Review article

Anthropogenic environmental change and the emergence of infectious diseases in wildlife

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Abstract

By using the criteria that define emerging infectious diseases (EIDs) of humans, we can identify a similar group of EIDs in wildlife. In the current review we highlight an important series of wildlife EIDs: amphibian chytridiomycosis; diseases of marine invertebrates and vertebrates and two recently-emerged viral zoonoses, Nipah virus disease and West Nile virus disease. These exemplify the varied etiology, pathogenesis, zoonotic potential and ecological impact of wildlife EIDs. Strikingly similar underlying factors drive disease emergence in both human and wildlife populations. These are predominantly ecological and almost entirely the product of human environmental change. The implications of wildlife EIDs are twofold: emerging wildlife diseases cause direct and indirect loss of biodiversity and add to the threat of zoonotic disease emergence. Since human environmental changes are largely responsible for their emergence, the threats wildlife EIDs pose to biodiversity and human health represent yet another consequence of anthropogenic influence on ecosystems. We identify key areas where existing expertise in ecology, conservation biology, wildlife biology, veterinary medicine and the impact of environmental change would augment programs to investigate emerging diseases of humans, and we comment on the need for greater medical and microbiological input into the study of wildlife diseases. © 2001 Elsevier Science B.V. All rights reserved.

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1. Introduction

Emerging infectious diseases (EIDs) are defined as diseases that have recently increased in inci-

dence or geographic range, recently moved into new host populations, recently been discovered or are caused by newly-evolved pathogens (Lederberg et al., 1992; Morse, 1993; Daszak et al., 2000a). This broad definition encompasses a range of human diseases that form a significant threat to public health (Krause, 1992; Binder et al., 1999). These include pandemic diseases caus-

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ing high morbidity or mortality (e.g. A.I.D.S.), diseases caused by pathogens that have evolved drug resistance (e.g. tuberculosis, *Staphylococcus aureus* infection, malaria) and pathogens that have caused local outbreaks, but form a particular public health threat due to their high case fatality rates (e.g. Ebola virus disease, hantavirus pulmonary syndrome) or lack of preventive or therapeutic regimen (e.g. Hendra virus disease, Nipah virus disease).

The key event in the emergence of most EIDs is a change in host–parasite ecology (in this review we include infectious prokaryotes and viruses as 'parasites' — see Anderson and May, 1979) resulting from changes in human demography, behaviour or social structure (Garnett and Holmes, 1996; Dobson and Carper, 1996). These essentially ecological changes (Schrag and Wiener, 1995) act within a background of pathogen evolution to allow increased transmission between individual hosts, increased contact with new host populations or species and selection pressure leading to the dominance of pathogen strains adapted to these new environmental conditions. Evolution clearly plays a key role in the emergence of drug-resistant microbe strains, but even here, ecological (including human behavioral) factors have usually fostered their emergence (Krause, 1992; Lederberg et al., 1992).

Wildlife populations have long been considered a link in the chain of pathogen emergence, by forming the reservoirs from which zoonotic pathogens (pathogens transmissible between animals and humans) may emerge. Most of the human EIDs listed by the Institute of Medicine (Lederberg et al., 1992) are zoonotic, either in the sense that they cause disease in both animals and humans (e.g. re-emerging rabies, plague), are caused by pathogens with wildlife reservoir hosts in which they rarely or never cause disease (e.g. Hendra virus disease and many vector-borne diseases such as Lyme Disease), or are caused by pathogens that have recently evolved from wildlife ancestors (e.g. AIDS). Recent analysis demonstrates that although only 49% of human diseases are zoonotic, 73% of human EIDs are caused by zoonotic pathogens (Taylor and Woolhouse, 2000). Despite these public health implications,

important gaps in our understanding of disease emergence remain. In particular, do anthropogenic changes to wildlife habitat (e.g. habitat destuction, fragmentation, encroachment) increase the flow of pathogens from wildlife reservoirs and thereby foster disease emergence in humans? How do these environmental changes affect complex host–parasite relationships for vector-borne diseases? The impact of human environmental change on wildlife populations is clear, with an unprecedented rate of wildlife habitat loss, increasing human encroachment into wildlife territory, significant habitat fragmentation and loss of biodiversity (Pimm et al., 1995). To understand the role of environmental changes in disease emergence requires an integration of diverse branches of biology such as ecology, wildlife biology, conservation biology, invasion biology, wildlife veterinary medicine and microbiology into current studies of human and domestic animal disease emergence.

As a step towards this integration, our current review highlights a group of wildlife diseases that can be considered emerging, using the same criteria that define human EIDs. Some of these wildlife EIDs represent a significant threat to biodiversity by causing or promoting local (population) or global (species) extinctions. The emergence of wildlife diseases may further increase the zoonotic threat to human health by amplifying transmission rates of zoonotic pathogens.

2. Wildlife EIDs

In a previous paper (Daszak et al., 2000a) we identified a number of infectious diseases of terrestrial wildlife that fit the criteria for 'emerging' diseases (Lederberg et al., 1992; Morse, 1993). These are part of a larger cohort of wildlife EIDs that affect terrestrial, freshwater and marine habitats, causing either localized outbreaks (epizootics) or affecting populations on international or global scales (panzootics). Here, we highlight three recently-described, emerging diseases (or groups of diseases) that reflect the range of pathogen types, host habitats and impacts on wildlife and human populations.

2.1. Amphibian chytridiomycosis

Amphibians have undergone population declines on a global scale over the past few decades (Blaustein and Wake, 1990; Drost and Fellers, 1996). Recent analyses suggest that this phenomenon began in the 1960s (Houlahan et al., 2000). However, not all populations are in decline (Pechmann et al., 1991) and, where declines occur, populations of some species remain stable. Amphibian population declines are distinct from the widely-publicized issue of amphibian deformities, which affect only a small number of sites in modified agricultural or urban habitat and are unassociated with mass mortalities and declines (Johnson et al., 1999). After a decade of work, a number of causal hypotheses (habitat loss, increases in ultraviolet-B irradiation, global climate change, chemical pollution, introduced predators, acidification, unidentified environmental 'stressors' leading to immunosuppression) have been proposed, with varying degrees of experimental and observational support, to explain these declines (e.g. Beebee et al., 1990; Blaustein et al., 1994; Gamradt and Kats, 1996; Means et al., 1996; Carey et al., 1999). However, a series of important population declines remained enigmatic (Richards et al., 1993; Lips, 1998). These occurred during the 1980s and 1990s in the tropical forests of Queensland, Australia and Central America, so-called 'pristine' areas that are relatively removed from human impact. It was hypothesized that these declines were caused either by human environmental change acting on a global scale (Blaustein and Wake, 1990; Weygoldt, 1989) or by the introduction of an unknown infectious agent (Laurance et al., 1996).

Veterinary examination of carcasses collected at sites of mass mortality in Australia, and later in Panama, revealed infection by a previously unknown non-hyphal fungus belonging to the phylum Chytridiomycota (Berger et al., 1998). This chytrid fungus infects the keratinized layers of the epithelium in adult amphibians, as well as the keratinized mouthparts of tadpoles and has since been named *Batrachochytrium dendrobatidis* (Longcore et al., 1999). It was shown to be pathogenic in experimental infections of Australian frogs (Berger et al., 1998), and Koch's postulates were later proved using an isolate cultured from a captive frog in a North American zoo (Longcore et al., 1999; Pessier et al., 1999). It remains uncertain how the organism causes death, but the hyperkeratotic reaction of the epidermis to infection may impede supplementary respiration or osmoregulation through the skin (a common feature of amphibian biology) and toxins may be released during infection. The recent discovery of this disease, its absence in archival specimens, its recent and ongoing geographic expansion and its high impact (previous impact at this level could not have been sustained) mark it as an emerging disease. Chytridiomycosis, usually associated with amphibian population declines, has now been reported from Europe, North and South America, Western and Eastern Australia, Costa Rica, Panama and New Zealand (Daszak et al., 1999; Bosch et al., 2000; Waldman et al., 2000). Two hypotheses for the origin of chytridiomycosis emergence are supported by observational evidence. First, global climate change has been proposed as the cause of an increase in number of dry days and length of dry periods at the Monteverde cloud forest site in Costa Rica (Pounds et al., 1999). This drying of the habitat may have caused crowding of amphibians in remnant humid areas, increasing the transmission rate of *B. dendrobatidis*. Second, the wave-like spread of declines in Australia and Central America, the catastrophic rate of population declines associated with chytridiomycosis and the wide host range of this pathogen suggest that the disease has been introduced into these areas from an area of enzootic infection elsewhere (Daszak et al., 1999). The potential for anthropogenic introduction is supported by the finding of *B. dendrobatidis*-infected animals in the international trade of amphibians for pet stores, ornamental pond-stocking, zoos, laboratories, consumption as food by humans and in amphibian species introduced into Australia and North and South America (Daszak, pers. obs.; Pessier et al., 1999; Berger et al., 1998; Mutschmann et al., 2000).

Preliminary ecological analysis suggests that the impact of this disease is heightened by the ability of *B. dendrobatidis* to persist both as a saprobe

and via infection of larval mouthparts. Such persistence would lower the threshold density for the disease and allow it to cause catastrophic declines and local (population) extinctions (Daszak et al., 1999). The heightened impact on certain montane rainforest species may be due to the pathogen's predilection for cool temperatures (Longcore et al., 1999), coupled with the low fecundity and specialized niches that characterize these hosts and which predict for a reduced ability to survive a catastrophic population loss induced by epizootic disease (Williams and Hero, 1998). Chytridiomycosis is an extreme example of a wildlife EID, affecting animals on a panzootic scale, causing mass mortalities, population declines, local and possibly species extinctions and infecting multiple species in a class of vertebrates.

2.2. Diseases of marine invertebrates and vertebrates

Diseases of marine environments have increased markedly in incidence over the past few decades (Epstein, 1998; Harvell et al., 1999). These include a number of coral diseases that have recently emerged, increasing in incidence and severity of impact and for which the 'signatures' of previous epizootics are absent (Harvell et al., 1999; Hayes and Goreau, 1998). For example, in the Caribbean an unknown pathogen almost entirely removed the black-spined sea urchin (*Diadema antillarum*) population between 1982 and 1983 (Lessios, 1988), *Aspergillus sydowii* infections have caused mass die-offs of sea fans (Geiser et al., 1998) and a range of other coral diseases of known and unknown etiology have been recently reported (e.g. Ritchie and Smith, 1998; Williams and Bunkley-Williams, 1990; Porter et al., in press (a); Porter et al., in press (b)). Some analyses suggest a correlation between disease incidence and proximity to the Florida mainland — a region affected by pollution, silt run-off, diversion of water flow and other anthropogenic factors (Boyer et al., 1999). Marine pollutants may stimulate growth of bacteria and invasion into tissues (Mitchell and Chet, 1975) and therefore enhance the impact of a range of coral diseases (Peters, 1984; Peters et al., 1997; Porter et al., 1999).

The marine environment has fostered some notable EIDs of vertebrates, some of which may have emerged as a result of human activity. In 1995, an epizootic of herpesvirus disease caused mass mortality of pilchards over more than 5000 km of the Australian coastline and 500 km of the New Zealand coastline (Whittington et al., 1997). No other species of fish were affected, and the disease spread with and against the current, leading to rafts of dead pilchards up to 3 by 1 km in size floating offshore. Mortality was associated with the presence of replicating herpesvirus in the gill epithelium (Hyatt et al., 1997). The lack of a history of large-scale pilchard mortality in Australia, the focal origin of the outbreak and the rapid spread of disease over large areas suggest a recently introduced pathogen. Whittington et al. (1997) proposed two potential mechanisms of introduction: ballast water discharge by ocean-going ships or the importation of over 10 000 tonnes per annum of non-quarantined frozen mixed by-catch species into Australian waters from North and South America and Japan for use in the bluefin tuna farming industry. In 1998 a similar epizootic occurred, the origin and epizootiology of which resembled that of the 1995 outbreak. To the best of our knowledge, no preventative measures were undertaken subsequent to the 1995 or the 1998 epizootic.

Less direct human activity may explain the emergence of a range of diseases in marine mammals. Newly recognized morbilliviruses have caused a number of epizootics in dolphins, porpoises and seals in recent years, but an outbreak of phocine distemper virus in European harbor seals may have resulted from forced migration of infected harp seals following human depletion of their foodstocks by overfishing elsewhere (Dietz et al., 1989; Heidejorgensen et al., 1992). Canine distemper virus (CDV), a pathogen of domestic dogs that readily undergoes interspecies transmission (Harder and Osterhaus, 1997) was probably introduced to Crabeater seals in Antarctica from expedition sled dogs (Bengston et al., 1991) and has also been recovered from a Siberian seal, in the form of a strain genetically identical to those infecting domestic dogs in the same region (Mamaev et al., 1995). More recently, epizootic CDV

has been reported as causing mass mortalities in the Caspian seal (Kennedy et al., 2000). The ability of pathogens to cross the land–sea barrier is not new, but as the rate of contact between humans or their domesticated animals and marine wildlife increases and the deposition of soil run-off and sewage effluent rises, such events may also be increasing. Recent reports of toxoplasmosis, a disease transmitted by cats, in a spinner dolphin (Migaki et al., 1990) and Beluga whales (Mikaelian et al., 2000); of influenza B virus, a pathogen previously found only in humans, in a harbor seal (Osterhaus et al., 2000), and of soil-borne pathogens, such as *Coccidioides immitis* in California sea lions (Fauquier et al., 1996) and *A. sydowii* in sea fans (Smith et al., 1996) support this hypothesis.

2.3. Newly emergent zoonoses: Nipah virus disease and West Nile virus disease

In 1998, an outbreak of severe encephalitis in pig-farm workers in Peninsular Malaysia resulted in the first human fatalities attributable to Nipah virus disease, a zoonotic EID caused by a newly described member of the family *Paramyxoviridae*, subfamily *Paramyxovirinae* (Chua et al., 2000a). During this and subsequent outbreaks in Malaysia and Singapore, over 265 cases of febrile encephalitis and 106 fatalities were reported (Anon., 1999a; Chua et al., 2000a). The symptoms and epidemiology were distinct from Japanese encephalitis, a pathogenic viral disease endemic to the region (Chua et al., 1999; Goh et al., 2000). In particular, a high proportion of patients had direct contact with pigs, either in pig-rearing facilities or slaughter houses (Chua et al., 1999; Paton et al., 1999). Pigs at these farms exhibited a pronounced respiratory and neurological syndrome, sometimes causing sudden death (Mohd Nor et al., 2000). This disease has been termed 'porcine respiratory and neurological (or encephalitis) syndrome' or 'barking pig syndrome' (Mohd Nor et al., 2000). The movement of Nipah virus through the large (> 2 million) Malaysian pig population may have been augmented by an increased trade in pigs and transmission between farms by domestic dogs and cats (human to human transmission is thought not to occur). Clues to the origins of this virus were gleaned when antibodies to Nipah virus were found in two species of frugivorous bats in Malaysia (Johara et al., 1999). Recently, the virus has been isolated from pteropid fruitbats (Chua et al., 2000b), along with another new virus belonging to the subfamily *Paramyxovirinae* (Tioman virus) (Enserink, 2000). This pattern of aclinical infection in a fruitbat reservoir host — followed by disease in domestic animal 'amplifier' hosts with subsequent transmission to humans ('dead-end' hosts) resembles that of Hendra virus but differs from another zoonotic virus from fruitbats, Australian bat lyssavirus, which is transmitted directly to humans. Hendra virus (Murray et al., 1995) is closely related to Nipah virus (around 78% N gene sequence homology — Chua et al., 2000a), is fatal to horses and humans and can, under experimental conditions, infect cats and guinea pigs (Williamson et al., 1998). It appears that encroachment of human populations into wildlife habitat is the prime cause for the emergence of these diseases, with emergence also depending on the presence of domestic animal 'amplifier' hosts. Changes in agriculture and human behaviour also have an influence on emergence. In the case of Nipah virus, it appears that changes in the distribution of flora associated with intensively managed farms (i.e. introduction of fruiting trees) increased the probability of viral transmission between co-evolved (fruitbats) and naïve (pig) hosts. To date, it is not known if Hendra virus can infect pigs; if this is the case, then Hendra virus may be of higher medical importance than it is currently thought.

West Nile virus disease is a zoonosis which causes disease in both human and other animal hosts. West Nile virus (WNV) is an arthropod-borne flavivirus, related to Japanese encephalitis virus, St. Louis encephalitis virus and others. It is transmitted within its natural (sylvatic) cycle by *Culex* spp. mosquitoes and other arthropods (Hubálek and Halouzka, 1999; Komar, 2000). WNV infects humans and horses (and in one case a domestic cat) incidentally, causing (sometimes fatal) encephalitis in humans (Tsai et al., 1998) and more rarely a syndrome characterized by

paralysis and convulsions in horses (Komar, 2000). WNV disease has been reported from Asia, Oceania and Africa and has recently emerged in Europe and America (Anderson et al., 1999; Lanciotti et al., 1999). Recent outbreaks of human disease in Algeria, Central Europe and Russia are thought to have been related to migration of infected birds. However, the cause of the recent appearance of this virus in the northeastern USA (the first report of WNV from the New World) remains unclear. The epidemic of WNV disease in New York State was preceded by a large epizootic in wild and captive (zoo) birds, particularly of crows (Anderson et al., 1999; Steele et al., 2000). The close phylogenetic relationship of a North American isolate of WNV and an isolate collected from a goose in Israel, suggests that WNV was imported from a European or Mid-East focus via infected birds, human travelers or infected mosquitoes. Regardless of the vector, the introduction of WNV into North America most probably was a direct result of human activity.

3. Underlying causes of emergence

Analysis of the underlying causes of wildlife disease emergence reveals striking parallels with human EIDs (Fig. 1). In most cases these driving factors are simply different presentations of the same anthropogenic environmental change and a product of the globalization of agriculture, com-

Human EIDs	Domestic Animal EIDs	Wildlife EIDs
International travel & commerce	Global introduction of domestic animals	Introduction of domestic & wild animals to new habitats
Demographics: population changes, migration, encroachment into wildlife habitat, cultural changes	Increased animal population density due to intensive farming	Concurrent human & domestic animal population expansion and encroachment. Reduced available habitat.
Changes to agriculture & food processing	Intensive farming practices	Agriculture
Climate: ENSO	Climate: ENSO	Climate: ENSO
Global climate change	Global climate change	Global climate change

Fig. 1. Common underlying causal themes in the emergence of human, domestic animal and wildlife EIDs* reflect shared habitat, shared pathogens, and shared consequences of anthropogenic environmental change. The table lists the most frequently cited causes of emergence for EIDs within these three groups†. The mechanisms driving human EID emergence (left column) are closely paralleled by those driving EID emergence in domestic animals and wildlife (listed correspondingly in the second and third columns). The underlying causes of almost all domestic animal and wildlife EIDs are anthropogenic, and often simply different presentations of the same form of environmental change. These changes are broadly related to the globalization of agriculture and commerce, changes in demography, increasing biological invasions and introductions, and human and domestic animal encroachment into wildlife habitat (Vitousek et al., 1997).

Medical technology (e.g. transfusion)	Veterinary technology (e.g. antibiotic use)	Biological technology (e.g. biocontrol)
Microbial adaptation	Microbial adaptation	Microbial adaptation
Breakdown in public health infrastructure	Breakdown in veterinary health infrastructure	Poor control of international animal and plant traffic
Increased interaction with vectors & contact with wildlife	Increased interaction with vectors & contact with wildlife	Increased interaction with vectors & contact with humans and domestic animals
Immunosuppression (elderly population, HIV infection)	Increased susceptibility (stress, inbreeding, FIV infection)	Increased host density in captive populations leading to heightened susceptibility to disease. Hypothesized immunosuppression of wild populations by unknown environmental "stressors". Infection by immunosuppressive viruses (e.g. FIV, SIV and morbilliviruses).
Increased surveillance	Increased surveillance	Increased surveillance

*Emerging infectious diseases are defined according to Daszak et al. (2000a). Wildlife EIDs are those which cause morbidity or mortality in wildlife.

[†]Information in the current table is taken from an extensive review of the literature for each disease group.

Fig. 1. (*Continued*)

merce and human travel. Two major drivers of wildlife EID emergence can be identified, both of which are the result of human activity and often act jointly to foster emergence (Daszak et al., 2000a). First, a number of high-impact wildlife EIDs have emerged due to 'spill-over' of pathogens from domestic animals into wildlife populations. Because domestic animals often outnumber the wildlife hosts of shared pathogens (e.g. canine distemper and rabies in domestic and African wild dogs), they act as maintenance hosts, enabling the pathogen to avoid the threshold density effect and

drive the smaller population of wild animals to virtual extinction (Cleaveland and Dye, 1995). When wildlife pathogens threaten to 'spill-back' into domestic animals (e.g. brucellosis in bison and domestic cattle), these issues may become politically charged, as conservationists and commercial interests clash (Dobson and Meagher, 1996). The second, and perhaps most common, factor driving the emergence of wildlife EIDs is the anthropogenic movement of pathogens into new geographic locations — a phenomenon we have termed 'pathogen pollution' (Cunningham, 1996; Daszak et al., 2000a), a disease equivalent of anthropogenic biological invasions and introductions (Vitousek et al., 1997).

Pathogen pollution is rooted in the unprecedented globalization of the transport of domestic animals and their products, wild animals (for pets, hunting, food or conservation), contaminated produce and materials. These movements have been linked to a number of wildlife EIDs such as elephant herpesvirus disease in zoos (Richman et al., 1999), West Nile virus disease in the USA, crayfish plague in Europe (Alderman, 1996), red squirrel poxvirus disease in the UK (Sainsbury et al., 2000), avian malaria and poxvirus disease in Hawaii (Van Riper et al., 1986) and varroasis in honeybees globally (Oldroyd, 1999), among many others. Pathogen introductions have a particularly high impact when naive host populations are targeted and introduced pathogens may contribute to the competitive success of the invading carrier hosts by a process known as 'apparent competition' (Hudson and Greenman, 1998).

Pathogens may also be spread or amplified following the introduction of uninfected hosts into new geographic areas, when these hosts allow disease emergence. This has occurred in the case of varroasis on a global scale (following continuous expansion of disease-free European honeybee stocks into enzootic areas of Asia) and bovine tuberculosis in New Zealand (following the introduction of brushtail possums from Australia, which became a new reservoir for the disease in New Zealand) (Oldroyd, 1999; Viggers et al., 1993). This anthropogenic movement of uninfected hosts leading to disease emergence may also be viewed as pathogen pollution in its broadest definition: human-mediated pathogen invasion.

The impact of other anthropogenic environmental changes on wildlife disease emergence is less well-studied. Human landscape changes that remove portions of host populations (e.g. habitat alteration or destruction), alter host migration patterns (e.g. habitat fragmentation) or increase host density are likely to impact disease emergence (Dobson and May, 1986). For example, some direct life-cycle nematodes of herbivores have evolved long-lived, free-living larval stages that enable transmission between migratory groups in the wild. The formation of game reserves and blocking of natural migration routes may lead to increased transmission rates and disease problems from these parasites (Dobson and May, 1986). Provisioning for wild birds at backyard feeders in the UK and USA has led to the emergence of bacterial disease and mycoplasmal conjunctivitis respectively, due to increases in host density, contact rates or the variety of species in close contact at feeding stations (Kirkwood, 1998; Hartup et al., 1998). Protection of the northern elephant seal in the USA in the absence of its former predators has led to a rapid increase in host density and range, and a perceived threat of disease emergence in wildlife and humans (Brownell et al., 2000; Daszak et al., 2000b). Complex interplay between the ecology and population biology of different hosts, vectors and changing environments may have surprising consequences for disease emergence. A shift in agriculture to the midwestern USA early in the 20th century and the resultant reforestation of former arable farmland in the northeastern USA probably fostered Lyme disease emergence (Barbour and Fish, 1993). The complexity of Lyme disease transmission, however, means that cyclic variations in population density of the introduced gypsy moth, *Lymantria dispar*, may profoundly affect Lyme disease risk in humans. The moth larvae feed on oak foliage, therefore cycles of moth density lead to cycles in oak deforestation and mast production. This, in turn, results in population cycles of both white-footed mice and Lyme disease spirochete-infected black-legged ticks which are vectors for the disease (Jones et

al., 1998). Recent work on Lyme disease hypothesizes that regional host biodiversity differences help to explain disease distribution (Ostfield and Keesing, 2000). If this proves true, it has an important further implication: that anthropogenic biodiversity loss *per se* may in some cases drive disease emergence. For Lyme disease, Ostfield and Keesing (2000) suggest that habitat fragmentation on agricultural land causes loss of biodiversity that may increase local risk of Lyme disease. On a global scale, the role of habitat fragmentation, biodiversity loss and other landscape changes in disease emergence are in need of serious assessment.

Direct negative impacts of chemical pollution on wildlife have been documented (e.g. organochlorine toxicity in raptors, lead toxicity in wildfowl). These may lead to changes in species abundance that heighten disease risks (Dobson and May, 1986). It has also been hypothesized that toxic chemical accumulation at subclinical levels may cause immunosuppression that increases host susceptibility to infectious diseases. Causal links, however, often remain unproven. For example, epidemiological data and feeding experiments support a role of PCBs and other immunotoxicants in some phocine distemper virus outbreaks (Ross et al., 2000), while other data refute this rôle (O'Shea, 2000).

4. Impact of wildlife EIDs

Wildlife EIDs are responsible for mass mortalities, local (population) extinctions and global (species) extinctions (Cunningham and Daszak, 1998; Daszak and Cunningham, 1999). This direct loss of biodiversity due to infectious disease may lead to further impacts on ecosystems via 'knock-on' effects. For example, the introduction of rinderpest into Africa in the late 19th century resulted in massive changes in grazing pressure and a perturbation of succession in the savanna flora that persists to date (Dobson and Crawley, 1994). The effective removal of *Diadema antillarum* (a major herbivore) from the Caribbean marine ecosystem produced catastrophic, wide-ranging impacts on its algal food base and its coral habitat (Lessios, 1988). Such knock-on effects may lead to extinction of species further up the food chain that remain uninfected by the pathogen, such as may have occurred for the eelgrass limpet, *Lottia alveus*, following an outbreak of eelgrass wasting disease on the eastern US seaboard (Daszak and Cunningham, 1999).

Our analysis suggests that human environmental modification drives the emergence of human, domestic animal and wildlife EIDs. In particular, the continued expansion of human populations brings us into contact with a large pool of known and unknown zoonotic pathogens (Mahy and Brown, 2000). The economic impact of zoonotic EIDs may be significant. The Nipah virus disease outbreaks in Malaysia resulted in the slaughter of over 1 million pigs, a loss of around 60% of Malaysian pig farms, 36 000 jobs and US$ 120 million in exports. The risk of zoonotic disease emergence may prove costly in a number of other ways. Firstly, public perception of disease risk may increase the cost of treatment, as occurred following the identification of a single rabid kitten in a New Hampshire pet store in 1994, for which at least 665 people received postexposure prophylaxis, at a cost of around $1.1 million (Noah et al., 1996). Secondly, removal of potential disease reservoirs is a costly and complex issue. As urban sprawl expands human populations into prairie dog habitat in Colorado, these reservoirs of plague are culled or relocated at great expense and two recent cases of human blastomycosis are thought to have been acquired during relocation programs (Anon., 1999b). Wildlife EIDs with no direct human involvement may also have significant impacts on human health or well-being. They may threaten populations normally harvested by humans, such as occurred in Australia during the outbreak of pilchard herpesvirus disease, at a cost of A$12 million to the Australian fishing industry. Loss of biodiversity itself has economic ramifications (Costanza et al., 1997) and its conservation is of increasing economic interest and ethical concern. Thus, the threat of EID outbreaks in wildlife populations leads to complicated (and costly) conservation issues, such as pressure to control the expansion of previously threatened California sealions to prevent future disease outbreaks (Brownell et al., 2000; Daszak et al., 2000b).

Wildlife EIDs threaten biodiversity, human health and well-being in a complex, inter-related manner. To understand their impact will require broad integration and an understanding of how changes in human behaviour, agriculture, demography and economy alter pathogen transmission within the human–domestic animal–wildlife continuum (Daszak et al., 2000a). In particular, wildlife EIDs raise awareness of the following gaps in our understanding of disease emergence.

1. Despite a growing body of knowledge (Vitousek et al., 1997) concerning the impact of global anthropogenic environmental change on terrestrial, aquatic and marine habitats, the extent of its influence on wildlife disease ecology is under-researched. For example, little definitive work on the role of sublethal toxin accumulation on disease emergence has been published. The role of human landscape changes such as habitat fragmentation or destruction on disease emergence is also relatively unknown. Recent work on the effects of habitat fragmentation on species distributions, on the impact of invasive or introduced species on ecosystems, and the level of biodiversity loss across ecosystems may prove particularly useful in understanding disease emergence.

2. The translocation of hosts, parasites ('pathogen pollution') or both by humans is a significant driver of disease emergence and should be considered a form of global anthropogenic environmental change in the same way as biological invasions (Vitousek et al., 1996). Significant efforts to prevent disease translocation are urgently required. These should be underpinned by further research to define the scope of pathogen pollution, its cost to biodiversity, to public health and to the economy of nations.

3. There is a dearth of knowledge on the etiology and pathogenesis of wildlife diseases, particularly those of less-charismatic vertebrates and invertebrates, of non-game animals and in marine ecosystems. Surveys of wildlife parasite and pathogen biodiversity may become a significant tool for identifying the agents of wildlife EIDs and predicting their future emergence. Coral EIDs are a particulalry poorly understood group, having been investigated primarily within an ecological context, without significant input from veterinary scientists or microbiologists. Most coral diseases are of unknown etiology and have still not been rigorously classified (Richardson, 1998).

4. We have a detailed understanding of the pathogenesis of zoonotic EIDs impacting human populations, but a relative lack of knowledge of the role environmental changes play in driving their emergence from wildlife reservoirs.

5. Conclusions

We have described a group of wildlife diseases that can be classified as emerging in the same way as human EIDs. These represent a link in the chain of emergence of human and domestic animal diseases, with pathogens, habitats and environmental changes shared between these populations. Parallels between causes of emergence across these groups of diseases demonstrates an important concept: that human environmental change may be the most significant driver of wildlife, domestic animal and human EIDs.

Previously we have argued that the investigation and study of EIDs requires an integrated, multidisciplinary approach (Daszak et al., 2000a). This was highlighted recently by the emergence of WNV disease in the USA. A substantial amount of pathological information pointing towards a WNV diagnosis had been collected by zoo veterinarians working on an outbreak in captive birds, prior to the identification of WNV disease in humans. Future collaboration between wildlife ecologists, conservation biologists, environmental biologists, veterinarians and medical scientists, medical microbiologists and related disciplines should be particularly fruitful, especially for workers who are able to view disease emergence in its broadest terms.

Acknowledgements

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