PHILOSOPHICAL TRANSACTIONS B

rstb.royalsocietypublishing.org

Review

Cite this article: Cunningham AA, Daszak P, Wood JLN. 2017 One Health, emerging infectious diseases and wildlife: two decades of progress? *Phil. Trans. R. Soc. B* **372**: 20160167.
http://dx.doi.org/10.1098/rstb.2016.0167

Accepted: 3 April 2017

One contribution of 12 to a theme issue 'One Health for a changing world: zoonoses, ecosystems and human well-being'.

Subject Areas:
ecology, environmental science, health and disease and epidemiology

Keywords:
One Health, ecosystem services, emerging infectious disease, zoonoses, wildlife disease, policy

Author for correspondence:
Andrew A. Cunningham
e-mail: a.cunningham@ioz.ac.uk

†These authors contributed equally to the study.

THE ROYAL SOCIETY PUBLISHING

One Health, emerging infectious diseases and wildlife: two decades of progress?

Andrew A. Cunningham[1,†], Peter Daszak[2,†] and James L. N. Wood[3,†]

[1]Institute of Zoology, Zoological Society of London, Regent's Park, London NW1 4RY, UK
[2]Ecohealth Alliance, 460 West 34th Street, New York, NY 10001, USA
[3]Department of Veterinary Medicine, Disease Dynamics Unit, University of Cambridge, Madingley Road, Cambridge CB3 0ES, UK

AAC, 0000-0002-3543-6504

Infectious diseases affect people, domestic animals and wildlife alike, with many pathogens being able to infect multiple species. Fifty years ago, following the wide-scale manufacture and use of antibiotics and vaccines, it seemed that the battle against infections was being won for the human population. Since then, however, and in addition to increasing antimicrobial resistance among bacterial pathogens, there has been an increase in the emergence of, mostly viral, zoonotic diseases from wildlife, sometimes causing fatal outbreaks of epidemic proportions. Concurrently, infectious disease has been identified as an increasing threat to wildlife conservation. A synthesis published in 2000 showed common anthropogenic drivers of disease threats to biodiversity and human health, including encroachment and destruction of wildlife habitat and the human-assisted spread of pathogens. Almost two decades later, the situation has not changed and, despite improved knowledge of the underlying causes, little has been done at the policy level to address these threats. For the sake of public health and well-being, human-kind needs to work better to conserve nature and preserve the ecosystem services, including disease regulation, that biodiversity provides while also understanding and mitigating activities which lead to disease emergence. We consider that holistic, One Health approaches to the management and mitigation of the risks of emerging infectious diseases have the greatest chance of success.

This article is part of the themed issue 'One Health for a changing world: zoonoses, ecosystems and human well-being'.

1. Introduction

By the 1970s, the human burden of infectious diseases in the developed world was substantially diminished from historical levels, largely due to improved sanitation and the development of effective vaccines and antimicrobial drugs [1]. The emergence of a series of novel diseases in the 1970s and 1980s (e.g. toxic shock syndrome, Legionnaire's disease), culminating with the global spread of HIV/AIDS, however, led to infectious disease rising back up the health policy and political agendas [2]. Public concern about emerging infectious diseases (EIDs) has been heightened because of the perception that infectious diseases were previously under control, because of their often rapid spread (e.g. severe acute respiratory syndrome; SARS), because they often have high case fatality rates (e.g. Ebola virus disease) and because the development of drugs and vaccines to combat some of these (e.g. HIV/AIDS) has been slow and costly. By the 1990s, authors had begun to review similarities among these diseases and identify patterns in their origins and emergence [3,4]. Similarities included a skew to zoonotic pathogens originating in wildlife in tropical regions (e.g. Ebola virus), and that emergence was associated with environmental or human behavioural change and human interaction with wildlife (e.g. HIV/AIDS) or with domestic animals which had interactions

with wildlife (e.g. Nipah virus) [5–7]. Emergence was found to be exacerbated by increasing volumes and rates of human travel and globalized trade [8].

By the end of the 1990s, the study of EIDs was a staple of most schools of public health, a key focus of national health agencies, a book topic and the title of a scientific journal [3]. Novel diseases continued to emerge, often from unexpected reservoirs and via new pathways. For example, between 1994 and 1998, three new zoonotic viruses (Hendra, Menangle and Nipah viruses) emerged from pteropodid bats in Australia and southeast Asia [9]. Each of these was transmitted via livestock (horses or pigs), and each belonged to the *Paramyxoviridae*. Around this time, emerging diseases were identified in a series of well-reported die-offs in wildlife, including canine distemper in African lions (*Panthera leo*) in the Serengeti, chytridiomycosis in amphibians globally, pilchard herpesvirus disease in Australasia and West Nile virus in corvids and other birds in New York [10–13]. Pathogens were also implicated for the first time in species extinctions, or near-extinctions, e.g. canine distemper in the black-footed ferret (*Mustela nigripes*), chytridiomycosis in the sharp-snouted day frog (*Taudactylus acutirostris*) and steinhausiosis in the Polynesian tree snail, *Partula turgida* [14–16]. Novel diseases and their emergence in people and wildlife were reviewed, and commonalities in the underlying causes of emergence discussed, in a paper published at the end of the decade [17]. Here, we re-examine some of the key conclusions of that paper, review how the field has progressed 17 years on and identify some of the remaining challenges to understanding and mitigating the impacts of disease emergence in and from wildlife.[1]

2. Disease threats to wildlife

Prior to 2000, wildlife diseases were mostly studied to improve zoo animal survival and welfare, with little published on the diseases of free-living wildlife unless they affected heavily hunted species (e.g. deer in North America) or were considered a threat to livestock health (e.g. tuberculosis, rinderpest). While non-infectious diseases had been widely recognized as important drivers of species declines (e.g. DDT poisoning of raptors [18,19]), only a small number of researchers investigated infectious disease as a factor in, often covert, wildlife population regulation [20,21]. The role of infectious diseases in mass mortality events or population declines was often considered controversial or secondary to other factors [22], and their role in species extinctions often disputed [23,24]. The first definitive identification of disease as a cause of species extinction was published in 1996 following the demise of the last population of the Polynesian tree snail *P. turgida* due to a microsporidian infection [16]. This added to evidence that infectious agents had caused the extinction in the wild of the black-footed ferret, the extinction of around one-third of Hawaiian honeycreepers and the slime mould-induced decline of eelgrass (*Zostera marina*) beds in the USA, leading to extinction of the eelgrass limpet (*Lottia alveus*) [14,25–27]. During the 1990s, wildlife mortality events caused by infectious diseases were reported in zoos, in wildlife translocation programmes and in other conservation programmes [28–32]. Perhaps the most important of these was the discovery of amphibian chytridiomycosis, caused by the chytrid fungal pathogen *Batrachochytrium dendrobatidis*, which was first recognized in the 1990s and has since been implicated in the decline or extinction of over 200 species of amphibian [11,15,33,34]. This disease continues to threaten amphibians globally and has been described as 'the worst infectious disease ever recorded among vertebrates in terms of the number of species impacted, and its propensity to drive them to extinction' [35].

Amphibian chytridiomycosis appears to have emerged contemporaneously in Australia and Central America, associated with large-scale die-offs and extinction events, although in retrospect it might have been causing amphibian mortalities and declines in North America prior to this [36]. Proving that a disease is a cause of population declines in wildlife requires longitudinal population and pathogen data, which are often very difficult to collect. Thus, a series of papers disputing the role of chytridiomycosis in amphibian declines ensued, with most suggesting that this disease either emerged secondarily to other factors, or that it was not the cause of declines/extinctions [37–40]. Long-term datasets have since been published which provide convincing evidence that amphibian chytridiomycosis alone can cause mass mortalities leading to population declines [41]. Policy measures to control amphibian chytridiomycosis, however, have been slow to be enacted, with the first international policy measure (listing of chytridiomycosis by the World Organisation for Animal Health) occurring in 2010 [42] and with the implementation of measures recognized to mitigate the spread of this disease still not being enacted by the international community [43].

Public and political reaction to the more-recent emergence of white nose syndrome (WNS) in North American bats provides evidence that the conservation implications of wildlife EIDs are becoming more widely accepted. The causative agent of WNS is the fungus *Pseudogymnoascus destructans* which colonizes the skin of a range of temperate-zone bats, often causing death during hibernation [44]. Only 1 year after the initial discovery of the disease in the USA in January 2007, visitors to bat caves across the country were being advised to reduce visits and to implement biosecurity measures, and by 2009, caves in over 20 states were closed to the public. The disease has been the focus of a series of grants, formation of multi-disciplinary research partnerships and significant efforts to identify pathogenesis, transmission pathways and potential control measures [45,46].

Although there is a growing recognition of the impact of pathogens on wildlife, the significance of infectious disease as a cause of historical extinctions is likely underestimated due to a previous relative lack of infectious disease focus and diagnostic capability [47]. Collaboration among ecologists, conservation biologists and veterinary pathologists is relatively recent and increased pathological and epidemiological involvement in studies of the causes of wildlife declines are critically needed to identify and understand disease threats to wildlife and how to mitigate them.

3. Zoonotic disease emergence from wildlife

In addition to identifying an apparently growing trend of disease threats to wildlife, Daszak *et al*. [17] highlighted wildlife as the source of a series of high-impact, recently emerging pathogens affecting people. These authors reiterated the widely proposed hypothesis that most emerging pathogens

originate in wildlife and spillover into human hosts due to a range of ecological, demographic and socio-economic changes [1,3,48]. Prior to 2000, these wildlife-origin pathogens were known to include Ebola and Marburg virus, HIV-1 and HIV-2, Sin Nombre virus, Nipah, Hendra and Menangle virus, West Nile virus, *Borrelia burgdorferi* and others. Since then, other human diseases have emerged from wildlife, including Middle East respiratory syndrome (MERS) and different subtypes of avian influenza, and further advances have been made in our understanding of patterns of zoonotic disease emergence. A series of papers analysed a database of all known human EIDs and confirmed that the majority are of animal origin, with viruses being a particularly important group [49–52]. Further analysis of an updated version of this database identified that EIDs had increased in frequency (even accounting for increased numbers of researchers), with the proportion of those emerging from wildlife hosts increasing substantially over the last four decades of the twentieth century [53].

The emergence of bat-origin viral EIDs of people during the 1990s was highlighted by Daszak *et al*. [17]. Since then, it has been shown that bats are reservoir hosts of a striking number of zoonotic viruses, including high-profile pathogens with high case fatality rates, such as Nipah and Hendra paramyxoviruses, filoviruses, SARS-like coronaviruses and possibly also MERS coronavirus [54,55]. This led some authors to propose that bats harbour a disproportionate number of emerging zoonoses compared with other mammalian groups [55–57]: a hypothesis that has been supported by two separate analyses of mammal virus datasets [58,59]. Understanding why bats host so many zoonotic pathogens that cause lethal diseases in humans and how spillover from bats to humans occurs is important in order to control these, and possibly as-yet-undiscovered, diseases [58,60–63].

4. Drivers of disease emergence

There are likely to be multiple causes of novel disease emergence, but the human-mediated transport of pathogens (often in infected hosts) or vectors across geographical or ecological boundaries, a process termed 'pathogen pollution', has been identified as a major driver of this in wildlife [64] and also in plants [65]. The anthropogenic spread of pathogens has been responsible for the emergence of a series of high-profile wildlife EIDs, including the two known agents of amphibian chytridiomycosis, *B. dendrobatidis* and *B. salamandrivorans* [66,67]. Subsequent research indicates that this is only part of the story, as it appears that the global pandemic lineage of *B. dendrobatidis* arose from a single hybrid origin via an ancestral meiosis, possibly via the anthropogenic mixing of allopatric lineages [68,69]. There is a substantial volume of research that shows how, once evolved, this virulent lineage has been introduced globally via the international trade in amphibians and via the human-assisted introduction of invasive species [66,70–75].

In recent years, a body of literature has developed the concept of the ecosystem service of disease regulation. While still controversial, and probably not universal [76], this proposes that natural biodiversity limits the exposure and impact of many pathogens, including those that are zoonotic, through a dilution or buffering effect, thus limiting opportunities for pathogen spillover from wildlife to people [77]. When biodiversity is depleted (usually by human activities), this ecosystem service is impaired and zoonotic pathogens are more likely to emerge, as has been shown for hantavirus [78] and for *B. burgdorferi*, the causative agent of Lyme disease [79,80]. Also, alteration of species complements (again, usually due to anthropogenic impacts), rather than loss of biodiversity *per se*, can alter infection dynamics and lead to increased zoonotic disease risk [81]. Our understanding of the interactions between ecosystem change, disease regulation and human well-being, however, is in its infancy.

Almost 20 years since the threats to conservation and human health that wildlife EIDs represent was first highlighted, there has been little effort to put in place policies to reduce risk. Detecting and preventing the importation of infected hosts is widely used to prevent importation of many domestic animal diseases of economic or public health importance. Some countries even enact this principle for the movement of people, whereby they conduct (often cursory) surveillance for infected persons arriving at their international borders, particularly during human pandemics [82,83]. The World Health Organisation provides guidance and training on this through its International Health Regulations (http://www.who.int/ihr/en/). Rules and regulations for international trade, including of animals and their products, are created and enforced by the World Trade Organisation (WTO), which has the remit of ensuring 'that trade flows as smoothly, predictably and freely as possible' (www.wto.org). The WTO agreement on sanitary and phytosanitary measures was enacted on 1 January 1995 with the aim of protecting human, animal and plant life from disease-causing agents. While countries have discretion in what should be included, they are guided by the World Organisation for Animal Health (OIE) list of diseases of international importance. Although the OIE has a remit of protecting biodiversity, only two pathogens are listed for this purpose: *B. dendrobatidis* and *Ranavirus* [42]. Most countries, therefore, use import controls to only protect against domestic animal diseases of obvious public health or economic importance, such as rabies and foot and mouth disease; diseases restricted to wildlife are not included even when OIE-listed.

In addition, trade agreements often prohibit barriers to international animal movements for the purposes of infectious disease control. For example, countries within the European Union have little ability to prevent the spread of pathogens via within-EU trade unless as part of a specific EU disease control programme. Even where technically legal under WTO rules, there appears to be reluctance by countries to unilaterally impose restrictions on non-listed diseases in case they create an economic disadvantage or are subsequently found to be in breach of international trade regulations. It is possible that the international spread of amphibian chytridiomycosis would have been reduced if such measures had been implemented for this disease [43]. Perhaps learning from this, in January 2016, the USA banned the importation of salamanders following the emergence of *B. salamandrivorans* in order to protect native wildlife from this novel pathogen [84]. Such protective action was enacted relatively rapidly following the discovery of *B. salamandrivorans* as a novel lethal fungus infecting and killing captive and wild salamanders in Europe [67,85,86]. Hopefully, this will open the doors to the imposition of trade controls for other diseases and by other nations in

order to protect biodiversity from the anthropogenic spread of pathogens.

Challenges remain to understanding the wildlife origins of zoonotic EIDs. It is often difficult, time-consuming, logistically challenging and very expensive to identify the origins of newly emerged pathogens of humans. For example, viruses similar to HIV/AIDs were discovered in non-human primates in the early 1980s, but identification of the true progenitor viruses in chimpanzees took almost a decade of additional research [87]. Similarly, the origins of Ebola and Marburg viruses have been investigated for over 30 years. To date, however, despite indications that bats are the natural reservoir hosts of these viruses, clear evidence has only been found for Marburg virus infection in bats in limited locations [88–90]. Identifying putative reservoir host(s) is just the beginning. In order to identify actions to prevent or mitigate future zoonotic spillover, both an understanding of the ecology of the pathogen in its natural host(s) and of human–host interactions are required [63]. For example, substantial efforts have been conducted to understand immunological, behavioural and ecological characteristics of bats as part of a strategy to control zoonotic spillover from bats [91–93]. Long-term, multi-disciplinary studies that systematically investigate the ecology of zoonotic pathogens in their wildlife hosts along with the risk characteristics for spillover are critical to better predict and prevent future pandemics [63]. Such a study, which included years of field data collection on fruit tree distribution, pig farm management, viral dynamics and satellite telemetry of fruit bats, analysis of climate trends, experimental infection of bats under BioSafety Level-4 conditions and mathematical modelling of virus infection dynamics, identified the intensification of the pig industry as the driver of the zoonotic emergence of Nipah virus in Malaysia [94]. These results informed government policies to separate pigs from bats via the removal of fruit trees from pig farms and the relocation of farms away from forested areas [95], since when no further Nipah virus disease outbreaks have occurred in Malaysia.

5. Endemic zoonoses from wildlife

EID events have been the focus of intense research over the past two decades, even though the numbers of people diagnosed with them are often relatively small. This disproportionate focus on EIDs probably relates to the dislike of human society for uncertainty, or put more simply, fear of the unknown. This may lead to perverse scenarios in which fear of disease can have a greater impact than the direct impact of the outbreak itself. For example, during a recent Ebola virus epidemic in West Africa, more people are estimated to have died from malaria due to their avoidance of healthcare facilities, where they feared they might catch Ebola, than the thousands that died from the virus itself [96].

Indeed, when one considers the overall impact of zoonotic diseases on the human population, the largest (diagnosed) burden is associated with well known and fully recognized (in the industrial north), but neglected, diseases such as brucellosis, rickettsioses and Rift Valley fever [97]. This predictable burden falls heavily on the global poor—poverty being the major risk factor for most zoonoses, which in turn causes some communities to suffer disproportionately from the burden of zoonotic disease [97]. The neglect of such diseases includes diagnostic neglect (and confusion with other conditions such as malaria [98]) and historic and current research neglect; all of which feeds into therapeutic neglect. The delivery of the United Nations sustainable development goals, which should result in much reduced poverty and improved health, will in themselves reduce the substantial burden of zoonotic disease.

6. Whither One Health

One Health is the term used when approaches to tackling disease (particularly zoonoses) consider all components that might lead to, or increase, the threat of disease. These include environmental and ecological/wildlife components as well as domestic animal and human factors. The last encompasses behavioural as well as medical issues, including cultural, political and other socio-economic drivers that might result in disease occurrence or spread. The review by Daszak *et al*. [17] was perhaps the first 'One Health' review of emerging diseases, in that it brought together veterinary, ecological, conservation and human medical perspectives on disease emergence. The field of One Health has expanded substantially since 2000, diversifying to produce new journals, such as *One Health*, *EcoHealth* and *The Lancet Planetary Health*, the One Health Platform, the International Association of Ecology and Health, the Planetary Health Alliance and a series of One Health institutions in the USA, Europe, Australia and increasingly also in developing countries. The success of this multi-disciplinary approach has been driven largely by the synergistic impact of combining detailed and logistically challenging field sciences (e.g. ecology, field biology) with analytical approaches (e.g. epidemiological modelling, pathogen phylogenetic analysis) and laboratory science (e.g. serology, pathogen diagnostics, immunology). Challenges remain, however. Importantly, while the conservation, ecological and veterinary professions are increasingly engaged with One Health, substantial elements of the medical profession are not aware of, or involved in, this approach.

Despite their neglect, a number of zoonotic diseases are eminently controllable or manageable by One Health approaches, including infectious causes of abortion in livestock, which frequently result in febrile human disease, and human rabies transmitted via dog bites. Control or prevention is best achieved through integrated public health, veterinary medicine, animal management and ecological approaches. One particular challenge for this is in the case of some zoonotic infections that do not cause clinical signs in their animal hosts, one of the most common examples of which is *Campylobacter* spp. infection of poultry, which globally is the most frequent cause of food poisoning in humans [99]. Is it, then, the responsibility of farmers and vets to ensure that people do not become infected, or of public health practitioners or the general public through improved kitchen hygiene and behaviours? Here, this would involve reduced infection of poultry (the role of farmers and veterinarians), reduced contamination of meat (the responsibility of veterinary public health workers) and preventive measures in the kitchen (hygiene and proper cooking), which are the domain of public health workers and the public [99].

One Health approaches are required at the policy and governance levels, too. Responsibility for preventing and treating zoonotic disease, in both a developing and

developed world setting, for example, often falls in between government Ministries of Health and Agriculture (and for wildlife, Ministries of Environment and Forestry) and this can structurally prevent the simplest of solutions from being implemented. An important example is rabies in humans transmitted through dog bites which kills around 60 000 people annually [100] and causes fear in many more in rabies endemic regions. The disease is easily preventable (and arguably open to eradication) through repeated annual or biannual mass vaccination of dogs [101]. In many countries with a high burden of rabies in dogs, considerable sums are spent by the public and Ministries of Health annually on post-exposure prophylaxis (PEP—often given after dog bites whether or not the animal was known to be rabid). The expense of this repeated treatment usually dictates that far more is spent on treatment than would be required to vaccinate all dogs in the same region. However, in many countries, the dog is regarded as a pest and not an agricultural animal for which Ministries of Agriculture have responsibility. In others, the dog does fall under the Agricultural Ministry, but these Ministries are typically far less well resourced compared with Ministries of Health, thus rabies, which does not relate to food animals, is not prioritized. The obvious solution is for a synergized One Health approach with the Ministries of Health supporting prophylactic vaccination programmes for dogs delivered by their typically far less well-resourced Ministries of Agriculture. This, however, rarely seems to happen and continued expenditure on bite management and PEP continues. One Health programmes addressing rabies have been extremely successful when appropriately resourced [102,103]; however, they often fail to influence national government policy and are rarely adopted long term [104].

7. Policies for prevention and control

In addition to the high costs of dealing with endemic zoonoses, such as rabies, emerging and re-emerging zoonoses can have substantial economic impacts. The cost implications of zoonotic EIDs were highlighted by Daszak *et al*. [17] as a rationale for policy measures, but methods for calculating the economic consequences of disease emergence have not advanced in the interim. Despite clearly high financial impacts associated with some EIDs, few detailed economic analyses of their impact have been undertaken. Estimates of the cost of the 2003 SARS outbreak, for example, range from $5 to $50 billion, while the true costs of most EIDs have never been estimated [105]. Pike *et al.* [105] approached the problem of disease emergence in the same way as the climate change phenomenon. They used the increasing frequency of emerging disease events reported by Jones *et al*. [53] to analyse two strategies to deal with the rising costs of EIDs over time: adaptation, whereby we adopt a business-as-usual approach and continue to cause increased EID events, then target control programmes after emergence; and mitigation, whereby we deal with the underlying drivers (e.g. wildlife trade, deforestation) and reduce the frequency of EID events. Pike *et al.* [105] show that mitigation strategies are more cost effective in the long term, with a 10-fold return on investment, and that these need to be enacted on a global scale within the current generation or the cost of EIDs becomes unaffordable.

What would these global strategies entail? We highlight three approaches. First, a series of emerging diseases have been linked to the wildlife trade, or consumption of wildlife (e.g. SARS, Ebola). The health implications of the trade in wildlife have not been widely used to implement controls, or advocate for reduction in consumption, and may be a more effective message than its conservation impacts. This needs to be done judiciously, however, as disease spillover is a rare event and both bushmeat hunters and consumers will be wary of public health messages that do not fit with their experiences [93,106].

Second, a revision of an earlier analysis of global drivers of disease emergence [53] shows that land-use change correlates strongly with the emergence of zoonoses from wildlife (P Daszak 2017, unpublished observation). In Malaysia, analyses of the economic cost of diseases that emerge due to land conversion for palm oil production (e.g. malaria, leptospirosis) are currently being used to advise industry where to reduce long-term impact. Identifying land-use changes that lead to disease emergence informs policies for mitigation strategies. This could be done, for example, via the incorporation of wildlife and zoonotic disease threats in environmental impact studies, an approach for the prevention of disease emergence suggested by Daszak *et al*. [17].

Third, targeted global surveillance programmes to identify novel pathogens of zoonotic potential before they emerge may increase our capacity to reduce their risk of emergence. For example, a series of laboratories now specialize in identifying novel viruses from wildlife hosts, e.g. bats [107–113]. The USAID Emerging Pandemic Threats programme specifically targets emerging disease hotspots to identify novel viruses from bats, rodents and primates, to characterize high-risk behaviours in people and to identify potential mitigation strategies [60]. While these programmes have already identified over 1000 new viruses from viral families with known zoonoses in the last few years, challenges remain in how to identify those with the highest (or any) risk of zoonotic emergence. This indicates that a change in approach is required, building on rapidly expanding databases of pathogen sequences, phenotypic characteristics and host–pathogen interactions. For example, the rapid incorporation of novel viral sequences into diagnostic tests may lead to more rapid identification of related, previously unknown, pathogens that emerge in outbreaks. Using this approach, combined with a One Health perspective that targets the underlying drivers of emergence, could result in the identification of pathogens that already are spilling over from wildlife hosts sporadically at low levels, enabling measures to be taken to reduce pandemic risk.

8. Conclusion

Since the synthesis paper by Daszak *et al*. [17] highlighted emerging disease threats of, and from, wildlife and the main drivers underlying these, further advances have been made in our understanding of the origin, size and potential scope of these threats. Endemic zoonoses, however, continue to be relatively neglected, often with a lack of local and international realization of the extent to which they impact human health and well-being. This is partly due to issues surrounding local capacity and knowledge and partly because, unlike EIDs, they are not seen as a threat to people in the developed world. Both EIDs and endemic zoonoses, however, can be tackled using a One Health approach, including the

identification and mitigation of human activities that lead to disease emergence and spread. One Health approaches to dealing with disease threats from and to wildlife are still relatively young and untried, but all evidence points to them being most successful and cost-effective if developed and implemented in full by all relevant parties, including policy-makers and the medical profession.

Authors' contributions. A.A.C. conceived the idea for the article. All authors contributed equally to the writing of the manuscript.

Competing interests. We declare we have no competing interests. A.A.C. and J.L.N.W. are Guest Editors of the issue.

Funding. A.A.C. and J.L.N.W. were funded by ESPA (Ecosystem Services for Poverty Alleviation), supported by NERC (Natural Environment Research Council), DFID (Department for International Development) and ESRC (Economic and Social Research Council) (NEJ001570-1), and by the European Commission Seventh Framework Programme under ANTIGONE, Project Number 278976. A.A.C. was supported by a Royal Society Wolfson Research Merit award. J.L.N.W. is supported by the Alborada Trust. P.D. was supported by the United States Agency for International Development (USAID) Emerging Pandemic Threats PREDICT project (cooperative agreement number GHN-A-OO-09-00010-00).

Endnote

[1]In this paper, when we discuss wildlife, we refer to non-domesticated animals regardless of taxon.

References

1. Lederberg J, Shope RE, Oaks SC (eds). 1992 *Emerging infections: microbial threats to health in the United States*. Washington, DC: National Academy Press.
2. Smolinski MS, Hamburg MA, Lederberg J (eds). 2003 *Microbial threats to health: emergence, detection, and response*. Washington, DC: National Academies Press.
3. Morse S. 1995 Factors in the emergence of infectious diseases. *Emerg. Infect. Dis.* **1**, 7–15. (doi:10.3201/eid0101.950102)
4. Krause RM. 1994 Dynamics of emergence. *J. Infect. Dis.* **170**, 265–271. (doi:10.1093/infdis/170.2.265)
5. Chua KB *et al.* 2000 Nipah virus: a recently emergent deadly paramyxovirus. *Science* **288**, 1432–1435. (doi:10.1126/science.288.5470.1432)
6. Weiss RA, McMichael AJ. 2004 Social and environmental risk factors in the emergence of infectious diseases. *Nat. Med.* **10**, S70–S76. (doi:10.1038/nm1150)
7. Hahn BH. 2000 AIDS as a zoonosis: scientific and public health implications. *Science* **287**, 607–614. (doi:10.1126/science.287.5453.607)
8. Morse SS. 1993 *Emerging viruses*. New York, NY: Oxford University Press.
9. Field HE, Mackenzie JS, Daszak P. 2007 Henipaviruses: emerging paramyxoviruses associated with fruit bats. *Curr. Top. Microbiol. Immunol.* **315**, 133–159.
10. Lanciotti RS *et al.* 2002 Complete genome sequences and phylogenetic analysis of West Nile virus strains isolated from the United States, Europe, and the Middle East. *Virology* **298**, 96–105. (doi:10.1006/viro.2002.1449)
11. Berger L *et al.* 1998 Chytridiomycosis causes amphibian mortality associated with population declines in the rain forests of Australia and Central America. *Proc. Natl Acad. Sci. USA* **95**, 9031–9036. (doi:10.1073/pnas.95.15.9031)
12. Roelke-Parker ME *et al.* 1996 A canine distemper virus epidemic in Serengeti lions (*Panthero leo*). *Nature* **379**, 441. (doi:10.1038/379441a0)
13. Hyatt AD, Hine PM, Jones B, Whittington R, Wise T, Crane M. 1997 Epizootic mortality in the pilchard (*Sardinops sagax neopilchardus*) in Australia and New Zealand in 1995. II. Identification of a herpesvirus within the gill epithelium. *Dis. Aquat. Org.* **28**, 17–29. (doi:10.3354/dao028017)
14. Thorne ET, Williams ES. 1988 Disease and endangered species: the black-footed ferret as a recent example. *Conserv. Biol.* **2** 66–74. (doi:10.1111/j.1523-1739.1988.tb00336.x)
15. Schloegel LM, Hero JM, Berger L, Speare R, McDonald K, Daszak P. 2006 The decline of the sharp-snouted day frog (*Taudactylus acutirostris*): the first documented case of extinction by infection in a free-ranging wildlife species? *Ecohealth* **3**, 35–40. (doi:10.1007/s10393-005-0012-6)
16. Cunningham AA, Daszak P. 1998 Extinction of a species of land snail due to infection with a microsporidian parasite. *Conserv. Biol.* **12**, 1139. (doi:10.1046/j.1523-1739.1998.97485.x)
17. Daszak P, Cunningham AA, Hyatt AD. 2000 Emerging infectious diseases of wildlife—threats to biodiversity and human health. *Science* **287**, 443–449. (doi:10.1126/science.287.5452.443)
18. Hickey JJ, Anderson DW. 1968 Chlorinated hydrocarbons and eggshell changes in raptorial and fish-eating birds. *Science* **162**, 271–273. (doi:10.1126/science.162.3850.271)
19. Ratcliff DA. 1967 Decrease in eggshell weight in certain birds of prey. *Nature* **215**, 208–210. (doi:10.1038/215208a0)
20. Hudson P, Greenman J. 1998 Competition mediated by parasites: biological and theoretical progress. *Trends Ecol. Evol.* **13**, 387–390. (doi:10.1016/S0169-5347(98)01475-X)
21. Hudson PJ, Dobson AP, Newborn D. 1998 Prevention of population cycles by parasite removal. *Science* **282**, 2256–2258. (doi:10.1126/science.282.5397.2256)
22. Harvell CD *et al.* 1999 Emerging marine diseases—climate links and anthropogenic factors. *Science* **285**, 1505–1510. (doi:10.1126/science.285.5433.1505)
23. McCallum H. 2005 Inconclusiveness of chytridiomycosis as the agent in widespread frog declines. *Conserv. Biol.* **19**, 1421–1430. (doi:10.1111/j.1523-1739.2005.00217.x)
24. McCallum H, Dobson A. 1995 Detecting disease and parasite threats to endangered species and ecosystems. *Trends Ecol. Evol.* **10**, 190–194. (doi:10.1016/S0169-5347(00)89050-3)
25. Van Riper III C, Van Riper SG, Goff LM, Laird M. 1986 The epizootiology and ecological significance of malaria in Hawaiian land birds. *Ecol. Monogr.* **56**, 327–344. (doi:10.2307/1942550)
26. Daszak P, Cunningham A. 1999 Extinction by infection. *Trends Ecol. Evol.* **14**, 279. (doi:10.1016/S0169-5347(99)01665-1)
27. Carlton JT, Vermeij GJ, Lindberg DR, Carlton DA, Dudley EC. 1991 The first historical extinction of a marine invertebrate in an ocean basin—the demise of the eelgrass limpet *Lottia alveus*. *Biol. Bull.* **180**, 72–80. (doi:10.2307/1542430)
28. Viggers KL, Lindenmayer DB, Spratt DM. 1993 The importance of disease in reintroduction programmes. *Wildlife Res.* **20**, 687–698. (doi:10.1071/WR9930687)
29. Woodford MH. 1993 International disease implications for wildlife translocations. *J. Zoo Wildl. Med.* **24**, 265.
30. Lyles AM, Dobson AP. 1993 Infectious disease and intensive management: population dynamics, threatened hosts, and their parasites. *J. Zoo Wildl. Med.* **24**, 315–326.
31. Cunningham AA. 1996 Disease risks of wildlife translocations. *Conserv. Biol.* **10**, 349–353. (doi:10.1046/j.1523-1739.1996.10020349.x)
32. Nettles VF. 1996 Reemerging and emerging infectious diseases: economic and other impacts on wildlife—transport of animals sometimes spreads infections, while other outbreaks are a mystery. *ASM News* **62**, 589–591.
33. Skerratt LF, Berger L, Speare R, Cashins S, McDonald KR, Phillott AD, Hines HB, Kenyon N. 2007 Spread of chytridiomycosis has caused the rapid global decline and extinction of frogs. *Ecohealth* **4**, 125–134. (doi:10.1007/s10393-007-0093-5)
34. Cunningham AA. 1998 A breakthrough in the hunt for a cause of amphibian declines. *Froglog* **30**, 3.
35. Amphibian Conservation Summit. 2005 *Amphibian Conservation Summit, Washington DC, 17–19*

September 2005. Declaration. See http://irceb.asu.edu/amphibians/pdf/ACAP%20Summit%20Declaration.pdf.

36. Daszak P, Berger L, Cunningham AA, Hyatt AD, Green DE, Speare R. 1999 Emerging infectious diseases and amphibian population declines. *Emerg. Infect. Dis.* **5**, 735–748. (doi:10.3201/eid0506.990601)
37. Pounds JA. 2001 Climate and amphibian declines. *Nature* **410**, 639–640. (doi:10.1038/35070683)
38. Carey C, Alexander MA. 2003 Climate change and amphibian declines: is there a link? *Divers. Distrib.* **9**, 111–121. (doi:10.1046/j.1472-4642.2003.00011.x)
39. Stallard RF. 2001 Possible environmental factors underlying amphibian decline in eastern Puerto Rico: analysis of US government data archives. *Conserv. Biol.* **15**, 943–953. (doi:10.1046/j.1523-1739.2001.015004943.x)
40. Kiesecker JM, Blaustein AR, Belden LK. 2001 Complex causes of amphibian population declines. *Nature* **410**, 681–684. (doi:10.1038/35070552)
41. Lips KR *et al.* 2006 Emerging infectious disease and the loss of biodiversity in a neotropical amphibian community. *Proc. Natl Acad. Sci. USA* **103**, 3165–3170. (doi:10.1073/pnas.0506889103)
42. Schloegel LM, Daszak P, Cunningham AA, Speare R, Hill B. 2010 Two amphibian diseases, chytridiomycosis and ranaviral disease, are now globally notifiable to the World Organization for Animal Health (OIE): an assessment. *Dis. Aquat. Org.* **92**, 101–108. (doi:10.3354/dao02140)
43. Hudson MA *et al.* 2016 Dynamics and genetics of a disease-driven species decline to near extinction: lessons for conservation. *Sci. Rep.* **6**, 30772. (doi:10.1038/srep30772)
44. Blehert DS *et al.* 2009 Bat white-nose syndrome: an emerging fungal pathogen? *Science* **323**, 227. (doi:10.1126/science.1163874)
45. Lorch JM *et al.* 2011 Experimental infection of bats with *Geomyces destructans* causes white-nose syndrome. *Nature* **480**, 376–378. (doi:10.1038/nature10590)
46. Lee JJ. 2015 Killer fungus that's devastating bats may have met its match. *National Geographic*, 27 May 2015. See http://news.nationalgeographic.com/2015/05/150527-bats-white-nose-syndrome-treatment-conservation-animals-science/.
47. Smith KF, Sax DF, Lafferty KD. 2006 Evidence for the role of infectious disease in species extinction and endangerment. *Conserv. Biol.* **20**, 1349–1357. (doi:10.1111/j.1523-1739.2006.00524.x)
48. Krause RM. 1992 The origins of plagues: old and new. *Science* **257**, 1073–1078. (doi:10.1126/science.257.5073.1073)
49. Taylor LH, Latham SM, Woolhouse ME. 2001 Risk factors for human disease emergence. *Phil. Trans. R. Soc. Lond. B* **356**, 983–989. (doi:10.1098/rstb.2001.0888)
50. Woolhouse MEJ, Taylor LH, Haydon DT. 2001 Population biology of multihost pathogens. *Science* **292**, 1109–1112. (doi:10.1126/science.1059026)
51. Woolhouse MEJ. 2002 Population biology of emerging and re-emerging pathogens. *Trends Microbiol.* **10**, S3–S7. (doi:10.1016/s0966-842x(02)02428-9)
52. Woolhouse MEJ, Gowtage-Sequeria S. 2005 Host range and emerging and reemerging pathogens. *Emerg. Infect. Dis.* **11**, 1842–1847. (doi:10.3201/eid1112.050997)
53. Jones KE, Patel NG, Levy MA, Storeygard A, Balk D, Gittleman JL, Daszak P. 2008 Global trends in emerging infectious diseases. *Nature* **451**, 990–993. (doi:10.1038/nature06536)
54. Memish ZA *et al.* 2013 Middle East Respiratory Syndrome coronavirus in bats, Saudi Arabia. *Emerg. Infect. Dis.* **19**, 1819–1823. (doi:10.3201/eid1911.131172)
55. Calisher CH, Childs JE, Field HE, Holmes KV, Schountz T. 2006 Bats: important reservoir hosts of emerging viruses. *Clin. Microbiol. Rev.* **19**, 531–545. (doi:10.1128/CMR.00017-06)
56. Dobson AP. 2005 What links bats to emerging infectious diseases? *Science* **310**, 628–629. (doi:10.1126/science.1120872)
57. Wang L-F, Shi Z, Zhang S, Field H, Daszak P, Eaton BT. 2006 A review of bats and SARS: virus origin and genetic diversity. *Emerg. Infect. Dis.* **12**, 1834–1840. (doi:10.3201/eid1212.060401)
58. Luis AD *et al.* 2013 A comparison of bats and rodents as reservoirs of zoonotic viruses: are bats special? *Proc. R. Soc. B* **280**, 20122753. (doi:10.1098/rspb.2012.2753)
59. Olival KJ, Hosseini PR, Zambrana-Torrelio C, Ross N, Bogich TL, Daszak P. In press. Host and viral traits predict zoonotic spillover from mammals. *Nature*.
60. Morse SS, Mazet JAK, Woolhouse M, Parrish CR, Carroll D, Karesh WB, Zambrana-Torrelio C, Lipkin WI, Daszak P. 2012 Prediction and prevention of the next pandemic zoonosis. *Lancet* **380**, 1956–1965. (doi:10.1016/S0140-6736(12)61684-5)
61. Luis AD, O'Shea TJ, Hayman DTS, Wood JLN, Cunningham AA, Gilbert AT, Mills JN, Webb CT. 2015 Network analysis of host-virus communities in bats and rodents reveals determinants of cross-species transmission. *Ecol. Lett.* **18**, 1153–1162. (doi:10.1111/ele.12491)
62. O'Shea TJ, Cryan PM, Cunningham AA, Fooks AR, Hayman DTS, Luis AD, Peel AJ, Plowright RK, Wood JLN. 2014 Bat flight and zoonotic viruses. *Emerg. Infect. Dis.* **20**, 741–745. (doi:10.3201/eid2005.130539)
63. Wood JLN *et al.* 2012 A framework for the study of zoonotic disease emergence and its drivers: spillover of bat pathogens as a case study. *Phil. Trans. R. Soc. B* **367**, 2881–2892. (doi:10.1098/rstb.2012.0228)
64. Cunningham AA, Daszak P, Rodríguez JP. 2003 Pathogen pollution: defining a parasitological threat to biodiversity conservation. *J. Parasitol.* **89**, S78–S83.
65. Anderson PK, Cunningham AA, Patel NG, Morales FJ, Epstein PR, Daszak P. 2004 Emerging infectious diseases of plants: pathogen pollution, climate change and agrotechnology drivers. *Trends Ecol. Evol.* **19**, 535–544. (doi:10.1016/j.tree.2004.07.021)
66. Fisher MC, Walker SF, Garner TWJ. 2009 The global emergence of *Batrachochytrium dendrobatidis* in space, time, and host. *Ann. Rev. Microbiol.* **63**, 291–310. (doi:10.1146/annurev.micro.091208.073435)
67. Martel A *et al.* 2014 Recent introduction of a chytrid fungus endangers Western Palearctic salamanders. *Science* **346**, 630–631. (doi:10.1126/science.1258268)
68. James TY *et al.* 2009 Rapid expansion of an emerging fungal disease into declining and healthy amphibian populations. *PLoS Pathog.* **5**, e1000458. (doi:10.1371/journal.ppat.1000458)
69. Farrer RA *et al.* 2011 Multiple emergences of genetically diverse amphibian-infecting chytrids include a globalized hypervirulent recombinant lineage. *Proc. Natl Acad. Sci. USA* **108**, 18 732–18 736. (doi:10.1073/pnas.1111915108)
70. Gilbert M *et al.* 2012 Amphibian pathogens in Southeast Asian frog trade. *Ecohealth* **9**, 386–398. (doi:10.1007/s10393-013-0817-7)
71. Liu X, Rohr JR, Li YM. 2013 Climate, vegetation, introduced hosts and trade shape a global wildlife pandemic. *Proc. R. Soc. B* **280**, 20122506. (doi:10.1098/rspb.2012.2506)
72. McKenzie VJ, Peterson AC. 2012 Pathogen pollution and the emergence of a deadly amphibian pathogen. *Mol. Ecol.* **21**, 5151–5154. (doi:10.1111/mec.12013)
73. Peel AJ, Hartley M, Cunningham AA. 2012 Qualitative risk analysis of introducing *Batrachochytrium dendrobatidis* to the UK through the importation of live amphibians. *Dis. Aquat. Org.* **98**, 95–112. (doi:10.3354/dao02424)
74. Schloegel LM *et al.* 2012 Novel, panzootic and hybrid genotypes of amphibian chytridiomycosis associated with the bullfrog trade. *Mol. Ecol.* **21**, 5162–5177. (doi:10.1111/j.1365-294X.2012.05710.x)
75. Wombwell EL, Garner TWJ, Cunningham AA, Quest R, Pritchard S, Rowcliffe JM, Griffiths RA. 2016 Detection of *Batrachochytrium dendrobatidis* in amphibians imported into the UK for the pet trade. *Ecohealth* **13**, 456–466. (doi:10.1007/s10393-016-1138-4)
76. Salkeld DJ, Padgett KA, Jones JH. 2013 A meta-analysis suggesting that the relationship between biodiversity and risk of zoonotic pathogen transmission is idiosyncratic. *Ecol. Lett.* **16**, 679–686. (doi:10.1111/ele.12101)
77. Johnson PTJ, Thieltges DW. 2010 Diversity, decoys and the dilution effect: how ecological communities affect disease risk. *J. Exp. Biol.* **213**, 961–970. (doi:10.1242/jeb.037721)
78. Suzan G, Marce E, Giermakowski JT, Mills JN, Ceballos G, Ostfeld RS, Armien B, Pascale JM, Yates TL. 2009 Experimental evidence for reduced rodent diversity causing increased hantavirus prevalence. *PLoS ONE* **4**, e5461. (doi:10.1371/journal.pone.0005461)
79. Ostfeld RS, Keesing F. 2000 Biodiversity and disease risk: the case of Lyme disease. *Conserv. Biol.* **14**, 722–728. (doi:10.1046/j.1523-1739.2000.99014.x)

80. LoGiudice K, Ostfeld RS, Schmidt KA, Keesing F. 2003 The ecology of infectious disease: effects of host diversity and community composition on Lyme disease risk. *Proc. Natl Acad. Sci. USA* **100**, 567–571. (doi:10.1073/pnas.0233733100)
81. Kilpatrick AM, Daszak P, Jones MJ, Marra PP, Kramer LD. 2006 Host heterogeneity dominates West Nile virus transmission. *Proc. R. Soc. B* **273**, 2327–2333. (doi:10.1098/rspb.2006.3575)
82. Cetron M, Landwirth J. 2005 Public health and ethical considerations in planning for quarantine. *Yale J. Biol. Med.* **78**, 325–330.
83. Waterman SH, Escobedo M, Wilson T, Edelson PJ, Bethel JW, Fishbein DB. 2009 A new paradigm for quarantine and public health activities at land borders: opportunities and challenges. *Public Health Rep.* **124**, 203–211.
84. Bean MJ. 2016 *Injurious wildlife species; listing salamanders due to risk of salamander chytrid fungus—Document 81 FR 1534*, pp. 1534–1556. Federal Register, US Fish & Wildlife Agency. See https://www.fws.gov/policy/library/2016/2016–00452.html.
85. Martel A *et al.* 2013 *Batrachochytrium salamandrivorans* sp. nov causes lethal chytridiomycosis in amphibians. *Proc. Natl Acad. Sci. USA* **110**, 15 325–15 329. (doi:10.1073/pnas.1307356110)
86. Cunningham AA *et al.* 2015 Emerging disease in UK amphibians. *Vet. Rec.* **176**, 468. (doi:10.1136/vr.h2264)
87. Gao F *et al.* 1999 Origin of HIV-1 in the chimpanzee *Pan troglodytes*. *Nature* **397**, 436–441. (doi:10.1038/17130)
88. Amman BR *et al.* 2012 Seasonal pulses of Marburg virus circulation in juvenile *Rousettus aegyptiacus* bats coincide with periods of increased risk of human infection. *PLoS Pathog.* **8**, e1002877. (doi:10.1371/journal.ppat.1002877)
89. Amman BR *et al.* 2015 Oral shedding of Marburg virus in experimentally infected Egyptian fruit bats (*Rousettus aegyptiacus*). *J. Wildl. Dis.* **51**, 113–124. (doi:10.7589/2014-08-198)
90. Jones MEB, Schuh AJ, Amman BR, Sealy TK, Zaki SR, Nichol ST, Towner JS. 2015 Experimental inoculation of Egyptian Rousette bats (*Rousettus aegyptiacus*) with viruses of the Ebolavirus and Marburgvirus genera. *Viruses* **7**, 3420–3442. (doi:10.3390/v7072779)
91. Kamins AO, Rowcliffe JM, Ntiamoa-Baidu Y, Cunningham AA, Wood JLN, Restifl O. 2015 Characteristics and risk perceptions of Ghanaians potentially exposed to bat-borne zoonoses through bushmeat. *Ecohealth* **12**, 104–120. (doi:10.1007/s10393-014-0977-0)
92. Mannerings AO, Osikowicz LM, Restif O, Nyarko E, Suu-Ire R, Cunningham AA, Wood JLN, Kosoy MY. 2016 Exposure to bat-associated *Bartonella* spp. among humans and other animals, Ghana. *Emerg. Infect. Dis.* **22**, 922–924. (doi:10.3201/eid2205.151908)
93. Wood JLN, Cunningham AA, Suu-Ire RD, Jephcott FL, Ntiamoa-Baidu Y. 2016 Ebola, bats and evidence-based policy: informing Ebola policy. *Ecohealth* **13**, 9–11. (doi:10.1007/s10393-015-1050-3)
94. Pulliam JR *et al.* 2012 Agricultural intensification, priming for persistence and the emergence of Nipah virus: a lethal bat-borne zoonosis. *J. R. Soc. Interface* **9**, 89–101. (doi:10.1098/rsif.2011.0223)
95. Epstein JH, Field HE, Luby S, Pulliam JRC, Daszak P. 2006 Nipah virus: impact, origins, and causes of emergence. *Curr. Infect. Dis. Rep.* **8**, 59–65. (doi:10.1007/s11908-006-0036-2)
96. Plucinski MM *et al.* 2015 Effect of the Ebola-virus-disease epidemic on malaria case management in Guinea, 2014: a cross-sectional survey of health facilities. *Lancet Infect. Dis.* **15**, 1017–1023. (doi:10.1016/s1473-3099(15)00061-4)
97. Halliday JEB, Allan KJ, Ekwem D, Cleaveland S, Kazwala RR, Crump JA. 2015 Endemic zoonoses in the tropics: a public health problem hiding in plain sight. *Vet. Rec.* **176**, 220–225. (doi:10.1136/vr.h798)
98. Jephcott FL, Wood JLN, Cunningham AA. 2017 Facility-based surveillance for emerging infectious diseases; diagnostic practices in rural West African hospital settings: observations from Ghana. *Phil. Trans. R. Soc. B* **372**, 20160544. (doi:10.1098/rstb.2016.0544)
99. Silva J, Leite D, Fernandes M, Mena C, Gibbs PA, Teixeira P. 2011 *Campylobacter* spp. as a foodborne pathogen: a review. *Front. Microbiol.* **2**, 200. (doi:10.3389/fmicb.2011.00200)
100. Hampson K *et al.* 2015 Estimating the global burden of endemic canine rabies. *PLoS Negl. Trop. Dis.* **9**, e0003709. (doi:10.1371/journal.pntd.0003709)
101. Cleaveland S *et al.* 2014 The changing landscape of rabies epidemiology and control. *Onderstepoort J. Vet. Res.* **81**, E1–E8. (doi:10.4102/ojvr.v81i2.731)
102. Morters MK *et al.* 2014 Achieving population-level immunity to rabies in free-roaming dogs in Africa and Asia. *PLoS Negl. Trop. Dis.* **8**, e3160. (doi:10.1371/journal.pntd.0003160)
103. Morters MK, McNabb S, Horton DL, Fooks AR, Schoeman JP, Whay HR, Wood JLN, Cleaveland S. 2015 Effective vaccination against rabies in puppies in rabies endemic regions. *Vet. Rec.* **177**, 150–154. (doi:10.1136/vr.102975)
104. Cleaveland S, Lankester F, Townsend S, Lembo T, Hampson K. 2014 Rabies control and elimination: a test case for One Health. *Vet. Rec.* **175**, 188–193. (doi:10.1136/vr.g4996)
105. Pike J, Bogich TL, Elwood S, Finnoff DC, Daszak P. 2014 Economic optimization of a global strategy to reduce the pandemic threat. *Proc. Natl Acad. Sci. USA* **111**, 18 519–18 523. (doi:10.1073/pnas.1412661112)
106. Kümpel NF, Cunningham AA, Fa JE, Jones JPG, Rowcliffe JM, Milner-Gulland EJ. 2015 *Ebola and bushmeat: myth and reality*. NWFP Update 5: Bushmeat. Rome, Italy: FAO. See http://forestry.fao.msgfocus.com/q/1bqqKZHedDwdxkXJuzD/wv.
107. Müller MA *et al.* 2007 Coronavirus antibodies in African bat species. *Emerg. Infect. Dis.* **13**, 1367–1370. (doi:10.3201/eid1309.070342)
108. Drexler JF *et al.* 2009 Henipavirus RNA in African bats. *PLoS ONE* **4**, e6367. (doi:10.1371/journal.pone.0006367)
109. Weiss S *et al.* 2012 Hantavirus in bat, Sierra Leone. *Emerg. Infect. Dis.* **18**, 159–161. (doi:10.3201/eid1801.111026)
110. Drexler JF *et al.* 2012 Bats host major mammalian paramyxoviruses. *Nat. Commun.* **3**, 796. (doi:10.1038/ncomms1796)
111. Yang Y, Du L, Liu C, Wang L, Ma C, Tang J, Baric RS, Jiang S, Li F. 2014 Receptor usage and cell entry of bat coronavirus HKU4 provide insight into bat-to-human transmission of MERS coronavirus. *Proc. Natl Acad. Sci. USA* **111**, 12 516–12 521. (doi:10.1073/pnas.1405889111)
112. Ge X-Y *et al.* 2013 Isolation and characterization of a bat SARS-like coronavirus that uses the ACE2 receptor. *Nature* **503**, 535–538. (doi:10.1038/nature12711)
113. Menachery VD *et al.* 2015 A SARS-like cluster of circulating bat coronaviruses shows potential for human emergence. *Nat. Med.* **21**, 1508–1513. (doi:10.1038/nm.3985)