Review

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Emerging infectious diseases of wildlife: a critical perspective

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We review the literature to distinguish reports of vertebrate wildlife disease emergence with sufficient evidence, enabling a robust assessment of emergence drivers. For potentially emerging agents that cannot be confirmed, sufficient data on prior absence (or a prior difference in disease dynamics) are frequently lacking. Improved surveillance, particularly for neglected host taxa, geographical regions and infectious agents, would enable more effective management should emergence occur. Exposure to domestic sources of infection and human-assisted exposure to wild sources were identified as the two main drivers of emergence across host taxa; the domestic source was primary for fish while the wild source was primary for other taxa. There was generally insufficient evidence for major roles of other hypothesized drivers of emergence.

Introduction

Be they impacting people, agriculture, or wildlife, emerging infectious diseases (disease-causing agents that rapidly increase in geographical range, host range, or prevalence) are acknowledged to be occurring at an increased rate globally [1–3]. Management to successfully mitigate these threats requires identifying and understanding their drivers. However, it is increasingly recognized that many reports of currently and recently emerging disease-causing agents may have insufficient supporting evidence to substantiate their status as such [4,5]. In such cases, frequently limited resources for research and management may be misallocated with respect to where they could make the most valuable impact. In addition, the 'noise' generated by spurious cases may obscure accurate assessments of emergence drivers and thus be misleading in considerations of suitable and effective management actions to decrease risk of emergence.

Here we conduct to the best of our knowledge the most critical review and assessment to date of the current and recent vertebrate wildlife emerging infectious disease literature (see Box 1 for the methodology used). Our aim is threefold. First, we separate agents for which there is sufficient evidence of emergence from those for which there is insufficient evidence to support such a conclusion and interrogate the patterns observed with respect to host and agent taxa and the timing and geography of emergence. Second, based on only those agents with sufficient evidence, we objectively identify and rank in terms of importance the causes and drivers of disease emergence in vertebrate wildlife, to provide robust guidelines for management to mitigate such threats to wild populations (see Box 2 for all of the potential drivers of disease emergence indicated by the full review). Third, we provide direction to researchers regarding where efforts would be best focused to further increase our understanding of, and thus our ability to prevent, such disease emergence.

Amphibians and reptiles

Nine disease-causing infectious agents of amphibians and reptiles were identified with evidence of potential emergence from 2000 onward (Table S1 in the supplementary material online). Amphibians were the most affected group (with six potential agents) followed by turtles (with three). Almost half of the potential emergences are ongoing from the past century and there is no obvious temporal bias in those reported this century (Figure 1). There is also no strong evidence of any agent taxon bias, microparasite versus macroparasite bias, or geographical region bias within this set.

Of the nine agents identified, only three (all microparasites) were considered to have sufficient evidence of their current emergence (Figure 1 and Table 1): *Batrachochytrium dendrobatidis* causing the disease chytridiomycosis in adult amphibians [6,7]: the related *Batrachochytrium salamandrivorans* in fire salamanders in The Netherlands [8]; and ranavirus causing mass mortalities of larval amphibians [9,10] (Table S1 in the supplementary material online). Both *B. dendrobatidis* and ranavirus are ongoing emergences from the past century, with the spread of ranavirus both within North America and to England and the continued global spread of *B. dendrobatidis*. *B. salamandrivorans* is a recent emergence.

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Box 1. Literature review methodology

To provide an initial set of candidate disease-causing infectious agents that are potentially emerging or continuing to emerge, we applied a consistent set of search terms to the Web of Science and Google Scholar databases and the Google search engine. This set comprised all combinations of pathogen*, parasite*, or disease* with each of the following in turn: emergence, emergent, emerging, resurgent, new, novel, expanding range, change*, shift*, switch*, expansion*, introduction*. Searches were run separately for different host taxon sets (amphibians and reptiles, birds, eutherian mammals, fish, marsupials and monotremes) and the findings interrogated to generate a list of disease-causing agents for which there is some evidence of emergence from 2000 onward in wild host populations. Reports of newly discovered agents, expanding host or geographical ranges, increasing disease impacts, or increasing prevalence of known disease-causing agents were all considered indicators of potential emergence. However, cases where the evidence presented was entirely speculative or was (without other supporting evidence) the first host record for a known generalist parasite, the first geographical record for a known widespread parasite, reported in a single individual, or a new species or strain in a group known for its diversity across hosts were excluded. Agents with no known disease impacts were also excluded, as were agents with evidence for emergence in domestic populations only and reported diseases with putative but not yet confirmed causative agents.

To consider the drivers of emergence, a more conservative set of agents was derived from the 'potentially emerging' list. Specifically, we considered only those agents with sufficient evidence of emergence. This refinement was necessary since including cases that are not actually emerging (which considering the full list of potentially emerging agents would likely do) would obscure our assessment of the relative importance of various drivers. By contrast, the potential loss of one or two actually emerging agents, while minimizing 'false positives' in this way, would not obscure our assessment as long as any potential biases generated by this approach are considered. We used the following criteria: evidence (not solely correlational) that the agent causes disease impacts at either the individual or the population scale, together with either: (i) evidence of a consistent trend of increase (or a maintained increase) in disease impact, host species range, or geographical range; or (ii), for epizootic outbreaks, evidence that the outbreaks observed are likely not snapshots of natural long-term variation in disease dynamics. For this second criterion, where agents have undergone monotonic increases in host or geographical range (or disease incidence or impact), we considered at least one survey of relevant host, spatial, and temporal extent prior to the potential emergence that shows a significantly lower level of infection to be sufficient. Where agents have sporadic outbreak dynamics, we required enough evidence of prior absence or difference to be confident that such outbreaks have not occurred previously (dependent on the size and duration of those currently/recently observed) for sufficiency.

Note that under our criteria the documentation of a new species or strain of infectious agent that is known to be variable in ecological timeframes is not considered emerging, unless such variation allows that agent to infect a novel host species or hosts in a new geographical region or at a novel level of infection or impact. Every time a new strain of the common cold appears, we do not consider the cold to be emerging; rather, it is part of the natural dynamics that characterizes this disease.

Birds

Eight disease-causing infectious agents of birds were identified with evidence of potential emergence from 2000 onward (Table S2 in the supplementary material online), counting Lineages 1 and 2 of West Nile virus as distinct emergences. Six of these agents solely or mainly impact passerine birds while two impact waterfowl. A large proportion were initially reported from North America or Europe, also with a temporal bias in reporting; while two are ongoing from the past century, the other six were all first reported during 2000–2004 (Figure 1). All of the agents in this set are microparasites (with over half of them being viruses).

Of the eight agents identified, five (four viruses and one bacterium) were considered to have sufficient evidence of their current emergence (Figure 1 and Table 1): the ongoing spread of West Nile virus Lineage 1 [11,12] and *Mycoplasma gallisepticum* [13,14] in North America; the emergence and spread of avian influenza A (H5N1) across Asia, Europe, and Africa [15] [World Health Organization (2011) *H5N1 Avian Influenza: Timeline of Major Events* (http://www.who.int/influenza/human_animal_interface/en/)]; and the emergence and spread of both West Nile virus Lineage 2 [16,17] and Usutu virus [18,19] in Europe.

Eutherian mammals

Eighteen disease-causing infectious agents of eutherian mammals were identified with evidence of potential emergence from 2000 onward (Table S3 in the supplementary material online). While the most common host orders were the Carnivora (both terrestrial and aquatic) and Cetacea, potential emergences were reported from a wide host range. As with birds, a large proportion of potential emergences were initially reported from North America or Europe, again with a temporal bias in reporting; while four potential emergences are ongoing from the past century, ten were first noted during 2000–2004 compared with only three during 2005–2009 and one during 2010–2014 (Figure 1). The potentially emerging agents are strongly biased toward microparasites (particularly viruses and bacteria). Note that H5N1 is considered emerging in birds (with occasional spillover to eutherian mammals [20–23]) and thus is not counted here.

Of the 18 agents identified, only five (four microparasites and one macroparasite) were considered to have sufficient evidence of their current emergence (Figure 1

Box 2. Potential drivers of disease emergence indicated by the full review

- Host species or population exposure to an infectious agent from other wild populations. Such exposure can occur through movement of the exposed host, or agent movement. It can be human-assisted or natural, and may be facilitated by agent adaptation to new hosts or vectors after initial emergence.
- Host species or population exposure to an infectious agent from domestic populations. Agents in domestic populations to which wildlife can be exposed may be the result of agent movement into and among populations, high host densities, transmission among species, and agent evolution in domestic populations.
- Host clustering/overcrowding/stress due to habitat modification.
- Host poor nutrition/food supply variation/dietary shifts.
- Host thermal stress/climate change.
- Waning host immunity/immune variation/immune system modulators.
- Host genetic instability/low genetic diversity/inbreeding.
- Favorable climate for vectors/other drivers of increasing vector numbers.
- Note that several of these drivers are not necessarily independent (e.g., waning host immunity may be caused by stress, poor nutrition, or genetic issues).

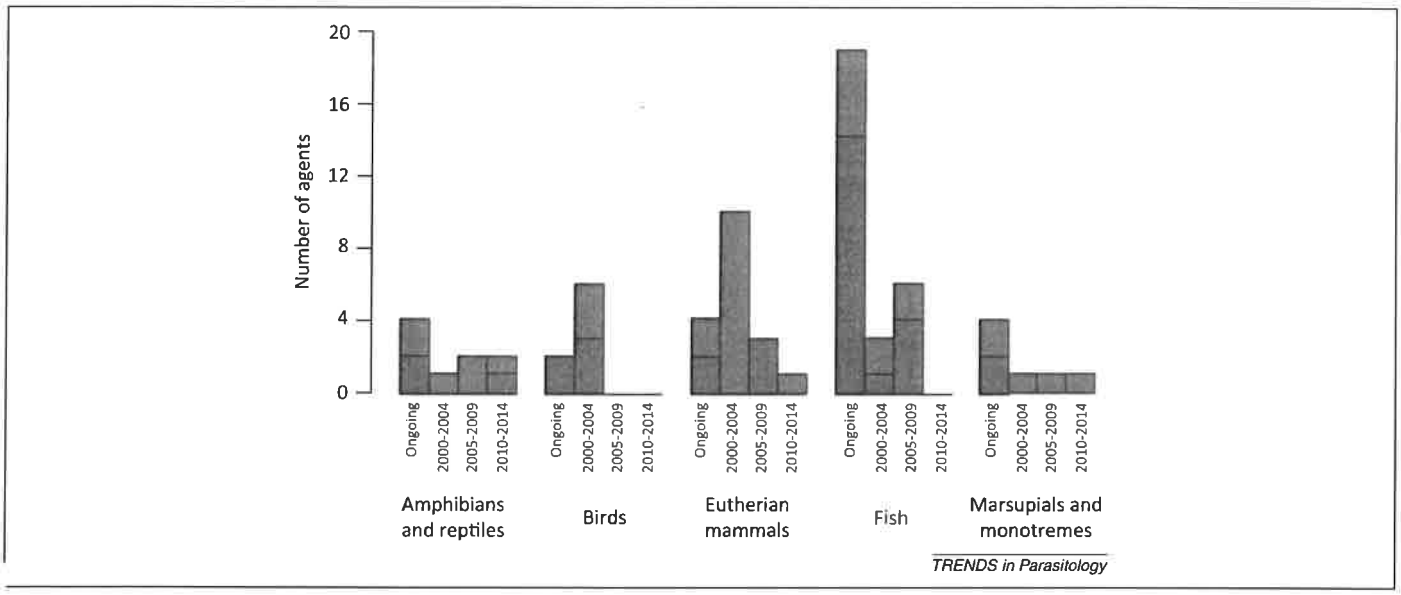

Figure 1. Disease-causing emerging infectious agents of vertebrate wildlife that are either potentially emerging in wild populations from 2000 onward (blue) or for which there is sufficient evidence of emergence (red), by host taxon group. See Box 1 for criteria. Agents are split into those for which emergence is ongoing from the past century and those for which emergence was first recorded in 2000–2004, 2005–2009, or 2010–2014.

and Table 1): the ongoing spread of squirrelpox virus through Great Britain [24–26]; and, in North America, the dramatic outbreaks of *Pseudogymnoascus destructans* (causing white-nose syndrome) in bats [27,28], the expanding range of chronic wasting disease (CWD) in ungulates [29], and the emergence of both canine distemper virus (CDV) [30] and sarcoptic mange [31] in the newly established wolf population in Yellowstone National Park.

Fish

Twenty-eight disease-causing infectious agents of fish were identified with evidence of potential emergence from 2000 onward (Table S4 in the supplementary material online). Agents were recorded across freshwater, estuarine, and marine host species, but with a bias toward economically important fish in temperate waters (salmonids, cyprinids, and catfish) and those that are farmed for food production or ornamental trade. There is very poor representation of emerging infectious diseases of tropical fish, in both freshwater and marine environments, as well as pelagic marine environments in general. Nineteen of the potential emergences are ongoing from the past century, three were first reported during 2000–2004 and six during 2005–2009 (Figure 1). While there is again a microparasite bias, the taxonomic spread of agents is broad.

Of the 28 agents identified, 19 (still microparasite biased but with a broad taxonomic spread) were considered to have sufficient evidence of their current emergence (Figure 1 and Table 1): the ongoing spread of cyprinid herpesvirus [32], epizootic hematopoietic necrosis virus (EHNV) [33], European catfish virus (ECV) [33], infectious spleen and kidney necrosis virus (ISKNV) [33], red seabream iridovirus (RSIV) [33], viral hemorrhagic septicemia virus (VHSV) [34], largemouth bass virus [35], *Aeromonas salmonicida* [36], *Aphanomyces invadans* (with a first record in Africa in 2006) [37], *Myxobolus cerebralis* [38], *Neoheterobothrium hirame* [39], *Caligus* spp. [40], *Lepeoptheirus salmonis* [41], and *Lernaea cyprinacea* [42] all extending into broader geographical ranges; and the emergence and spread of spring viremia of carp virus (SVCV) [43] and *Ichthyophonus* [44] in North America, *Sphaerothecum destruens* [45] and red vent syndrome caused by *Anisakis simplex* [46] in Europe, and *Edwardsiella ictaluri* in freshwater sweetfish in Japan [47].

Marsupials and monotremes

Seven disease-causing infectious agents of marsupials and monotremes were identified with evidence of potential emergence from 2000 onward (Table S5 in the supplementary material online); four are ongoing from the past century and there is no obvious temporal bias in those reported this century (Figure 1). Six of these agents impact marsupials (spanning kangaroos, wallabies, bandicoots, wombats, and the Tasmanian devil) and one impacts monotremes (platypus in Tasmania). Of the six marsupial-infecting agents, five are recorded from Australia and one from South America. Six of the seven agents in this set are microparasites, in addition to sarcoptic mange (considered an independent emergence to that noted for eutherian mammals).

Of the seven agents identified, only two (one microparasite and one macroparasite) were considered to have sufficient evidence of their current emergence (Figure 1 and Table 1): devil facial tumor disease (DFTD) in Tasmania [48]; and sarcoptic mange across Australia [49,50]. DFTD is an ongoing emergence in the Tasmanian devil from the 1990s with continued spread from east to west [51], while sarcoptic mange (also an ongoing emergence) has increased its host range to include macropods such as wild swamp wallabies and agile wallabies [49,50].

Taxonomic identity of emerging infectious agents

In the complete set of agents with sufficient evidence of emergence (N = 34), there is a clear skew toward microparasites across all host taxa; 76% were microparasites and 14% macroparasites, with over half of the

Table 1. Disease-causing infectious agents of vertebrate wildlife with sufficient evidence of emergence in wild populations from 2000 onward (see Box 1 for criteria)[a]

Parasite/disease	Host	Locality	Emergence dynamics	Cause
Microparasites				
Viruses				
A(H5N1) influenza	Primarily birds; some eutherian mammals	Eurasia, Africa	Continental spread from emergence in Hong Kong in 2002	Spillover from domestic poultry into wild birds; mutation into highly pathogenic strain; spread via wild bird dispersal/migration and human movement of birds; mammal cases generally presumed to be due to spillover from wild birds or poultry in the diet of captive animals
Canine distemper virus (CDV)	Wolves	Yellowstone National Park, USA	Emergence in newly established population in 2005–2008	Possible spillover from coyote and red fox
Cyprinid herpesvirus (also koi herpesvirus)	Cyprinid fish	Widespread	Emerged 1990s; ongoing spread through North America, Europe, Asia, Australia	Spread via global trade of goldfish and transport of carp; potential interaction with warm-water stress and spillover/spillback effects
Epizootic haematopoietic necrosis virus (EHNV)	Redfin perch	Australia	Ongoing; emergence and spread in wild populations	Spread via trade/transport of fish
European catfish virus (ECV)	Perch, sheatfish, catfish	Europe	Ongoing emergence and continued spread	Spread via trade/transport of fish
Infectious spleen & kidney necrosis virus (ISKNV)	Fish	Asia	Ongoing emergence in many areas of Asia	Spread via trade and movement of ornamental and food fish
Largemouth bass virus	Largemouth bass	Southern USA	Ongoing; spread out of the southeast USA since 2000	Spread by boat movement
Ranavirus	Amphibians	North America, England	Ongoing; evidence for spread in North America and to England	Spread through trade/transport of amphibians; sporadic local nature suggests various sufficient causes including waning immunity
Red sea bream iridovirus (RSIV)	Fish	Asia	Ongoing emergence in many areas of Asia	Initial (1990) spillover from wild fish; spread via trade and movement of ornamental and food fish
Spring viraemia of carp virus (SVCV)	Carp	North America	Emergence and possibly spread in North America from 2002	Introduction from Europe or the Middle East via trade/shipping of carp and koi (ornamental fish)
Squirrelpox virus	Red squirrel	Scotland, Ireland	Continual spread from emergence location in England	Spillover from introduced grey squirrels; presumed introduction to Ireland with introduced grey squirrels
Usutu virus	Birds	Europe	Emergence in and local spread from Austria in 2001	Agent spread from Africa by wild bird migration; local spread through wild bird dispersal; favorable climate for vectors
Viral hemorrhagic septicemia virus (VHSV)	Fish	North America	Ongoing spread in eastern North America	Hydrodynamic spread (spread via commercial shipping implicated in original emergence)
West Nile virus Lineage 1	Birds	North America	Ongoing continental spread from emergence in NY, USA in 1999	Introduction from Israel in 1999 by air transport; continental spread through wild bird dispersal; strain adaptation to vectors
West Nile virus Lineage 2	Birds	Europe	Local spread from emergence in Hungary in 2004	Introduction from Africa by wild bird migration; local spread through wild bird dispersal; favorable climate for vectors
Bacteria				
Aeromonas salmonicida	Fish (lamprey)	Canada	Ongoing spread to new locations and species (lamprey in Lake Ontario)	Spillover and spillback between wild and aquaculture populations
Edwardsiella ictaluri	Sweetfish	Japan	Emergence in Japanese freshwaters, in wild sweetfish, in 2007	Molecular evidence indicates introduction from Asia; hypothesized unintentional introduction to wild with fish release into rivers
Mycoplasma gallisepticum	Birds	USA	Introduction to western (native) house finch populations (from eastern) in 2002	Spillover from domestic poultry/novel strain (1994); continental spread through wild bird dispersal; facilitated by clustering at bird feeders
Fungi (and fungus like)				
Aphanomyces invadans	Fish	USA, Australia, Asia, Africa	Ongoing spatial spread in USA, Australia, Asia; reached Africa in 2006	Spatial spread via zoospores through river systems, coastal estuaries, and flood plains

Table 1 (*Continued*)

Parasite/disease	Host	Locality	Emergence dynamics	Cause
Batrachochytrium dendrobatidis	Amphibians	Worldwide	Ongoing population decline and extirpation in multiple species	Spread via globalization of amphibian trade and lack of biosecurity; origin possibly southern Africa or Asia
Batrachochytrium salamandrivorans	Fire salamander	The Netherlands	Emergence in 2013 causing 96% population decline	Spread via globalization of amphibian trade and lack of biosecurity; likely origin Asia
Ichthyophonus	Fish (American shad)	North America	First recorded epizootic in Columbia River watershed in 2007	Interaction with invasive host species leading to pathogen amplification; spillback concern to sympatric salmonids
Pseudogymnoascus destructans	Bats	North America	Spreading rapidly in the eastern and central USA and Canada since 2007	Introduction from Europe; continental spread by bat-to-bat transmission
Sphaerothecum destruens	Cyprinids, bream, salmonids, roach	Europe	Spreading through Europe and the UK since 2005	Spread to Europe via introduced North American salmon; spillover in Europe from non-native Asian species (the cyprinid *Pseudorasbora parva*)
Prions				
Chronic wasting disease (CWD)	Ungulates	North America	Expanding geographical range in North America ongoing from the past century	Possible spread among contiguous cervid and moose populations from the Colorado epicenter and associated with human movement of elk and deer
Cancer				
Devil facial tumor disease (DFTD)	Tasmanian devil	Tasmania	Expanding geographical range across the state ongoing from the past century	Good evidence that it is a newly emerged transmissible cancer; genetic instability and low genetic diversity likely to have facilitated emergence
Macroparasites				
Myxozoa				
Myxobolus cerebralis	Fish	Western North America	Ongoing spread particularly in Pacific Northwest and Alaska	Spread via introduction/transport of salmonids
Monogenea				
Neoheterobothrium hirame	Flounder	Japan	Ongoing spread through coastal Japan after introduction in 1999	Spread via introduction from North America, then host switching
Copepods				
Caligus spp.	Salmonids	Canada	Ongoing spread; increased abundance on juveniles; emergence in Canada	Spillback from salmon aquaculture
Lepeoptheirus salmonis	Salmon and trout	North Atlantic and Northeast Pacific coast	Increases ongoing from 1990 (starting in Europe) to 2005	Evolutionary escape from chemical therapuetants in aquaculture; spread and spillback from domestic reservoir
Lernaea cyprinacea	Freshwater fish	Western Australia	Ongoing spread and host range expansion across Australia	Spread via introduced cyprinids
Nematodes				
Anisakis simplex	Atlantic salmon	Northeast Atlantic	Simultaneous emergence of red vent syndrome across Norway and UK in 2007	Unclear; possible link to climate change or change in parasite–host interaction
Mites				
Sarcoptes scabiei	Wolves	Yellowstone National Park	Emergence in newly established population	Possible spillover from coyote and red fox, or domestic dog
	Marsupials	Australia	Ongoing expanding host range, including agile wallaby and swamp wallaby	Unclear

[a]Note that H5N1 is considered as emerging in birds (with occasional spillover to eutherian mammals) and West Nile virus Lineage 1 and 2 are considered independent emergences, as is *S. scabiei* in wolves and marsupials.

microparasites being viruses (Figure 2). Exceptions to the dominance of viral agents were observed in the marsupials and monotremes host taxa set (with one mite and one transmissible cancer being the only agents with sufficient evidence of emergence) and in the amphibians and reptiles set (with one virus and two fungi with sufficient evidence). This pattern of microparasite (and viral) dominance is similar to that observed by Daszak *et al.* in their earlier summary of emerging infectious diseases of wildlife [1]. However, macroparasites may be underrepresented

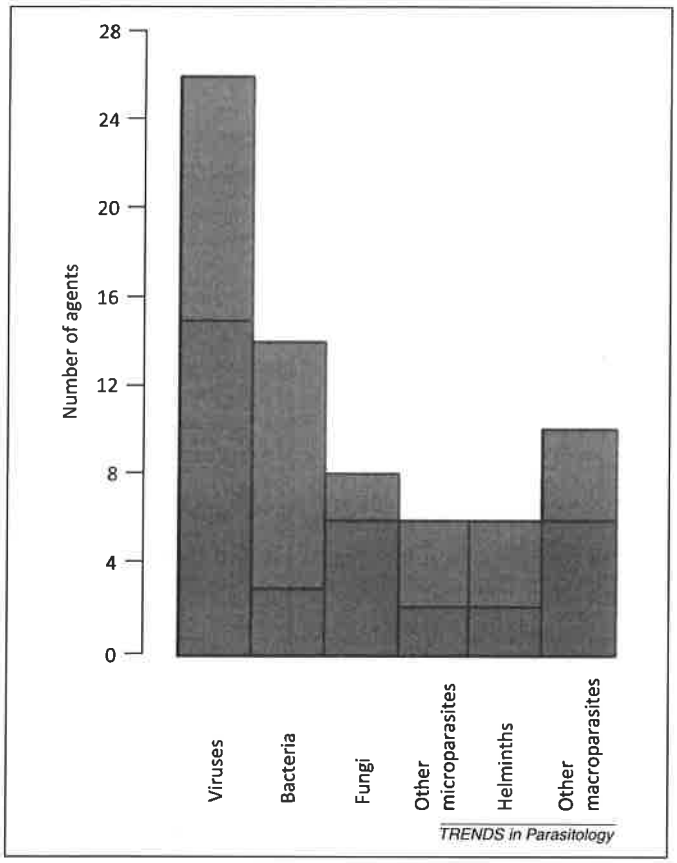

Figure 2. Disease-causing emerging infectious agents of vertebrate wildlife that are either potentially emerging in wild populations from 2000 onward (blue) or for which there is sufficient evidence of emergence (red), by agent taxon group. See Box 1 for criteria.

in both reviews since their impacts are generally more cryptic (for example, causing morbidity rather than mortality) [52]. For the macroparasite emergences with good evidence, observed emergence dynamics are frequently less dramatic than those of microparasites – increases in abundance, host range, or geographical range as opposed to epizootic outbreaks with associated mortality (Table 1).

Host taxon patterns of agent emergence

Reports from fish account for over half of the agents with sufficient evidence of emergence, with other host taxa accounting for approximately 5–15% each (Table 2). This may reflect a real greater rate of disease emergence in fish, a higher level of surveillance of fish populations, or simply that there are more species of fish in the world than of the other host taxa (Table 2). However, in terms of emergences with sufficient evidence per species known, it is the marsupial and monotreme host taxon set that is greatly overrepresented (Table 2). Although this may reflect a higher level of surveillance of a charismatic and relatively rare host group, it may also reflect intrinsic host issues. For example, the Dasyuridae (of which the Tasmanian devil is a member) are afflicted by genetic instability, while many marsupials and monotremes have been through past population bottlenecks and/or suffer current population fragmentation, both of which may increase their susceptibility to disease emergence [53,54]. At the other end of the scale, the amphibian and reptile host taxon set is underrepresented in terms of emergences with sufficient evidence per species known (Table 2). This may reflect that amphibians and reptiles are relatively neglected vertebrate taxa in terms of research and surveillance or could be a result of the particularly dramatic impacts of chytridiomycosis (and to a lesser extent ranavirus) across a broad host and geographical range precluding other emerging agents. Biases were also observed within the host taxon sets used, potentially reflecting variation in research and surveillance efforts.

Temporal and geographical patterns of agent emergence

For the agents with sufficient evidence of emergence, almost two-thirds were agents that initially emerged in the past century that were/are continuing to spread from 2000 onward. Of the remainder, four were first reported during the period 2000–2004, seven during 2005–2009, and only one during 2010–2014 (undoubtedly at least partly reflecting a time lag in clinical identification and publication in the peer-reviewed literature). Interestingly, there is a high proportion of potentially emerging agents first reported during 2000–2004 for which there is not sufficient evidence of emergence (Figure 1). One possible explanation for this is the influence that the emergence of (or agent identification for) several high-profile diseases in the late 1990s (including chytridiomycosis, DFTD, West Nile virus, mycoplasmal conjunctivitis, cyprinid herpesvirus, and CWD) had on this field of study. This raise in profile may have translated into both an increase in work focused on such and an increase in the numbers of agents claimed to be emerging regardless of the quality of the supporting evidence. This signal is particularly strong in

Table 2. Numbers of disease-causing infectious agents of wildlife documented as either potentially emerging or with sufficient evidence of emergence from 2000 onward. Emergences are detailed both across host taxa and in relation to the known number of species per host tax on set (see Box 1 for criteria)

Host tax on set	Known species per set	Potentially emerging agents		Agents with sufficient evidence of emergence	
	(approximate)	Total cases observed	Per known species	Total cases observed	Per known species
Amphibians & reptiles	17350	9	0.0005	3	0.0002
Birds	10000	8	0.0008	5	0.0005
Eutherian mammals	4000	18	0.0045	5	0.0013
Fish	27300	28	0.0010	19	0.0007
Marsupials and monotremes	339	7	0.0206	2	0.0059

the eutherian mammal host taxon set and in reports from North America and Europe (Tables S1–S5 in the supplementary material online), probably reflecting both host taxon and geographical biases in research and surveillance effort. An underrepresentation of reports from some geographical areas, particularly South America and Africa, remains evident in the sufficient evidence set (Table 1).

Drivers of agent emergence

There is a clear difference between fish and the other host taxa in which drivers played the greatest role in the emergence of disease-causing infectious agents (Figure 3). For the cases in fish with sufficient evidence (N = 19; Table 1), host exposure to infectious agents from domestic populations (aquaculture, stocks for release, and the ornamental fish trade) is recognized as the primary cause in 14 (74%). With exposure to domestic populations playing a lesser role in agent emergence in other host taxa (see below), this strongly implies that the overrepresentation of emergences in fish noted above is at least partly due to this driver. The disease issues within the domestic populations are due to a combination of factors (Table S4 in the supplementary material online), including agent movement into and among populations, high host densities, transmission among species, and some suggestion of agent evolution (for example, the emergence of *L. salmonis* being possibly associated with the evolutionary escape of strains from chemical therapuetants [41]). We do not tease apart any further the causes of disease issues within domestic populations (to which wild populations can be exposed) in this review. However, one pattern of note is the high level of macroparasite emergence in fish, which is highly likely to be linked to aquaculture practices (three-quarters of all macroparasites with sufficient evidence of emergence are reported from fish; Table 1).

For the other five cases with sufficient evidence of emergence in fish, the cause of red vent syndrome emergence in Atlantic salmon due to *A. simplex* is unknown [46], while host exposure to infectious agents from other wild populations is recognized as the driver of the remaining four (21%); one is due to natural source host movement (*Ichthyophonus* amplified in the Columbia River American shad population by infected migratory individuals from the Northeast Pacific [44]), two are due to human-assisted wild source host movement (*N. hirame* from North America to Japan [39], and *L. cyprinacea* from east to west Australia [42]), and one is due to human-assisted agent movement (boat movement among lakes in the Southern USA causing largemouth bass virus spread [35]). The only other driver hypothesized for the 'good evidence' set in fish is an interaction with warm-water stress for the emergence of cyprinid herpesvirus in wild populations (primarily due to domestic exposure) [32]. However, for this, and the other drivers suggested for potentially emerging agents only (Table S4 in the supplementary material online), there is generally insufficient evidence to support a major role in post-2000 disease emergence.

Across all other host taxa combined, exposure to infectious agents from domestic populations is recognized as the primary cause of three (20%) of the 15 agents with sufficient evidence that they are emerging or continuing to emerge from 2000 onward (Table 1): initial H5N1 emergence was facilitated by multispecies aggregations in markets [World Health Organization (2011) *H5N1 Avian Influenza: Timeline of Major Events* (http://www.who.int/influenza/human_animal_interface/en/)]; *M. gallisepticum* was sourced from poultry [13,14]; and initial CWD emergence is hypothesized to be the result of exposure to a domestic population (a novel strain from scrapie in domestic sheep) [55]. However, this driver is eclipsed by exposure to infectious agents from other wild populations being recognized as the primary cause of ten of these cases (Figure 3), five of which (33% of the total 15) are due to human-assisted wild source host movement (Table 1). In this manner, Old-to-New World agent spread has occurred for *B. dendrobatidis* [56–58] while ranavirus has spread in the opposite direction [59,60], *B. salamandrivorans* is a hypothesized introduction to Europe from Asia [8], West Nile virus Lineage 1 was introduced to North America from Israel [61–64], and squirrelpox virus was introduced to the

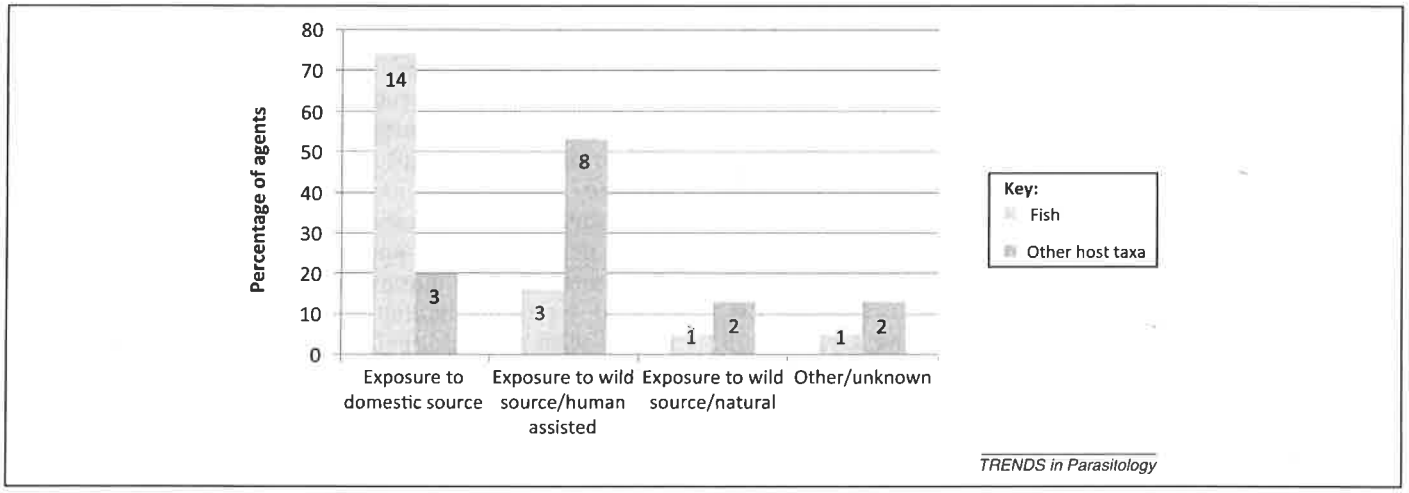

Figure 3. Primary drivers of emergence for the disease-causing emerging infectious agents of vertebrate wildlife for which there is sufficient evidence of emergence in wild populations from 2000 onward. Cases are split between those occurring in fish and those occurring in other vertebrate host taxa. Numbers of cases are indicated in bars. See Box 2 for criteria.

British Isles from North America through which it continues to spread [65]. The importance of human-assisted wild source host movement in disease emergence is also apparent in non-wild populations; for example, the introduction of monkeypox to North America in 2003 via a shipment of rodents from Africa rapidly spread via the pet trade to impact captive prairie dogs (and cause human zoonotic infections) across the continent [66].

Another of the ten wild exposure cases in non-fish taxa (7% of all 15 cases with sufficient evidence) is due to human-assisted agent movement (*P. destructans* from Europe to North America) [67], while a further two (13%) are due to human-assisted host movement (both CDV and sarcoptic mange emergence in the new Yellowstone National Park wolf population through exposure to sympatric wild canids) [30,31]. The emergence of Tammar wallaby sudden death syndrome in Australia, ongoing from 1998, indicates that this mechanism also occurs in other host taxa; this emerging disease (as with monkeypox, not included in our tables here as it was recorded impacting captive animals only) is believed to be due to the translocation of Tammar wallabies into the range of a pathogenic orbivirus [68]. The last two of the ten wild exposure cases in non-fish taxa (13% of the 15 cases with sufficient evidence) are due to natural wild source host movement (Usutu virus and West Nile virus Lineage 2, both from Africa to Europe via wild bird migration) [61,63].

The remaining two of the 15 agents with sufficient evidence that they are emerging or continuing to emerge in non-fish taxa from 2000 onward yet to be discussed are DFTD in Tasmanian devils and sarcoptic mange in Australian marsupials (Table 1). While the cause of sarcoptic mange emergence in Australian marsupials is unknown (predisposing host conditions such as age, illness, injury, nutritional inadequacy, and/or stress due to captivity or habitat disruption have been considered [49]), strong evidence indicates that DFTD emerged *in situ* within the species [69], with it being hypothesized that such emergence could occur due to genetic instability in the Dasyuridae (all of the family are prone to cancers) and/or low genetic diversity [associated with population bottlenecks during the last glacial maximum and an El Niño Southern Oscillation (ENSO) event 5–8 kiloyears before present (ky BP)] [53,54]. However, with only one example (7% of the 15 cases with sufficient evidence), the generality of this mechanism appears limited.

Other factors indicated or hypothesized to facilitate emergence in the sufficient evidence set for non-fish taxa are favorable climate conditions for the insect vectors of bird viruses (West Nile virus Lineages 1 and 2 and Usutu virus) [11,62,63], waning immunity against ranavirus outbreaks in frogs [70], land-use changes providing favorable host and vector habitats for West Nile virus Lineage 1 in North America [12], host clustering at garden bird feeders for *M. gallisepticum* [13,14], and possible exposure to DNA-altering carcinogens for DFTD [71]. However, for these, and the other drivers suggested for potentially emerging agents only (Tables S1–S3 and S5 in the supplementary material online), there are generally insufficient data to support any major role in post-2000 disease emergence. Although novel strain evolution is not indicated here as a cause of disease emergence (outside it being one potential factor creating disease risk in domestic populations to which wild vertebrates can be exposed), it is frequently a consequence of disease emergence in wildlife that leads to greater spread and impact: the evolution of A(H5N1) from H5N1 in waterfowl [15] [World Health Organization (2011) *H5N1 Avian Influenza: Timeline of Major Events* (http://www.who.int/influenza/human_animal_interface/en/)], West Nile Virus Lineage 1 adapting to infect local vectors more efficiently in North America [62], and the increase in virulence of *M. gallisepticum* post-emergence [72].

Evidence of absence or absence of evidence?

The more compelling examples of wildlife disease emergence are those with multiple lines of evidence all supporting the hypothesis of emergence and the proposed drivers. For example, the cases for chytridiomycosis, DFTD, West Nile virus, mycoplasmal conjunctivitis white-nose syndrome, and A(H5N1) emergence are al supported by a combination of high-quality spatiotemporal surveillance data, molecular evidence of emergence source, and outbreak investigations that together are more than convincing that a true disease emergence has occurred (Tables S1–S5 in the supplementary material online). These cases are all good-quality examples of how a disease emergence can be thoroughly characterized and unsurprisingly show an overrepresentation of geographical areas, host taxon groups, and infectious agents (e.g., zoonoses) for which more research resources are available. However, while such detailed characterization is indeed beneficial, the key element lacking for most of the potentially emerging agents listed in Tables S1–S5 for which there is not good evidence of emergence is sufficient evidence for the prior absence of an agent (or a prior difference in disease dynamics) to convince us that a change has occurred. This sends a clear message: while it is too late to improve the status of *a priori* surveillance for agents that may have already emerged, our ability to better establish whether new emerging diseases of wildlife (or new ongoing emergence) are occurring in the future will rely on the establishment of better surveillance operations now to provide the necessary prior evidence of absence or difference. Of primary importance are steps to address the obvious gaps in research and surveillance effort: relatively 'neglected' host taxa such as amphibians and reptiles, relatively neglected geographical regions such as Africa and South America, and relatively neglected agents such as those that are not also human zoonoses or threaten domestic stock. Not only would better *a priori* surveillance provide faster and more effective guidance for responsive disease management should emergence occur [73,74], we would be able to greatly increase our pool of agents with sufficient evidence of emergence. This would allow far more powerful analyses of drivers of emergence than that conducted here, providing clear guidance for or against the involvement of the large set of emergence drivers that are currently only hypothesized. In doing so, this will allow more effective pre-emptive management to be put in place for the prevention of future wildlife disease emergences.

Concluding remarks

Our consideration of wildlife disease emergence (Box 3) is undoubtedly biased; the requirement of 'sufficient evidence' for our assessment of drivers will have a bias toward agents for which emergence is easier to demonstrate; that is, those with obvious disease as opposed to more cryptic impacts, those showing monotonic increases in host or geographical range (or disease incidence or impact) as opposed to more sporadic outbreak dynamics, and those that are localized and specialists as opposed to those that are globally distributed and generalists. These considerations may explain, for example, why the proportion of potentially emerging bacterial agents with sufficient evidence of emergence is relatively low (due to sporadic dynamics and/or a frequent generalist nature) or why the same proportion across all agents in birds and fish is relatively high (due to a high level of surveillance for particular species sets through activities such as recreational fishing, bird watching, and aquaculture). However, while future steps to address such biases may provide the necessary evidence for other drivers, the evidence for two drivers of disease emergence in vertebrate wildlife being predominant (exposure to infectious agents from domestic populations and human-assisted exposure to infectious agents from other wild populations; Box 3) being by far the most important drivers of emergence is unlikely to change, given their consistent representation across host taxa, agent taxa, and geographical locations (Table 1).

Our findings indicate that the greatest gains to be made in the prevention of future wild fish disease emergence is in the improvement of biosecurity systems and management to prevent infectious agent exposure at the domestic/wildlife interface, with such exposure identified as the initiating cause of 74% of the ongoing or new such emergences from 2000 onward. Of secondary importance to wild fish health is management to prevent human-assisted infectious agent exposure from other wild populations [75] due to either source host movement or agent movement (identified as the cause of a further 16%). By contrast, tackling exposure from other wild populations occurring through pathogen pollution [76], pathogen spillover [77], or pathogen spill back [78] dynamics would target the greatest preventive gains to be made for other wild vertebrates, with such exposure (predominantly due to source host movement, but also due to host movement and agent movement) identified as the initiating cause of 66% of the ongoing or new such emergences from 2000 onward. In this context, managing the domestic/wildlife interface would also be a priority (identified as the cause of a further 20%). These conclusions have implications across many spheres of activity, from global trade to the recent interest in assisted wildlife colonization [79]. One only needs look at the disease impacts facing the aquaculture industry and wild fish populations after past decades of relatively unrestricted movement to appreciate the potential consequences if such risks are not sufficiently managed.

Box 3. Summary of key points

- Many reports of emergence of disease-causing infectious agents of vertebrate wildlife from 2000 onward have insufficient supporting evidence to substantiate their status as such. The key element lacking for most of the potentially emerging agents that cannot be confirmed is sufficient data on the prior absence of the agent (or a prior difference in disease dynamics); improving such evidence will allow better analyses of drivers of emergence.
- There is a bias of research effort away from 'neglected' host taxa such as amphibians and reptiles, neglected geographical regions such as Africa and South America, and neglected agents such as those that are not also human zoonoses or threaten domestic stock. Improved surveillance, particularly to address these gaps, would provide faster and more effective guidance for responsive disease management should emergence occur.
- Exposure to infectious agents from domestic sources and human-assisted exposure to infectious agents from other wild populations were the two main drivers of emergence: the domestic source was primary for fish; the wild source was primary for other taxa. Natural exposure to infectious agents from other wild populations has also occurred for fish and other host taxa, with insufficient evidence for major roles of other drivers.
- Our findings indicate that the greatest gains to be made in the prevention of future vertebrate wildlife disease emergence are in the areas of biosecurity systems and management to prevent: (i) infectious agent exposure at the domestic/wildlife interface (of primary importance for fish); and (ii) the human-assisted movement of both wild hosts into non-native regions (be they sources of infection or hosts exposed to infection) and resilient infective stages from wild hosts.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.pt.2015.01.007.

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