

Wild great apes as sentinels and sources of infectious disease

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Abstract

Emerging zoonotic infectious diseases pose a serious threat to global health. This is especially true in relation to the great apes, whose close phylogenetic relationship with humans results in a high potential for microorganism exchange. In this review, we show how studies of the microorganisms of wild great apes can lead to the discovery of novel pathogens of importance for humans. We also illustrate how these primates, living in their natural habitats, can serve as sentinels for outbreaks of human disease in regions with a high likelihood of disease emergence. Greater sampling efforts and improvements in sample preservation and diagnostic capacity are rapidly improving our understanding of the diversity and distribution of microorganisms in wild great apes. Linking non-invasive diagnostic data with observational health data from great apes habituated to human presence is a promising approach for the discovery of pathogens of high relevance for humans.

Keywords: Chimpanzee, disease ecology, emerging infectious diseases, gorilla, zoonoses

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Introduction

Emerging infectious diseases frequently originate from zoonotic transmission events implicating wildlife reservoirs [1], with the highest risk for emergence being seen in tropical countries with high biodiversity and low infrastructure [2]. Consequently, the characterization of microorganisms infecting wildlife in the tropics is central to the development of global health surveillance systems that are capable of identifying putative pathogens before they enter the human population [1,2]. However, where should we start? Technically, it is possible to broadly screen various wildlife and vector species for known and unknown pathogens (e.g. viruses in mosquitoes [3], bats, or rodents [4]). But what does the identification of various (new) microorganisms tell us? Are they of pathogenic importance for humans? For some microorganisms, we may be able to assume zoonotic and pathogenic potential, owing to relatedness to known pathogens, but a true understanding of the risk associated with each microorganism requires comprehensive study of a large number of

well-characterized samples from healthy and symptomatic humans from affected areas. This time-consuming and expensive process often yields results only after a pathogen has emerged from a wildlife source and become established in the human population. Fortunately, we can identify hosts with a high potential for carrying microorganisms of importance for humans, on the basis of host demography, ecology, and behaviour. Focusing on such high-potential hosts will improve our chances of understanding a microorganism with emergence potential well before it becomes established in humans [5]. Ideally, candidates should be high-density animal species exhibiting extensive niche overlap with humans in tropical regions, such as rodents or bats.

Another criterion for selecting target species is to focus on hosts with close evolutionary relatedness to humans. Close relatedness between species (including primates) has been shown to increase the likelihood of cross-species transmission events [5,6], and closely related species are also more likely to develop similar clinical signs when infected with specific pathogens [7].

Therefore, wild great apes are promising candidates for screening for potential new zoonotic agents. First, they are the closest evolutionary relatives of humans, facilitating microorganism transmission, which has, for example, led to the emergence of human immunodeficiency virus-1 [8]. Second, it has been shown that great apes suffer from acute and chronic diseases of high importance to humans, such as those caused by Ebola viruses and simian immunodeficiency viruses (SIVs), respectively [9,10], and can therefore serve as 'indicator species' for pathogens of clinical importance for humans. Third, there is intense exposure of humans to body fluids and tissues of great apes, as all non-human primate species are commonly hunted in West and Central Africa [11].

However, all great ape species are highly endangered, and invasive studies including anaesthesia or even euthanasia, such as those performed on bats or rodents, are ethically impossible [12]. The only invasive samples that it is ethically possible to collect are samples obtained from animals found dead (in bushmeat markets or in the wild) or samples collected in the course of interventions necessary to save the lives of great apes (e.g. hunting snare removal) [7]. In order to study microorganisms in living wild great apes, methods have been developed to allow the detection of microorganisms and antibodies in materials that can be collected non-invasively from these animals without disturbing them. Such investigations rely mostly on faeces, but in some cases also on urine or food wadges [7,13,14], and have led to the identification of at least 12 families and 17 genera of viruses infecting wild great apes (Table 1).

In this review, we summarize the current knowledge regarding the diversity of enzootic and non-enzootic microorganisms of African wild great apes (almost no information is available on microorganisms of Asian wild great apes), with a special emphasis on great apes as sentinels for emerging infectious diseases. We also highlight important gaps in our knowledge and areas in urgent need of further investigation. We did not consider data obtained from captive or semi-captive wild ape populations, which is not to say that they are uninformative, but rather that they are more likely to be biased [15]. Additionally, a comprehensive overview of this field of research would be beyond the scope of a review, so we have restricted our overview to the best-studied microorganisms at the ape-human interface.

Great Ape Enzootic Microorganisms

Most microorganisms identified in wild great apes appear to be enzootic (that is, they persistently infect wild great ape populations), and are not known to be associated with acute

disease. This is nicely exemplified by viruses such as adenoviruses, hepadnaviruses, herpesviruses, parvoviruses, picornaviruses, polyomaviruses, and retroviruses (Table 1), and also applies to most gastrointestinal helminths, protozoa, and bacteria [7,16,17].

In contrast, nothing is known about enzootic pathogens spreading through populations and causing acute disease or death. For humans, various acute disease-causing endemic pathogens are known, e.g. smallpox or measles viruses. One may argue that this is simply because of a lack of surveillance in wild ape populations; however, habituated communities of wild great apes are distributed across each species range, and some of these communities have been under continuous observation for decades, so massive die-offs would not have gone unnoticed. A more likely explanation is that great ape demography (i.e. low-density, fragmented populations) is not compatible with the sustained circulation of such acute disease-causing pathogens. Known acute diseases in wild great apes originated either from other species (Ebola) [10], the environment (anthrax) [18], or humans (respiratory diseases) [19] (see below).

Great Ape Non-enzootic Microorganisms

A number of microorganisms infect wild great apes by environmental means, at least in the initial phases of what might later become an epizootic sustained by within-species transmission. Such infections occur through the consumption of contaminated food items, direct (e.g. biting) or indirect (e.g. aerosols) contact with other species, or vector bites (arthropods).

Microorganisms acquired through contaminated food

Obviously, all wild great apes are exposed to foodborne microorganisms, including other animal microorganisms found on vegetation as a consequence of contamination by infected animal fluids. Although diets typically exhibit significant variation at multiple scales (e.g. within subspecies or species, within regions, or over seasons for a specific population), the most striking differences are seen between the omnivorous genus *Pan* (chimpanzees and bonobo) and the herbivorous genus *Gorilla*. Chimpanzee and bonobo diets consist primarily of fruit and other plant parts, but animals, including other non-human primates, are eaten on occasion, with interesting regional differences in diet composition [20,21]. Thus, chimpanzees and bonobos are more likely to acquire microorganisms from other non-human primate species than are gorillas, which have never been observed to consume non-human primates or other animals in the wild [22,23].

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Both lethal and non-lethal microorganisms have been shown to be acquired from contaminated fruits or vegetation in wild great apes. For example, studies in Uganda have demonstrated transmission of non-pathogenic *Escherichia coli* from humans and goats to wild gorillas, as well as from humans to wild chimpanzees, probably simply through overlap and contamination of habitat [17,24]. Also, phylogenetic and anecdotal evidence exists for similar transmission of adenoviruses [24]. The clinical relevance of such infection remains unknown.

However, the examples of Ebola haemorrhagic fever and anthrax demonstrate the degree to which infectious diseases can impact on wild ape populations. Ebola viruses have caused massive die-offs of gorillas and chimpanzees [25,26]. The most likely sources of Ebola and other filoviruses are fruit bats [27] contaminating fruits with saliva or other bodily excretions [10].

Anthrax has also been identified as a cause of death for wild chimpanzees [28] and gorillas (*G. gorilla*) [18]. Interestingly, the bacterial agent isolated from these infections falls outside the known variation of *Bacillus anthracis*, and instead is a member of the *Bacillus cereus* family; however, it encodes all factors needed to cause the disease 'anthrax'. Therefore, this strain was termed *B. cereus* biovar *anthracis* [29,30]. All members of the *Bacillus* group are classically acquired through point-source infections via ingestion of contaminated vegetation, soil, or animal carcasses. As great apes do not scavenge, such infections were probably acquired from contaminated fruits or leaves [18].

In addition to vegetation-borne microorganisms, wild chimpanzees and bonobos are exposed to microorganisms of their prey. Among ape-hunted species, monkeys are of the greatest interest, both as the most frequently consumed prey and as the prey with the greatest phylogenetic proximity to great apes. Unfortunately, comprehensive investigation of pathogen transmission via predator-prey dynamics is lacking, and evidence of cross-species transmission is mainly restricted to retroviruses.

Simian foamy viruses (SFVs), which have no known clinical effect on primates, show a distinctive pattern of long-term co-speciation with primates [31]. Hence, one would presume that cross-species transmission would be theoretically easy to pinpoint by phylogenetic means. In this regard, two wild chimpanzees in the Taï National Park (Côte d'Ivoire) have shown evidence of co-infection with chimpanzee-specific SFV and the SFV of their favourite prey, the red colobus monkey (*Piliocolobus badius*) [32]. In another study, one faecal sample (out of nearly 400) from a wild chimpanzee was found to be positive for an undetermined cercopithecoid species SFV [33]. Considering the intense contact between chimpanzees and their prey [22,34], transmission of monkey SFVs to chimpanzees seems to occur at a very low rate.

Simian T-lymphotropic viruses type I (STLV-Is) also show evidence of cross-species transmission. In contrast to SFVs, STLV-Is show low host specificity [35]. Consequently, sympatric host species tend to harbour STLV-Is whose sequences are interspersed in phylogenetic analyses. For example, chimpanzees from the Taï National Park (Côte d'Ivoire) are infected with STLV-I strains belonging to three distinct clades, one probably constituting the 'original' chimpanzee STLV-I clade and the others comprising STLV-I strains from the sympatric red colobus and sooty mangabeys (*Cercocebus atys*) [36]. Relatively frequent cross-species transmission seems to occur for this virus, but the slow evolutionary rates of STLV-Is [37] prevent exact determination of the frequency of transmission events.

Finally, SIVcpz has been shown to stem initially from inter-species transmission from monkeys to chimpanzees, as this virus is a recombinant of two different monkey SIVs [38]. However, exposure to prey SIVs does not automatically lead to infection, as demonstrated by the absence of red colobus SIVs in wild chimpanzees, who prey heavily on SIV-infected red colobus monkeys [34].

Microorganisms acquired through aerosols

To date, no respiratory pathogen has been conclusively shown to enzootically infect wild great apes. All published evidence of aerosol-transmitted respiratory microorganisms have involved either human respiratory syncytial virus or human metapneumovirus. These viruses were transmitted in settings where ape-human overlap was extensive, such as tourism (gorilla) and research projects (chimpanzees) [13,19,39]. Furthermore, *Streptococcus pneumoniae* and *Pasteurella multocida* of undetermined origin have been found in several cases as co-infections with the viral infections mentioned above [40–42].

Because it is likely that respiratory pathogens can be spread easily, it will be important to determine whether wild great apes are hosts or intermediate amplifiers of any of these pathogens.

Microorganisms acquired through aggressive interactions

The possibility of transmission of microorganisms through aggressive interactions between great apes (such as biting) has rarely been investigated. However, retroviral transmission from chimpanzees to gorillas is suggested by SIVgor nesting within the diversity of SIVcpz [43,44]. Another example suggestive of transmission via gorilla-chimpanzee aggressive interactions is provided by cytomegalovirus strains in chimpanzees and gorillas each nesting within the diversity of the other in two different clades, indicating transmission in both directions [45].

Microorganisms acquired through arthropod vectors

The only vector-borne microorganism known from wild great apes is *Plasmodium*. Various malarial parasites infect chimpanzees and gorillas [46–48]. The effect of plasmodial infections on wild great ape health remains unknown, but current efforts to integrate non-invasive faecal rectal temperature measurements [49] with observational and non-invasive diagnostic data [48] may yield interesting results.

Transmission of Great Ape Microorganisms to Humans

Because of their generally higher prevalence, enzootic microorganisms are likely to have a higher risk of transmission than non-enzootic ones. To date, the only known example of a non-enzootic great ape-borne pathogen is Ebola virus, for which wild great apes can serve as intermediate amplifiers [10]. This contrasts with frequent reports of great ape SFV and STLV-I transmission to humans (notably in high-risk populations such as great ape-bitten hunters; reviewed in [50]). Also, among enzootic microorganisms, prevalence seems to be a key parameter. For example, transmission of great ape SIVs, which are found at only a low prevalence, with few communities infected [51], to humans appears to be exceptionally rare [8]. Therefore, large-scale mapping of the prevalence of wild great ape enzootic microorganisms will be required to assess the risk of transmission to humans. Obviously, prevalence is not the only determining parameter, as shown by the example of plasmodia, which are highly prevalent in great apes but were apparently only transmitted once to humans, giving rise to *Plasmodium falciparum* [47].

Transmission mode will also be important in defining exposure and consecutive transmission risk for different pathogens. For example, the transmission of many microorganisms requires blood-to-blood contact, such as can occur in bushmeat hunting and butchering. However, these bushmeat practices are not homogeneously encountered throughout Africa. Conversely, transmission of vector-borne microorganisms such as plasmodia requires the presence of appropriate vectors. Other microorganisms could also be transmitted as a simple consequence of habitat overlap (e.g. via faecal–oral transmission; Table 1).

Summary

Despite minimal surveillance for microorganisms in wild great ape populations to date, these species are associated with a number of pathogens that are highly relevant to

humans, including retroviruses, filoviruses, plasmodia, and *B. cereus* biovar *anthracis*. However, transmission can occur in both directions, as demonstrated by the examples of human respiratory viruses and gastrointestinal bacteria being transmitted from humans to wild great apes [13,17,19,39]. Only indirect evidence of transmission exists for other pathogens with high potential to threaten wild ape populations, such as measles viruses and scabies [12].

Future studies integrating long-term observational health data with non-invasive diagnostics for great apes will be essential in improving our understanding of the epizootology and biology of pathogens in great apes. The example of SIVcpz nicely illustrates the benefits of such monitoring of habituated wild great apes. Like other SIVs, SIVcpz was long considered to be non-pathogenic in its natural host. Only through longitudinal sampling linked to observational health data was its pathogenicity in chimpanzees revealed [9,52]. Such observational health data are critically needed to understand the pathogenic potential of other great ape pathogens, such as *Plasmodium*. In addition, studies of wild chimpanzees and bonobos are of particular interest for the study of cross-species transmission events associated with predator–prey interactions and the possible resulting recombination and reassortment events that may lead to altered properties of the microorganisms (Table 1). More generally, longitudinal studies may allow for the investigation of adaptive processes associated with pathogen host switches [5,53].

Finally, great apes may give us the unique opportunity to determine the original diversity of hominin microbiomes (including our own), because, in contrast to humans, they continued to live isolated in their pristine habitats, unexposed to our global microbiome. The protection of the great apes and their habitat is a pre-condition for such studies.

Transparency Declaration

No conflict of interest to be declared.

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