



Emerging and Re-Emerging Disease of Wildlife Threat to Biodiversity and Human Health

Kranti Sharma^{1*}, Vandana Bhagat Namita Shukla² and Mukesh Sharma²

¹Kamdenu Panchgavya Research and Extension Centre, Anjora Durg, India

²Daushree Vashudev Chandrakar Kamdhenu University, Anjora Durg, India

***Corresponding Author:** Kranti Sharma, Assistant Professor, Kamdhenu Panchgavya Research and Extension Centre, Anjora Durg, India.

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Abstract

The past two decades have seen the emergence of pathogenic infectious diseases such as acquired immunodeficiency syndrome, multidrug-resistant tuberculosis, and tick-borne diseases, which represent a substantial global threat to human health. Other contributing factors may include habitat changes caused by humans and resource depletion, causing the displacement of traditional wild hosts. These new animal and human health changes will require innovative measures to improve vigilance. International hazard, identification, risk communication and risk management strategies will become increasingly important as new diseases emerge in the future. Organizations such as the World Organization for Animal Health (OIE), the Food and Agriculture Organization of the UN and the World Health Organization will have a decisive role to play in the detection and management of emerging diseases.

Keywords: Wild Life; Global; International Hazard; Disease Emerge; Organizations; Animal Health

Introduction

There are huge numbers of wild animals distributed throughout the world and the diversity of wildlife species is immense. Each landscape and habitat has a kaleidoscope of niches supporting an enormous variety of vertebrate and invertebrate species, and each species or taxon support an even more impressive array of micro and macro parasites. Infectious pathogens that originate in wild animals have become increasingly important throughout the world in recent decades, as they had substantial impacts on human health, agricultural production, wildlife-based economies and wildlife conservation. Infectious diseases have continuously affected human health and there has been a relentless appearance of various infectious disease outbreaks reported, including the plague that scourged Europe during the Middle Ages [1]. In the twentieth century, by increasing public health knowledge and interventions, the burden of infectious diseases was reduced, particularly in more

developed countries [2]. Furthermore, the later industrialization and urbanization brought great improvements in sanitation, house structural development (such as windows screened with netting), and vector control. These measures collectively ameliorated the transmission rates by reducing contact with particular infectious agents [3]. In addition, the discovery of penicillin in 1928, as well as the development of various vaccines, ushered the era of treatment and prevention in public health, significantly contributing to the eradication of certain infectious diseases [4].

Most of them linked to the sharp and exponential rise of global human activity. Among these causal factors are the burgeoning human population, the increasing frequency and speed of local and international travel, the increase in human assisted movement of animals and animal products, changing agricultural practices that

favors the transfer of pathogens between wild and domestic animals, and range of environmental changes that alter the distribution of wild hosts and vectors and thus facilitate the transmission of infectious agents. Two different patterns of transmission of pathogens from wild animals to humans are evident among these emerging zoonotic diseases. In one pattern, actual transmission of the pathogens to human is a rare event, but once it has occurred human to human transmission maintain the infection for some period of time or permanently. Some examples of pathogens with this pattern of transmission are human immune deficiency virus/acquired immunodeficiency syndrome, influenza A Ebola virus and severe acute respiratory syndrome. In the second pattern, direct or vector-mediated animal to human transmission is the usual source of human infection. Wild animal populations are the principal reservoirs of the pathogens and human to human disease transmission is rare. Examples of pathogens with this pattern of transmission include rabies and other lyssaviruses, Nipah virus, west Nile virus, Hanta virus and the agents of lyme borreliosis, plague, tularemia, leptospirosis and ehrlichiosis. These zoonotic diseases from wild animal sources all have trends that are rising sharply upwards [5].

Disease threat to wild animal

Prior to 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 noninfectious diseases had been widely recognized as important drivers of species declines (e.g. D.D.T poisoning of raptors [6,7], only a small number of researchers investigated infectious diseases as a factor in, often convert, wildlife population regulation [8,9].

The role of infectious diseases in mass mortality events or population declines was often considered controversial or secondary to other factors [10], and their role in species examinations often disputed [11,12]. 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 infections [13].

Parallel between human and wildlife EIDs extend to early human colonization of the globe and the dissemination of exotic pathogens. In the same way that Spanish conquistadores introduced smallpox and measles to the Americas, the movement of domestic and other animals during colonization introduced their own suite of pathogens. The African rinderpest panoptic of the late 1880s and 1890s is paradigm for the introduction, spread, and impact of virulent exotic pathogens on wildlife populations [4,6]. This highly pathogenic morbillivirus disease, enzootic to Asia, was introduced in to Africa in 1889. The panoptic front traveled 5000 km in 10 years, reaching the Cape of good Hope by 1897, extirpating more than 90% of Kenya's buffalo population and causing secondary effects on predator's populations and local extinction of the tsetse fly. Population of some species remain depleted and the persistence of rinderpest in eastern Africa continues to threaten bovid populations.

Pandemic of cholera, influenza, and other diseases seriously impact human populations. Such clear cut panzootic outbreaks of diseases in wildlife are probably prevents, but a lack of awareness and reporting, particularly during the earlier decades of European expansion. Almost certainly belies their true extent.

Common causal theme

The increasing number of wildlife EIDs may reflect increasing vigilance, but parallels between causal factors driving the emergence of human and wildlife EIDs suggest that this trend is valid [14]. Disease emergence most frequently results from a change in ecology of host pathogen or both [15]. Human population expansion has driven the emerges of EIDs via increasing population density, especially in urban areas (dengue, cholera) and encroachment in to wildlife habitat (Ross River virus disease) [16]. This encroachment may have been a key factor in Africa for the global emergence of Marburg and Ebola viruses and human immunodeficiency virus (HIV) [17].

Pressure of human encroachment on shrinking wildlife habitat also cause increased wildlife population densities and the emergence of wildlife EIDs [18]. The international movement of live-

stock and modern agricultural practices and bovine spongiform encephalitis (BSE) in Europe. Similar situations occur in wildlife populations managed either *in situ* or in captivity. The extent of *in situ* management may be substantially underestimated. Recent analysis suggests that 15,000 tons of pea-nuts are fed annually to United Kingdom garden birds. This form of provisioning has led to the emergence of infection by *Salmonella typhimurium* DT 40 and *Escherichia coli* 086: k61 in Britain and *Mycoplasma gallisepticum* in the United States because of a high density and diversity of birds at feeding stations [19]. The maintenance of brucellosis in bison in the Grand Teton National park (United States) is related to the presence of disease in managed sympatric elk [20]. Even changes in arable farming may lead to disease emergence, such as the shift in agriculture from the eastern United State to the Midwest, which allowed reforestation of New England, providing the conditions for Lyme disease emergence [21]. Anthropogenic global climate change is likely to cause major changes to the geographic range and incidence of arthropod-borne infectious disease. Expansion of mosquito vector geographical ranges has been proposed to explain the reemergence of malaria and dengue in South America, Central Africa and Asia during the 1980s and 1990s [22]. Similarly, the biting midge vector for African horse sickness (AHS) and blue tongue has recently invaded Europe and North Africa [23].

Spill-over and “Spill-Back”

The transmission of infectious agents from reservoir animal populations (often domesticated species) to sympatric wildlife, termed spill-over, underpins the emergence of a range of wildlife EIDs. Spill-over is a particular threat to endangered species, because the presence of infected reservoir hosts can lower the pathogen's threshold density and lead to local (population) extinction [24-26]. Population of Africa wild dog (*Lycaon ictus*) has been declining since the 1960s. This species is now endangered and, with a fragmented population of less than 5000, is susceptible to stochastic events such as disease outbreaks. Wild dogs became extinct in the Serengeti in 1991, concurrent with epizootic canine distemper in sympatric domestic dogs [27,28]. Rabies has also caused mortality of wild dogs, and a viral variant common in sympatric domestic dogs has been identified from one such incidence. The geographic expansion of human populations and the consequent

encroachment of domestic dog carriers may explain the emergence and impact of Rabies in wild dogs in the Serengeti [29].

Spill over epizootic outbreaks represent a serious threat both to wildlife and via. Reverse spill-over (“spill-back”), to sympatric populations of susceptible domesticated animals. Brucellosis was probably introduced in to American with cattle. In Yellowstone National Park (United States), the presence of disease in elk and bison is considered a potential threat to domesticated cattle grazing at park boundaries [30]. Other examples of spill-over infection include Sarcastic mange in foxes (Europe) and wombats (Australia) and bovine's tuberculosis (global). The latter threatens to spill back to domestic livestock [24,25] and ultimately, to humans.

Emerges Owing to host or parasite translocations

The translocation of wildlife for conservation, agriculture and hunting occurs on a global scale, with an inherent risk of exposure of wildlife species to exotic infectious agents [24,25]. Translocation and introduction of animals to new geographic regions correspond to increased human global travel and commerce as underlying factors for infectious disease emergence [14,31]. The translocation of fish and possibly amphibians may have driven the emergence of rana virus epizootics as threats to freshwater fish and wild herpeto fauna. Similarly, a rabies epizootic in the mid-Atlantic region of the United State resulted from translocation of infected raccoons from a southeastern U.S. enzootic focus. The introduction of potentially hosts in to new graphic areas without co-introduction of pathogen can also result in disease emergence. For examples, varroosis a disease of honeybees caused by the mite *Varroa jacobsoni*, spread globally (except Australia) After the European honey-bee except (*Apis mellifera*) was introduced in to Asia.

This type of emergence is a particularly concern to conservation program me that bring allopatric endangered species in to close proximity or that alter basic host-parasite variables such as population density and structure [31,32]. Molecular analysis of a newly discovered herpes virus associated with disease in captive elephants indicate that a normally benign herpesvirus of the African elephant can be lethal to its Asian cousin [32]. Zoo animals in the United kingdom to food contaminated by the BSE agent. Scrapie

like spongiform encephalopathy thought to result from exposure to the BSE agent have been confirmed in 58 zoo animals of 17 species [4]. Recommendation have been published to preempt the potentially disastrous consequences to wildlife, agriculture and public health should BSE be introduced in to free-living wildlife [33].

Risk factors for disease emergence in conservation programme

For example, epizootic toxoplasmosis, with high mortality rate, has occurred in captive lemurs, New world primates and Australian marsupials. These animals evolved in the absence of *Toxoplasma Gondii*, and only recently, after human intervention (translocation), they have been exposed to the parasite. The feeding of contaminated neonates mice to captive callitrichid primates (marmosets and tamarins) led to the emergence of callitrichid hepatitis caused by a variant of the zoonotic pathogen, lympho-cytic choriomeningitis virus (LCMV). The zoonotic risk of LCMV is mirrored by the transfer of pathogens from humans to wildlife species. For example, measles contracted from humans threatens wild mountain gorillas habituated to tourists and polio virus has killed chimpanzees in the Gombe National Park in Tanzania.

Captive breeding programme aims to maintain genetically viable, healthy populations for subsequent release in to the wild. The potential transfer of pathogens in to previously unexposed wild populations in often sensitive, protected areas represents a serious challenge to conservation efforts. This can impinge on release programs even when no apparent disease is observed. The release of captive reared field crickets (*Gryllus campestris*) was suspended in England after the discovery of unidentified, potentially exotic parasites that were not associated with ill-health, but that posed a diseased threat to sympatric wild species at release sites [12].

The loss of host-specific parasites from endangered species in captive breeding programme is also a substantial threat to biodiversity conservation. In addition to ethical obligation to conserve parasite assemblages along with their more favored hosts [12]. The maintenance of established host-parasite relation may be im-

portant for the overall well-being of the host species both at an individual level (maintenance of immunity) and at a population level (maintenance of genetic diversity) [24-26].

Emergence without overt Human involvement

Correlation between emergence of human disease such as (cryptosporidiosis, haemorrhagic fever, cholera and malaria) and weather pattern (flooding the EL Nino Southern Oscillation (ENSO)) are common [34]. These patterns may also causes changes in parasite prevalence and intensity and host mortality rates in wild animals such as the 3 to 4 year cycles of population crashes in feral sheep on the St. Kilda archipelago, Scotland and major epizootics of AHS every 10 to 15 years in South Africa. There is increasing evidence that the frequency and severity of these events are influenced by anthropogenic effects on climate.

Drivers of disease emergence

There likely to be multiple causes of novel disease emergence, but the human mediated transport of pathogens (often infected host) or vectors across geographical or ecological boundaries, a process termed "pathogen pollution" has been identified as a major driver of this in wildlife and also in plants [35]. 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. salamaandrorivans*. 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 all opathiclineages. 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 [36].

Almost 20 years since the threats to conservation and human health that wildlife EIDs represents was first highlighted, there has been little effort to put in place policies to reduce risk. Detecting and preventing the importance of infected hosts is widely used

to prevent importance 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 person arriving at their international borders, particularly during human pandemics. The World Health organization 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 Organization (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 phyto sanitary measures are 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 Organization for Animal health (OIE) list of disease of international importance. Although the OIE has a remit of protecting biodiversity, only two pathogens are listed for this purpose: B. dendrobatidis and Ranavirus. Most centuries, therefore, use import controls to only protect against domestic animal diseases of obvious public health or economic importance even when OIE-listed.

Policies for prevention and control

In addition to the high costs of dealing with endemic zoonosis, such as rabies, emerging and re-emerging zoonoses can have substantial economic impacts. The cost implications of zoonotic EIDs were highlighted by various author. 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, ranges from 5 dollars to 50 dollar billion, while the true cost of most EIDs have never been estimated [37].

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