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International Journal for Parasitology 35 (2005) 1279–1294
www.elsevier.com/locate/ijpara

Invited review

# Navigating parasite webs and parasite flow: Emerging and re-emerging parasitic zoonoses of wildlife origin

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Received 2 April 2005; received in revised form 25 July 2005; accepted 25 July 2005

**Abstract**

Wildlife are now recognised as an important source of emerging human pathogens, including parasites. This paper discusses the linkages between wildlife, people, zoonotic parasites and the ecosystems in which they co-exist, revisits definitions for 'emerging' and 're-emerging', and lists zoonotic parasites that can be acquired from wildlife including, for some, estimates of the associated global human health burdens. The paper also introduces the concepts of 'parasite webs' and 'parasite flow', provides a context for parasites, relative to other infectious agents, as causes of emerging human disease, and discusses drivers of disease emergence and re-emergence, especially changes in biodiversity and climate. *Angiostrongylus cantonensis* in the Caribbean and the southern United States, *Baylisascaris procyonis* in California and Georgia, *Plasmodium knowlesi* in Sarawak, Malaysia, Human African Trypanosomiasis, *Sarcoptes scabiei* in carnivores, and *Cryptosporidium, Giardia* and *Toxoplasma* in marine ecosystems are presented as examples of wildlife-derived zoonotic parasites of particular recent interest. An ecological approach to disease is promoted, as is a need for an increased profile for this approach in undergraduate and graduate education in the health sciences. Synergy among scientists and disciplines is identified as critical for the study of parasites and parasitic disease in wildlife populations. Recent advances in techniques for the investigation of parasite fauna of wildlife are presented and monitoring and surveillance systems for wildlife disease are discussed. Some of the limitations inherent in predictions for the emergence and re-emergence of infection and disease associated with zoonotic parasites of wildlife are identified. The importance of public awareness and public education in the prevention and control of emerging and re-emerging zoonotic infection and disease are emphasised. Finally, some thoughts for the future are presented.


*Keywords:* Wildlife; Zoonoses; Emerging disease; Disease ecology

## 1. Introduction

Historically, appreciation of the ubiquity and significance of links between infectious diseases in human and animal hosts developed slowly, and domestic animals were naturally the initial focus of interest as sources of zoonoses (Hardy, 2003). More recently, however, with ongoing discoveries of new infectious agents and diseases, it has become clear that free-ranging animals and birds are a major source of emerging human pathogens (Daszak et al., 2000).

Among the emerging and re-emerging infectious agents that people can acquire from wildlife, viruses and bacteria are the subject of intensive study because of their frequently severe clinical effects in people and because of the complexities of treatment and control. For parasites, other than major pathogens, including *Trypanosoma, Leishmania* and *Cryptosporidium*, there seems less global concern. This may be because while some of these parasites have high regional prevalence, their effects are less dramatic, with fewer sudden outbreaks involving substantial human mortality. Interestingly, of two recently published overviews of wildlife and emerging zoonoses, one included no parasites (Bengis et al., 2004), and the second mentioned only *Echinococcus multilocularis* (Kruse et al., 2004).

The aims of this paper are: (i) to discuss definitions of 'emerging' and 're-emerging' as they apply to zoonotic parasites derived from wildlife; (ii) to identify parasites for

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doi:10.1016/j.ijpara.2005.07.003

which wildlife have been reported as sources of human infection; (iii) to review new information on interesting wildlife-derived emerging and re-emerging zoonoses not included elsewhere in this issue: and (iv) to review recent literature on fundamental themes for the associations between emerging and re-emerging zoonotic parasites, their human and wildlife hosts, and the ecosystems in which they co-exist.

## 2. Towards definitions for emerging and re-emerging

Persistent among the published definitions of 'emerging infections' is that from the 1992 US Institute of Medicine (IOM) report *Emerging Infectious Diseases: Microbial Threats to Health in the United States*, which states that they are: "those whose incidence in humans has increased within the past two decades or threatens to increase in the near future. Emergence may be due to the spread of a new agent, to the recognition of an infection that has been present in the population but has gone undetected, or to the realisation that an established disease has an infectious origin. Emergence may also be used to describe the reappearance (or 're-emergence') of a known infection after a decline in incidence" (Lederberg et al., 1992). For parasitic zoonoses transmitted from wildlife, these definitions could be amended as follows: an infection or disease is emerging when there is an increased incidence of human infection or disease associated with a parasite of wildlife origin; and (i) a new parasite of wildlife origin causing human infection or disease; or (ii) a new causal association between human infection or disease and a parasite of wildlife origin; or (iii) a new route of transmission to people of a parasite of wildlife origin; or (iv) a new geographic location for human infection or disease associated with a parasite of wildlife origin; and an infection or disease is re-emerging when there is an increased incidence of human infection or disease associated with a parasite of wildlife origin after a period of reduced incidence.

These revised definitions distinguish between infection and disease and apply to both. They also imply pre-existing baseline data on parasite occurrence to which newly observed features of infection and disease in human populations can be compared. For parasitic zoonoses acquired from wildlife, these occurrence data are often incomplete. It may be problematic, therefore, to distinguish between 'genuine' emergence or re-emergence, where there really is a new parasite and/or infection and/or disease and/or epidemiological pattern, and 'apparent' emergence or re-emergence, where the infection and/or disease and/or pattern is old, but is newly recognised as a result of increased detection (Randolph et al., 2001; Kutz et al., 2003).

## 3. Wildlife as sources of zoonotic parasites

For most of the emerging and re-emerging parasitic zoonoses reviewed in this issue, wildlife are known to be a potential source of human infection, and for some are

Table 1
Parasitic zoonoses of wildlife origin acquired by parasite flow through vectors

| Parasite | Global human health burden[a] | Wildlife host(s) | Route(s) of human infection |
|---|---|---|---|
| Nematodes | | | |
| *Brugia malayi* and *Brugia* spp. | 13[b]; 5[c]; 0[d] | Non-human primates and cats (*B. pahangi*) | Infective larvae from mosquitoes |
| *Dirofilaria tenuis* | | Raccoons | Infective larvae from mosquitoes |
| *Dirofilaria ursi* | | Bears | Infective larvae from mosquitoes |
| *Dirofilaria subdermata* | | Porcupines | Infective larvae from mosquitoes |
| *Dipetalonema* spp. | | Unknown | Infective larvae from mosquitoes |
| *Loa loa* | 13[b] | Non-human primates | Infective larvae from mosquitoes |
| *Thelazia* spp. | | Rabbits | Infective larvae from muscid flies |
| Protozoa | | | |
| *Babesia microti* | | Rodents and deer | Sporozoites from ticks |
| *Babesia* spp. | | Ruminants and other mammals | Sporozoites from ticks |
| *Leishmania* spp. | 2[c]; 51,000[d] | Various mammals | Promastigotes from sand flies |
| *Plasmodium knowlesi* | | Non-human primates | Sporozoites from mosquitoes |
| *Plasmodium simium* | | Non-human primates | Sporozoites from mosquitoes |
| *Trypanosoma brucei rhodesiense* | 1.5[c]; 48,000[d] | Antelope and other ruminants, carnivorous mammals | Metacyclic forms from tse-tse flies |
| *Trypanosoma cruzi* | 0.67[c]; 14,000[d] | Ruminants and other mammals | Metacyclic forms from faeces of triatomine bugs |

Tables 1–4 were prepared using data from: Palmer et al. (1998); Acha and Szyfres (2001); and Krauss et al. (2003).

[a] Human infections with many of these parasites can be acquired from other people and/or domestic animals, as well as wildlife; there are no readily available published data indicating the relative significance of each of these three possible sources.

[b] Estimated global totals of human infections (millions) for 1999 (data assembled by Crompton, 1999).

[c] Estimated global Disability Adjusted Life Years (DALYS) (millions) for 2002 (data from WHO, 2004).

[d] Estimated global mortality for 2002 (data from WHO, 2004).

Table 2
Parasitic zoonoses of wildlife origin acquired by parasite flow through food, water and other drinks

| Parasite | Global human health burden[a] | Wildlife host(s) | Route(s) of human infection |
|---|---|---|---|
| Nematodes | | | |
| *Angiostrongylus cantonensis* | | Rodents | Infective larvae in gastropod intermediate hosts, fish, crab or crayfish paratenic hosts, or on vegetables or in water |
| *Angiostrongylus costaricensis* | | Rodents | Infective larvae in gastropod intermediate hosts or on vegetables |
| Anisakids | | Piscivorous reptiles, fishes, birds and mammals | Infective larvae in fish intermediate or paratenic hosts or in cephalopod, scallop, or shrimp paratenic hosts |
| *Dioctophyma renale* | | Carnivorous mammals | Infective larvae in fish or frog paratenic hosts |
| *Dracunculus insignis* | 0.08[b] | Raccoons, mink and other carnivorous mammals | Infective larvae in copepod intermediate hosts in water |
| *Gnathostoma* spp. | | Canids and felids | Infective larvae in copepod, fish, frog or gastropod intermediate hosts, or in avian, rodent, amphibian, or reptile intermediate or paratenic hosts |
| *Lagochilascaris minor* | | Rodents | Infective larvae in rodent intermediate hosts? |
| *Toxocara canis* | | Foxes and other canids | Infective eggs in environment or on vegetables, or infective larvae in mammalian paratenic hosts |
| *Trichinella* spp. | | Carnivorous and omnivorous mammals, birds and reptiles | Infective larvae in animal tissues |
| Cestodes | | | |
| *Diphyllobothrium* spp. | 9[b] | Piscivorous birds and mammals, including marine mammals | Plerocercoids in fish intermediate hosts |
| *Diplogonoporus grandis* | | Whales | Plerocercoids in fish intermediate hosts |
| *Mesocestoides lineatus* | | Birds and carnivorous mammals | Tetrathyridia in reptile and amphibian intermediate hosts ? |
| *Spirometra* spp. | | Carnivorous mammals | Plerocercoids in fish, amphibian or reptile intermediate hosts, or invasion by plerocercoids from intermediate hosts used in poultices |
| Trematodes | | | |
| *Clonorchis sinensis* | 7[b] | Piscivorous mammals | Metacercariae in fish intermediate hosts |
| Echinostomes | 0.15[b] | Mammals and birds | Metacercariae in mollusc, fish or tadpole intermediate hosts |
| *Fasciola hepatica*; *Fascioloa gigantica* | 2.4[b] | Ruminants and equids | Metacercariae on vegetation grown in water and consumed as food, or in water |
| *Gastrodiscoides hominis* | | Non-human primates | Metacercariae on vegetation grown in water and consumed as food |
| *Haplorchis yokogawai* | | Piscivorous mammals and birds | Metacercariae in fish intermediate hosts |
| *Heterophyes* spp. | 0.24[b] | Piscivorous mammals and birds | Metacercariae in fish intermediate hosts |
| *Metagonimus yokogawai* | 0.66[b] | Piscivorous mammals and birds | Metacercariae in fish intermediate hosts |
| *Nanophyetus salmincola* | | Piscivorous mammals and birds | Metacercariae in fish intermediate hosts |
| *Opisthorchis* spp. | 10[b] | Piscivorous mammals | Metacercariae in fish intermediate hosts |
| *Paragonimus* spp. | 20[b] | Crustacean-eating mammals | Metacercariae in crabs and other crustacean intermediate hosts or mammalian paratenic hosts |
| Protozoa | | | |
| *Cryptosporidium* spp. | | Mammals, birds, fish, reptiles, and amphibians | Sporulated oocysts in a variety of sources |
| *Giardia* spp. | | Mammals, birds, and amphibians | Cysts in a variety of sources |
| *Toxoplasma gondii* | | Mammals and birds | Ingestion of sporulated oocysts in water or of bradyzoites or tachyzoites in intermediate hosts |
| Arthropods | | | |
| Pentastomids | | Canids and snakes | Infective eggs on food or in water |

[a] Human infections with many of these parasites can be acquired from other people and/or domestic animals, as well as wildlife; there are no readily available published data indicating the relative significance of each of these three possible sources.
[b] Estimated global totals of human infections (millions) for 1999 (data assembled by Crompton, 1999).

among the more important sources (Tables 1–4). For example: cougars may have contributed to the 1994 water-borne outbreak of human toxoplasmosis in Victoria, BC (Bowie et al., 1997); wildlife are important reservoirs for human leishmaniasis (Jacobson et al., 2003) and for *Trypanosoma cruzi* (Rodriguez Coura et al., 2002; Yabsley and Pittman Noblet, 2002; Miles et al., 2003); *Cryptosporidium* and *Giardia* are widely distributed in wildlife, though there is uncertainty concerning the significance of this source for people (Fayer, 2004; Thompson, 2004); human trichinellosis acquired from wild animals remains a problem (Schellenberg et al., 2003); wild canids are potential sources of human infection with *Echinococcus granulosus*, *E. multilocularis, E. vogeli*

Table 3
Parasitic zoonoses of wildlife origin acquired by parasite flow through the environment

| Parasite | Global human health burden[a] | Wildlife host(s) | Route(s) of human infection |
|---|---|---|---|
| Nematodes | | | |
| *Anatrichosoma cutaneum* | | Primates and opossums | Ingestion of infective eggs in exudate from skin lesions? |
| *Baylisascaris procyonis* | | Raccoons | Ingestion of infective eggs from environment |
| *Capillaria aerophila* | | Canids and felids | Ingestion of infective eggs from environment or infective larvae in earthworm paratenic hosts |
| *Capillaria hepatica* | | Rodents | Ingestion of infective eggs from environment |
| *Gongylonema pulchrum* | | Non-human primates, carnivores and rodents | Ingestion of infective larvae in beetle intermediate hosts |
| *Oesophagostomum* and *Ternidens* spp | | Non-human primates | Ingestion of infective larvae from environment |
| *Strongyloides stercoralis* | | Non-human primates | Ingestion or skin penetration of infective larvae from environment (Ingestion or skin penetration) |
| *Mammomonogamus* spp. | | Ruminants | Ingestion of infective eggs or larvae on vegetables or in water, or infective larvae in invertebrate paratenic hosts |
| *Toxocara canis* | | Foxes and other canids | Ingestion of infective eggs in environment or on vegetables, or infective larvae in mammalian paratenic hosts |
| *Trichostrongylus* spp. | | Ruminants | Ingestion of infective larvae from environment |
| *Trichuris vulpis* | | Canids | Ingestion of infective eggs from environment |
| Cestodes | | | |
| *Bertiella studeri* | | Non-human primates | Ingestion of cysticercoids in free-living mite intermediate hosts |
| *Dipylidium caninum* | | Canids and felids | Ingestion of cysticercoids in flea intermediate hosts |
| *Echinococcus* spp. | 2.7[b] | Canids and felids | Ingestion of infective eggs from environment |
| *Hymenolepis* spp. | 75[b] | Rodents | Ingestion of cysticercoids in beetle, flea and moth intermediate hosts |
| *Inermicapsifer* spp. | | Rodents | Ingestion of cysticercoids in arthropod intermediate hosts |
| *Raillietina* spp. | | Rodents | Ingestion of cysticercoids in arthropod intermediate hosts |
| *Spirometra* spp. | | Carnivorous mammals | Ingestion of plerocercoids in amphibian and reptile intermediate hosts, or invasion by plerocercoids from intermediate hosts used in poultices |
| *Taenia* spp. | | Canids and felids | Ingestion of infective eggs from environment |
| Trematodes | | | |
| *Dicrocoelium dendriticum* | | Ruminants and lagomorphs | Ingestion of metacercariae in ant intermediate hosts |
| *Schistosoma* spp. | 201[b]; 1.7[c]; 15,000[d] | Mammals and birds | Skin penetration by cercaria in water |
| Protozoa | | | |
| *Blastocystis* spp. | | Reptiles, amphibians, arthropods | Ingestion of infective cysts from environment |
| *Cryptosporidium* spp. | | Mammals, birds, fish, amphibians, and reptiles | Ingestion of sporulated oocysts from a variety of sources |
| *Giardia* spp. | | Mammals, birds, and amphibians | Ingestion of cysts from a variety of sources |
| *Toxoplasma gondii* | | Mammals and birds | Ingestion of sporulated oocysts from faeces of felids |
| Arthropods | | | |
| *Auchmeromyia luteola* | | Mammals | Skin invasion by larvae hatched from eggs laid in soil |
| *Chrysomya* spp. | | Mammals | Wound invasion by larvae hatched from eggs laid in wounds or on mucous membranes |
| *Cochliomyia hominivorax* | | Mammals and birds | Wound invasion by larvae hatched from eggs laid in wounds or on mucous membranes |
| *Cordylobia anthropophagia* | | Mammals and birds | Skin invasion by larvae hatched from eggs laid in soil invade skin |
| *Gasterophilus* spp. | | Equids | Skin invasion by larvae hatched from eggs laid on hairs |
| *Hypoderma tarandi* | | Caribou and reindeer | Skin invasion by larvae hatched from eggs laid on hairs |
| *Oestrus* spp. | | Mammals | Invasion of nasal chambers and sinuses by larvae hatched from eggs deposited in nose |
| Pentastomids | | Canids and snakes | Ingestion of infective eggs on food or in water |

[a] Human infections with many of these parasites can be acquired from other people and/or domestic animals, as well as wildlife; there are no readily available published data indicating the relative significance of each of these three possible sources.
[b] Estimated global totals of human infections (millions) for 1999 (data assembled by Crompton, 1999).
[c] Estimated global Disability Adjusted Life Years (DALYS) (millions) for 2002 (data from WHO, 2004).
[d] Estimated global mortality for 2002 (data from WHO, 2004).

Table 4
Parasitic zoonoses of wildlife origin acquired by parasite flow from infested hosts by direct contact or through fomites

| Parasite | Global human health burden[a] | Wildlife host(s) | Route(s) of human infection |
|---|---|---|---|
| Arthropods | | | |
| *Cheyletiella* spp. | | Mammals | Contact with infested mammals or via fomites |
| *Dermanyssus* | | Birds | Contact with infested birds or via fomites |
| *Dermatophagoides* spp. | | Rodents, bats and birds | Contact with infested mammals or birds or via fomites |
| Fleas | | Mammals and birds | Contact with infested mammals or birds |
| *Macronyssus* spp. | | Rodents | Contact with infested mammals or via fomites |
| *Ornithonyssus* | | Birds | Contact with infested birds or via fomites |
| *Sarcoptes scabiei* | | Mammals | Contact with infested mammals or via fomites |
| *Trixacarus caviae* | | Guinea Pigs | Contact with infested mammals or via fomites |

[a] Human infections with many of these parasites can be acquired from other people and/or domestic animals, as well as wildlife; there are no readily available published data indicating the relative significance of each of these three possible sources.

and perhaps *E. shiquicus*, as are wild felids for *Echinococcus oligarthrus* (Eckert and Deplazes, 2004; Xiao et al., 2005); and many of the fish and other aquatic fauna that harbour infective stages of zoonotic parasites can be considered wildlife, even those that are farmed. For many of these parasites wildlife share their role as potential sources of human infection with domestic animals and with other people.

Primarily because of the growth rate of the global human population, the developed world's expanding appetite for resources of all types has led to dissolution of many ecological barriers important in the natural control of disease. More specifically for parasitic zoonoses acquired from wildlife, human intrusions into many wildlife habitats, and the reverse, have resulted in a shift in the interface between wildlife and people from often sporadic and fragile to more permanent and substantial, providing significant opportunities for parasite transmission (Daszak et al., 2001).

## 4. Routes of zoonotic transmission: parasite webs and parasite flow

The presence of parasites in any ecosystem generates complex parasite webs within the system, and it is through these webs that zoonotic parasites move from wildlife to people (Fig. 1). Typically, each species of parasite has its own web, parts of which may be shared with other parasites, for example, multiple tick-transmitted pathogens infecting a host species within a geographic area. Some parasite webs include alternative transmission routes, and which of these is used in a particular situation depends on overall ecosystem structure and function. For example *Trichinella* may be acquired by people in a particular region from more than one wildlife species.

Pivotal to an appreciation of the function of parasite webs is an understanding of parasite flow ('flow of pathogens' Daszak et al., 2001): by which routes, under what conditions and at what levels does the parasite flow among its various hosts, of the same or different species (including vectors), and between the hosts and the environment. Some parasite webs are intricate and uncertain, with fragility of parasite flow; others are more robust, although in some cases only temporarily. For zoonoses of wildlife origin, either type of web can facilitate sufficient parasite flow to bring about human infection and perhaps disease.

Parasite flow from wildlife to people can occur by a variety of routes, including through an arthropod vector (e.g. *Leishmania*), through contaminated food (e.g. *Trichinella* in meat, *Giardia* on fruit and vegetables), or contaminated water (e.g. *Cryptosporidium* and *Toxoplasma*), through the environment (e.g. *Echinococcus* and *Baylisascaris*), or through an infested wildlife host

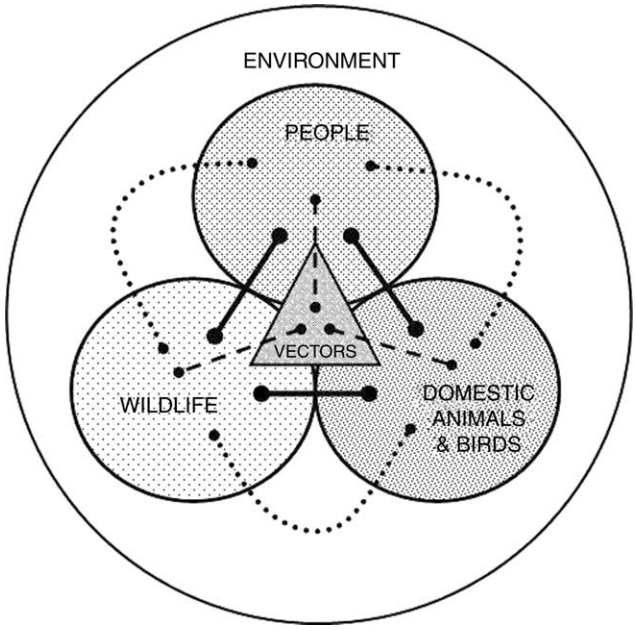


Fig. 1. Representation of a basic parasite web comprising multiple possible routes of parasite flow. Dashed lines - flow through vectors; dotted lines - flow through the environment, including through contaminated food and water; solid lines - flow through direct contact or through consumption of infected tissues. These diagrams can be amended to illustrate webs for specific parasites and to show greater detail.

(e.g. *Sarcoptes*) (Tables 1–4). Parasite flow from wildlife to people through the environment includes two perhaps less obvious routes: through the feces of migratory birds in which *Cryptosporidium* spp. and *Giardia* spp. are found (Kassa et al., 2004); and through the pelage of mammals, from which eggs of *Toxocara canis* and other parasitic nematodes have been recovered (Wolfe and Wright, 2004) and which, epidemiological evidence suggests, may contain infective oocysts of *Toxoplasma gondii* (Frenkel and Parker, 1996).

## 5. A global context

The landmark paper by Daszak et al. (2000) reviewed an array of emerging infectious diseases (EIDs) affecting people and discussed some of the mechanisms underlying emergence and the potential consequences of EIDs for human, domestic animal and wildlife health, and for biodiversity. Of the 18 EIDs identified, only three are parasitic: cryptosporidiosis, sarcoptic mange, and avian malaria. Of these only the first is considered a zoonosis of major importance, but the significance of wildlife relative to other possible sources of human *Cryptosporidium* infection remains uncertain (Fayer, 2004).

A subsequent analysis of 1415 species of human pathogens included 217 viruses and prions, 538 bacteria and rickettsia, 307 fungi, 66 protozoa and 287 helminths (Taylor et al., 2001). Of these, 868 (61%) are known to be zoonotic, including 132 (75%) of the 175 associated with diseases considered to be emerging. This analysis also suggested that zoonotic pathogens are twice as likely to be associated with emerging diseases as are those that are not zoonotic, and that viruses and then protozoa are the more probable causes of emerging disease. Among zoonotic protozoa, those transmitted to people by indirect contact (from food or the environment) included the highest proportion of emerging species (43%), second only to similarly transmitted viruses (46%), followed by protozoa transmitted by vectors (19%) (Taylor et al., 2001).

A similar study of 1922 pathogens (parasites, fungi, bacteria and viruses) determined that 620/1415 (43.8%) of those infecting people also infect wildlife (compared to 39.1% also infecting domestic animals), and 373/1415 (26.4%) infect people, domestic animals and wildlife (Cleaveland and Laurensen, 2001). Among zoonotic pathogens transmitted to people from domestic and/or wild animals, carnivores were identified as the most likely source of human infection (43.0% of pathogens), then ungulates (39.3%), then rodents (22.5%) then non-human primates (12.9%). This analysis also suggested that an emerging pathogen of people or domestic animals is most likely to be a virus, probably an RNA virus, with protozoa the next most likely, ahead of bacteria, helminths and fungi.

In a discussion of the characteristics and population biology of emerging and re-emerging pathogens, *Cryptosporidium parvum* and *Cyclospora cayetanensis* were the only parasites in a list of 32 such agents recognised since 1973, and RNA viruses were again identified as the most likely causes of EIDs (Woolhouse, 2002). Despite this viral predominance, several of the features of a typical EID listed in this paper are applicable to parasites: (i) 'zoonotic, with a reservoir host range that is taxonomically and ecologically broad'; (ii) 'transmitted by vectors, especially by biting flies that are generalist feeders'; and (iii) 'found in areas that are experiencing ecological, demographic or social change' (Woolhouse, 2002).

## 6. Drivers of emergence and re-emergence

In 2003 the IOM updated its 1992 report on emerging infectious diseases, and included 13 'factors in emergence': microbial adaptation and change; human vulnerability; climate and weather; changing ecosystems; economic development and land use; human demographics and behaviour; technology and industry; international travel and commerce; breakdown of public health measures; poverty and social inequality; war and famine; lack of political will; and intent to harm (Lederberg and Morgan, 2003). Subsequently, focussing on land use and infectious disease occurrence, Patz et al. (2004) listed the physical environment, movement of host populations and pathogens, trade, agriculture, and urbanisation as having the greatest potential impact on public health, and identified 34 human infectious diseases with strong links to landscape changes, including eight parasites (malaria, onchocerciasis, trypanosomiasis, filariasis, leishmaniasis, hookworm, schistosomiasis, and cryptosporidiosis), at least five of which can be acquired by people from wildlife. Approximately half of the diseases listed are vector-transmitted, including five of the parasites. Thus land use and the other factors identified by the IOM and by Patz et al. are relevant to the emergence or re-emergence of parasitic zoonoses of wildlife origin.

The IOM factors in emergence are key connectors between changes in ecological and/or societal structure and/or function and disease emergence or re-emergence, but a closer look reveals a second tier of drivers, more directly related to parasite webs and parasite flow from wildlife to people. This second tier includes changes in: (i) abiotic components of the ecosystem, especially climate and hydrology, and in vegetation; (ii) faunal structure of the host component of an ecosystem, for example, ebbs and surges in the various constituent species, including vectors; (iii) host geographic distributions—including those for domestic animals and people—at local to continental scales, including animal translocations, and human intrusions into wildlife habitat, and vice-versa; (iv) faunal structure of the parasite component of an ecosystem, and in parasite prevalence, abundance and virulence; (v) health status of host populations, including people at risk, particularly their level of general disease resistance and their ability to

develop protective immunity, which may vary with age structure, nutritional status and co-existing disease; (vi) the host population's previous exposure to the parasite, which may alter susceptibility to infection relative to naïve hosts, as well as the parasite's pathological effects; (vii) animal and/or human behaviour at a local or regional scale, particularly changes leading to increased contact between wildlife and people; and (viii) specific measures to prevent exposure of people to parasites, as might occur as a result of anthropogenic blunders, detected or undetected. Clearly, the relative significance of each of these drivers will vary with the parasite and with ecosystem structure and function at a particular time.

## 7. Biodiversity

A knowledge of the phylogeny of hosts and parasites, and of the history of the biodiversity of an ecosystem, can provide important clues to understanding the emergence and re-emergence of parasites and other pathogens (Brooks and Hoberg, 2000). Emerging and re-emerging infectious diseases are commonly viewed as threats to biodiversity because of their potentially erosive effects on host populations, with the risk of extirpation or extinction of host species (Daszak et al., 2000). This concern has led to a special interest in pathogens that can infect multiple species within an ecosystem, a characteristic of many wildlife-derived parasitic zoonoses, and particularly on the influence of the pathogens on the population health of the various hosts. For example, Dobson (2004) used a theoretical model for the population dynamics of multi-host pathogens to suggest that: (i) high host diversity within an ecosystem may amplify epidemic infectious disease outbreaks where pathogen transmission is density dependent, and buffer outbreaks where transmission is frequency dependent or by vectors; (ii) disease outbreaks can be controlled when the host species most important for transmission is identified and removed from the transmission web (e.g. by vaccination); (iii) pathogen transmission between host species at a level lower than that within a host species facilitates pathogen persistence; and (iv) if these transmission levels shift towards equality, and one host species is better able to recover from disease outbreaks, other hosts species and the pathogen may become extinct. Dobson (2004) also explored the implications of these results for the modified patterns of disease occurrence resulting from climate change, and suggested that the extension of frequency-dependent or vector transmitted pathogens from tropical areas, where rich host biodiversity may buffer disease outbreaks, to temperate regions, with relatively limited host biodiversity, may lead to outbreak amplification. These effects may result from climate change, or from alterations in the geographic distribution of parasites arising from other causes.

Changes in the geographic distributions of hosts and their parasites may also result in mingling of two or more host species in which the parasite has different effects. This was elegantly explored by Tompkins et al. (2001) using experimental infections with *Heterakis gallinarum*. In ring-necked pheasants growth parameters were unaffected, while grey partridges experienced reductions in weight gain, food consumption and caecal activity. Tompkins et al. (2001) suggested that competition between these two host species may be mediated by *H. gallinarum* and that its adverse effects may lead to extirpation of the 'susceptible' host species. They cautioned, however, that if the parasite has sufficient pathogenicity for pheasant populations, leading to reductions in the force of infection for both hosts, the effects on partridges could be mitigated and the two species could co-exist.

Parasite biodiversity can have other important influences on ecosystem structure and function. For example, individual hosts and mono-specific host populations usually harbour more than one species of parasite, as well as fungi, bacteria, viruses and prions. For wildlife very little is known of the interactions among these various pathogens, nor about the effects of the interactions on host population health. Among human pathogens, the interactions between HIV and several parasites, especially malaria and leishmaniasis, have been extensively studied (Desjeux and Alvar, 2002; Ter Kuile et al., 2004).

## 8. Climate change

The scenarios proposed for global climate change, including extreme weather events, strongly suggest impacts on infectious disease, including those associated with zoonotic parasites, although the nature and magnitude of these effects will depend on the nature of the change, the parasites involved, and the region (Fayer, 2000; Patz et al., 2000; Dobson et al., 2003; Sutherst, 2001; 2004). Climate change may alter hosts—including vectors, as well as parasites, vegetation, abiotic elements of the ecosystem, and many of the associations among these that affect the parasite web and parasite flow. Identification of all components of an ecosystem, or even of simply the parasites and hosts, potentially susceptible to climate change is difficult, but perhaps easier than predicting the impacts of change. For zoonotic parasites of wildlife, altered development and mortality rates of life-cycle stages affected by environmental conditions, and changes in vector ecology, including contact rates with human and animal hosts, are among the more obvious possible impacts. For wildlife and people, among the possible consequences of climate changes are altered host biodiversity, population structure and immunocompetence, and contact patterns between wildlife and people. But care is required in attempting to establish causal relationships between climate change and altered disease occurrence, as demonstrated for malaria in the East African highlands (Hay et al., 2002).

## 9. Some recent examples of emergence and re-emergence

This issue of the International Journal for Parasitology contains detailed updates for most of the major emerging zoonotic parasites transmissible from wildlife. What follows is an overview of recent reports of other such parasites that illustrate key features of emergence or re-emergence.

### 9.1. Angiostrongylus cantonensis

This is a genuine emergence resulting from increased incidence or apparent emergence resulting from increased detection.

This metastrongylid nematode is the most common cause of human eosinophilic meningitis, primarily in Southeast Asia and the Pacific Basin, but increasingly in other areas of the world, especially the Caribbean (Prociv et al., 2000). In 1996, a single probable case was reported in a Jamaican who had never left the island (Barrow et al., 1996). In 2000, 12 of 23 American tourists who had visited Jamaica for a week developed clinical signs and specific antibody consistent with eosinophilic meningitis 6–30 days after returning home (Slom et al., 2002). Subsequent surveys in various parts of the island showed 24/109 (22%) rats (*Rattus norvegicus* and *Rattus rattus*) had pulmonary infections with adult *A. cantonensis*, and 4/48 (8%) snails (*Thelidomus asper*, *Orthalicus jamaicensis* and *Dentellaria sloaneana*) contained larvae of the parasite (Lindo et al., 2002).

Risk of human infection with *A. cantonensis* follows ingestion of infective larvae in raw or poorly cooked gastropod intermediate hosts, or transport hosts such as freshwater prawns, frogs, fish or planarians, sometimes contaminating other foods, especially salads (Cross, 1998). In the outbreak in American tourists in Jamaica, disease was strongly associated with the consumption of a Caesar salad—made with lettuce imported from the United States—at a restaurant on the night before leaving the island (Slom et al., 2002). An intriguing question concerning the transmission of *A. cantonensis* to people is the possible role of infective larvae that have emerged from the gastropods, a phenomenon described for *Angiostrongylus costaricensis* (Ubelaker et al., 1980) and for other parasitic nematodes that utilise gastropod intermediate hosts (Kutz et al., 2000).

The extent and abundance of *A. cantonensis* in its various hosts in the Caribbean region and the southern United States, and in other areas outside Southeast Asia and the Pacific Basin, are not fully known, nor is its significance as a human pathogen in these areas of the world. A single human case has been reported from New Orleans (New et al., 1995), and more recently the parasite has been recovered from a lemur, a wood rat and four opossums in Louisiana (Kim et al., 2002). It is interesting that *A. cantonensis* has been added to the list of parasitic infections in the new edition of the primer on foodborne illnesses, published for physicians and health care professionals by the United States Centers for Disease Control and Prevention (Centers for Disease Control and Prevention, 2004).

### 9.2. Baylisascaris procyonis

This is a genuine emergence resulting from increased incidence or apparent emergence resulting from increased detection.

This ascarid nematode of raccoons is well recognised as a rare cause of serious, usually neurological, human disease in several areas of the United States, and one case has been reported from Germany (Sorvillo et al., 2002). *Baylisascaris procyonis* has also been associated with neurological disease in many domestic and wild animals and birds (Kazacos and Boyce, 1989), and may be a factor in the population health and survival of animals that share habitat with raccoons (LoGiudice, 2003).

Two recent studies in the United States have altered a paradigm for the geographic distribution of *B. procyonis*, and highlighted the parasite's potential infection pressure for people living in areas frequented by raccoons. First, adult parasites were found in 11/50 (22%) raccoons in a necropsy survey in the Atlanta area, searching not for *B. procyonis*, but for *Dracunculus insignis* (Eberhard et al., 2003). This report is the first documented occurrence of *B. procyonis* in the southeastern United States, where the parasite was thought either to be very rare, or absent. In a second study in California, where there have been at least four cases of clinically significant human *B. procyonis* infection, feces were collected from 215 raccoon latrines on 164 residential properties in three communities in the northern part of the state (Roussere et al., 2003). These latrines are specific areas used by raccoons for defecation, and are recognised as important potential sources of human infection with *B. procyonis* (Page et al., 1999). Eggs of the parasite were found in feces from 44 to 53% of latrines, and in 16 to 32% of latrines eggs were fully larvated and assumed to be infective. In two of the communities, infective eggs were found in approximately 50% of latrines, and in the third in approximately 25%. Latrine densities on the properties in the three communities were 3.5, 6.2 and 8.8 per acre. Between 30 and 750 g of feces were recovered from individual latrines. The high levels of environmental contamination with *B. procyonis* described for these residential neighbourhoods in California is one of several factors that increase the potential for the parasite to become a more significant emerging pathogen. The geographic distribution of raccoons is expanding, particularly in northeastern North America, and their numbers in urban and suburban areas are increasing (Riley et al., 1998). For the parasite, the geographic distribution is incompletely known, prevalence, intensity and fecundity (45 million eggs daily) in raccoons are often high, and eggs can survive in the environment for long periods (Kazacos, 2001). These features mean that human infection with

*B. procyonis* may be more common than is clinical disease and disease in people may be under-diagnosed (Sorvillo et al., 2002).

### 9.3. Plasmodium knowlesi

This is a genuine emergence resulting from a parasite in a 'new' host.

Species of *Plasmodium* associated with human malaria can infect non-human primates, and *Plasmodium* species from these hosts have been recovered from people, but both types of host switching were thought to be very rare (White, 2004). Recently, however, *P. knowlesi*, for which macaques are believed to be the primary hosts, was found by PCR in 120/208 (58%) of human malaria cases in the Kapit division of Sarawak, Malaysia, between March 2000 and November 2002 (Singh et al., 2004). *Plasmodium knowlesi* was the only malaria species found in 106 of these 120 patients.

This is the first published report of a large number of cases of human malaria associated with a species of *Plasmodium* from non-human primates. Several features of the report are interesting from the perspective of the detection of a newly emerging disease. First, for all 120 patients, PCR-based identification of the parasite was inconsistent with that based on morphology in stained blood films; most of the *P. knowlesi* cases had been diagnosed morphologically as *Plasmodium malariae* (which was not detected in any of the patients by PCR), the others as *Plasmodium falciparum* or *Plasmodium vivax*. Second, careful investigations following the initial PCR diagnosis of *P. knowlesi* demonstrated significant clinical and epidemiological differences between this and the other species of *Plasmodium* infecting people in the area. Among these, the absence of clustering of cases in association with the long houses in which the people live suggested that the source of these infections might have been macaques rather than people. Third, several experimental, clinical and deductive observations, dating back to 1932, suggested the possibility of human infections with *P. knowlesi*, particularly in Malaysia (Singh et al., 2004). Among these, two isolated human cases of *P. knowlesi* were initially diagnosed morphologically as *P. falciparum* and *P. malariae*, the first then as *P. malariae* (Chin et al., 1965). Since the report from Sarawak, a single human case of P. knowlesi, confirmed by PCR, has been described from Thailand (Jungwutiwes et al., 2004).

A sequence of events similar to that leading to the discovery of *P. knowlesi* as an emerging cause of human malaria will probably occur in the future, particularly with the accelerating development of new diagnostic technology. In terms of emerging disease, what is particularly relevant about this discovery is the historical record of this parasite's potential for human infection, and the distinctive clinical and epidemiological characteristics of the human *P. knowlesi* infections.

### 9.4. Trypanosoma brucei rhodesiense and Trypanosoma brucei gambiense

In Africans, these infections are a genuine re-emergence resulting from reappearance after a decline in incidence; in tourists in Africa, they are a genuine emergence resulting from increased incidence or apparent emergence resulting from increased detection.

Human African Trypanosomisasis (HAT) caused by *Trypanosoma brucei rhodesiense* or, more commonly, *Trypanosoma brucei gambiense* is re-emerging in sub-Saharan Africa (Stich et al., 2003). *T. b. gambiense* transmits within the human population, apparently with only occasional animal infections, whereas cattle and, to a lesser extent, other domestic species and wildlife can be important reservoirs of *T. b. rhodesiense* for people (Welburn and Odiit, 2002). Reasons for this resurgence of HAT are complex, and include weakening of control programs put in place during the last century, the emergence of HIV/AIDS and other major health problems—and the resulting re-allocation of limited resources, the complexities of treatment and clinical management of HAT, and societal disruptions generated by conflict (Smith et al., 1998).

The re-emergence of these trypanosomes is having significant impacts on the health and well being of the peoples of Africa. For example, the situation is worsening in Uganda for both sub-species (Ochan, 2004), and in Sudan, following near elimination in the 1960s, *T. b. rhodesiense* has become a crisis (World Health Organisation, 2004a,b). While there is a clear role for domestic animals and wildlife as reservoirs for *T. b. rhodesiense*, it has been suggested that animals—primarily pigs—may maintain infection with *T. b. gambiense* in areas where it has been almost eliminated from the human population, and become a potentially significant source of human infection (Welburn and Odiit, 2002).

Occasional cases of HAT occur outside Africa. For *T. b. gambiense*, most are in Africans who have emigrated from endemic areas and who return for some reason. For *T. b. rhodesiense*, patients are most commonly tourists who are thought to acquire the parasite while visiting game parks in east Africa (Chretien and Smoak, 2005). For example, a 2002 report described nine cases, one of which was fatal, in European and South African tourists, all of whom who had recently visited parks in Tanzania (Ripamonti et al., 2002; Jelinek et al., 2002). It is interesting that the links between these cases were found through TropNetEurop, a clinic-based international sentinel surveillance network (http://www.tropnet.net/) (Jelinek et al., 2002).

### 9.5. Sarcoptes scabiei

This is a genuine emergence resulting from increased incidence or apparent emergence resulting from increased detection.

*Sarcoptes scabiei* has been recovered from approximately 100 species of free-ranging mammals, and human infections acquired from wildlife—usually as a result of direct contact—have been reported from several parts of the world (Bornstein et al., 2001). While it probably unlikely that scabies from wildlife sources will become a major human health problem, the natural history of *S. scabiei* in free-ranging mammals, particularly the potentially devastating epizootics (Bornstein et al., 2001), suggest a risk for people in contact with infected animals. Of special concern are situations such as that for wild canids in Scandinavia where, in Sweden, *Sarcoptes* is thought to have halved the red fox population in the decade following its initial introduction in the mid 1970s. Scabies is also a potentially significant threat to the health, and sometimes existence, of endangered or isolated wildlife populations (Pence and Ueckermann, 2002). There have been attempts recently to use molecular techniques to differentiate between isolates of *S. scabiei* from different hosts and geographic regions (Skerratt et al., 2002; Berrilli et al., 2002), but some of the conclusions from this work are controversial (Morrison et al., 2003).

### *9.6.* Protozoan zoonoses in marine ecosystems: *Cryptosporidium, Giardia,* and *Toxoplasma*

There are genuine emergences resulting from increased incidence or apparent emergence resulting from increased detection.

A 2004 special issue of *Veterinary Parasitology* highlighted the increasing interest in water as a source of parasitic zoonoses (Gajadhar and Allen, 2004). Of particular recent concern is the presence of zoonotic protozoa in marine ecosystems in various parts of the world, especially *Cryptosporidium* and *Giardia* in shellfish, and *Giardia* and *Toxoplasma* in a variety of marine mammals (Fayer et al., 2004). The relative roles of the marine ecosystem itself and of freshwater run-off from land in the maintenance of these parasites in marine hosts are not fully understood. Notwithstanding uncertainties concerning genotypic identities, *Cryptosporidium* and *Giardia* are clearly a potential risk to people consuming shellfish or using the sea for recreation, and foodborne *Toxoplasma* is a concern for people dependent on marine mammals for food (McDonald et al., 1990).

## 10. Disease ecology: an emerging or re-emerging science

The complexities of the relationships among infectious agents, including parasites, their human and animal hosts and the environment have renewed movement towards an all-encompassing approach to the study of infectious disease. Such an approach is sometimes referred to as disease ecology (Real, 1996), although a clear definition of this term remains elusive and it is sometimes used in a sense indistinguishable from disease epidemiology. Disease ecology is more, and implies consideration of all possible contributors to the behaviour of disease within an ecosystem. To some it also implies the application of distinctly ecological techniques to disease investigation, which brings significant insights and benefits different from those associated with the more traditional clinical and epidemiological approaches (Smith et al., 2005). This ecological perspective is not new, for 30 years ago John Whitlock, the Cornell parasitologist, assembled evidence to remind us of the well-established understanding that in the past at least partial control of many preventable illnesses in human or animal populations had been achieved by making changes to the environment, in the absence of specific measures against the causative agents, few of which had been identified (Whitlock, 1974).

Among the pivotal elements of the study of disease ecology is an understanding the innate structure and function of the ecological barriers that limit disease, their flexibilities, and the mechanisms and consequences of their strengthening, weakening, or destruction. Disease ecology thus demands the incorporation of many areas of expertise, and there is a strong basis for optimism, among researchers at least, that in recent years this multidisciplinary approach is being more widely adopted (Patz et al., 2004; Daszak et al., 2004).

High priorities now should be the more widespread inclusion of disease ecology as a distinct theme, alongside microbiology, epidemiology and public health, in health sciences curricula with, wherever possible, combined learning experiences for medical, veterinary and other students. Also, where appropriate, the education of graduate students should ensure an understanding of the use of ecological techniques in the study of 'disease', or at least of the benefits of an ecological approach. Several colleges of veterinary medicine and medicine are already moving in this direction for undergraduates through opportunities in 'ecosystem health' (Lannigan, 2004), including the veterinary colleges in Canada (Waltner-Toews et al., 2004). Moving these initiatives from the periphery of the curriculum and introducing them more widely will present several attitudinal and practical challenges (Howard, 2004), and may require paradigms to be discarded, perhaps along with some traditional course material. These changes should, however, greatly strengthen society's future ability to deal effectively with a wide array of diseases of people, domestic animals and wildlife, especially those caused by infectious agents.

## 11. Synergy for access and analysis

As already outlined, essential for success in an ecological approach to disease is synergy among key players, bringing together different perspectives and areas of expertise.

This approach to the study of parasitic zoonoses in wildlife should include three special considerations. First, accessing wildlife for the collection of data and/or samples is often complex and expensive, particularly in remote areas. Other than in disease-crisis situations, biologists and others interested in the overall status of populations probably have greater contact with wildlife than do veterinarians and others for whom health and disease are the primary interests. In exercises such as population surveillance, therefore, there are many opportunities for health and disease-focussed researchers to work synergistically with biologists and others to collect information and specimens. Second, where wildlife species are essential for the physical and cultural well-being of local people, for example, through subsistence hunting, harvesters and others within the local communities are potential sources of traditional knowledge and other uniquely useful information, especially where specific training is offered to these individuals on the recognition of health and disease in wildlife (see http://wildlife2.usask.ca/web_devel/web_dev/Sahtu/index.php). Lastly, when wildlife populations are the subject of disease-focussed research, veterinarians, population biologists, epidemiologists, and other researchers with complementary expertise can learn much from each other, especially in the planning stages of the investigations.

## 12. Defining the parasite fauna of wildlife

Alongside the frequent difficulties in accessing wildlife, especially those that are remote or secretive, is the paucity of information regarding the host and geographic distributions of their zoonotic parasites, and the lack of validation of some of the diagnostic tests used for these infections in wildlife, particularly those based on serology. Fortunately, newer laboratory techniques show considerable promise in advancing monitoring and surveillance capabilities. For example: a coproantigen test has recently been employed to explore the epidemiology of *E. multilocularis* in foxes in eastern France (Pleydell et al., 2004); examination of serosanguinous fluid from the chest cavities of 250 dead lynx for *Toxoplasma* antibodies has been used to investigate the parasite's epidemiology (Zarnke et al., 2001); DNA sequencing of larvae from fecal samples has been used to redefine the geographic distribution of *Parelaphostrongylus odocoilei* in thinhorn sheep in western North America (Jenkins et al., 2005); and in people in Sarawak, a PCR was the basis of the discovery of human infections with *P. knowlesi* (Singh et al., 2004). Molecular techniques have also proved very useful in separating morphologically indistinguishable species and genotypes for a range of parasites, particularly in elucidating host specificities for *Cryptosporidium*, *Giardia* and other zoonotic protozoans capable of infecting multiple host species. Techniques based on nucleic acid analysis, for example broad-range PCR, representational difference analysis and expression library screening, can also be used to scan for unknown or unsuspected organisms in a variety of materials (Relman, 1999). For some zoonotic parasites these newer techniques make possible a specific diagnosis in live rather than dead animals, a significant advantage when working with wildlife.

## 13. Expect the unexpected, or the expected?

The discovery of *E. multilocularis* in Norway (Henttonen et al., 2001) is a good example of the surprises inherent in the study of zoonotic infectious disease in wildlife. In an analysis of the unexpected nature of emerging diseases in general, Stephen et al. (2004) applied descriptors for emerging phenomena in business: they are often unpredictable and counterintuitive; seemingly minor changes in the action of one component of the system can have profound effects on the whole system; and there is not necessarily a logical link between the actions of a single component and the outcome at the group or system level (Bonabeau, 2002). These descriptors imply a high probability of surprise, and add a useful dimension to thinking about emerging and re-emerging zoonotic parasites in wildlife. They also underline the importance of the all-encompassing perspective on disease.

Application of the descriptors from business to emerging disease raises some interesting questions related to predictions of pathogen and disease occurrence in human populations. The recent discovery of a series of patients with *P. knowlesi* provides an appropriate model. As described above, probable human infections with this simian parasite had been described in the past, albeit rarely, and the recent report from Sarawak indicates that the clinical and epidemiological features of this infection in people are different from those of other species of *Plasmodium* more commonly associated with human malaria. Thus while the exact sequence of events leading to this host switching in Sarawak has not yet been determined, were zoonotic parasite flow by *P. knowlesi* and the emergence of human disease in any way predictable, even in the absence of access to 'developed' health care? It seems almost certain that there are other seemingly uncommon zoonotic parasites and other pathogens for which known or suspected human infections represent only 'the tip of the iceberg'.

## 14. Monitoring and surveillance

Assessing the status of infectious agents, including parasites, in wildlife populations is a foundation of disease monitoring and, if subsequent intervention is planned, surveillance (Salman, 2003). Such assessments may be passive or active and retrospective or prospective. They require one or more points of assembly for wildlife disease

data, on appropriate geographic scales, and ideally they should facilitate proactive detection and management of emerging and re-emerging infections and diseases in wildlife, especially those of major concern for human or animal health.

In North America and in Europe there are several formally organised, regional or national monitoring/surveillance systems (MOSS) for wildlife disease (Morner et al., 2002), for example, the Canadian Cooperative Wildlife Health Centre (CCWHC) database (Leighton et al., 1997) that now incorporates more than 100,000 disease occurrence records from diagnostic laboratories across the country. Complementing this is the Canada Database of Animal Parasites (CDAP), comprising national occurrence records for selected parasites of domestic animals, wildlife and people, including several zoonotic infections, assembled from publications, diagnostic laboratory reports and other sources (Polley et al., 2000). Another interesting approach to parasitic disease surveillance of potential use for wildlife is that of Trotz-Williams and Trees (2003), who used published reports to map both the geographic distribution and the annual incidence (presented in the paper as 'force of infection') for *Leishmania infantum*, *Dirofilaria immitis*, *Dirofilaria repens*, *Babesia canis* and *Ehrlichia canis* in dogs and cats across Europe. Other than *B. canis*, these parasites can flow by vector transmission from wildlife to people. Also useful as sources of information on wildlife disease are archived collections of parasites, tissues and other specimens that provide especially an historical context for current events related to disease emergence and re-emergence (Brooks and Hoberg, 2000; Hoberg, 2002).

Information assembled through MOSSs for wildlife has been helpful in the investigation of major health problems, and this is essential for the systems to be worthwhile. A MOSS can also be very useful for generating baselines for an emerging or re-emerging infectious disease, to which recent changes in geographic and host distributions, disease incidence and other characteristics can be compared. Additionally, retrospective analyses of information from the systems may reveal previously undetected features of host-parasite associations. It should be noted, however, that other than a few diseases of urgent concern, most wildlife MOSSs assemble information passively and retrospectively, and deal primarily with the dead animals. This approach may introduce bias into health assessments, particularly at the population level.

The over-riding determinants of the value of a MOSS for wildlife disease are: (i) completeness and validity of the data; (ii) database design—which should be standardised ideally at least at the national level, and which must facilitate effective and efficient entry and retrieval of information: and (iii) cost-effectiveness—which for parasitic zoonoses depends partly on its usefulness for predicting, detecting and managing significant disease problems in wildlife and in people.

## 15. Predictions of emergence and re-emergence

Among zoonoses transmitted from wildlife, the discovery rate of new parasites is currently low relative to new bacteria and viruses. Thus we have greater familiarity with the organisms. Despite this it is sometimes difficult to identify, in advance or with hindsight, the events leading to emergence or re-emergence of a wildlife-derived zoonotic parasite in a particular ecosystem. In some instances, an increase in the incidence of human infection or disease associated with a wildlife-derived parasite may occur almost relentlessly over a wide geographic area, and in other cases may affect only a few individuals or a single community. For most of the emerging parasites discussed in this issue we know, therefore, that host switching from wildlife, leading to human infection and perhaps disease can occur, but we cannot predict when or how it will occur. For re-emerging parasites we know that human infection can occur, and effective predictions that it will occur may be easier when some of the factors leading to a resurgence of disease can be identified in advance, for example, the deliberate or inadvertent suspension of demonstrably effective control measures.

Predictions of disease emergence or re-emergence usually depend on some form of risk assessment, of which there are two major components: risk of infection and severity of impact. For some emerging and especially re-emerging zoonotic parasites of wildlife we may be able to estimate severity of impact on human health, at least for individuals. The complex structure of many parasite webs and our often-limited knowledge of the factors affecting parasite flow leading to human infection make the assessment of risk of infection difficult. Another issue complicating risk assessment for some zoonotic parasites of wildlife is the absence of simple methods to differentiate currently infected, historically infected, infectious, and resistant wildlife hosts, and to determine the significance of each category for parasite flow.

Predictions of parasite flow from wildlife to people depend in part on identification of the most likely, but not necessarily all possible, sources of infection. For some parasites this is relatively straightforward, for example, raccoon latrines in areas of high prevalence and abundance of *B. procyonis*. More difficult to deal with are parasites with relatively low prevalence in their wildlife host, their vectors, or the environment. For example, while *Trichinella* or *Toxoplasma* may be known to occur in a host species in a particular region, it may be very difficult to accurately predict the risk of human infection associated with the consumption of meat from these hosts. For these two parasites, the occasional published reports of human disease, especially in hunters and others who utilise game meats and other wildlife for food, lead to questions about the true incidence of these infections, and of the diseases they cause, in at-risk human populations (Ross et al., 2001). For *Trichinella* and *Toxoplasma*, and other parasites acquired

from food of wildlife origin, the ability to accurately predict risk of infection is also influenced by cultural traditions for food preparation that may affect parasite viability (Forbes et al., 2003).

The absence of adequately detailed data on key aspects of the ecology of many zoonotic parasites in wildlife usually means that only general predictions of the risk of human infection are possible. This and other problems with risk assessment related to zoonotic transmission are a major constraint for the design and implementation of effective prevention and control measures for these parasites.

## 16. Public awareness

Public education can be very helpful for the prevention and control of human infections with many of the parasitic zoonoses acquired from wildlife. Usually efforts are focussed on making people aware of possible sources of the parasites and on measures to lessen or remove the risk of human infection. This type of awareness enhancement is more easily achieved in the developed world, but there are significant opportunities elsewhere. A pioneering example is the testing program for *Trichinella* in harvested walruses recently initiated in the Canadian Arctic following re-emergence of human trichinellosis (Proulx et al., 2002). Education and prevention programs of this type for people who depend on wildlife for food are greatly enhanced when the information presented is readily understandable by the target audience, is consistent with traditional knowledge, and where the benefits are clearly evident.

## 17. Looking forward

While at present some emerging and re-emerging wildlife-derived zoonotic parasites and diseases are perhaps behind the forefront of societal concern, planning for the effective management of these diseases will result in benefits for global ecosystem integrity and for human health and well being. Identifying priorities for society's scientific resources at this complex intersection of people, domestic animals, wildlife and pathogens is difficult, but the following certainly merit attention: (i) increase society's awareness of the links between infectious diseases in wildlife (and domestic animals) and people, at local to global scales; (ii) continue to enhance the ecological approach to health and disease, and especially the education of those who work or will work in human or animal health, very broadly defined; (iii) encourage pro-active exploration and assessment of potentially major influences on parasite flow from wildlife to people, especially climate change, human intrusion into wildlife habitats—and vice-versa, and other potential causes of ecosystem disruption; (iv) ensure the development and retention of maximally effective management for emerging and re-emerging infections and diseases in all types of hosts; (v) further develop and maintain the capability for effective monitoring and surveillance of infection and disease in wildlife; (vi) further develop and maintain the capability for effective multi-method detection and characterisation of previously known and unknown parasites; (vii) maximise synergy among scientists who study different aspects of wildlife; (viii) explore new approaches to risk assessment for parasite flow from wildlife to people, including increased pro-active assessment where there is new and significant intrusion by people into wildlife habitat, or vice-versa; (ix) explore new approaches to public education where possible, and especially those related to prevention of zoonotic transmission; and (x) ensure preparedness for the biosecurity implications of these parasites.

Successful response to these priorities is easier, but not easy, in more developed areas of the world. Globally, many emerging and re-emerging zoonotic parasites acquired from wildlife are considered of relatively low significance for human health compared to malnutrition, malaria, HIV/AIDS and other infections, and overall childhood mortality. For the individuals and communities and areas affected, however, they are sometimes very important, and in many instances effective campaigns for recognition, diagnosis, prevention, control and even prediction can be designed and implemented on these smaller scales.

As Picasso's friend, the American writer Gertrude Stein, lay dying in France she asked her desolated companion, Alice B. Toklas, "What is the answer?" After a brief silence came Gertrude's own response "In that case, what is the question?" Is this perhaps an effective coda for our efforts to survive emerging and re-emerging infectious disease?

## Acknowledgements

The development of this review benefited greatly from conversations with Dr Susan Kutz and Dr Emily Jenkins, Research Group for Arctic Parasitology, Dr Alvin Gajadhar, Head, Centre for Animal Parasitology, Canadian Food Inspection Agency, and especially with Dr Eric Hoberg, Chief Curator, US National Parasite Collection, United States Department of Agriculture.

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