REPORT

Was a 'hyperdisease' responsible for the late Pleistocene megafaunal extinction?

S. Kathleen Lyons,[1]* Felisa A. Smith,[1] Peter J. Wagner,[2] Ethan P. White[1] and James H. Brown[1]

[1]*Department of Biology, University of New Mexico, Albuquerque, NM 87131, USA*
[2]*Department of Geology, Field Museum of Natural History, Chicago, IL 60605, USA*
Present address: S. Kathleen Lyons, National Center for Ecological Analysis and Synthesis, University of California – Santa Barbara, Santa Barbara, CA 93101, USA.
Correspondence: E-mail: lyons@nceas.ucsb.edu

Abstract

Numerous hypotheses have been proposed to explain the end Pleistocene extinction of large bodied mammals. The disease hypothesis attributes the extinction to the arrival of a novel 'hyperdisease' brought by immigrating aboriginal humans. However, until West Nile virus (WNV) invaded the United States, no known disease met the criteria of a hyperdisease. We evaluate the disease hypothesis using WNV in the United States as a model system. We show that WNV is size-biased in its infection of North America birds, but is unlikely to result in an extinction similar to that of the end Pleistocene. WNV infects birds more uniformly across the body size spectrum than extinctions did across mammals and is not size-biased within orders. Our study explores the potential impact of WNV on bird populations and provides no support for disease as a causal mechanism for the end Pleistocene megafaunal extinction.

Keywords

Disease hypothesis, end-Pleistocene extinctions, mammals, megafauna, size-biased extinctions, West Nile virus.

Ecology Letters (2004) **7**: 859–868

INTRODUCTION

The mammalian faunas of Australia and the New World are depauperate today. Before the arrival of humans, each continent contained an assemblage of large-bodied mammals that rivaled that of modern Africa. The New World contained numerous species of mastodons, ground sloths, camels, and horses; marsupials the size of rhinoceros inhabited Australia (Smith *et al.* 2003). Approximately, 12 000 years ago in the New World (Fiedel 1999; Thorne *et al.* 1999) and 46,000 years ago in Australia (Roberts *et al.* 2001), the largest species in each fauna went extinct (Martin & Klein 1984; MacPhee 1999; Lyons *et al.* 2004). The ultimate causes of this extinction fall into three general categories: (1) environmental change, which attributes the extinction of the megafauna to changes in climate and vegetation (Guilday 1967; Graham & Lundelius 1984), (2) human predation, which attributes the extinction to hunting by newly arrived aboriginal humans (Martin 1967, 1984), and (3) disease, which attributes the extinction to diseases brought by newly arrived aboriginal humans (MacPhee & Marx 1997). The first two hypotheses have been exhaustively debated in the literature (e.g. Martin & Klein 1984; MacPhee 1999 and references therein). Here, we focus on the disease hypothesis.

The disease hypothesis attributes the extinction of large mammals during the late Pleistocene to indirect effects of the newly arrived aboriginal humans (MacPhee & Marx 1997). It proposes that humans or their commensals introduced one or more highly virulent diseases into vulnerable populations of native mammals, eventually causing extinctions. The failure of several prior immigrations of mammals into North America from Eurasia throughout the Cenozoic to yield widespread extinction necessarily implicates humans or their commensals as the disease vector. Small-bodied species are postulated to have a greater population resilience due to their life history traits (e.g. shorter gestation time, greater population sizes, etc.), causing the extinction event to be biased toward larger-sized species (MacPhee & Marx 1997).

If a disease was indeed responsible for the end-Pleistocene extinctions, then there are several criteria it must satisfy (see Table 7.3 in MacPhee & Marx 1997). First, the pathogen must have a stable carrier state in a reservoir species. That is, it must be able to sustain itself in the environment when there are no susceptible hosts available to infect. Second, the pathogen must have a high infection rate, such that it is able to infect virtually all individuals of all ages and sexes encountered. Third, it must be extremely lethal, with a mortality rate of *c.* 50–75%. Finally, it must

have the ability to infect multiple host species without posing a serious threat to humans. Humans may be infected, but the disease must not be highly lethal or able to cause an epidemic.

A major problem with the disease hypothesis is that, until recently, there was no known modern disease that met all of the criteria except perhaps rabies which has a low incidence of infection (see Table 7.4 in MacPhee & Marx 1997). Further, the chance of finding evidence of such a disease in extinct animals is slim. The recent introduction and spread of West Nile virus (WNV) (Flaviviridae: *Flavivirus* Japanese Encephalitis Antigenic Complex) in the United Sates may provide a good proxy for examining the efficacy of the disease hypothesis.

Here, we explore the hypothesis that disease caused the end Pleistocene extinctions of megafauna in North America. We begin by evaluating the utility of using WNV as a model system by comparing characteristics of the virus and its mode of infection with those required by a hyperdisease. We then evaluate two possible ways in which a disease could cause a size-biased extinction event: (1) size-biased infection, and (2) size-biased extinction risk once infected. We do this by characterizing the pattern of infection of WNV and its likely mortality schedule and comparing it to the unique size signature of the late Pleistocene mammalian extinction.

METHODS

Flaviviruses are responsible for many diseases such as dengue fever, yellow fever, encephalitis, and hemorrhagic fevers (Center for Disease Control 2004d). WNV is endemic to Africa, and was first reported in the New World in New York City in August of 1999. WNV is zoonotic, with a fairly complex life cycle. Birds are the primary vertebrate hosts; transmission occurs through feeding by infected mosquitoes. Understanding of the life cycle of WNV is complicated by the extremely diverse array of insect vectors involved in transmission. Thus far, 43 species of mosquitoes from nine genera have tested positive for WNV (Center for Disease Control 2003a).

MacPhee & Marx (1997) identified four criteria as necessary prerequisites for a hyperdisease to induce size-biased extinctions. We conducted an exhaustive search using both literature and electronic web sources to obtain data on infection and mortality rates, known reservoirs, and hosts of WNV across the US since its introduction in 1999. We used these data to evaluate the potential of WNV as a candidate hyperdisease.

To determine whether WNV could lead to the significant size-biased extinction seen at the terminal Pleistocene, we evaluated the size signature of the disease in the United States. The expected pattern of extinction of large-bodied forms, and little-to-no appreciable impact on small to medium bodied species could result from either size-biased infection rates or mortality, or both. We were able to quantitatively examine the first possibility and to indirectly examine the latter. We obtained the list of bird species reported to be West Nile positive from the Center for Disease Control (CDC) and the National Wildlife Health Center (Center for Disease Control 2004a; National Wildlife Heath Center 2004). We obtained a list of all birds occurring in the continental US from the Breeding Bird Survey of North America (Sauer *et al.* 2000; USGS Patuxent Wildlife Research Center 2004). This list represents the pool of bird species that WNV could potentially infect and includes any species found within the continental United States during the summer between 1966 and 2000. Although the CDC also reports exotic birds that have been infected in the United States, these were excluded from our analyses as we were interested only in natural, free-living, non-captive populations. Two bird species, the California condor (*Gymnogyps californianus*) and the whooping crane (*Grus Americana*), were eliminated from analyses because they are essentially extinct in the wild and have populations of *c.* 90 and 300 individuals, respectively (California Department of Fish and Game 2004; Whooping Crane Conservation Association 2004). Elimination of these two species is conservative with respect to the results because both birds are very large (4.0 and 3.77 log units) and the condor is uninfected. There are few very large birds, so eliminating these species increases the proportion of infected birds in the largest bin. This in turn favors results corroborating the hyperdisease hypothesis. We obtained independent body mass estimates for all species from the CRC handbook of avian body masses (Dunning 1993); these were averaged across sex and geographic locality. We used these data to construct body size distributions of infected and non-infected birds. Smith *et al.* (2003) present a species list and an estimate of body mass for mammals present before and after the terminal Pleistocene extinction event that we use for comparison.

We analyzed the size signature of the distributions in several ways. First, we binned body size into 0.25 log units, and compared the body size distributions of infected and non-infected birds using Kolmorgorov–Smirnov two-sample tests. Second, we computed the proportion of infected birds in each size category and examined the relationship between infection rate and body size using likelihood ratio tests. The null hypothesis was that infection rate was uniform across body size. We considered both linear and non-linear increases in infection rates and body size as alternative hypotheses. We performed similar analyses for the relationship between extinction rate and body size in mammals. The results for birds were compared to the results for mammals using likelihood ratio tests. Third, Kolmorgorov–Smirnov two sample tests were used to

determine if, at the ordinal level, the body size distribution of infected birds was significantly different from the distribution for non-infected birds. Orders with less than two infected or non-infected species were excluded from analyses. Fourth, we scaled mammalian body size to that of birds by combining species into 0.45 log unit bins and calculating extinction rates for each category. The resultant pattern for mammal extinctions was compared with the figure for bird infection rates to determine if similar curves were obtained when body size for mammals was scaled to that of birds.

The overall body size distributions for both late Pleistocene mammals and contemporary birds were compared with the distributions of extinct mammals and infected birds, respectively. For each taxon, we then removed the extinct or infected species to produce a 'survivor' plot. For birds, this assumed that infection rate predicted extinction risk. The resultant distributions were compared using Kolmorgorov–Smirnov two sample tests to determine if survivors differed from the original species pool. We also statistically compared the infected or extinct species with both survivors and the original species pool. Descriptive statistics were calculated for infected, non-infected and the overall bird distribution; Lyons *et al.* (2004) present equivalent data for mammals.

Size-biased mortality rates among infected species also could induce a size-biased extinction. However, the introduction of WNV into the United States has not yet led to any bird extinctions. Moreover, data on mortality rates for infected birds are sparse because only dead birds are tested for WNV. Without comparable data on live populations, it is impossible to determine whether mortality rates vary with body size across species. Therefore, we evaluated the possibility of a post-infection size bias indirectly in several ways. First, we evaluated the degree to which mortality rates must be size-biased across species to produce a size-biased extinction like that of the end-Pleistocene. Second, we examined the size specificity of an extinction event known to have been caused by avian Malaria in Hawaii over historic times. Data on bird species in Hawaii were obtained from the Bishop Museum website (Bishop Museum 2002) and included endemics and regular migrants. The list of extinct species for Hawaii was obtained from Van Riper *et al.* (1986). Body masses for Hawaiian birds were compiled from a variety of literature sources, field guides and species accounts (e.g. Madge & Burn 1988; Dunning 1993; Alsop 2001). Occasional migrants and introduced species were excluded from the analysis. Finally, we examined several confounding life history characteristics that have been proposed to lead to higher extinction risk. For example, we examined the relationship between body size and geographic range for late-Pleistocene mammals using body sizes data from Smith *et al.* (2003) and geographic range data from Lyons (2003). Species lists and body size data for the continental United States and Hawaii are available upon request from the senior author.

RESULTS AND DISCUSSION

Is West Nile a reasonable proxy for a hyperdisease?

Criterion no. 1: Does WNV have a reservoir species with a stable character state?

Several wide-ranging or migratory bird species in North America have been identified as competent reservoirs for WNV including blue jays, common grackles, house finches, American crows, and house sparrows (Milius 2003). Competent reservoirs are species that attain viral levels high enough to pass on the disease, but still survive. There is some controversy concerning the particular species identified as reservoirs. Crows and jays, for example, have a high mortality rate to WNV, making them less suitable as a reservoir species (McLean *et al.* 2001; Center for Disease Control 2003c). Other species such as humans and chickens are considered dead-end hosts because infected individuals do not build up enough virus in the bloodstream for effective transmission (Center for Disease Control 2004b). Regardless of the identity of the particular species that are competent reservoirs for WNV, the first requirement of a stable carrier state for the hyperdisease is likely met by one or more wide-ranging native North American birds.

Criterion no. 2: Does WNV have a high infection rate in susceptible species and affect all age groups?

In the Old World, where WNV is long established and widespread, susceptibility to WNV varies among species and among age classes (Lanciotti *et al.* 1999). However, in the New World, WNV appears to have the high and non-specific infection rate required of a hyperdisease. During the year 2000, 33% of all birds tested in New York state were WNV positive (Kramer & Bernard 2001). In Oklahoma in 2002, 50% of crows were estimated to be positive for WNV (Milius 2003). That same year, testing across the US indicated that crows had an infection rate of 77% and other birds (i.e. 93 species in total) had infections rates of *c.* 40% (Centre for Disease Control 2002). The rapid spread of the disease across the United States also testifies to its high infection rate. Following detection in New York City in August of 1999, by 2002 the disease had spread to California and 42 other states (Centre for Disease Control 2002).

A Yaremych *et al.* (2004) study on New World crows suggests that WNV is capable of infecting all age classes (Yaremych *et al.* 2004). They found no significant differences in infection rates of males vs. females or with age class. Other researches have reported WNV in adult chickens as well as chicks (Milius 2003).

Criterion no. 3: Is WNV hyperlethal in new hosts with a mortality rate exceeding 50–75% among infected individuals?

Exact estimates for the mortality rate due to WNV infection are rare. However, WNV appears to meet the criteria. In laboratory tests using American crows, West Nile had a 100% mortality rate (McLean *et al.* 2001). A recent field study on the same species suggested a 68% mortality rate (Yaremych *et al.* 2004). Unfortunately, mortality rates for other species are not currently available. However, WNV probably fits the third criterion for a hyperdisease.

Criterion no. 4: Does WNV have the ability to infect multiple species without having epidemic potential in humans?

Diseases that meet the first three criteria of a hyperdisease are not unknown (see Table 7.4 in MacPhee & Marx 1997). Indeed, MacPhee & Marx (1997) identified 12 diseases out of a possible 20 that were highly infectious, highly lethal, and had a stable carrier state. However, only two of the 20 were capable of infecting multiple species without having epidemic potential in humans. Most diseases that are capable of crossing species barriers do so only within families or orders (e.g. Myxomatosis and Rinderpest). WNV is an exception. To date, WNV has infected 160 species of birds and has been found in taxa as diverse as mammals and reptiles (Center for Disease Control 2003c; Milius 2003; Center for Disease Control 2004a; National Wildlife Heath Center 2004). Moreover, WNV is unlikely to cause an epidemic among humans. Infection and mortality rates are low and humans are a dead-end host (Center for Disease Control 2003b, 2004c) consistent with the explicit criterion of MacPhee & Marx (1997). In the New World, the vast majority of 'naïve' humans who contract West Nile disease survive; the mortality rate for 2003 is *c.* 2.6% (Center for Disease Control 2004b). Clearly, West Nile meets the final criterion of a hyperdisease.

The hyperdisease hypothesis requires that the taxon of interest be naïve with respect to the invading disease. Although many US birds are migratory and less likely to be genetically naïve, they typically migrate within the New World and should be naïve with respect to diseases from the Old World. Moreover, modern birds may not be the closest ecological analogs to large terrestrial continental mammals; however, life history traits correlate with body size in birds like they do in mammals. Thus, the conditions necessary for a hyperdisease to cause an extinction are present in modern birds as well as Pleistocene mammals. Because WNV appears to meet all of the criteria identified for a hyperdisease (*sensu* MacPhee & Marx 1997), we use it as an model system to examine the disease hypothesis for the end-Pleistocene extinctions.

Does WNV have the potential to cause a size-biased extinction?

A unique aspect of the extinction in the late Pleistocene is the strong size bias. On every continent for which an extinction has been documented, only large-bodied vertebrates were affected, whereas small to medium bodied species remain relatively unscathed (Martin & Klein 1984; MacPhee 1999; Lyons *et al.* 2004). No other mammalian extinction event during the Cenozoic has been so strikingly size selective (Alroy 1999). Even recent historical extinctions caused by European settlements and expansions have not led to a size-biased extinction (Lyons *et al.* 2004). Clearly, any explanation for the late Pleistocene continental extinctions must account for the bias toward animals of large body size and a lack of impact on small and medium species. If disease were the causal mechanism, a size-biased extinction could result if either infection or mortality rates were size-biased.

Our results suggest that WNV is size selective in its infection rates. The body size distribution of infected birds was significantly different from that of non-infected birds (Fig. 1a; $\chi^2 = 16.283$; $P = 0.0006$) with infected birds having higher mean and median body sizes (Table 1). For both birds and mammals, the relationship between body size and infection/extinction was curvilinear. However, WNV did not selectively target large bodied birds while ignoring small to medium bodied birds. The form of the relationship was significantly different. Unlike the mammalian extinction which has relatively low extinction rates at small to medium body sizes and an abrupt increase when some threshold size is reached, infection rates for WNV increase continuously with bird body size (contrast Fig. 2a,c). The best-fit mammal curve is distinctly sigmoidal and rises rapidly through the large sizes (Fig. 2c). In contrast, the bird curve appeared to asymptote at an infection rate of *c.* 0.6 (Fig. 2a). Indeed, an exponential function predicts avian infections slightly better than the curvilinear Hill equation [f(infection) $= 0.174$(log mass)$^{0.795}$, ln L $= -39.335$]; however, the best exponential function for mammals is not asymptotic [f(extinction) $= 0.0187$(log mass)$^{2.01}$] and is appreciably worse than the best Hill function (ln L $= -39.544$). Because all birds in the three largest body size bins are positive for WNV, the best fit bird curve seems to provide a bad fit (Fig. 2a). However, we stress that the likelihood tests assess the expected numbers of infected species (Fig. 2b), and because of the low numbers of large birds, the observed numbers are only slightly greater than the expected numbers. The small sample size for large bird species means that 100% infection is not improbable under almost any infection rate. Although a non-linear association between infection and body size is significantly more likely than a linear or uniform association, the expectations of all three hypotheses (Fig. 2b) are much
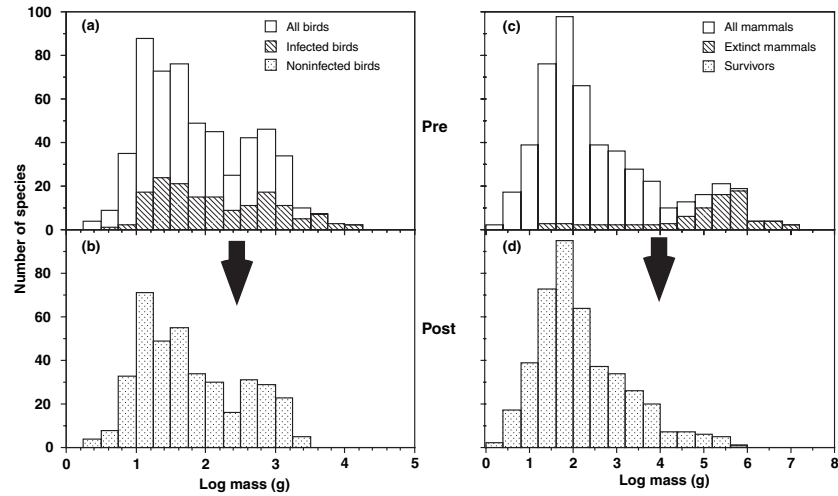
Figure 1 Body size distributions for contemporary birds (panels a and b), and late Pleistocene mammals (panels c and d). In each case, the top panels represent the overall species pool before the infection (a), or extinction (c) event; the lower panels represent the 'survivors' (b and d). The late Pleistocene extinction led to significant losses of large bodied species, but did not appreciably impact small or medium bodied species. In contrast, if the pattern of West Nile virus infection corresponds to the extinction risk, there are substantial impacts not only on the very largest birds, but also on medium and small birds as well. A Kolmorgorov–Smirnov two sample tests suggests that the pre and post distributions for mammals are highly significantly different ($P < 0.0001$); but that the pre and post distributions for birds are non-significant ($P > 0.4$). See text for details.

Table 1 Descriptive statistics for the body size frequency distributions for all birds in the United States, non-infected birds and native birds infected with West Nile virus (WNV). Exotic species infected with WNV were excluded from the analyses

	All birds	Non-infected birds	Birds infected with WNV
n	548	388	160
Mean body mass	1.88	1.77	2.14
Median	1.73	1.64	2.02
Standard deviation	0.77	0.73	0.81
Variance	0.59	0.53	0.66
Standard error	0.03	0.04	0.06
Minimum	0.44	0.44	0.54
Maximum	4.06	3.47	4.06
Range	3.62	3.03	3.51
Skewness	0.47	0.44	0.40
Kurtosis	−0.70	−0.89	−0.82

more similar to each other than are the best analogous hypotheses for associations between extinction and mammalian body size (Fig. 2d). Moreover, the greatest number of infected birds were contained in the 1.25 – 1.5 log units size bin (*c.* 17 – 32 g; Fig. 2b) and 50% of all infections were in birds less than 2 log units (< 100 g), in line with the overall distribution of birds. In contrast, the greatest number of extinct mammals were contained in the 5.4 – 5.85 log units sizes bin (*c.* 250 000 – 708 000 g; Fig. 2d) and 50% of all

extinctions were in mammals greater than 5.4 log units. This was significantly different from the overall distribution of mammals.

A unique aspect of the end Pleistocene extinction is that the largest species within each order were disproportionately affected (Lyons *et al.* 2004). WNV does not replicate this pattern within avian orders (Table 2). Of the eleven orders examined, the body size distributions of infected and non-infected species were significantly different in only two orders: the Passeriformes and the Strigiformes (Table 2). For the passerines, the difference between the mean and median for infected and uninfected birds was small (i.e. infected birds: mean = 1.515 log units, median = 1.477 log units; uninfected birds: mean = 1.317 log units, median = 1.261 log units). Moreover, passerines tend to be small to medium bodied, ranging from *c.* 0.7 to 3.1 log units (5–1200 g, respectively), whereas the overall pool of US birds ranges from *c.* 0.4 to 4 log units (2.5–10 000 g, respectively). Strigiformes are larger than passerines, but are medium-bodied in general. They range from *c.* 1.6 to 3.25 log units (41 – 1800 g, respectively). Although Passeriformes is the most species rich order of birds in the US, the size bias in infection rate within this order and within the strigiforms cannot produce an extinction like that of the end Pleistocene. Within each order, extinction risk due to WNV must be much greater for larger bodied birds to result in an ordinal extinction similar to that of late Pleistocene mammals. Moreover, this raises the possibility that the
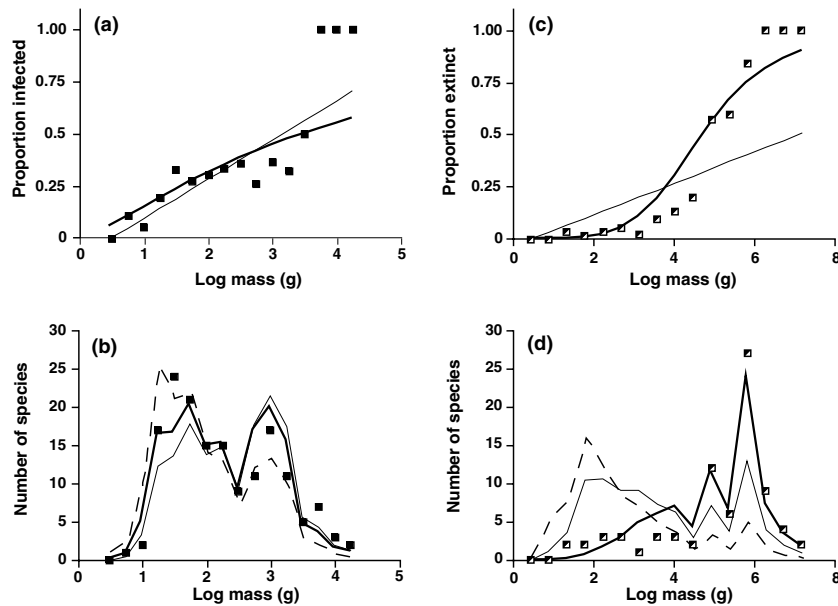
Figure 2 Proportion of the overall species pool (panels a and c) infected (filled squares) or extinct (semi-filled squares), and the number of species (panels b and c) infected or extinct plotted as a function of body size. Ln likelihood ratio tests indicate that rates vary non-linearly with body size for both birds (panels a and b) and mammals (panels c and d), but in different ways. Non-linear curves were fit using Hill equations which take the form: $y = X^n/[(X50)^n + X^n]$, where $X50$ is the point along the *x*-axis at which $y = 0.5$ and n is a fitted exponent. For birds, the best uniform rate estimate was $f(\text{infection}) = 0.292$ (ln L = −50.415). The best linear estimate (panel a, thin line) was $f(\text{infection}) = 0.187(\log \text{mass})$, (ln L = −39.873) and the best non-linear estimate (panel a, thick line) was $f(\text{infection}) = (\log \text{mass})^{1.34}/[3.420^{1.34} + (\log \text{mass})^{1.34}]$, (ln L = −37.551). For mammals, the best uniform rate estimate was $f(\text{extinction}) = 0.154$ (ln L = −129.34). The best linear estimate (panel c, thin line) was $f(\text{extinction}) = 0.075(\log \text{mass})$, (ln L = −68.70), and the best non-linear estimate (panel c, thick line) was $f(\text{extinction}) = (\log \text{mass})^{4.65}/[4.597^{4.65} + (\log \text{mass})^{4.65}]$, (ln L = −32.30). As expected from their close likelihood values, the different rate models predicted similar numbers of infected bird species with each performing well (panel b: uniform, dashed line; linear, thin line; non-linear, thick line). In contrast, the different rate models predicted different numbers of extinct mammal species with only the non-linear model performing well (panel d: uniform, dashed line; linear, thin line; non-linear, thick line).

Table 2 Comparisons of the body size distributions of infected and non-infected birds within each order using Kolmorgorov–Smirnov two sample tests

Orders	Infected vs. Non-infected			
	χ^2	P	Infected (*n*)	Non-infected (*n*)
Anseriformes	4.16	0.250	6	23
Charadiiformes	1.44	0.971	9	39
Ciconiiformes	1.78	0.822	6	12
Columbiformes	3.33	0.379	7	4
Falconiformes	1.52	0.936	21	8
Galliformes	6.09	0.095	5	14
Gruiformes	1.60	0.899	2	8
Passeriformes	12.40	0.004	72	209
Pelecaniformes	4.91	0.172	3	8
Piciformes	0.82	> 0.99	4	18
Strigiformes	9.80	0.015	11	8

χ^2 is the chi-square value from KS tests; P is the significance level; *n* is the number of species infected or not in a particular order.

entire correlation between size and infection rate is a phylogenetic artifact. That is, if one or more large-bodied clades is susceptible to WNV for reasons other than size, then we still would get the patterns documented here (e.g. Raup & Gould 1974).

Our results suggest that the pattern of WNV infection should not result in a size-biased extinction similar to that seen in mammals at the terminal Pleistocene (Fig. 1). Comparison of the overall body size distributions of both birds and mammals before and after either infection or extinction, illustrated that these events would impact the distributions in significantly different ways. For mammals, the extinction event resulted in a distribution of survivors that was fundamentally different from the original species pool. A Kolmorgorov–Smirnov two-sample test confirmed that the distributions pre- and post-extinction were significantly different (Fig. 1c,d; $\chi^2 = 11.856$, $P < 0.005$). Conversely, if WNV infections reflect mortality and extinction risk, the resulting distribution of surviving bird species would not be significantly different from the original

species pool (Fig. 1a,b; $\chi^2 = 2.78$, $P > 0.45$). Clearly, a size signature similar to that of mammalian fauna could only result if there are substantial differences in bird mortality and species extinction risk as a function of body size (compare Fig. 1a,b).

If the rate of WNV infection reflects extinction risk, then clearly the very largest birds (> 3.5 log units; 3162 g) have the highest probability of extinction. However, any viable hypothesis for the end-Pleistocene extinction must necessarily explain not only the extinction of the largest mammals, but also the absence of a substantial impact on small to medium species. Unfortunately, most studies ignore small to medium species (e.g. Guilday 1967; MacDonald 1984; Alroy 2001; Johnson 2002; Brook & Bowman 2004). When the overall infection rates of WNV are plotted as a function of body size, there is a large impact across virtually the entire body size spectrum (Fig. 2a,b). This is in sharp contrast to the pattern of mammalian extinctions where small species were largely unaffected (Fig. 2c,d). Whereas, the average infection rate for birds in the lower half of the body size spectrum is 25% (0.4–2.0 log units, 2.8–100 g), the corresponding extinction rate for mammals is under 4% (0.3–3.42 log units, 2–2630 g). The differences between the bird and mammal curves are especially evident when mammal body size is rescaled to that of birds (Fig. 3). Mortality rates would have to scale positively with body size if the pattern of WNV infection were to result in anything approaching that of the mammal curve. The log probability of the WNV infection curve resulting in an extinction pattern like that of the mammals is −149.4. Indeed, rough calculations indicate that there would have to be a sixfold difference in mortality rate between the smallest half of the body size spectrum and the largest species to result in a pattern similar to that reported for the late Pleistocene megafauna extinction (Fig. 3).

Even proponents of the hyperdisease hypothesis allow that there is no *a priori* reason why small body size *per se* should confer resistance to extinction. Indeed, the disease hypothesis does not propose that disease must be size-biased in its incidence of infection, but that the resulting extinction should be size-biased because the life history characteristics of large-size species increase their extinction risk (MacPhee & Marx 1997). Few factors thought to cause extinction target large-bodied species to the exclusion of small-bodied species. Instead, many hypotheses for the late Pleistocene extinctions invoke differences in life history characteristics to explain the size-selectivity (e.g. Guilday 1967; MacDonald 1984; MacPhee & Marx 1997; Johnson 2002). Large-bodied species are thought to be more vulnerable because they have low intrinsic rates of population increase (r_{max}) and low population densities. Conversely, under a disease scenario, species with low population densities might have lower infection rates,
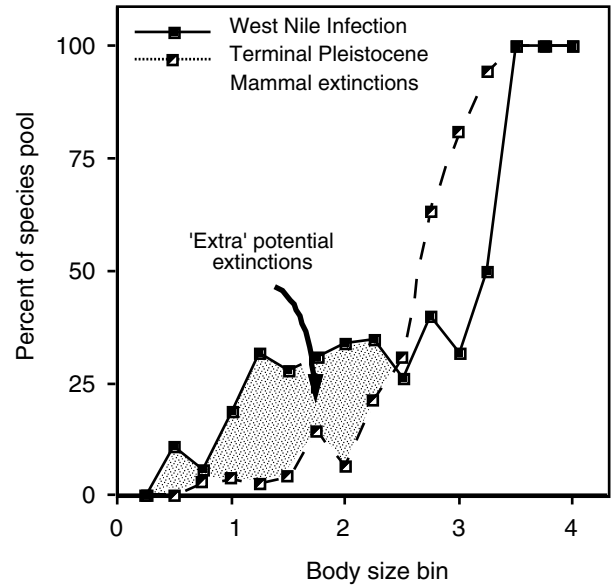

Figure 3 The percent of the overall species pool represented by a particular body size bin; filled square, birds infected by West Nile virus (WNV); semi-filled square, mammals of that size category that went extinct at the end Pleistocene. The *x*-axis is rescaled for mammals since they contain a much larger range of body masses. Thus, each bin represents 0.43 log units for mammals (range: 0.3–7 log units, or *c.* 2 g–10 000 kg), and 0.25 log units for birds (range: 0.25–4 log units, or *c.* 2.8–11 kg). The size signature of the terminal Pleistocene mammal extinctions is distinct from the pattern that would result if WNV leads to equivalent mortality rates across body size categories. The log probability of the WNV infection curve resulting in an extinction pattern like that of the mammals is −149.4.

especially if they do not aggregate. Nonetheless, both population density and intrinsic rate of increase scale with mass; as $M^{-3/4}$ and $M^{-1/4}$, respectively (Fenchel 1974; Damuth 1981; Peters 1983; Calder 1984; Schmidt-Nielsen 1984; Damuth 1987). Because these relationships hold across environmental and taxonomic groups, they should also hold over evolutionary time. Thus, large bodied mammal species from the Cenozoic should have had relatively small population densities and low intrinsic rates of increase. However, if large-bodied species were truly more vulnerable to extinction, then most *natural* extinction events in mammals should have been size biased. They were not. Although there is a weak relationship between size bias and the intensity of the extinction event, in general, the mean body size has been similar for victims and survivors of extinction events throughout the Cenozoic (Alroy 1999). Moreover, if life history traits associated with large body size confer increased extinction risk, then large-bodied species should have higher origination and extinction rates relative to small-bodied species over evolutionary time. Background
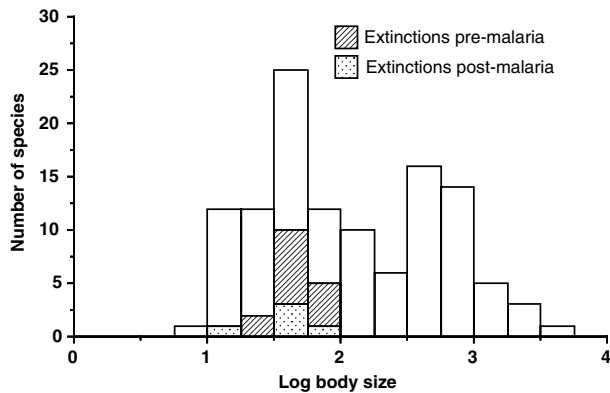
Figure 4 Comparison of the body mass distributions for all birds in Hawaii (white bars) with those that went extinct before (stripped bars) and after the introduction of malaria (shaded bars).

extinction rates for mammals show no relationship with body size (Alroy 2002). This lack of size-bias in deep time extinction events makes it unlikely that population density and fecundity alone could generate a size-biased extinction. Moreover, a recent, well documented, extinction event in Hawaiian birds clearly illustrates that life history characteristics alone are not sufficient to predict extinction risk (Fig. 4). Given these life history arguments and allometric relationships, one would predict the largest species to be at highest risk from avian malaria. We found the opposite pattern to be true: the species that went extinct were all of small body size (i.e. 10–100 g; Fig. 4).

Size-biased mortality might not necessarily result from low fecundity and population density because of differences in extinction risk associated with life history traits. Another, often overlooked, life history characteristic associated with extinction risk is the extent of the geographic range. Species with small or limited geographic ranges might be more prone to extinction than species with large geographic ranges. For example, Jablonski (1986) demonstrated an association between small geographic range and extinction risk in the fossil record for bivalves. A similar relationship might exist for mammals and birds. Several authors have demonstrated that geographic range varies predictably with contemporary mammalian body size, as *c.* $M^{1/4}$ (Peters 1983; Brown 1995) although there is considerable variation in the relationship. This relationship has been found for other taxa (Brown & Maurer 1987; Brown 1995; McGill & Collins 2003). Geographic range size and body mass for late Pleistocene mammals shows a similar scaling relationship ($Y = 4.241 + 0.201 \times$ Mass; $R^2 = 0.055$; $P = 0.002$). The pattern is not simple, however. Although large mammals tend to have large ranges, small mammals have ranges than span the entire spectrum. For example,

rodents of similar body size may have ranges that differ by more than five orders of magnitude. Such 'triangular' shaped relationships are common (Brown 1995). Thus, a small-bodied species with a small geographic range might actually be more vulnerable to extinction than a widely ranging, large-bodied species under the scenario of a hyperdisease. This point has been overlooked by researchers that invoke life history to explain the size bias of the end Pleistocene extinction.

SUMMARY AND CONCLUSIONS

If the scenario proposed by MacPhee & Marx (1997) is correct, then humans arrived in North America with a highly infectious commensal or 'typhoid Mary' reservoir. This hyperdisease rapidly infected the populations of native fauna whose lack of exposure and adaptation to the disease made them especially vulnerable, drastically lowering population densities, and driving entire species, and in some cases genera, extinct. It is assumed that large-bodied species were especially vulnerable, because they did not have the reproductive capacity to recover from deadly epidemics. The disease hypothesis has garnered considerable attention because of its novel interpretation of the late Pleistocene extinctions (MacPhee 1999 and references therein; Alroy 2001; Johnson 2002; Lyons *et al.* 2004). Here, we have demonstrated that this scenario is highly unlikely. Based on analyses of the only known disease conforming to the criteria, it appears that neither WNV infection nor extinction risk would likely lead to the unique signature of the late Pleistocene extinctions. Moreover, the differential impact on small to medium bodied species across all birds and within orders (Figs 1–3; Table 2), coupled with confounding predictions about the vulnerability resulting from various life history characteristics, and the pattern of extinction resulting from avian malaria (Fig. 4) greatly undermine the validity of the disease hypothesis as an explanation for the terminal Pleistocene extinctions. Indeed it is probable that a hyperdisease would target mammals of all sizes, a prediction considerably at odds with the observed pattern. Thus, even a disease that meets all the criteria of a hyperdisease is an unlikely explanation for a size-biased extinction event like that of the late Pleistocene.

ACKNOWLEDGEMENTS

REFERENCES

Alroy, J. (1999). Putting North America's end-Pleistocene megafaunal extinction in context: large scale analyses of spatial patterns, extinction rates, and size distributions. In: *Extinctions in Near Time: Causes, Contexts, and Consequences* (ed. MacPhee, R.D.E.). Kluwer Academic/Plenum, New York, pp. 105–143.

Alroy, J. (2001). A multispecies overkill simulation of the end-Pleistocene megafaunal mass extinction. *Science*, 292, 1893–1896.

Alroy, J. (2002). Walking the gangplank: ecological correlates of extinction in fossil mammals abundance, body mass, ecomorphology, extinction, Mammalia. Available at: http://www.gsa.confex.com/gsa/2002AM/finalprogram/abstract_43926.htm.

Alsop, F.J. III (2001). *Birds of North America: Western Region. Smithsonian Handbooks*. DK Publishing Inc., New York.

Bishop Museum (2002). Hawaiian bird checklist (digital). Available at: http://www2.bishopmuseum.org/HBS/checklist/query.asp?gr$P$ = Bird.

Brook, B.W. & Bowman, D. (2004). The uncertain blitzkrieg of Pleistocene megafauna. *J. Biogeogr.*, 31, 517–523.

Brown, J.H. (1995). *Macroecology*. University of Chicago Press, Chicago, IL.

Brown, J.H. & Maurer, B.A. (1987). Evolution of species assemblages – effects of energetic constraints and species dynamics on the diversification of the North-American avifauna. *Am. Nat.*, 130, 1–17.

Calder, W.A. III (1984). *Size, Function, and Life History*. Harvard University Press, Cambridge, MA.

California Department of Fish and Game (2004). California condor population size and distribution (digital). Available at: http://www.dfg.ca.gov/hcpb/species/t_e_spp/tebird/Condor%20Pop%20Stat.pdf.

Center for Disease Control (2003b). Transmission (digital). Available at: http://www.cdc.gov/ncidod/dvbid/westnile/qa/transmission.htm.

Center for Disease Control (2003c). Vertebrate ecology (digital). Available at: http://www.cdc.gov/ncidod/dvbid/westnile/birds&mammals.htm.

Center for Disease Control (2004a). Bird species (digital). Available at: http://www.cdc.gov/ncidod/dvbid/westnile/birdspecies.htm.

Center for Disease Control (2004b). Case Coun03_detailed (digital). Available at: http://www.cdc.gov/ncidod/nvbid/westnile/surv&controlCaseCount03_detailed.htm.

Center for Disease Control (2004c). Flowchart: West Nile virus transmission cycle (digital). Available at: http://www.cdc.gov/ncidod/dvbid/westnile/cycle.htm.

Center for Disease Control (2004d). Virology: classification of West Nile virus (digital). Available at: http://www.cdc.gov/ncidod/dvbid/westnile/virus/htm.

Centre for Disease Control (2002). Provisional surveillance summary of the West Nile virus epidemic – United States, January–November, 2002. *Morb. Mort. Wkly Rep.*, 51, 1129–1133.

Centre for Disease Control (2003a). Mosquito species (digital). Available at: http://www.cdc.gov/ncidod/dvbid/westnile/mosquitoSpecies.htm.

Damuth, J. (1981). Population-density and body size in mammals. *Nature*, 290, 699–700.

Damuth, J. (1987). Interspecific allometry of population-density in mammals and other animals – the independence of body-mass and population energy-use. *Biol. J. Linn. Soc.*, 31, 193–246.

Dunning, J.B. (1993). *CRC Handbook of Avian Body Masses*. CRC Press, Boca Raton, FL.

Fenchel, T. (1974). Intrinsic rate of natural increase – relationship with body size. *Oecologia*, 14, 317–326.

Fiedel, S.J. (1999). Older than we thought: implications of corrected dates for Paleoindians. *Am. Antiquity*, 64, 95–115.

Graham, R.W. & Lundelius, E.L. Jr. (1984). Coevolutionary disequilibrium and Pleistocene extinctions. In: *Quaternary Extinctions: A Prehistoric Revolution* (eds Martin, P.S. & Klein, R.G.). University of Arizona Press, Tucson, AZ, pp. 223–249.

Guilday, J. (1967). Differential extinction during late-Pleistocene and Recent times. In: *Pleistocene Extinctions: The Search for a Cause* (eds Martin, P.S. & Wright, H.E. Jr.), Yale University Press, New Haven, CT, pp. 121–140.

Jablonski, D. (1986). Background and mass extinctions – the alternation of macroevolutionary regimes. *Science*, 231, 129–133.

Johnson, C.N. (2002). Determinants of loss of mammal species during the late Quaternary 'megafauna' extinctions: life history and ecology, but not body size. *Proc. R. Soc. Lond. B*, 269, 2221–2227.

Kramer, L.D. & Bernard, K.A. (2001). West Nile virus infection in birds and mammals. In: *West Nile Virus: Detection, Surveillance, and Control*. New York Academic Sciences, New York, NY, pp. 84–93.

Lanciotti, R.S., Roehrig, J.T., Deubel, V., Smith, J., Parker, M. & Steele, K. (1999). Origin of the West Nile virus responsible for an outbreak of encephalitis in the northeastern United States. *Science*, 286, 2333–2337.

Lyons, S.K. (2003). A quantitative assessment of the range shifts of Pleistocene mammals. *J. Mammal.*, 84, 385–402.

Lyons, S.K., Smith, F.A. & Brown, J.H. (2004). Of mice mastodons and men: human mediated extinction on four continents. *Evol. Ecol. Res.*, 6, 339–358.

MacDonald, J.N. (1984). The reordered North American selection regime and late quaternary megafaunal extinctions. In: *Quaternary Extinctions: A Prehistoric Revolution* (eds Martin, P.S. & Klein, R.G.). University of Arizona Press, Tucson, AZ, pp. 404–439.

MacPhee, R.D.E. (1999). *Extinctions in Near Time: Causes, Contexts, and Consequences*. Kluwer Academic/Plenum, New York, NY.

MacPhee, R.D.E. & Marx, P.A. (1997). Humans, hyperdisease, and first-contact extinctions. In: *Natural Change and Human Impact in Madagascar* (eds Goodman, S.M. & Patterson, B.D.). Smithsonian Institution Press, Washington, DC, pp. 169–217.

Madge, S. & Burn, H. (1988). *Wildfowl: An Identification Guide to the Ducks, Geese, and Swans of the World*. Christopher Helm (Publishers) Ltd., Bromley, Kent.

Martin, P.S. (1967). Prehistoric overkill. In: *Pleistocene Extinctions: The Search for a Cause* (eds Martin, P.S. & Wright, H.E. Jr.). Yale University Press, New Haven, CT, pp. 75–120.

Martin, P.S. (1984). Prehistoric overkill: The global model. In: *Quaternary Extinctions: A Prehistoric Revolution* (eds Martin, P.S. & Klein, R.G.). University of Arizona Press, Tucson, AZ, pp. 354–403.

Martin, P.S. & Klein, R.G. (1984). *Quaternary Extinctions: A Prehistoric Revolution*. University of Arizona Press, Tucson, AZ.

McGill, B. & Collins, C. (2003). A unified theory for macroecology based on spatial patterns of abundance. *Evol. Ecol. Res.*, 5, 469–492.

McLean, R.G., Ubico, S.R., Docherty, D.E., Hansen, W.R., Sileo, L. & McNamara, T.S. (2001). West Nile virus transmission and ecology in birds. In: *West Nile Virus: Detection, Surveillance, and Control*. New York Academic Sciences, New York, NY, pp. 54–57.

Milius, S. (2003). After West Nile virus: what will it do to the birds and beasts of North America? *Sci. News Online*, 163, 1–8.

National Wildlife Heath Center (2004). *Species Found Positive for WNV in Surveillance Efforts* (digital). Available at: http://www.nwhc.usgs.gov/research/west_nile/west_nile.html.

Peters, R.H. (1983). *The Ecological Implications of Body Size*. Cambridge University Press, Cambridge, UK.

Raup, D.M. & Gould, S.J. (1974). Stochastic simulation and evolution of morphology – towards a nomothetic paleontology. *Syst. Zool.*, 23, 305–322.

Roberts, R.G., Flannery, T.F., Ayliffe, L.K., Yoshida, H., Olley, J.M., Prideaux, G.J. *et al.* (2001). New ages for the last Australian megafauna: continent-wide extinction about 46 000 years ago. *Science*, 292, 1888–1892.

Sauer, J.R., Hines, J.E., Thomas, I., Fallon, J. & Gough, G. (2000). *The North American Breeding Bird Survey, Results and Analysis 1966–1999*. USGS Patuxent Wildlife Research Center. Available at: http://www.mbr-pwrc.usgs.gov/bbs/.

Schmidt-Nielsen, K. (1984). *Scaling, Why is Animal Size so Important?* Cambridge University Press, Cambridge, UK.

Smith, F.A., Lyons, S.K., Ernest, S.K.M., Jones, K.E., Kaufman, D.M., Dayan, T. *et al.* (2003). Body mass of late quaternary mammals. *Ecology*, 84, 3403.

Thorne, A., Grun, R., Mortimer, G., Spooner, N.A., Simpson, J.J., McCulloch, M. *et al.* (1999). Australia's oldest human remains: age of the Lake Mungo 3 skeleton. *J. Hum. Evol.*, 36, 591–612.

USGS Patuxent Wildlife Research Center (2004). North American breeding bird Survey ftp dataset (digital). Available at: http://www.pwrc.usgs.gov/bbs/retrieval/menu/cfm.

Van Riper, C., Van Riper, S.G., Goff, M.L. & Laird, M. (1986). The epizootiology and ecological significance of malaria in Hawaiian land birds. *Ecol. Monogr.*, 56, 327–344.

Whooping Crane Conservation Association (2004). How many whooping cranes are there in the world? (digital). Available at: http://www.whoopingcrane.com/wccaflockstatus.htm.

Yaremych, S.A., Warner, R.E., Mankin, P.C., Brawn, J.D., Raim, A. & Novak, R. (2004). West Nile virus and high death rate in American crows. *Emerg. Infect. Dis.*, 10. Available at: http://www.cdc.gov/ncidod/EID/vol10no4/03-0499.htm.