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### Cover Page Footnote

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CRIB NOTES

# Disease Introduction by Aboriginal Humans in North America and the Pleistocene Extinction



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## ABSTRACT

*While overhunting and climate change have been the major hypotheses to explain the late-Pleistocene New World megafaunal extinctions, the role of introduced disease has only received brief attention. Here, we review pre-Columbian diseases endemic to aboriginal Americans and evaluate their potential to cause large-scale mortality in Pleistocene mammals. Of the probable communicable diseases present in pre-Columbian times, we regard anthrax and tuberculosis as viable candidates. These two diseases demonstrate characteristics that could have made them deadly to immunologically naïve populations. Introduced disease, as a primary cause or interacting with overhunting and climate change, could have contributed to the decline and extirpation of Pleistocene megafauna.*

## INTRODUCTION

The cause of the Pleistocene megafaunal extinction in the Americas has been debated for decades with two major hypotheses emerging as likely candidates: overhunting by aboriginal Americans (i.e., overkill hypothesis, Koch and Barnosky 2006; Martin and Wright 1967) and climate change (Barnosky et al. 2004), or interaction of these two processes (Haynes 2009). However, both of these hypotheses are criticized as being inadequate to explain the extinction pattern. The climate variation at the end of the Pleistocene was similar to previous climatic shifts that did not correspond to major mammalian extinctions (Koch and Barnosky 2006). While the overkill hypothesis has become more accepted in recent times, it has been argued that small populations of technologically limited humans

would have had difficulty exterminating species with continental-wide distributions. In addition, there is archaeological evidence for hunting of only six extinct genera of Pleistocene mammals (Koch and Barnosky 2006, but see Surovell and Waguespack 2008), while the extinction encompassed over 100 genera.

An additional factor that has only been briefly discussed, and in some cases dismissed entirely, is introduction of disease to the New World via human colonization, referred to in some publications as hyperdisease (Koch and Barnosky 2006; Lyons et al. 2004; MacPhee and Marx 1997). Aboriginal populations in the Americas were not free of communicable diseases in pre-Columbian times (Martin and Goodman 2002), and it is likely they introduced diseases that could affect wild animal

populations. As we have seen in recent times, the human introduction of disease can have severe effects on various species. For example, Chytridiomycosis, a disease caused by *Batrachochytrium dendrobatidis* has apparently extirpated or caused substantial declines of 30% of frog species across the globe (Vredenburg et al. 2010). Other examples include white-nose syndrome (*Pseudogymnoascus destructans*), currently devastating North American bat species (Bleher et al. 2009), avian malaria (van Riper 1986), introduced fungal infections that have severely reduced tree species in North America (Anagnostakis 1987; Brasier 1991), and possibly the extinction of Neanderthals with the arrival of modern humans in Europe (Houldcroft and Underdown 2016). The introduction of novel diseases and subsequent declines in large mammals has also been seen in historical times with the emergence of the rinderpest virus (*Morbillivirus*, Dobson 1995).

In this paper, we review the potentially human-introduced diseases that were present before the time of New World European settlement, and identify those that could have caused substantial mortality in native mammals during the Pleistocene, animals that presumably would have been immunologically naïve. Of the ten diseases that were present or suspected to be present in Pre-Columbian times (Martin and Goodman 2002), we find one candidate that we assess as a high risk and one that we assess as a moderate risk to late-Pleistocene megafauna (Table 1).

## LIST OF DISEASE CANDIDATES

### 1. Anthrax (*Bacillus anthracis*)

Anthrax is a highly contagious disease that causes high mortality in many animals, including humans, and is spread by the ingestion or inhalation of spores and ingestion of infected flesh. Grazing animals are particularly susceptible, especially herding species. Humans frequently spread anthrax via infected animal products, particularly skins (Marston et al. 2011). Humans traveling across the Bering Land

Bridge during the Pleistocene would have certainly carried large numbers of animal skins for clothing, some of which could have been infected with anthrax spores. Although anthrax evolved in Eurasia, its appearance in the North America corresponds to definitive human colonization and spread, about 13,000 years BP, and predates the extinction event by less than 2000 years (Kenefic et al. 2009).

### 2. Tuberculosis (*Mycobacterium tuberculosis*)

Tuberculosis was clearly present in Pre-Columbian human populations (Martin and Goodman 2002), although the origin and specific strain is a subject of debate (Bos et al. 2014). *Mycobacterium* DNA and associated skeletal deformities have been found in both North and South American human populations dating before European contact (Ortner 2003; Salo et al. 1994). Human tuberculosis also infects many species of animals, and of particular interest for the Pleistocene, proboscideans (i.e., elephants). In extant elephants, tuberculosis is a highly contagious disease with substantial mortality rates (Mikota et al. 2001). The disease is also easily spread between humans and elephants through respiratory aerosols (Lacasse 2007). Since Pre-Columbian humans clearly hunted elephants (Haynes 2002; Waters et al. 2011), and were therefore in close contact, it is plausible that they could have spread *Mycobacterium* to wild populations. Bone lesions indicative of tuberculosis have been found in late-Pleistocene mastodons (*Mammuthus*, Rothschild and Laub 2006), gomphotheres (de Souza Barbosa 2013), and both bone lesions and *Mycobacterium* DNA in the extinct bison (*Bison antiquus*, Rothschild et al. 2001; Rothschild and Martin 2003). In these cases, a large proportion of skeletal remains showed evidence of *Mycobacterium* infection, over 50% in *Mammuthus* (Rothschild and Laub 2006). *Mycobacterium* also affects many other animals (Steele 1980, Table 1), so it could have become widespread throughout American ecosystems.

However, the evidence described above is equivocal. DNA evidence indicates that pre-Columbian

**TABLE 1. Human pre-Columbian diseases endemic to the New World, non-human fauna affected, origin date, and assessment of risk to Pleistocene fauna.**

Disease	Fauna Affected	Origin Date in New World	Assessment of Risk
<i>Bacillus anthracis</i> (Anthrax)	Most mammals	13,000 B.P.	High
<i>Mycobacterium tuberculosis</i> (Tuberculosis)	Ungulates, Proboscideans, Rodents, Canids Bird, Reptiles	17,000 B.P.	Moderate
<i>Bordetella pertussis</i> , <i>B. paraptussis</i> , <i>B. bronchiseptica</i> (Pertussis, Whooping cough)	Many mammals	~15,000 B.P.	Uncertain
<i>Francisella tularensis</i> (Tularemia)	Rodents, Lagamorphs	pre-Pleistocene	Low
<i>Sarcomastigophora</i> (Giardia)	Rodents, Ungulates Canids	Unknown, probably ancient	Low
<i>Orthohepadnavirus</i> (Hepatitis B)	Primates	~15,000 B.P.	Low
<i>Simplexvirus</i> (Herpes)	Primates	~15,000 B.P.	Low
<i>Enterovirus c</i> (Polio)	Primates	~15,000 B.P.	Low
<i>Lyssavirus Rabies</i>	Mammals (mostly carnivores)	uncertain, probably post-Columbian	Low/None
<i>Treponema pallidum</i> (Syphilis)	Primates	~15,000 B.P.	None

populations were infected with a strain of *Mycobacterium* most closely related to seals and sea lions (Bos et al. 2014), and may have been transmitted to coastal human populations and not brought directly from Eurasia. With the exception of proboscidean and bovids, there is little evidence of bone lesions consistent with *Mycobacterium* in other large Pleistocene mammals (Rothschild and Martin 2006). Furthermore, bone lesions, although similar to tuberculosis, can also be caused by other infections, especially fungal (HersHKovitz et al. 1998). However, the *Mycobacterium* variety isolated from late-Pleistocene bison most closely matches human forms of tuberculosis, specifically *M. africanum* and *M. tuberculosis* (Rothschild et al. 2001). Therefore, the role of tuberculosis remains inconclusive, but should be studied further.

### **3. Pertussis (*Bordetella pertussis*, *B. paraptussis*, and *B. bronchiseptica*).**

Pertussis (whooping cough) is a highly contagious bacterial disease that causes respiratory disease in humans, but relatively low mortality (Bromberg 1994). It was apparently present in populations that migrated across the Bering Land Bridge (Martin and Goodman 2002). Pertussis seems to have evolved before the evolution of modern humans (Parkhill et al. 2003), and therefore aboriginal Americans could have conceivably carried it into the New World. The modern human form has no animal reservoir (Bromberg 1994). Humans can also contract the common animal strain, *B. bronchiseptica* (Stefanelli et al. 1997), which also is the likely progenitor of the human forms of the disease (Bjornstad and Harvill

2005). If aboriginal humans carried this variety to the Americas, they could have exposed native animals to a novel disease. However, the evolution and distribution of *Bordetella* is uncertain and more research needs to be undertaken (Bjornstad and Harvill 2005, Diavatopoulos et al. 2005). We therefore view the risk of *Bordetella* Pleistocene mammals as uncertain, but a factor worthy of further study.

#### **4. Rabies (*Lyssavirus*).**

Rabies is a highly contagious viral disease that can affect all mammals and is invariably fatal to placental mammals. Researchers have suggested that rabies was present in the New World in pre-Columbian times (Vos et al. 2011), and it has been proposed as a possible candidate causing megafaunal extinctions (Lyons et al. 2004; MacPhee and Marx 1997). Molecular data indicate modern rabies strains arrived in colonial times, although the possibility of ancient bat rabies varieties in the Americas that are now extinct is suggested (Hughes et al. 2005). Furthermore, although smaller Carnivora and bats often spread rabies, it is rare in megafauna and almost never spreads through large herbivore populations. Because of its probable late arrival in the New World and low transmission among most megafauna, we view its risk to Pleistocene animals as low.

#### **5. Syphilis (*Treponema pallidum*, several subspecies).**

Syphilis, in the non-sexually transmitted form (the diseases Pinta, Bejel, and Yaws, Antal et al. 2002), was present in Native American populations in pre-Columbian times, while the sexually transmitted form was introduced by European contact. It is rare in other animals and has only been reported in other primates. We therefore consider it a low to non-existent risk to Pleistocene megafauna.

#### **6. Tularemia (*Francisella tularensis*).**

Tularemia is a bacterial infection occasionally found in humans, but in North America it most commonly

affects lagomorphs and aquatic rodents (Hornick 1994; Mörner 1992). It is spread through direct contact and via arthropod bites. It appears to have originated in North America, or at least been present for several million years (Hornick 1994), and it is therefore more likely that humans acquired it when colonizing North America, instead of introducing it to a new environment. For these reasons, we do not consider it a good candidate for causing the Pleistocene extinctions event.

#### **7. Giardia (*Sarcomastigophora*).**

Giardia is a protozoan disease that affects many mammals and birds, and is primarily spread through contaminated water (Feng and Xiao 2011). It can cause moderate to severe diarrhea, although mortality rates are low for most species. Giardia is an unusual organism, showing traits of both prokaryotes and eukaryotes, and probably evolved millions of years ago (Kabnick and Peattie 1991). Its origin in the New World is thought to be very ancient and may have had a near global distribution before the evolution of humans. The combination of ancient origin and relatively low mortality among animals infected suggests it was not a factor in the Pleistocene extinction.

#### **8. Hepatitis B (*Orthohepadnavirus*).**

Hepatitis B was present in aboriginal American populations, but is only capable of infecting other primates (Plotkin et al. 2012), so we find it unlikely to be an extinction factor.

#### **9. Herpes (*Simplexvirus*).**

*Simplexvirus* was probably present in aboriginal Americans, but only infects humans and non-human primates (Goodman 1994), and is unlikely to be an extinction factor.

#### **10. Polio (*Enterovirus C*).**

*Enterovirus C* was likely present in aboriginal



Americans that crossed the Bering Land Bridge. However, it infects only primates (Simoes 1994), and is unlikely to be an extinction factor.

### 11. *Theoretical diseases.*

We do not argue that the list described above is complete. Many other possible diseases, now extinct or never detected in the archaeological record, could have caused high mortality in Pleistocene megafauna. The first settlers of the Americas brought domestic dogs from Asia with them (Leonard et al. 2002), which could have also carried new disease to the Americas. However, most potentially lethal diseases that affect dogs and other Carnivora appear of recent evolutionary origin or were introduced at the time of European contact, including canine rabies (introduced 600 BP, Bourhy et al. 2008), parvovirus (Carmichael 2005), and canine distemper (Norrbj et al. 1985).

## DISCUSSION

We find two diseases that fit our criteria for being a possible proximate or contributing cause for the end-Pleistocene mass extinction of New World megafauna: anthrax and tuberculosis. These diseases appear to have been first introduced at the end of the Pleistocene Epoch (10-20,000 years BP), could have been easily spread by migrating humans, and can infect and cause substantial mortality in relatives of extinct megafauna. MacPhee and Marx (1997) provide four characteristics for a hyperdisease to function as a cause for an extinction: 1) reservoir species that presents a stable carrier state, 2) high potential for causing infection among susceptible species, 3) hyper-lethal affect in new hosts, and 4) ability to infect multiple species, but not seriously affect human groups. Our two candidate diseases more or less fit these characteristics, although a disease's hyper-lethal potential is difficult to determine for extinct host species.

Anthrax is a disease briefly discussed in the literature regarding its potential to affect Pleistocene megafauna. Its origin in North America has been

dated at approximately 13,000 years BP (Kenefic et al. 2009), and its origination point is from eastern Asia similar to early North American human settlers. There is general consensus that humans had colonized North America by 15,000 years BP, earlier than the evidence for anthrax presence in North America (Goebel et al. 2008). However, it is also likely that early settlers remained near coastal areas initially, and only later spread into interior North America where some of the largest populations of megafauna existed. We therefore do not argue that it spread rapidly throughout the Americas, but may have spread slowly as human populations expanded.

We find it highly possible that early humans in the Americas could have widely spread anthrax without even being directly infected, since the spores can easily be spread on animal skins (Marston et al. 2011), and this warrants further study. The spores can also survive up to 40 years, increasing their likelihood of surviving a human migration event. Although inhalation or ingestion in humans is often fatal, it is common to be exposed to spores for some time without causing active illness (CDC 2006). Anthrax is particularly dangerous in herbivorous herding animals. When animals die from anthrax and decay, spores enter the vegetation and soil and can be ingested by other grazers, perpetuating the infection (Dragon and Rennie 1995). The long survival time in soil means that infections can be easily re-established when migrating animals move in and out of an area. Anthrax also has one of the highest fatality rates in extant mammals (Shafazand et al. 1999, De Vos and Turnbull 2004), a factor likely to be further exacerbated in immunologically naïve populations. Non-herding animals (e.g., giant ground sloth, *Megatherium*) could have also been susceptible if they fed in areas inhabited by other anthrax-infected fauna.

Humans are also often a reservoir for tuberculosis since it is slow acting and typically causes mortality only after years of infection (Glickman and Jacobs 2001). Tuberculosis is highly infectious among many species of mammals. Although it is unclear about

its potential lethality to North American Pleistocene mammals, it can be particularly harmful to extant mammals (e.g., African buffalo, Michel et al. 2006). Studies suggest high prevalence in Mastodons during the late Pleistocene, with over 50% of skeletons detecting tuberculosis damage (Rothschild and Laub 2006). It has also been found in bison remains from the same time period (Rothschild et al. 2001).

We propose that disease is a plausible explanation that requires further investigation (either as the primary cause or through interactions with other proposed causes), especially for the extinction of widespread and very abundant mammals. For example, horses were extremely abundant and widespread throughout North America (Spencer et al. 2003). Yet there is only one documented case of pre-Columbian humans hunting horses (Waters et al. 2011). While climate change would have certainly affected horse populations and distributions, given that extant horses are generalists and can acclimate to a wide variety of habitats, it seems unlikely that climate change alone would cause their extinction. Because of the methods of transmission, high mortality rates, and ease of spread by humans, anthrax and tuberculosis are plausible suspects for the extinction of these animals. Other grazing animals such as camels, bovids, and saiga would have also been susceptible to these diseases.

The other diseases (besides tuberculosis and anthrax) that were endemic in aboriginal Americans (Table 1) do not appear to fit proposed criteria to cause the Pleistocene extinctions. Most are either limited to primates, or were present well before the arrival of humans in the New World. Because of co-evolutionary processes, we would not predict that diseases endemic to the New World for millions of years (e.g., *Giardia*) would cause mass extinctions.

We do not argue that the two proposed diseases were the single cause of the Pleistocene megafaunal extinction in the New World. In fact, several extinctions, especially on islands, are highly correlated

with human arrival and hunting culture (Duncan et al. 2002). However, there is no direct evidence for hunting of most Pleistocene extinct megafauna. Hunting has only been confirmed for mastodons (*Mammut* spp.), mammoths (*Mammuthus* spp.), gomphotheres (Family Gomphotheriidae), ground sloths (*Megalonyx jeffersonii*, Redmond et al. 2012), horses (*Equus* sp., 1 case), and camels (*Camelops* sp., 1 case), while over 100 species over 100kg went extinct around the time of human arrival (reviewed in Grayson and Meltzer 2002; Koch and Barnosky 2006). Whether the small number of archaeological sites demonstrating definitive hunting of megafauna is indicative of rarity, or is a consequence of preservation bias and actually evidence of widespread hunting, is a question of considerable debate (Surovell and Waguespack 2008). Considering that many of these species were very abundant (Guthrie 1968; Spencer et al. 2003; Springer et al. 2006), it would be expected that more archaeological evidence would be present if hunting was widespread (for a contrary view, see Surovell and Waguespack 2008).

Climate change was also occurring at the same time humans arrived, but that change was not dissimilar from multiple previous interglacial periods (Koch and Barnosky 2006). It should be noted that climate change and the overkill hypothesis are not mutually exclusive, since the interaction of the two factors could have increased probabilities of extinction (Haynes 2009; Koch and Barnosky 2006). We suggest that diseases could represent additional stresses on populations. Climate change causing rapid changes in habitat distribution, increased mortality from hunting, and increased mortality from disease could have been the combination of factors driving these species to extinction, while any individual factor would have been insufficient to cause extirpation.

Hyperdisease contributions to extinction might be particularly difficult to document. Novel disease can often cause high death rates and/or species extinctions upon initial introduction (e.g., Chytridiomycosis, Vredenburg et al. 2010), and



then remain in the environment at lower levels, infecting, but not causing high mortality among survivors. Therefore, current diseases may appear to be of low risk for extinction today, even if they caused large species losses in the past. However, genetic remains are likely to have survived in fossils (Rothschild et al. 2001). We urge further research into this potential extinction factor, including further searches for remnant DNA of these pathogens in the fossil remains of late Pleistocene mammals.

While disease introduction by humans is widely recognized as a current source of ecological disruption, we suggest that it could have been a source of environmental impact that extended well into the past. Human expansion and subsequent extinction events have usually been linked to direct hunting mortality. However, human interactions with their biotic environment are bound to be complex, including changes in pathogen-host ecology. The close interaction of humans with animals has often been associated with zoonotic diseases affecting human populations, while less attention has been focused on transmission from human to animals. We suggest that the impact of such interactions could have contributed to many extinctions linked to human arrival in new habitats and is an area that deserves further inquiry.

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