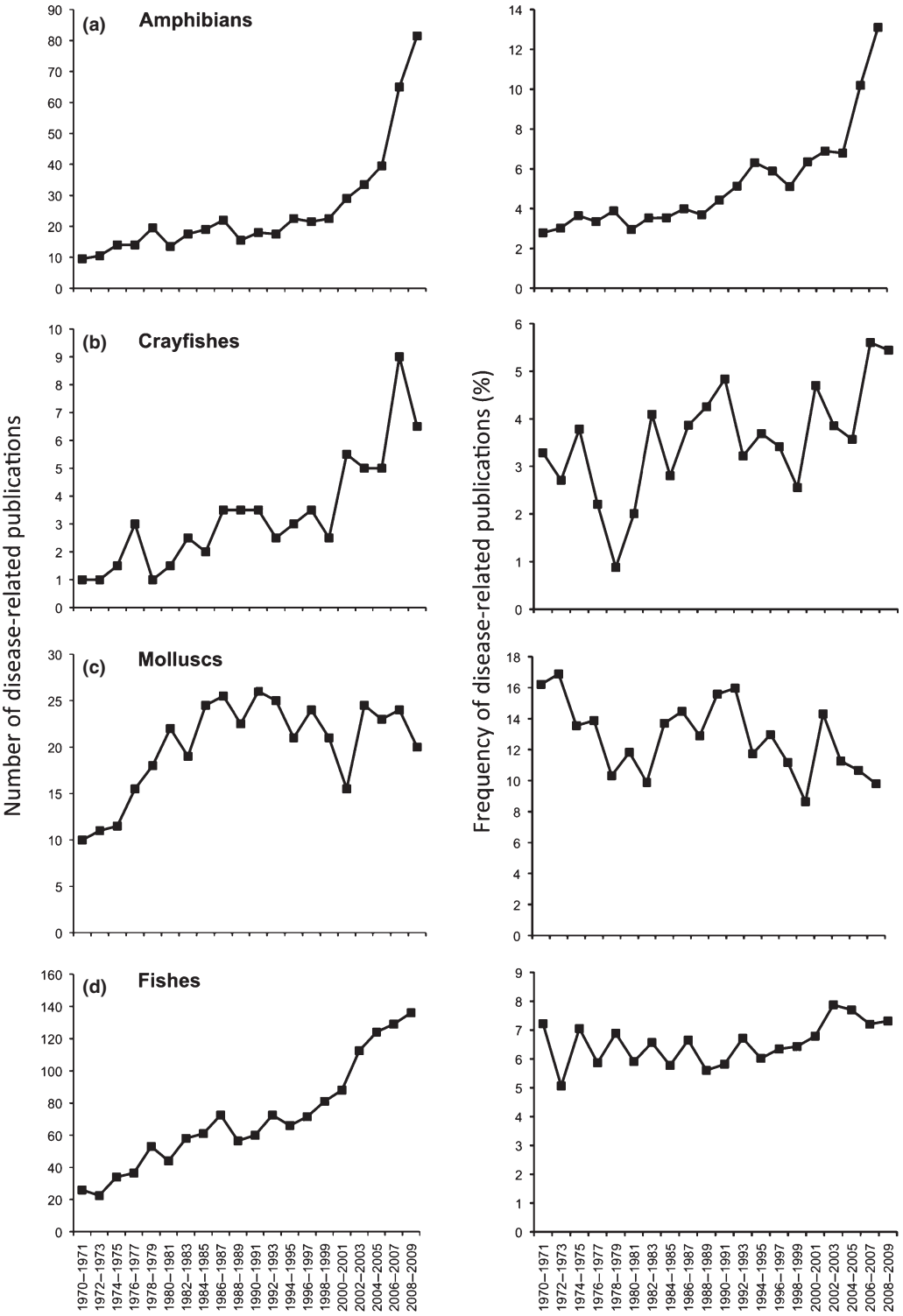

Fig. 1 Long-term (1970–2009) trends in disease-related publications from major freshwater groups, including (a) amphibians, (b) crayfishes, (c) molluscs, (d) fishes, (e) birds, (f) reptiles and (g) mammals. Graphs in the left column illustrate the total number of disease-related publications for each taxon as a 2-year average. Graphs in the right column depict the percentage of disease-related articles relative to the total number of articles published for a given taxon, also as a 2-year average value. Publication data derived from Web of Science.

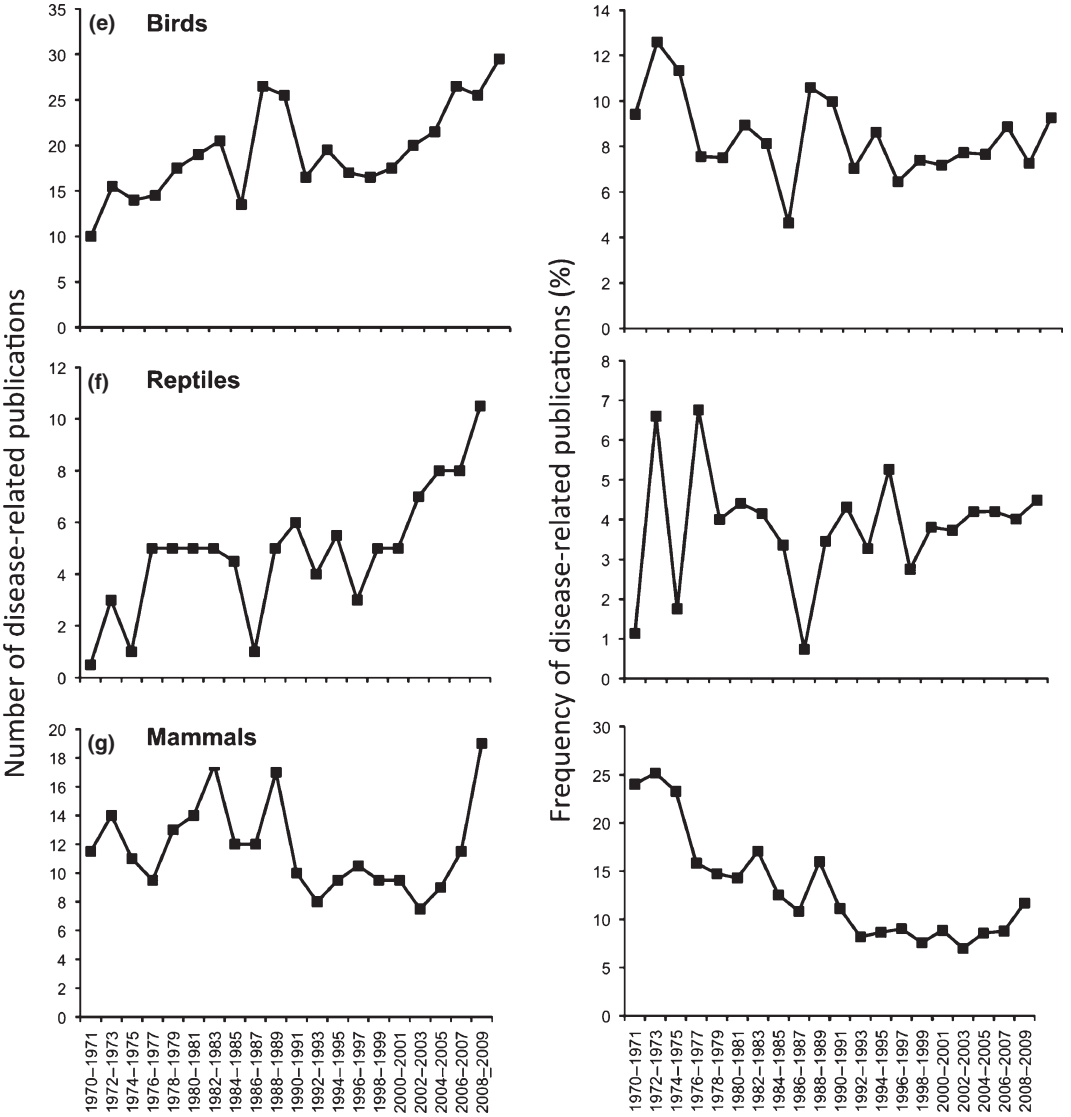

Fig. 1 (Continued).

superior fit to the data for amphibians, molluscs, mammals, fishes and birds, while significant autocorrelation structures were included in the models for amphibians, mammals, crayfishes, fishes and birds.

After correcting for total research effort, the proportion of disease-related articles also changed over time, and we found evidence of significant temporal patterns in disease research for five of the seven groups. Amphibians, crayfishes and fishes each exhibited long-term increases in the proportion of disease-related articles (Fig. 1; Amphibians R^2_{adj} = 0.80, P = 0.0021; Crayfishes R^2_{adj} = 0.10, P = 0.0098; Fishes R^2_{adj} = 0.13, P = 0.021). This trend was particularly pronounced in amphibians, for which the last 9 years had the highest proportion of disease-related articles in the amphibian data time-series (average of 2001–09 = 9.02% ± 0.99 SE). Results from the mollusc and mammal searches each revealed significant decreases or decreasing trends over time in the proportion of disease-related research articles (Fig. 1; Mollusc R^2_{adj} = 0.12, P = 0.04; Mammal R^2_{adj} = 0.62, P = 0.11). Reptiles and birds exhibited no long-term changes in disease publications (i.e. the slopes were not significantly different from zero). Based on AIC values, autocorrelation structures were included for crayfishes, molluscs and mammals while generalised additive models were selected for amphibians and mammals.

Table 1 Taxonomic classification of the types of parasites and pathogens reported in disease-related publications for each freshwater taxon. Presented is the percentage (%) of records involving each group of parasites as a function of host taxon. Because many publications report on more than one type of parasite, the sum of each column can exceed 100%

Pathogen type	Amphibians $n = 243$	Crayfishes $n = 127$	Molluscs $n = 163$	Fishes $n = 250$	Reptiles $n = 107$	Mammals $n = 125$	Birds $n = 214$
Virus	8.2	31.5	0.6	24.0	12.2	24.8	50.5
Bacteria	4.9	8.7	0	14.8	16.8	6.4	14.9
'Fungus'	41.2	41.0	0	6.4	2.8	6.4	1.4
Chytridiomycete	33.3	0	0	0	0	0	0
Microsporidian	0.4	18.1	0	3.2	0	0	0
Oomycete	2.9	21.6	0	1.6	0	0	0
Helminth	70.4	9.4	88.3	43.6	74.8	75.2	49.1
Digenean	22.6	8.7	87.7	8.0	19.6	20.8	8.9
Monogenean	3.7	0	0	9.6	5.6	0	0
Nematode	24.3	0	0.6	8.0	34.6	21.6	22.4
Cestode	7.4	0	0	10.8	6.5	16.8	12.6
Acanthocephalan	8.2	0	0	3.2	6.5	8.8	4.2
'Protists'	3.3	5.5	7.4	4.8	3.7	13.4	4.6
Ciliate	1.2	0.8	4.9	3.2	0	0	0
Apicomplexan	1.2	3.9	1.8	0	6.5	4.8	5.1
Trypanosome	0.8	0	0	0.4	0	1.6	0
Myxozoan (Cnidarian)	0.4	0	0	14.4	2.8	0	0.5
Oligochaete or leech	1.2	0	1.2	0.4	1.9	0	0.5
Arthropod	2.5	0	0.6	5.2	6.5	4.8	2.8
Copepod	0	0	0.6	2.4	0	0	0
Mite	0.8	0	0	0	0	0.8	0.5
Louse	0.4	0	0	2.8	0	0	1.4
Pentastome	0	0	0	0	5.6	0	0
Diptera	1.2	0	0	0	0	0.8	0.5
Tick/flea	0	0	0	0	0	2.4	0.5
Toxins and pollutants	0	0	0	0	3.7	0	0

Among the subset of articles published since 2000 that we examined in more detail, the predominant types of pathogens varied among host taxa. Most mollusc publications involved snails (86%) rather than mussels (10.8%) or clams (3.6%) ($n = 166$) (note that the total percentages can exceed 100% because many articles report on more than one host or pathogen type). Of those reports in which the type of pathogen was classified ($n = 163$), the majority (87.7%) involved digenetic trematodes, for which snails function as intermediate hosts (Table 1). Many of these were diseases of medical or veterinary importance, such as schistosomiasis and echinostomiasis in humans and fascioliasis in humans and livestock (Table 2). Other pathogens of significance to human health included *Cryptosporidium parvum* (Tyzzer), *G. lamblia* and hepatitis E virus. Crayfish diseases were dominated by viral and fungal parasites (Table 1), including white spot syndrome virus (22.6%), *A. astaci* (crayfish plague) (20%) and *Thelohania contejeani* (12.2%, cause of porcelain disease).

Among reports of amphibian disease, 72.7% of records involved frogs, 21.2% involved toads, 15.2% involved salamanders and 3% involved newts ($n = 198$). Reported pathogens of amphibians included viruses, bacteria, helminths, dipterans, protists, fungi and leeches (Table 1). The chytridiomycete *Batrachochytrium dendrobatidis* was the most commonly reported individual pathogen (35.5% of all amphibian-disease publications), whereas the helminths (nematodes, trematodes, cestodes and acanthocephalans) were the most frequent overall parasite group (70.4% of all amphibian-disease publications) (Table 2). Common genera of helminths recorded in amphibians included nematodes such as *Rhabdias* and *Oswaldocruzia* (Travassos) and trematodes such as *Echinostoma*, *Ribeiroia*, *Glypthelmins* (Stafford) and *Haematoloechus* (Looss). While some are pathogenic (e.g. *Ribeiroia* and *Echinostoma*) (Johnson & McKenzie, 2008), the effects of many helminths are either unknown or have little pathology. Other parasites included a diversity of protozoans (trypanosomes,

Table 2 Most commonly reported parasites and pathogens from each of the freshwater taxa considered. Listed are the pathogen, its taxonomic designation, the disease it causes (if applicable), and the percentage of disease-related publications referencing the pathogen. The mode of transmission is presented as a superscript letter. Note that inclusion in this table does not necessarily indicate that a given parasite is considered 'emerging', as our analysis focuses only on frequency of occurrence in publications, not changes over time. Unless otherwise stated, the disease listed refers to a condition occurring in the host group

Host group	Pathogen[†]	Taxonomy	Disease	Records (%)
Amphibians	*Batrachochytrium dendrobatidis* (Longcore *et al.*)[H]	Chytridiomycete	Chytridiomycosis	33.3
	Rhabdias spp.[C]	Nematode		9.2
	Ranavirus[H]	Virus		6.5
	Echinostomes[C]	Trematode	Echinostomiasis, oedema	5.3
	Ribeiroia ondatrae (Price)[C]	Trematode	Limb malformations	3.1
	Saprolegnia spp.[H]	Oomycete	Saprolegniasis	3.1
Crayfishes	White spot syndrome virus[H, V]	Virus	White spot syndrome	22.6
	Aphanomyces astaci (Schikora)[H]	Oomycete	Crayfish plague	20
	Thelohania contejeani (Henneguy)[C]	Microsporidian	Microspordiosis (porcelain disease)	12.2
	Alloglossoides spp.[C]	Trematode		4.3
	Psorospermium haeckeli (Haeckel)[C]	Uncertain	Psorospermiosis	3.5
Molluscs	*Schistosoma mansoni* (Sambon)[C]	Trematode	Human schistosomiasis	35.4
	Fasciola hepatica (L.)[C]	Trematode	Human and livestock fascioliasis	15.9
	Echinostomes[C]	Trematode	Human echinostomiasis	15.9
	Trichobilharzia spp.[C]	Tremaode	Avian schistosomes	2.1
	Chaetogaster spp.[H]	Oligochaete		1.4
Fishes	Infectious salmon anaemia virus[H]	Virus	Infectious salmon anaemia	6.4
	Infectious hematopoietic necrosis virus[H]	Virus	Hematopoietic necrosis	4.4
	Myxobolus cerebralis (Hofer)[C]	Myxozoan	Whirling disease	4.0
	Tetracapsuloides bryosalmonae (Canning *et al.*)[C]	Myxozoan	Proliferative kidney disease	3.2
	Spring viremia of carp virus[H]	Virus	Spring viremia of carp	2.4
Reptiles	*Mycobacterium*[H]	Bacterium	Tuberculosis	5.6
	Micropleura[C]	Nematode		4.7
	Sebekia[C]	Pentastomid		4.7
	Falcaustra	Nematode		4.7
	Mycoplasma[H]	Bacterium	Pneumonia	3.7
Mammals	Aleutian mink disease parvovirus[H]	Virus	Aleutian mink disease	22.5
	Strongyloides spp.[C]	Nematode		13.9
	Mucor amphibiorum (Frank *et al.*)[H]	Fungus	Ulcerative dermatitis	8.6
	Canine distemper virus[H]	Virus		8.6
	Skrjabingylus spp.[C]	Nematode	Sinus worms	6.9
	Schistosoma spp.[C]	Trematode	Schistosomiasis	6.9
Birds	Avian influenza A virus[H]	Virus	Highly pathogenic avian influenza	30.4
	Contracaecum[C]	Nematode		7.0
	West Nile virus[C]	Virus	Encephalitis	5.6
	Escherichia coli (Castellani & Chalmbers)[H]	Bacterium	Septicemia	4.7
	Salmonella[H]	Bacterium	Salmonellosis	4.7
	Newcastle disease virus[H]	Virus	Newcastle disease	3.7

[†]Mode of transmission for each pathogen: C = complex (requiring multiple hosts or free-living and parasitic stages); H = horizontal (spreading directly from host to host); V = vertical (from parent to offspring).

apicomplexans, ciliates and poorly described groups such as *Perkinsus*-like and *Ichthyophonus*-like organisms), myxozoans (classified as 'protozoa' although now viewed to be cnidarians; Jiménez-Guri *et al.*, 2007) and ranviruses (e.g. *Ambystoma tigrinum* virus, Frog virus 3, *Rana catesbeiana* virus-Z, *Rana grylio* virus) (Table 2).

Publications on fish diseases were dominated by two main host families: the Salmonidae (40.4%) and the Cyprinidae (22.8%). Other well-represented families of fish hosts included Cichlidae (5.2%), Ictaluridae (5.2%), Centrarchidae (4%), Gasterosteidae (3.6%) and Percidae (3.2%). Fish pathogens also exhibited a high diversity, including viruses, bacteria, fungi and several types of helminths, protists, myxozoan and arthropod parasites (Table 1). Viruses were the most common type of pathogen reported (24% of fish disease records), while bacteria (14.8%), myxozoans

(14.4%) and cestodes (10.8%) were also well represented (Table 1). The most common pathogens included salmon anaemia virus (6.4%) and infectious hematopoetic necrosis virus (4.4%) (Table 2). Among myxozoan parasites, *M. cerebralis*, which causes whirling disease and *Tetracapsuloides bryosalmonae*, which causes proliferative kidney disease, were involved in 4 and 3.2% of publications, respectively (Table 2). Commonly encountered genera of bacteria included *Aeromonas* (Stanier), *Flavobacterium* (Berget *et al.*), *Vibrio* (Pacini) and *Mycobacterium.*

In birds, the majority of disease-related publications involved either viral (50.5%), helminth (49.1%) or bacterial infections (14.9%) (Table 1). With respect to specific pathogens, avian influenza A virus was the single most commonly studied pathogen (30.4% of publications), particularly in reference to highly pathogenic avian influenza (HPAI). Other common pathogens included *Contracaecum* spp. nematodes (7.0%), West Nile virus (5.6%) and *Salmonella* spp. bacteria (4.7%) (Table 2). Ducks were the most frequently reported host group and were involved in 31.3% of publications.

For both freshwater reptiles and mammals, helminths were the most often reported parasites, comprising 74.8 and 75.2% of publications, respectively. Reptiles were also commonly infected with bacteria (16.8%), viruses (12.2%) and arthropods (6.5%), especially the pentastomes (see Table 1). In mammals, viruses (24.8%) and protists (12.8%) were each relatively common, with Aleutian mink disease parvovirus as the single most common individual pathogen. Interestingly, however, no specific pathogens comprised more than 8% of publications for reptiles (Table 2). Turtles were involved in 53.7% of reptile-related disease publications, whereas crocodilians were involved in 33%, snakes in 12% and lizards in <1%. For mammals, minks (33.3%), muskrats (9.3%) and river otters (9.3%) were the most common host groups in disease publications.

Discussion

Despite widespread concerns over disease emergence in wildlife (e.g. Daszak *et al.*, 2000; Harvell *et al.*, 2004), few efforts have specifically explored patterns related to freshwater diseases. Recent reviews focused on marine and terrestrial environments have reported evidence of increasing infections in some, but not all examined taxa (Harvell *et al.*, 1999; Epstein, 2001; Anderson *et al.*, 2004; Lafferty *et al.*, 2004). Baseline data on freshwater diseases in wildlife are largely lacking, and in spite of growing interest in freshwater biodiversity and conservation (e.g. Dudgeon *et al.*, 2006; Bailan *et al.*, 2008), the role of infectious diseases as a conservation threat remains understudied for most groups (e.g. Edgerton *et al.*, 2002; Collins & Crump, 2009; Winfield, 2009). Trends of freshwater-related diseases in humans have a mixed contemporary status. Following prolonged eradication efforts and treatment campaigns, some infections have declined dramatically, including dracunculiasis (Guinea worm disease), onchocerciasis (river blindness) and lymphatic filiariasis (elephantiasis) (Fenwick, 2006). However, other freshwater-related diseases are either stable or increasing in severity or distribution in association with drug resistance or environmental changes (Cotruvo *et al.*, 2004; Fenwick, 2006). Malaria and schistosomiasis, for instance, are two of the most common infections in sub-Saharan Africa outside of HIV/AIDS and collectively cause nearly 50 million years of life lost to death or disability annually (WHO 2004; Hotez *et al.*, 2007).

The epidemiological patterns of water-related diseases are frequently subsumed (or thought to be subsumed) under terrestrial disease systems; however, many properties of freshwater ecosystems with the potential to influence pathogen transmission and evolution differ distinctly from those found in marine or terrestrial environments. These distinctions include the following: the high rates of endemism and diversity in fresh waters, the small size and confined distribution of most freshwater systems, an elevated disturbance regime and high frequency of inputs from terrestrial habitats, an abundance of taxa with biphasic life histories and the tendency of freshwater systems to serve as interaction foci between aquatic and terrestrial organisms (see Dudgeon *et al.*, 2006). Such characteristics emphasise the importance of studies focused specifically on patterns of disease in fresh waters.

Results of our literature analysis provide some of the first quantitative evidence that certain freshwater taxa are exhibiting long-term increases in the frequency of disease-related publications, similar to findings from marine environments (Ward & Lafferty, 2004). After normalising for total research effort, which has increased for nearly all host groups since

1970, disease reports increased significantly in amphibians, crayfishes and fishes. This pattern was particularly pronounced for amphibian diseases, which began exhibiting a nonlinear increase in publication frequency in the late 1990s (see Fig. 1). These results are consistent with widespread concerns over amphibian population declines and the hypothesised role of infectious disease in amphibian population losses and extinctions (Stuart *et al.*, 2004; Skerratt *et al.*, 2007; Collins & Crump, 2009; Kilpatrick, Briggs & Daszak, 2010).

Importantly, however, not all groups exhibited an increase in disease publications over time. Freshwater mammals and molluscs each exhibited decreases in disease-related publications. Because many of the molluscan publications focused on human pathogens in the genus *Schistosoma*, this latter result might suggest long-term changes in either the prevalence of human schistosomiasis (e.g. associated with reductions in the cost of treatment) or in the number of researchers involved in tropical medicine (Fenwick, 2006). Why freshwater mammalian diseases would be on the decline is less clear, but this is also the group for which we recovered the fewest overall publications and is therefore information-limited.

For freshwater-related birds and reptiles, we found no significant temporal trend during the time frame of the analysis. Given recent interest in several high-profile diseases of freshwater birds including West Nile virus and avian influenza (Lanciotti *et al.*, 1999; Daszak *et al.* 2004; Olsen *et al.*, 2006), the lack of a detectable increase in bird diseases is surprising. Dieoffs associated with West Nile virus, duck plague, avian botulism, avian cholera and avian vacuolar myelinopathy have been widely reported over the last few decades (Horák & Kolárová, 2010; Franson & Friend, 1999; Converse & Kidd, 2001; Friend, 2002; Rocke *et al.*, 2005a; Blanchong, Samuel & Mack, 2006; Newman *et al.*, 2007). Disease is considered an important factor in explaining increases in waterfowl mass-mortality events (Friend, McLean & Dein, 2001; Rocke *et al.*, 2005b; Newman *et al.*, 2007). These trends may have been obscured in our dataset by a relatively high recovery of records focused on molecular rather than epidemiological studies of disease for birds. Wild waterfowl share a number of diseases with both domesticated birds and humans (Tsiodras *et al.*, 2008), making them good model organisms for the study of pathology or vaccine development. For example, 45% of the publications during the 1987 peak in disease-related publications (Fig. 1) are molecular studies of hepatitis B in ducks for the purpose of vaccine development. Further study of temporal trends in waterfowl mortality events is needed to confirm patterns in disease emergence and to assess the importance of other factors, including toxin exposure, habitat degradation and environmental change (Franson & Friend, 1999; Rocke *et al.*, 2005b).

While changes in scientific publications are not necessarily equivalent to changes in natural phenomena, increases in research effort are suggestive of changing patterns, particularly after controlling for total research effort and temporal autocorrelation. Ward & Lafferty (2004) provided evidence of the validity of this method by examining the increased publication frequency on rabies in North American raccoons, a disease for which the original introduction and spread are well documented. Qualitatively, we see a similar pattern in our own results with respect to amphibians. After its first description (Berger *et al.*, 1998; Longcore, Pessier & Nichols, 1999), the pathogenic chytridiomycete *B. dendrobatidis*, which accounted for 30% of disease-related publications in amphibians, exhibited a steady increase in the number and frequency of publications, paralleling the hypothesised global spread of this emerging pathogen (e.g. Lips *et al.*, 2006; Skerratt *et al.*, 2007). Similar increases were noted in the frequency of publications related to West Nile virus in birds following its introduction to the U.S.A. in 1999 and in studies of HPAI virus (H5N1) after its latest emergence in 2003–04 (Olsen *et al.*, 2006; LaDeau, Kilpatrick & Marra, 2007).

Our analysis provides one of the first quantitative, multi-taxa evaluations of long-term disease patterns in freshwater wildlife. Nevertheless, several caveats to this approach warrant discussion. First, coarse analyses of publication frequency such as this do not explicitly differentiate among papers describing small versus large epizootics. This can make it challenging to examine the impact of different diseases or the number of diseased and dying hosts associated with infection. For instance, type C botulism is estimated to have killed more than 15 000 water birds in California between 1978 and 2003, including 15% of the western population of white pelicans (Rocke *et al.*, 2005a). Nevertheless, this entire die-off was documented by only a small number of peer-reviewed scientific publications (see Friend *et al.*, 2001; Rocke *et al.*,

2005a). Second, this analysis makes no attempt to assess the disease-causing ability or emergent status of each parasite. Thus, while some infections cause mass mortality in host populations, as exemplified by botulism in the previous example, other publications document the endemic parasitofauna of healthy hosts. Many of the publications on helminths, for example, likely reflect research on endemic rather than epidemic infections. Hypothetically, ongoing declines in parasitological research could dampen or even mask patterns of emerging infectious diseases for a particular group. And finally, changes in amount of non-disease-related publications have the potential to alter the frequency of disease articles by changing the denominator. Increases in the number of conservation-related articles on molluscs (Lydeard *et al*., 2004), for example, could dampen detection of long-term changes in disease research.

We also noted sharp differences in both the types and diversity of pathogens affecting each host group. Diseases in crayfishes, for instance, were dominated by viruses and fungi, which were collectively involved in 70% of examined crayfish disease records. Infections in molluscs were overwhelmingly dominated by digenetic trematodes (87% of records). In amphibians, trematodes, nematodes and chytrid infections were involved in 80% of publications. Amphibians also exhibited a diverse suite of pathogens, possibly as a result of ecological factors (e.g. their biphasic life histories) or more intensive study and surveillance efforts associated with the widespread patterns of amphibian population declines (e.g. Stuart *et al*., 2004; Densmore & Green, 2007). Fish pathogens were remarkably diverse, with viruses as the dominant group (24% of records) and myxozoans, trematodes, cestodes, bacteria and nematodes each comprising between 8 and 15% of the remaining fish disease records. The high diversity of fish pathogens may reflect a combination of increased research effort associated with the economic importance of fisheries and an increased frequency of pathogen spillover associated with aquaculture (Heggberget *et al*., 1993; Poulin *et al*., 2010) and environmental change (Okamura *et al*., 2010).

Whether these observations represent natural patterns of pathogen infection and diversity among the examined host groups remains conjectural. The use of molluscs as intermediate hosts by trematodes that cause disease in humans and wildlife, for example, likely explains the dominance of these parasites within mollusc-related publications. In addition, the high frequency of viruses among crayfishes, fishes and birds could result from greater research effort and increased detection ability along with the economic or societal value of these hosts. Most emerging human diseases involve viral, bacterial, fungal or protistan pathogens (Taylor *et al*., 2001; Jones *et al*., 2008) for which treatments may not be well developed. Other groups that cause diseases in humans may be less likely to emerge because treatment options are available. If the same pattern holds for wildlife diseases, we might interpret the relative dominance by helminth infections in molluscs, reptiles and mammals as indicative of a low frequency of emerging infections. In contrast, the high levels of viral, bacterial, fungal and protist infections in fishes, crayfishes, amphibians and birds could reflect a greater fraction of emerging disease threats. Indeed, fishes, crayfishes and amphibians each exhibited long-term increases in the frequency of disease-related publications. Nevertheless, variation in disease surveillance and technology suggest such patterns must be interpreted with caution.

In light of the ongoing freshwater biodiversity crisis, in which many taxa have experienced severe declines in recent decades (e.g. Ricciardi & Rasmussen, 1999; Stuart *et al*., 2004; Dudgeon *et al*., 2006), we might actually expect diseases to show corresponding decreases (Marcogliese, 2005). Parasite transmission often depends critically on host density; when host density falls below a certain threshold, parasites can be eliminated from the system (McCallum, Barlow & Hone, 2001; Penczykowski *et al*., 2010). Ward & Lafferty (2004) suggested this might be one reason why the frequency of disease-related publications has decreased in marine fishes, which have been heavily harvested over the last century (Jackson *et al*., 2001). However, various forms of human activity can interact with disease to exacerbate declines in native species, even while host density continues to decrease (Daszak *et al*., 2000; de Castro & Bolker, 2005; Smith *et al*., 2006). In fresh waters, species invasions, agricultural and aquacultural intensification and habitat modification are among the most likely forms of environmental change affecting disease patterns. For example, introductions are responsible for some of the most spectacular emerging diseases in fresh waters, including amphibian chytridiomycosis (possibly

introduced from Africa to other continents), crayfish plague (introduced to Eurasia along with North American crayfish) and whirling disease (introduced to North America along with European brown trout) (Edgerton *et al.*, 2004; Weldon *et al.*, 2004; Koel *et al.*, 2005). Each of these infections has caused significant die-offs and/or economic losses in native taxa (Hedrick *et al.*, 1998; Edgerton *et al.*, 2004; Skerratt *et al.*, 2007). In addition to transmitting novel pathogens to native hosts, invasive species can alter disease transmission through dilution or amplification of native pathogens, or via indirect effects on native host immunity (Poulin *et al.*, 2010).

Likewise, aquacultural and animal husbandry practices have contributed to the emergence and spread of many fish, crayfish and waterfowl pathogens in natural systems (Reed *et al.*, 2003; Murray & Peeler, 2005). Aquacultural effects on disease can occur in association with translocation of animals to new regions (species introductions) or owing to the pathogen-conducive conditions created by maintaining large numbers of animals of multiple species in confined quarters (Krkosek, Lewis & Volpe, 2005). For example, the monogenean disease gyrodactyliasis and the bacterial disease furunculosis were both unobserved in wild Norwegian salmonid populations prior to 1970; expansion of aquacultural facilities and fish stocking in the region has correlated with the subsequent increases in disease-induced mortality in wild fish populations (Heggberget *et al.*, 1993). The accelerated growth of aquacultural production since the mid-1980s (e.g. Tilseth, Hansen & Moller, 1991; Rafael, 2008; Diana, 2009) could also help to explain the post-1990 increase in publications on fish diseases (Fig. 1). Similarly, high rates of crowding in domestic poultry operations can impair avian immune responses and facilitate mutation of pathogens that can then be spread to migrating waterfowl (Reed *et al.*, 2003; Webster & Hulse, 2004).

Finally, environmental changes to freshwater habitats, such as the alteration of natural flow regimes to create impoundments, eutrophication, increasing temperatures and habitat loss have frequently been linked to emerging diseases. Schistosomiasis, for example, which alternates between humans and freshwater snails, increased dramatically in association with construction of major dams in Africa during the latter half of the 20th century (Fenwick, 2006; Resh, 2009). Other parasites have also been linked to modification of freshwater habitats such as eutrophication or increasing temperatures (Okamura *et al.*, 2010; Lafferty, 1997; Johnson & Carpenter, 2008; Johnson *et al.*, 2010). Additionally, wetland habitat loss for migrating bird species can lead to increased crowding at stopover sites for migrating birds, facilitating pathogen transmission between individuals and species (Reed *et al.*, 2003; Blanchong *et al.*, 2006). Nevertheless, because not all of the identified pathogens in our results can be categorised as 'emerging', we cannot rule out the possibility that some of the observed trends reflect shifts in funding priorities or overall research interest rather than shifts in disease patterns driven by environmental changes.

Disease, water and the future

Freshwater ecosystems play a critical role in many disease patterns, and growing evidence suggests that future changes in water storage and availability, climate patterns and nutrient enrichment will affect the levels of water-related disease in human and wildlife populations (Daszak *et al.*, 2000; Marcogliese, 2001; Brooks & Hoberg, 2007; Johnson & Carpenter, 2008; Smith & Schindler, 2009). Water-related diseases already kill more than 5 million people per year with an estimated 2 billion suffering morbidity, and 2.2 billion lacking access to improved sanitation (WHO 2004; Resh, 2009). There are no reliable estimates of wildlife mortality associated with water-related diseases, but the figure is likely substantial based on observed bird, fish and amphibian die-offs alone. Warming temperatures could influence future patterns of disease in fresh waters by shifting host and pathogen ranges, changing pathogen or host replication rates, and altering host–parasite interactions, contributing to the emergence of some diseases and the decline of others (Okamura *et al.*, 2010; Paull & Johnson, in press). Other forms of environmental change such as drought, eutrophication and temperature-mediated changes in pathogen transmission or host immunity also have significant potential to alter patterns of infection and disease in a broad variety of host taxa and freshwater ecosystems (Altizer *et al.*, 2006; Smith & Schindler, 2009; Johnson *et al.*, 2010; Paull and Johnson, 2010). Such changes will undoubtedly cause increases in some pathogens and decreases in others, underscoring the importance of predictive

approaches to help guide the allocation of resources. Because increases in infectious disease risk have serious implications for human health, economic sustainability and conservation, it is essential that consideration of water-borne pathogen exposure is integrated into evaluations of global change and water quality.

Alongside conventional medical approaches focused on therapeutic treatment of affected individuals and identification of infection risk factors, there is an urgent need to recognise and address the complex and fundamental ecological interactions that drive disease emergence. Emerging infections often are directly or indirectly associated with shifting patterns in land use, which can alter the ecological and evolutionary relationships among humans, wildlife and the pathogens that move between them (Daszak *et al*., 2000; Dobson & Foufopoulos, 2001; Patz *et al*. 2004). More than 70% of emerging human diseases are vector-borne or zoonotic (Taylor *et al*., 2001; Woolhouse & Gowtage-Sequeria, 2005). Consequently, patterns of infection in humans are often intricately linked to the levels of infection in wildlife (e.g. American Veterinary Medical Association 2008), and effective disease management requires knowledge of the ecological factors that influence humans, wildlife hosts and their interactions (National Research Council 2001; Smith *et al*., 2006). It is thus crucial that the capacity of well-managed freshwater ecosystems for disease mitigation be formally quantified as an 'ecosystem service' (Swallow *et al*., 2009). By attaching economic value to the disease-preventing capacity of freshwater systems, the importance of this ecosystem service becomes more apparent.

Formal recognition of the role of freshwater ecosystems in controlling disease risk will facilitate the development of management strategies to reduce levels of freshwater diseases. While vaccines and treatment for some water-borne diseases are now widely available (Fenwick, 2006), treatments can be expensive, unavailable to the regions most in need, and often do little to prevent re-infection. Incorporation of ecological approaches to the study of disease can offer new insights about both emergence and prevention and will facilitate the concurrent examination of human and wildlife health (Cottingham, Chiavelli & Taylor, 2003; Guernier, Hochberg & Guegan, 2004; Jones *et al*., 2008). Some ways to manage for lower disease include reducing nutrient and pollutant runoff associated with agricultural intensification, managing aquacultural practices to minimise pathogen spillover, reducing translocations of non-native species and their parasites and establishing cooperative surveillance efforts focused on integrated monitoring of both human and wildlife pathogens. Likewise, there is a pressing need to more explicitly incorporate spatial scale into studies of water-related diseases, especially as many such diseases include both aquatic and terrestrial phases that operate at different spatial scales. The use of metacommunity analyses in combination with spatial epidemiology (Ostfeld, Glass & Keesing, 2005) and risk modelling (Thrush *et al*., 2010) are promising approaches and may help to integrate studies focused on freshwater, marine and terrestrial disease systems. Appropriate management of freshwater ecosystems could provide one of the most valuable of ecosystem services: disease control and prevention.

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Supporting Information

Additional Supporting Information may be found in the online version of this article:

Appendix S1. Search string used in Web of Science to identify articles related to each taxonomic group and diseases within each taxonomic group.

Appendix S2. Refinements of Web of Science searches on the basis of article types and subject areas.

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