Natural Change and Human Impact in Madagascar

EDITED BY
STEVEN M. GOODMAN
AND BRUCE D. PATTERSON

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table 7.4 that do not fit this requirement are Chagas' disease (exclusively in the Americas), simian hemorrhagic virus (exclusively African), and, probably, plague (Acha and Szyfres 1980).

4. *An ability to infect multiple species but not seriously affect human groups.* The last requirement is that the HP have an exceptionally lethal effect in a wide variety of species without, however, significantly threatening humans. (Humans should be either unaffected or inapparently affected, or lethal disease should occur, but without the potential to cause large epidemics.) Only two entries survive to this stage—leptospiroses and rabies. *Leptospira* is of interest because its numerous serovars are apparently able to infect virtually any mammal, and small-scale epidemics occasionally occur (Torten and Marshall 1994). Infections in *Homo sapiens* are dead-end, which is consistent with our criterion. By contrast, rabies is able to infect a narrower (if nevertheless remarkably large) range of mammals—but it is capable of sustaining widespread, hyperlethal epizootics in several species. Although rabies is highly lethal in humans, epidemics do not occur in *Homo sapiens* (Acha and Szyfres 1980).

Our purpose in differentially comparing disease organisms in this section is not to propose that rabies is actually the hyperdisease pathogen. Instead, our point is to determine whether any known pathogens meet reasonable hyperdisease criteria. Although rabies qualifies, it does so only marginally. Rabies infections in individuals of susceptible species almost always occur through direct contact, although in some experimental cases aerosol transmission has been demonstrated (Beran 1994). For rabies itself to be a plausible cause of complete extinction in the past, one has to imagine either that many extinct species had social behaviors that ensured unusual degrees of contact among animals or that the virus was able to infect indirectly as well as directly.

Discussion: Objections and Resolutions

> There is no science of palaeo-virology, so that conclusions concerning the evolutionary history and the antiquity of viral diseases must be based on inference.
> —F. Fenner and F. N. Ratcliffe (1965, p. 268), commenting on the antiquity of myxomatosis in the Americas

For an idea that has a brief but respectable pedigree (e.g., Osborn 1906; Haldane 1949; Emiliani 1993), it is somewhat surprising that disease as a cause of extinction has never been considered seriously by modelers of late Quaternary extinctions (cf. Edwards 1967). Even the perspicacious Dr. Molyneux (1697, p. 501), whose speculation on the extinction of *Megaceros giganteus* is quoted in the epigraph at the beginning of this chapter, ultimately concluded that the last giant deer were hunted to extinction by humans, "kill'd like other Venison as well for the sake of Food as Mastery and Diversion." In the encyclopedic *Quaternary Extinctions* (Martin and Klein 1984), only Diamond's (1984) paper makes anything other than incidental reference to disease as a possible cause of extinction. Nor is disease considered to be among the chief threats to vertebrate biodiversity at present. According to the WCMC survey (Groombridge 1992), which is admittedly conservative in allocating cause, only one modern-era vertebrate extinction was forced by disease—that of the New Zealand quail, *Coturnix novaezelandiae,* supposedly

7 The 40,000-Year Plague

Humans, Hyperdisease, and First-Contact Extinctions

ROSS D. E. MACPHEE AND PRESTON A. MARX

> By what means [the Irish giant deer,] formerly so common and numerous in this Country, should now become utterly lost and extinct, deserves our Consideration. . . . [Despite accepted explanations,] it seems more likely to me, this kind of Animal might become extinct here from a certain ill Constitution of Air in some of the past Seasons long since the Flood, which might occasion an *Epidemick Distemper*, if we may so call it, or *Pestilential Murren*, peculiarly to affect this sort of Creature, so as to destroy at once great Numbers of 'em, if not quite ruine the Species. And this is not so groundless an Assertion as at first it may appear. . . .
>
> —Thomas Molyneux (1697, p. 499), commenting on the extinction of the Irish giant deer (*Megaloceros giganteus*)

Understanding Late Quaternary Extinctions

Extinction is as much a part of the total pattern of life on this planet as is evolution. Indeed, without the winnowing effect of extinction, the production of new species by evolutionary forces would have long since led to biotic disaster by overburdening the world's available habitats. The extinction of any single species occurs at the death of the last member. In this narrowly actuarial sense, extinctions at the species level have presumably been the "same" at all times and places in earth history. However, this is not the only or even the most important level of analysis, because the fossil record clearly shows that the overwhelming major-

ity of extinctions have occurred in clusters, some of great size. The "Big Five" mass extinctions of marine invertebrates during the past 500 million years, for example, involved calculated species losses of up to 95% of total existing faunas and broadly affected many different phylogenetic groups (Jablonski 1994). Some of these mass extinctions may have been caused or set in motion by extraterrestrial factors, such as bolide impacts, but this possibility is not settled even for the locus classicus of biotic catastrophes, the K/T boundary mass extinction (cf. papers edited by Glen 1994; Eldredge 1991). Determining the factors that have forced massive extinctions throughout earth history remains a challenge of the first magnitude.

Coordinated extinctions have also occurred on a much smaller scale, in which losses were significant but tended to affect only one or a few groups. By far the most bizarre of these smaller events occurred in the late Quaternary (as defined here, the last 100,000 years). At different times on different continents and large islands, hundreds of land vertebrate species, especially large mammals, disappeared without replacement in a period lasting approximately 40,000 years. On the world's smaller islands, many more species, especially birds, were lost within the last 10,000 years. The uniqueness of these extinctions derives not only from the fact that losses were severe in some areas and negligible in others, but also from a famously portentous coincidence: they all occurred subsequent to the emergence of anatomically modern *Homo sapiens*.

The literature on the land vertebrate extinctions of the late Quaternary is extensive and includes two landmark summary volumes (Martin and Wright 1967; Martin and Klein 1984). Despite this level of attention, consensus on the prime cause or causes of these losses has yet to emerge. The explanations most frequently offered favor direct anthropogenic impacts (especially in the form of overhunting and habitat destruction by fire or other means), natural agents such as "climate change," or some combination of these two factors (e.g., cascades of interlocking extinctions due to loss of key megaherbivores, probably by overhunting; Owen-Smith 1988). The strengths and weaknesses of these explanations have been explored by a number of authors (e.g., Diamond 1984; Grayson 1984; Martin 1984b; Owen-Smith 1988; Stuart 1991, 1993; Burney 1993b, this volume; Flannery 1994b; see also Dewar, this volume). In case after case it seems beyond doubt that humans were on the scene, and recently so, at the time extinctions were occurring. But in the view of many workers, including the present authors, no satisfactory explanatory link has been forged between multiple species losses and the direct anthropogenic impacts that allegedly forced them (e.g., overhunting or extreme environmental transformation without sophisticated tools).

If people were not ultimately responsible for late Quaternary losses, then the alternative—the only alternative in this bipolar debate, if hybrid explanations are ignored—is to look for some evidence that the losses accrued from the operation of forces of nature. The search for causative agencies in climatic cycling has not been notably successful. For example, Grayson (1984) finds strength in the particularity of some accounts that link certain species losses to specific environmental occurrences, apparently on the premise that a solid scenario for one or a few extinctions implies wider applicability to many. The problem, however, is that an isolated instantiation tells us nothing general enough about the extinc-

tion process to carry over to the next instance of loss. Even if it were true that the extinction of mammoths was a result of increased seasonality at the end of the Pleistocene (Guthrie 1984; Lundelius 1988), there are no obvious logical implications in this statement for the disappearance of (for example) nesophontids in Cuba or bibymalagasy in Madagascar some 9,000–10,000 years later (MacPhee 1994; but see Burney, this volume). As Coope (1995) points out, glacial rather than interglacial conditions have been the rule for most of the Quaternary, and for only a fraction of that time (<2%) has climate been as warm as, or warmer than, it is today. For the climate change hypothesis to have a place at the table of prime causes, a mechanism with a potentially identifiable signature throughout the late Quaternary is required, and none proposed so far has withstood testing to any important degree (Martin 1984b).

Our position is that, even though direct impact and climate change arguments plausibly explain some extinctions of the late Quaternary, they lack sufficient generality to account for the nature of many losses that we hereafter identify as "first contact" (FC) extinctions. We offer the alternative proposition that, under certain circumstances, disease can cause the complete extinction of species.

During human history, exotic pathogens must have regularly challenged naive species as people and their commensals spread to new lands. We hypothesize that during this spread one or more newly introduced pathogens with certain transmission characteristics might have emerged as hypervirulent, hyperlethal diseases (hereafter, "hyperdiseases") in numerous susceptible species, leading to multiple bottlenecks, population crashes, and, ultimately, complete extinctions. A distinguishing feature of the hyperdisease hypothesis is the multiplier effect produced by the interlocking conjunctions of novel carriers, diseases, and victims—conjunctions that occur only once (at FC) in a given context.

Before scrutinizing the hyperdisease hypothesis in more detail, certain observations about extinction, extinctions, and explanations of extinction(s) need to be made. For a number of reasons we shall focus attention on losses that occurred among terrestrial mammals, although reference will be made to other vertebrate groups where pertinent. Furthermore, although this book, and the symposium from which it is derived, is concerned with natural and human-induced change in Madagascar, the biological ruination of this island must be examined in a comparative context. For this reason we shall not limit ourselves geographically, but employ Madagascar as one of several points of reference. Finally, although there are several versions of the anthropogenic direct impact argument (Marshall 1984), we shall primarily set our ideas against the expectations of "blitzkrieg"-style extinction as presented by Mosimann and Martin (1975) and Martin (1984a,b).

Late Quaternary Extinctions: Some Initial Propositions and Terms

Although there is considerable disagreement about the causes of late Quaternary extinctions, there is even less agreement on canons of evidence and procedures for testing conflicting hypotheses (Grayson 1984). However, in our opinion some general points about these extinctions are really not controversial. It is not controversial that a plethora of extinctions well above any plausible background rate (cf. May et al. 1995; Pimm et al. 1995) occurred after circa

40,000 yr B.P. on several continents, many shelf islands, and virtually every oceanic island, albeit at different times in different places and to vastly differing extents (Diamond 1984, 1989; Martin 1984a,b; Alcover et al. in prep.; MacPhee and Flemming in press). (Dates in "yr B.P." are usually uncalibrated radiocarbon years.) It is also not controversial that, in a given place, many and probably most extinctions were tightly grouped in time, as shown by carefully collected stratigraphic and radiocarbon evidence (e.g., Stuart 1991). Nor is it controversial that terrestrial vertebrates were overwhelmingly affected. On continents, land mammals suffered substantial losses, especially species whose body size exceeded 50 kg; on islands, all land vertebrate classes were usually affected, but the greatest losses appear to have occurred among birds (Diamond 1984, 1989; Steadman 1995). Finally, it is not controversial that there was no taxic replacement of lost faunal elements (Webb 1984; Alroy 1994).

If settled points about late Quaternary extinctions seem paltry relative to the number of investigations devoted to the topic, the reason for this is fairly clear. Because real extinctions are the biological sequelae of unique historical events, their causes cannot be recovered through precise repetition, as in a laboratory experiment. Instead, their nature must be retrieved indirectly and inductively, by confronting ideas and hypotheses about causes with quantities of superficially ill-matched, inconclusive, and often conflicting facts, in the hope that such a process will yield explanations of ever greater generality (Dretske 1971; Pappas and Swain 1978; Kitcher 1985). One way to make headway in this situation is to accept the view that some propositions have been sufficiently verified to warrant provisional acceptance. Using this criterion, one proposition that we provisionally accept is that the severest extinctions of the late Quaternary closely tracked the human diaspora, in both time and space, as originally proposed by Paul Martin (1967, 1984a,b, and elsewhere).

It might be argued that this proposition is hard to accept in certain important cases, like the extinctions in the Americas. Many workers accept the view that most or all of the terminal Pleistocene extinctions in the New World occurred soon after human arrival. However, the strictly chronometric evidence that can be quoted to support this opinion is very limited and possibly contradictory, if humans were in the Americas well before Clovis times (Gruhn and Bryan 1984). Many extinct American species lack any sort of associated radiometric record for estimating the date of their "last" appearances (Grayson 1989). True, the few that do have good records—such as Shasta sloths (*Nothrotheriops*) and sabertooth cats (*Smilodon*)—appear to overlap broadly in their "last" appearance dates, which center on circa 11,000 yr B.P. (Martin 1984a, 1990; Mead and Meltzer 1984; but see MacPhee et al. 1989). The problem of dating adequacy, although certainly crucial at one level (Meltzer and Mead 1985), is nevertheless secondary to the issue of cause. (To our knowledge, no one is taking the millenarian position that it is the dates themselves that killed.) If probable cause can be construed for this and other major extinctions, apparent differences in extinction schedules will eventually be sorted out.

Although the phrase "late Quaternary extinctions" is useful, it is imprecise because it can potentially refer to any loss occurring for any reason in the last 100,000 years. We need additional terms for characterizing losses under particular circumstances or temporal constraints.

Extinction and the Human Diaspora: Losses during "First Contact"

Analyzing losses under this heading is accomplished geographically, using as the climacteric the timing of FC. First contact in our sense refers to *initial human encounters with endemic species native to continents or islands where humans were never previously resident.* Rather than being a uniquely definable length of time, FC is an event or phase starting some time after initial human arrival and fuzzily bounded by the onset and termination of the acute faunal impact that constitutes the FC extinction event. Extinctions occurring in pre- and post-FC time are also of interest, but mainly for the comparative context they supply.

The few facts with any bearing on the matter suggest that FC extinction events have regularly gone to completion on a time scale between 1 year on small islands and 1500 years on large islands and continents (Diamond 1984; Martin 1984b; MacPhee and Burney 1991; Simons et al. 1995). Mosimann and Martin (1975) modeled regional extinctions (within 80-km radii) as occurring within ten years or less. However, complete extinctions at the species level would have taken longer, especially for species with wide distributions. Without wishing to be held too closely to dates that are perforce highly conjectural, to further the argument we will use the following FC spans for the areas of interest in this chapter: for the Americas, 12,000–10,500 yr B.P.; for the West Indies, 7000–5500 yr B.P.; and for Madagascar, 2000–500 yr B.P. The designated period for the Americas may be too short, whereas that for Madagascar may be too long. Defining any span at all for the West Indies may be futile at present, since the exiguous Antillean evidence lacks a clear FC extinction signature (MacPhee et al. 1989; Burney et al. 1994). The FC model works well for small Pacific islands (Steadman 1989, 1995; Steadman et al. 1991), but, as will be noted in more detail, it currently seems inapplicable or unworkable for Australia (Horton 1984; Flannery 1994b). These spans are meant to cover entire events, composed of many individual species losses. Presumably, the rapidity with which any individual species disappeared during a given FC event would have depended on its original population size, recruitment rate, distribution, social behavior, and like factors.

Extinction and European Expansion: Losses in the Modern Era

The period since A.D. 1500—hereafter referred to as the "modern era"—has been particularly momentous for the natural world, and it is important to recognize this in relation to extinction (Diamond 1984). Actually, 1492, or 1600, or 1768 (to memorialize the start of Cook's first major voyage of exploration), or any other defining moment chosen to mark the beginning of the modern era could be used without any loss of meaningful information. The real climacteric is the dramatic increase in empirical knowledge of the planet, including its natural history, that was occasioned by European expansion. An "event" of this sort cannot be dated to a range as narrow as a year, and we will simply use the beginning of the sixteenth century as a reasonable proxy.

We regard the difference between FC extinctions and those occurring before and after them as meaningful both biologically and interpretatively. As far as we can now tell, extinctions that occurred directly after the first arrival of people in "new" lands were vastly greater in scale and breadth

than any subsequent extinctions *in the same places*. This observation stands quite apart from any alleged causal factors recruited to explain why this pattern difference is repeatedly found.

The First-Contact Pattern

Critical features of FC extinctions can be briefly explored by reviewing fig. 7.1, which displays current understanding of where and when several major and some minor land vertebrate extinctions took place during the late Quaternary.

The pattern apparently begins in Australia–New Guinea ("Meganesia"), where a bout of mammal extinctions started at least 35,000 yr B.P., indefinitely long after first human settlement as early as 60,000 yr B.P. (Flannery 1994b). Because of unresolved chronological problems, the beginning and end points of the Meganesian extinctions remain very hazy, and how tightly the faunal losses and human FC were linked is disputed (e.g., compare Horton 1980, 1984 with Flannery 1994a). In addition, impacts on mammals were decidedly different in the two major landmasses comprising Meganesia: although the total number of Quaternary land mammal species in Australia was somewhat greater than that in New Guinea (Flannery 1994a,b), approximately 50 species died out before the modern era in Australia, whereas only 10 disappeared in New Guinea (P. Murray 1984; Flannery 1994b).

A better, if still controversial, case can be made for tight linkage at the venue of the next major extinction event, the Americas. At 12,500 yr B.P., according to the conventional archaeological framework accepted by many but by no means all workers, human groups first began to filter through the Americas from Asia. By 11,000 yr B.P., catastrophic depletion of the land mammal fauna of the New World, as judged by variably documented "last appearances" of taxa, was in full swing. As previously noted, the few species whose "last appearance" dates are supported by exceptionally good radiocarbon records establish that the severest effects occurred shortly before 10,000 yr B.P. (Meltzer and Mead 1985; Grayson 1989; Stuart 1991). The full calamity culminated in losses of approximately 85 genera throughout the New World, with more extinctions occurring in South America than in North America (Martin 1984a,b, 1990).

In the following Holocene epoch, the pattern continued, but now with short, sharp bursts of extinction activity being concentrated in the last remaining new places for human settlement—the world's islands (Olson 1989; Steadman 1989, 1995). A sampling of these later FC extinctions is appropriate. In the Greater Antilles, colonization by human groups beginning circa 7000 yr B.P. (Rouse 1992) preceded numerous but unfortunately very poorly dated extinctions (MacPhee et al. 1989; Burney et al. 1994). In the Lesser Antilles extinctions occurred as well, but they are likewise poorly dated (Pregill et al. 1994). Later, probably circa 2000–2500 yr B.P., people reached Madagascar for the first time (MacPhee and Burney 1991); by approximately A.D. 1500—the data are shaky—approximately two dozen vertebrate species

Figure 7.1. The dreadful syncopation: some representative examples. (After Martin 1984a and elsewhere.)

Afroeurasia: No major episodes of extinction during past 100,000 yr, although some losses occurred.

Meganesia: Humans arrive 40,000–60,000 yr B.P.; major extinction episode follows, but extends to circa 15,000 yr B.P. (or later?). FC effect is obscure.

Americas: Humans arrive 12,500 yr B.P.; major extinction episode terminates circa 10,500 yr B.P., few extinctions thereafter.

Mediterranea: Humans arrive 10,000 yr B.P.; major extinction episode terminates circa 4,000 yr B.P., few extinctions thereafter.

Antillea: Humans arrive 7000 yr B.P.; major episode of extinction follows, but extends to circa A.D. 1600 (or later?). FC effect is obscure.

Madagascar: Humans arrive 2000 yr B.P.; major episode of extinction terminates circa A.D. 1500, few extinctions thereafter.
Mascarenes (Ms): Humans arrive A.D. 1600; major episode of extinction terminates circa A.D. 1900 (FC occurred in modern era).
New Zealand: Humans arrive 800–1000 yr B.P.; major episode of extinction terminates circa A.D. 1500.

Commander Islands (C): Humans arrive A.D. 1741; Steller's sea cow extinct by A.D. 1768.
Wrangel Island (W): Humans arrive ?; mammoths survive to 4000 yr B.P.
Galapagos Islands (G): Humans arrive A.D. 1535; modern-era extinctions only.

were gone (Burney 1993a,b; Simons et al. 1995). Later still, islands so isolated that they were never settled prehistorically were finally occupied by the colonizing powers of Europe. The Mascarenes, discovered prior to but not occupied until the seventeenth century, lost approximately 30 species of reptiles, birds, and mammals in the next two centuries (Hachisuka 1953; Honegger 1981; Groombridge 1992). Literally hundreds of similar examples of FC extinctions on distant islands could be cited (Steadman 1995).

On present evidence, this dreadful syncopation—humans arrive, animals disappear—seems to have occurred to a greater or lesser degree on most habitable landmasses, major and minor, except the continents of earliest human evolution, Africa and Eurasia. As is well known, Martin's (1984a,b) explanation for the lack of large, concentrated losses on these continents is that megafaunal species adapted to the evolution of predatory humans by developing and maintaining suitable behavioral avoidance mechanisms. A less-noticed feature of this chronicle, however, is that after the period of first contact and initial substantial loss in affected areas, the pattern markedly changes: extinctions either no longer occur, or continue at a greatly reduced rate, and tend to impact different groups. Although there is some degree of variability (e.g., modern-era losses, or lack thereof, in North America compared to those in Australia), existing evidence establishes fairly strongly that the rate of identifiable loss at the species level dropped precipitously everywhere after FC episodes and stayed depressed until the present era (cf. losses listed by Groombridge 1992, 1993; Cole et al. 1994; Flemming and MacPhee 1995; MacPhee and Flemming in press). This point deserves closer scrutiny.

Lessons of Modern-Era Extinctions

The leading *threats* to the preservation of biodiversity are usually conceived to be habitat destruction, exploitation, and introduction of exotic species (Groombridge 1992). But are they also, historically, the leading *causes* of extinctions? For most conservation biologists, this amounts to a distinction without a difference; a threat is simply a cause in progress, which, if left unchecked, will ultimately lead to species losses. This view seems to imply that we actually know a great deal about the etiology of extinction, but the softness of recent estimates suggests otherwise. For example, the study by the World Conservation Monitoring Centre (WCMC) states that, of the total number of post-1600 extinctions of mollusks, birds, and mammals that have an assigned cause, 39% were perhaps due to exotic introductions and 36% to direct habitat destruction (Groombridge 1992, 1993). Hunting and deliberate exterminations may account for another 23%. However, these figures cover fewer than half of all extinctions that have occurred during this period, and, in the case of mammals alone, only 30% of extinctions since 1600 have a cited cause of disappearance.

Yet despite such uncertainties, modern-era extinctions are of great interest because they provide a standard against which to compare losses in earlier times, as the following points illustrate:

1. *In the modern era, only one species-level extinction in every eight has been in the megafaunal range; by contrast, three of every four losses were in the megafaunal range in the Americas 11,000 years ago.* Table 7.1A displays all mammalian species-level losses that can be ascribed to the period between A.D. 1500 and the present,

TABLE 7.1. Species Extinctions among Mammals: Critical Lists[a]

A. Species probably or certainly extinct after A.D. 1500[b]

Taxon	Common name	Locality	Last collected/seen
1. *Hydrodamalis gigas*	Steller's sea cow	Commander Islands	1768
2. *Hippotragus leucophaeus*	Blue buck	South Africa	1799
3. *Prolagus sardus*	Sardinian pika	Sardinia, Corsica	Eighteenth century
4. *Conilurus albipes*	White-footed rabbit-rat	Australia	1840s
5. *Pseudomys gouldii*	Gould's mouse	Australia	1857
6. *Pteropus subniger*	Réunion flying-fox	Mascarene Islands: Mauritius	1866
7. *Potorous platyops*	Broad-faced potoroo	Australia	1875
8. *Dusicyon australis*	Falklands dog	Falkland Islands	1876
9. *Oryzomys antillarum*	Jamaican rice-rat	West Indies: Jamaica	1877
10. *Megalomys luciae*	St. Lucia muskrat	West Indies: St. Lucia	Before 1881
11. *Lagorchestes leporides*	Eastern hare-wallaby	Australia	1890
12. *Oligoryzomys victus*	St. Vincent pygmy rice-rat	West Indies: St. Vincent	1892
13. *Pseudomys fieldii*	Alice Springs mouse	Australia	1895
14. *Acerodon lucifer*	Panay giant fruit bat	Philippines: Panay Island	1896
15. *Gazella rufina*	Algerian gazelle	Algeria	Nineteenth century
16. *Pteropus pilosus*	Palau flying-fox	Caroline Islands; Palau Islands	Nineteenth century
17. *Notomys longicaudatus*	Long-tailed hopping-mouse	Australia	1901
18. *Megalomys desmarestii*	Martinique muskrat	West Indies: Martinique	1902
19. *Chaeropus ecaudatus*	Pig-footed bandicoot	Australia	1907
20. *Rattus macleari*	Maclear's rat	Christmas Island	1908
21. *Rattus nativitatis*	Bulldog rat	Christmas Island	1908
22. *Macropus greyi*	Toolache wallaby	Australia	1927
23. *Nesoryzomys darwini*	Santa Cruz rice-rat	Galápagos Islands: Santa Cruz Island	1930
24. *Macrotis leucura*	Rabbit-eared bandicoot	Australia	1931
25. *Lagorchestes asomatus*	Central hare-wallaby	Australia	1932
26. *Leporillus apicalis*	Lesser stick-nest rat	Australia	1933
27. *Caloprymnus campestris*	Desert rat-kangaroo	Australia	1935
28. *Perameles eremiana*	Desert bandicoot	Australia	1935
29. *Thylacinus cynocephalus*	Thylacine	Tasmania	1936
30. *Monachus tropicalis*	Caribbean monk seal	Caribbean Sea	1952
31. *Geocapromys thoracatus*	Little Swan Island coney	Honduras: Little Swan Island	1955
32. *Onychogalea lunata*	Crescent nail-tail wallaby	Australia	1956
33. *Dobsonia chapmani*	Philippine bare-backed fruit bat	Philippines: Negros Island	1964
34. *Mystacina robusta*	Greater short-tailed bat	New Zealand: Big South Cape Island	1965
35. *Pteropus tokudae*	Guam flying-fox	Marianas: Guam	1968
36. *Leimacomys buettneri*	Groove-toothed forest mouse	Togo	Not seen in 1960s
37. *Uromys imperator*	Giant naked-tailed rat	Solomon Islands: Guadalcanal	Not seen in 1980s

(continued)

TABLE 7.1. Species Extinctions among Mammals: Critical Lists[a] *(continued)*

A. Species probably or certainly extinct after A.D. 1500[b]

Taxon	Common name	Locality	Last collected/seen
38. *Uromys porculus*	Little pig rat	Solomon Islands: Guadalcanal	Not seen in 1980s
39. *Sylvilagus insonus*	Omilteme cottontail	Mexico: Sierra Madre del Sur	Not seen in 1980s
40. *Solomys salamonis*	Naked-tailed rat	Solomon Islands: Florida Island	+ (1883)
41. *Oryzomys nelsoni*	Nelson's rice-rat	Mexico: Maria Madre Island	+ (1898)
42. *Nyctimene sanctacrucis*	Tube-nosed fruit-bat	Solomon Islands: Santa Cruz Island	+ (1931)
43. *Peromyscus pembertoni*	Pemberton's deer mouse	Mexico: San Pedro Nolasco Island	+ (1932)
44. *Notomys amplus*	Short-tailed hopping-mouse	Australia	+ (1936)
45. *Boromys offella*	—	West Indies: Cuba	*
46. *Brotomys voratus*	—	West Indies: Hispaniola	*
47. *Heteropsomys antillensis*	—	West Indies: Puerto Rico	*
48. *Capromys*, unnamed species	Cayman hutia	West Indies: Cayman Islands	*
49. *Geocapromys*, unnamed species 1[c]	Grand Cayman coney	West Indies: Cayman Islands	*
50. *Geocapromys*, unnamed species 2[c]	Cayman Brac coney	West Indies: Cayman Islands	*
51. *Hexolobodon phenax*	—	West Indies: Hispaniola	*
52. *Isolobodon montanus*	—	West Indies: Hispaniola	*
53. *Isolobodon puertoricensis*	—	West Indies: Puerto Rico	*
54. *Megaoryzomys curioi*	—	Galápagos Islands: Santa Cruz Island	*
55. *Megaoryzomys*, unnamed species[c]	—	Galápagos Islands: Isabela Island	*
56. *Nesoryzomys*, unnamed species 1[c]	Rabida Island rice-rat	Galápagos Islands: Rabida Island	*
57. *Nesoryzomys*, unnamed species 2[c]	Isabela Island rice-rat "A"	Galápagos Islands: Isabela Island	*
58. *Nesoryzomys*, unnamed species 3[c]	Isabela Island rice-rat "B"	Galápagos Islands: Isabela Island	*
59. *Nesophontes hypomicrus*	—	West Indies: Hispaniola	*
60. *Nesophontes micrus*	—	West Indies: Hispaniola	*
61. *Nesophontes paramicrus*	—	West Indies: Hispaniola	*
62. *Nesophontes zamicrus*	—	West Indies: Hispaniola	*
63. *Nesophontes*, unnamed species 1	—	West Indies: Cayman Islands	*
64. *Nesophontes*, unnamed species 2	—	West Indies: Cayman Islands	*
65. *Plagiodontia araeum*	—	West Indies: Hispaniola	*
66. *Plagiodontia ipnaeum*	—	West Indies: Hispaniola	*
67. *Quemisia gravis*	Quemi	West Indies: Hispaniola	*
68. *Solenodon marcanoi*	—	West Indies: Hispaniola	*

TABLE 7.1. Species Extinctions among Mammals: Critical Lists[a] *(continued)*

B. Taxa of uncertain validity or disappearance date[d]

Taxon	Locality	Reason
Crateromys paulus	Phillipines: Illin Island	SU
Geocapromys columbianus	Cuba	EU, SU
Hippopotamus madagascariensis[e]	Madagascar	EU
Hippopotamus lemerlei	Madagascar	EU
Kerivoula africana	Tanzania	SU
Malpaisomys insularis[f]	Canary Islands: Fuerteventura Island	EU
Megaladapis sp.	Madagascar	EU
Megalomys audreyae	West Indies: Barbuda	EU
Oryzomys sp.	West Indies: Barbados	SU
Nesophontes longirostris	West Indies: Cuba	SU
Nesophontes major	West Indies: Cuba	SU
Nesophontes submicrus	West Indies: Cuba	SU
Nesophontes superstes	West Indies: Cuba	SU
Nesoryzomys swarthi	Galápagos Islands: Santiago Island	SU
Notomys macrotis	Australia	SU
Notomys mordax	Australia	SU
Pitymys bavaricus	Germany	SU
Pteropus brunneus	Australia: Percy Island	SU
Solenodon arredondoi	West Indies: Cuba	SU

(continued on p. 180)

according to criteria used by MacPhee and Flemming (in press). The total comes to approximately five dozen species for which there is some positive evidence of extinction in the last 500 yr; another dozen or so are separately listed as "unresolved" as to time of extinction (table 7.1B). Other recently published lists differ marginally in their counts (e.g., Groombridge 1992, 1993; Cole et al. 1994), largely because there are no agreed-upon criteria for including or excluding taxa for which critical information is missing (cf. McCoy 1994).

The lists just cited are empirical in nature and therefore do not account for losses for which there is no direct evidence. For example, some small-mammal losses may still remain to be discovered in the terminal Pleistocene of North America, but experience suggests that such additions will be few. By contrast, it is likely that a number of taxa will be added to the modern-era total, if only because it will become increasingly imperative to use some device to enroll the very recently disappeared (e.g., taxa not seen in five decades and presumed extinct according to a rule adopted by the Convention on International Trade in Endangered Species [CITES]). It is predictable that nearly all such additions will be small mammals (Diamond's [1984] "minifaunal extinctions"). Including them will shift the modern-era ratio in a direction that will further underscore the apparent contrast between Pleistocene losses and recent ones. The contrast would be even starker if extinctions were listed at the level of genus rather than species (cf. lists compiled by Martin 1984b and Graham and Lundelius 1984).

The modern-era record of extinction is bound to be better than any paleontological record, if only because the systematist's

TABLE 7.1. Species Extinctions among Mammals: Critical Lists[a] *(continued)*

C. Taxa that should be excluded from list of modern-era extinctions[g]

Taxon	Locality	Reason
Amblyrhiza inundata	West Indies: Anguilla, St. Martin	CP
Brotomys contractus	West Indies: Hispaniola	S
Boromys torrei	West Indies: Cuba	S
Bubalus mephistopheles	China	CP
Cervus schomburgki	Thailand, Laos	E
Clidomys osborni	West Indies: Jamaica	CP
Clidomys parvus	West Indies: Jamaica	CP
Crocidura goliath	Africa: Central Africa	E
Elasmodontomys obliquus	West Indies: Puerto Rico	CP
Equus quagga	Africa: South Africa	ES
Eschrichtius gibbosus	North Atlantic	ES
Heteropsomys insulans	West Indies: Puerto Rico	S
Nesophontes edithae	West Indies: Puerto Rico	CP
Plagiodontia velozi	West Indies: Hispaniola	ES
Procyon gloveralleni	West Indies: Barbados	ES
Puertoricomys corozalus	West Indies: Puerto Rico	CP
Rhizoplagiodontia lemkei	West Indies: Hispaniola	CP
Scotophilus borbonicus	Mascarene Islands: Réunion	ES
Sphiggurus pallidus	West Indies (type locality unknown)	ES
Tolypeutes tricinctus	Brazil	E

[a]Details of species losses and criteria for inclusion or exclusion of taxa are given in a somewhat different format in MacPhee and Flemming (in press).

[b]Only those species are listed whose taxonomic distinctiveness and loss subsequent to A.D. 1500 can be documented (usually meaning that the species is well characterized taxonomically and has been recently sought but not found). Species surviving exclusively in captive or transplanted situations (e.g., survival in New Zealand of the parma wallaby, no longer in existence as a species-level taxon in Australia) are considered to be still extant. *Key:* dates in the format "1768," date of last collection or reliable sighting; dates in the format "+ (1936)," species known mammalogically, but extinction time uncertain (date in parenthesis = date of species description, which provides the maximum age at which extinction could have occurred); *, species known only paleontologically, but presumed to have died out subsequent to 1500 because bones have been found in association with those of introduced exotics (usually *Mus* or *Rattus* in New World contexts), or radiometrically dated to that period, or both.

[c]Some taxa tentatively listed in part A have not been formally described and therefore their apparent loss is technically not confirmable. *Nesoryzomys* is particularly problematic because of disagreement among authorities as to the number of valid species (see discussion by Musser and Carleton 1994).

[d]Taxa listed may have disappeared in the past 500 years but cannot be included unequivocally in part A because (SU), taxonomic status uncertain or no recent revisionary work on this species or its presumed relatives or (EU), no reliable information that extinction occurred after A.D. 1500.

[e]The Malagasy hippo species *madagascariensis* belongs in *Hippopotamus,* not *Hexaprotodon* (Steunes 1989).

[f]*Malpaisomys insularis* has been found in apparent association with *Mus* paleontologically, but the latter murid may have been introduced to the Canary Islands as much as 2000 years ago (Hutterer and Oromí 1993; Boye et al. 1992).

[g]Taxa listed do not qualify as post-1500 extinctions but have been included in lists of modern-era extinctions assembled by other workers (e.g., Cole et al. 1994). *Key:* CP, species became extinct before A.D. 1500 according to available evidence; E, name valid, species is extant; ES, name invalid, species is extant; S, systematic status unclear at time of writing (for resolution see MacPhee and Flemming [in press]).

ability to resolve species differences increases when specimens are represented by more than a sample of bones. Several species on the modern-era list of losses (e.g., *Perameles eremiana, Pseudomys fieldii, Dobsonia chapmani*) are difficult to separate osteologically from their closest affines even when good comparative material is available; in any typical paleontological context, these species would probably be systematically invisible. Although the "real" number of late Quaternary extinctions of mammals has probably been underestimated paleontologically, no satisfactory correction method appears to exist (for a treatment of this question relevant to Madagascar, see Tattersall 1993).

2. *Most of the groups that lost heavily in FC extinctions suffered few or no later losses.* Table 7.1 includes only a handful of artiodactyls; no perissodactyls (unless the doubtfully distinct *E. quagga* is counted); no proboscideans or xenarthrans; only two carnivorans; and so on. It thus markedly differs from the list for the terminal Pleistocene of North America, which features heavy losses among all these taxa. By further contrast, the loss list for modern-era extinctions is headed by rodents and bats; on the confirmed-extinction list (table 7.1A), these two groups together contribute 65% of the total number of modern-era losses. On Graham and Lundelius's (1984) list of extinct North American species, bats and microfaunal rodents account for only 17% of all losses in the terminal Pleistocene. These figures are of considerable interest: although island rodents were frequent casualties of FC extinctions, island bats were apparently not. Many local extirpations of bats have been and will no doubt continue to be documented (e.g., Morgan and Woods 1986; Steadman and Koopman 1995), but paucity of investigations does not seem to be the reason for relative underrepresentation of bat species in FC extinctions.

3. *The rate of loss of species has altered, probably more than once.* Whereas in the continental Americas terrestrial megafaunal species were substantially affected in the period of FC extinction around 11,000 years ago, this size group has suffered no confirmed losses at the species level since then. Supposed examples of "late" megafaunal losses in South America, like the alleged survival of *Mylodon* into the mid-Holocene in southern Patagonia (Marshall et al. 1984), have not withstood close examination (Markgraf 1985). According to the International Union for the Conservation of Nature and Natural Resources (IUCN) survey (Groombridge 1993), the only confirmed recent mammal losses at the species level in the Americas (exclusive of the West Indian islands) include a seal, *Monachus tropicalis* (Caribbean Sea) and four terrestrial species from shelf or near-shelf islands—a canid, *Dusicyon australis* (Falkland Islands); a hutia, *Geocapromys thoracatus* (Little Swan Island, Honduras); a rice rat, *Oryzomys nelsoni* (Maria Madre Island, Mexico); and a deer mouse, *Peromyscus pembertoni* (San Pedro Nolasco Island, Mexico). These losses (none of which is "continental" in the narrow sense) occurred in the nineteenth and twentieth centuries and are therefore very late within the modern era. What, if anything, should be made of the existence of the 10,000-year hiatus that precedes these minor exterminations, for which no species-level extinctions can be confirmed?

The problem of unrecognized extinct species has already been mentioned. Perhaps our records are simply inaccurate, and there were a number of extinctions in the Americas during the Holocene that have gone

unrecorded paleontologically. For the reasons cited earlier, some losses have surely been missed, but most of those probably pertain to small mammals. It may be objected that in any case the question is inappropriate, because during this period many American species have suffered massive reductions at the population/deme level and are arguably in imminent danger of complete extinction. This point is undoubtedly true, but to make it only reinforces the mystery: why did the number of complete extinctions at the species level drop so precipitously, if indeed catastrophic reductions at the population level kept occurring throughout the Holocene? A possible explanation is that the effective agency of earlier extinctions eventually ceased to operate on its former scale, whereas other factors may be responsible for recent depletions but have not yet prompted copious species-level extinctions.

Most large islands that had significant FC extinctions of mammals in prehistoric times have experienced few if any losses in the modern era (Alcover et al. in prep.): examples include New Guinea, the Balearics, and Sardinia. Madagascar may also belong on this list, depending on actual dates of disappearance of several subfossil taxa (Simons et al. 1995; Goodman and Rakotondravony 1996; S. M. Goodman pers. comm.; E. L. Simons pers. comm.). By contrast, islands discovered and occupied very late in human history, such as Mauritius and the Galápagos, provide a number of entries for modern-era vertebrate extinction and extirpation lists, as would be expected for lands that have only just passed through the FC filter (Arnold 1980; Cheke and Dahl 1981; Honegger 1981; Steadman et al. 1991; Groombridge 1992; Hutterer and Oromí 1993). However, overall the number of recent mammal losses on "new" islands is not large, because such islands tend to be comparatively small and isolated with few resident species.

This analysis purposely concentrates on mammals; patterns may be different for other groups. For the world avifauna, confirmed losses of full species are approximately twice that registered for mammals during the modern era (Groombridge 1992). However, Steadman (1995) estimated that the actual number of FC-style losses among birds might be 2000 or more, many of which would have occurred during the past 500 years. This number is an order of magnitude greater than mammalian losses as conventionally counted, and it would tend to imply that birds living on oceanic islands are less resistent to factors inducing extinction. It is therefore important to note that although Steadman (1995) contends that every island-bound bird population (e.g., the ubiquitous flightless rails of the Pacific) should be counted as a distinct entity, he does not address whether they can be physically distinguished from one another as separate species. We accept that there is rhetorical merit in bringing attention to population-level losses, since these are the logical prelude to complete loss of species (Ehrlich 1995). However, population-level losses are much harder to document in paleontological contexts, and for the present we shall continue to use the diagnosable species as the basic unit of analysis.

In the case of Madagascar, where no mammal species are known to have died out in the last four centuries, three birds have disappeared (*Aepyornis maximus, Tachybaptus rufolavatus,* and *Coua delalandei*) since A.D. 1600 according to the WCMC survey (Groombridge 1992). (*Aepyornis maximus* remains dubious as a modern extinction, because there is no accepted evidence supporting the survival of

any elephantbird species into modern times.) Goodman and Rakotozafy (this volume) have discovered or newly confirmed five more extinctions from subfossil remains. How many of these losses occurred after A.D. 1500–1600 is unclear, however, partly because of dating and association problems. Notwithstanding the fact that a significant proportion of the Malagasy avifauna is clearly highly endangered (Langrand 1990), the point remains that a strong case for numerous complete extinctions in the avifauna well after A.D. 1500 cannot yet be made. The few facts available seem to be just as consistent with a scenario of rapid losses among the birds at the same time as the large mammals and chelonians were disappearing in the period prior to the modern era.

4. *Some areas have experienced greater rates of loss than others have in the modern era.* The West Indies and Australia provide an important contrast to low losses in North America during the modern era: nearly 60% of all confirmed mammalian extinctions in the past 500 years took place in these two areas (table 7.1). In Australia, at least 17 confirmed extinctions have occurred in the last 150 years, most of them involving small to medium-sized ground-dwelling species (Burbidge and McKenzie 1989). This is probably a minimum count; some authors (e.g., Flannery 1994a,b) argue that nearly two dozen species have been lost, although we prefer to be conservative about the reality of several nominal taxa until appropriate revisionary work has been published. As in the case of North America, there is a long period for which there are no confirmed extinctions (~15,000 yr B.P. to circa A.D. 1800). This may be because none occurred, or more probably it is because there were comparatively few of them and they have gone unrecognized. (Local extirpations that occurred well before final disappearance of the species concerned, such as the loss of *Thylacinus cynocephalus* on the mainland of Australia within the last 4000 years, are not counted in our tally.)

Approximately 82% of all endemic terrestrial mammals in the West Indies have disappeared, but there are significant uncertainties about the date of final disappearance of many taxa (Morgan and Woods 1986; MacPhee et al. 1989; Woods and Eisenberg 1989). It is widely assumed that many taxa, including megafaunal sloths and rodents, became extinct shortly after aboriginal settlement, but the number of good-quality radiometric dates that can be cited to support this inference is negligible (MacPhee et al. 1989). Losses among West Indian tardigrades and caviomorphs are of interest in the context of global megafaunal extinctions, because very large species evolved within these groups (*Megalocnus rodens* from Cuba, estimated 200 kg body size [Paula Couto 1967]; *Amblyrhiza inundata* from Anguilla and St. Martin, estimated 80–150 kg body size [Biknevicius et al. 1993]). Associations between cultural detritus and remains of extinct animals have been reported for sites in Hispaniola (Miller 1929a,b) and Cuba (Suarez et al. 1984; Pino and Castellanos 1985). Although some of the associations may be judged suspect (P. S. Martin pers. comm.), it seems improbable that all or even the majority are. It is indisputable that humans were in Hispaniola and Cuba no later than circa 7000 yr B.P., because on both islands there are radiometrically dated archaeological sites of that age (see Burney et al. 1994; see also Rouse 1992). The youngest sloth-bearing cave fauna in the Greater Antilles may be Cueva Musulmanes in Holguín Province,

Cuba, although the only basis for so concluding is a date of 2410 ± 40 yr B.P. using the nonradioisotopic "collagen method" (Pino and Castellanos 1985). The youngest ^{14}C date for a sloth site in Hispaniola is 3715 ± 175 yr B.P., reported by Woods (1989; see also MacPhee and Woods 1982). This date indicates that sloths survived thousands of years after human arrival.

It is also reasonably clear that at least some losses in the West Indies—interestingly, mostly of microfaunal species—occurred relatively recently (Pregill et al. 1994). Skeletal remains of several of the endemic rodents and insectivores of the Greater Antilles have been found mixed together with introduced *Rattus,* sometimes in owl pellets in which prey hair is still intact (Miller 1929a, 1930). Although none of this material has yet been radiometrically dated, the circumstantial evidence warrants accepting these as modern-era losses (see table 7.1A). As many as 10–16 microfaunal species may have survived for a time after European occupation of the islands, thousands of years after initial aboriginal settlement. The West Indian extinctions may therefore parallel the Australian experience, if it is true that losses were spread out over several thousand years in both places.

In summary, areas affected by coordinated extinctions in the late Quaternary tend to present the following pattern: rapid and dramatic losses soon after human arrival, followed by a period of quiesence until the present century, when the pace of extinction may have accelerated again (Wilson 1992; Ehrlich 1995). Australia and the West Indies may represent exceptions to this pattern because their loss periods seem to have been greatly protracted compared to most other FC contexts, but the evidence needed to settle this point is inadequate at present. (Note that protracted extinction schedules in areas of high endemism experiencing great environmental impacts would run counter to prevailing theory [e.g., Pimm et al. 1995] concerning how such losses accrue.)

If, as seems reasonably certain, humans were on the scene when most late Quaternary extinctions were taking place in various parts of the world, it is natural to assume that they were implicated in some manner. However, the marked fall-off in the extinction rate in most places after the period of FC losses needs to be explained better than it has been to date. A comprehensive theory of late Quaternary extinction must provide for the entire pattern, not just the period of sweeping losses.

Blitzkrieg or Hyperdisease? A Comparison of Expectations and Implications

> I will lay aside impartiality. I think the overkill theorists have the more convincing argument for what happened in America 10,000 years ago. It seems likely that the Clovis people spread through the New World and demolished most of the large mammals during a hunters' blitzkrieg spanning several centuries.
> —E. O. Wilson (1992, p. 249), after comparing competing explanations of late Quaternary extinctions in the New World

When extremely lethal disease threatens a naive population, the infection rate and the mortality rate become virtually the same because essentially all infections terminate in death. Eventually, there will be no lethally infectable members of the species left. "No lethally infectable members left" does not mean that all individuals of the species vulnerable to the disease have already ex-

pired. In any real case, some individuals will never be infected and others will survive infection. However, if the interbreeding population size drops below a certain level, or if survivors are widely dispersed, in theory extinction could ensue strictly because the replacement rate is too low to sustain the species in the postpanzootic period. This, in a nutshell, is the expected outcome of a hyperdisease epizootic.

Martin's (1984b) blitzkrieg hypothesis has a similar expectation, but the mode of causality is different: all or nearly all megafaunal animals encountered by expanding human populations die as a consequence of overhunting. Indeed, the parallel that must now be acknowledged is that blitzkrieg is essentially descriptive of the epizootiology of a disease of unusual lethality. "Overkill" is simply the descriptive outcome under both hypotheses; in other words, it describes a result, not a process. Whether this result was obtained in the majority of FC contexts by real pathogens carried in by humans, their commensals, or synanthropics, or by *Homo sapiens* acting in the manner of a pathogen, are the competing explanations for the available facts that must now be confronted.

Although overkill is the fundamental pattern of both blitzkrieg and hyperdisease, they may be partly distinguished by examining how the available data accord with assumptions and expectations. In this section we explore this point by analyzing Martin's (1984b, p. 358) "eight attributes of late Pleistocene extinction which seem especially noteworthy." These attributes comprise Martin's catalogue of critical pattern features that must be accounted for by any explanation of late Quaternary extinction that purports to be comprehensive. In Martin's view (1984b, p. 360), "Taken together, the eight features of the late Pleistocene are compatible with a model of overkill. . . . They are not features one would necessarily encounter if phyletic replacement, faunal turnover, or climatic change were primary causes of late Pleistocene losses." In our view, hyperdisease is more compatible with the features Martin identifies than is blitzkrieg.

Why Were Large Species Differentially Affected?

Taken together, Martin's features 1, 2, and 5 (large mammals were decimated; continental rats survived, island rats did not; regional extinctions were diachronous) imply that there was a selective agent influencing late Quaternary extinctions. According to Haynes's (1991) evenhanded review of the history of publication on the matter, it has often been assumed that human hunters were largely responsible for the mass death assemblages of proboscideans and bison found in various parts of North America. Since these assemblages usually consist of animals of all ages (i.e., they are instantaneous or catastrophic assemblages), overkill supporters are forced to conclude that the humans doing the hunting either must have been indiscriminate in their kill choices or for some reason were interested in killing individuals from any and all age classes. McDonald (1984, p. 426), favoring the latter interpretation, argued that tools allowed paleo-Indians to "prey not only upon juveniles, aged, or infirmed individuals (groups usually culled by predators) but also healthy individuals, adults and entire herds of bison and possibly other herding prey." This provides a mechanism, of course, but not a reason: why expend energy in bringing down many times the number of animals that can be processed in a reasonable period of time (Silberhauer

1981)? If the "tool" is a buffalo jump or the equivalent, it can be argued that there was no excess expenditure of energy, since in effect the stampeding herd slaughtered itself. The trouble is that such a tool is valuable only for bringing down herding species within an obliging geomorphological landscape (cf. Frison 1988).

In the case of Madagascar the picture is even more obscure. It is plausible that the hippos, the elephantbirds, and possibly the giant tortoises formed substantial concentrations of individuals, at least from time to time, by analogy with East African hippos, South African ostriches, and Seychellois tortoises. But there are no large-scale kill sites (or at least none recognized as such) for these species (Dewar 1984). Equally, whereas one could selectively cite analogical evidence to buttress the view that many extinct lemurs formed long-lasting social groups, this explanation seems less likely for species like *Daubentonia robusta* (or, for that matter, *Cryptoprocta spelea, Hypogeomys australis,* and *Plesiorycteropus madagascariensis*).

From the perspective of the hyperdisease hypothesis, an animal's risk of infection is the chief selective factor. In general, risk increases with proximity to outbreak foci, but age is also critical because it is frequently the oldest and youngest members of a population or species that are at greatest risk. In natural populations, the number of truly "old" (=postreproductive) individuals will ordinarily be small to negligible. Their loss is not critical to population maintenance. Mammalian neonates, however, are immunologically immature and may be expected to suffer disproportionately from hypervirulent infection. In the context of late Quaternary extinctions, high wastage of newborn or young animals as a consequence of lethal disease might have had very severe effects on population survival, especially if the disease could resurface repeatedly from biological reservoirs, affecting susceptible individuals until most were dead (see "Modeling Hyperdisease"). This entire set of circumstances would seem to be more dangerous for large taxa, which characteristically have lower reproductive rates, longer gestations, and smaller progeny sizes. This is, of course, a variation on the reduced relative fitness argument (McDonald 1984), and, at the level of abstraction adopted here, overselection by hyperdisease has the same outcome as overselection by overhunting—i.e., extinction, especially of taxa that have the life history characteristics of large mammals.

A possible difference between blitzkrieg and hyperdisease concerns expectations regarding the effect of the selective agent on smaller species. Seriously incompatible with Martin's (1984b, p. 360) overkill model for North America would be the extinction of many "animals too small or ecologically resistant to be vulnerable to human impact." McDonald (1984) believes that paleo-Indians would have hunted smaller species from time to time, but "they probably preferred larger game." In fact, the big-game hunting model of direct impact extinctions practically mandates this preference: otherwise, there would be no rational explanation for the fact that species of large body size were everywhere the worst affected. But body size does not similarly constrain hyperdisease, because at the level of the individual there is no reason to believe that small body size would confer resistance. Yet the life history characteristics that generally go along with small body size (higher reproductive rates, shorter gestations, more young per litter) would certainly have conferred *resiliency* at the population/species level. The possibility, in FC episodes, that

TABLE 7.2. Body Mass Comparisons of Malagasy Terrestrial Amniotes

Body mass (kg)	Extinct taxa	Extant taxa	Extinction index (%)
≤ 5	*Daubentonia robusta* *Hypogeomys australis*	Numerous (100+ spp.)	~1
> 5–10	*Varecia insignis* *Mesopropithecus pithecoides* *Mesopropithecus globiceps* *Babakotia radofilaoi* *Archaeolemur majori* *Archaeolemur edwardsi* *Plesiorycteropus germainepetterae*	*Propithecus diadema* *Indri indri* *Cryptoprocta ferox* *Testudo radiata* *Testudo yniphora*	58
> 10–100	*Hadropithecus stenognathus* *Palaeopropithecus ingens* *Palaeopropithecus maximus* *Megaladapis edwardsi* *Megaladapis madagascariensis* *Cryptoprocta spelea* *Plesiorycteropus madagascariensis* *Crocodylus robustus* *Aldabrachelys abrupta*	*Potamochoerus larvatus* *Crocodylus niloticus*	82
> 100	*Archaeoindris fontoynonti* *Hippopotamus lemerlei* *Hippopotamus madagascariensis* *Aepyornis* (?5 spp.) *Mullerornis* (?2 spp.) *Aldabrachelys grandidieri*	—	100

small species were equally vulnerable to the factors that caused complete extinction of larger species cannot be dismissed merely by observing that the former managed to survive. Smaller species might also have been heavily affected, but survived bottlenecks because their life history strategies permitted high wastage of individuals.

With regard to species of small body size, it is not yet clear whether hyperdisease is consistent with more of the facts in need of explanation than is blitzkrieg. On islands, mammals in all size ranges became extinct during periods of FC loss, a finding that is hard to explain if hunting alone was the efficient cause of extinction. In the West Indies, for example, every land mammal above 2–3 kg is now extinct. The range is enormous, running from ~150–200 kg in the case of the largest sloths and rodents to <100 g in the case of the smallest of the shrewlike insectivores (*Nesophontes* spp.). The range is significantly narrower in the case of Madagascar (table 7.2). All mammals having body sizes >12 kg are extinct except for the bush pig (*Potamochoerus larvatus*), which is essentially indistinguishable from its East African counterpart and is probably a human import (but see Kingdon 1979). At present there are no confirmed instances of extinction among the insectivores and smaller rodents and lemurs (MacPhee 1994), but this point has never been adequately investigated and may be incorrect (S. M. Goodman pers. comm.). Unlike the case with Madagascar, no recent worker studying West Indian losses has suggested that human hunting was the perva-

sive cause of extinction, except perhaps in the case of the sloths. For the smaller fauna, the introduction of exotics and climate change are favored agents of destruction (see Morgan and Woods 1986). Nevertheless, Martin (1984b) prefers blitzkrieg and "more gradual" anthropogenic effects to explain Antillean extinctions. For hyperdisease to have been a contributory or even the main factor, one has to imagine multiple, extraordinarily lethal panzootics involving many species.

Certain serovars or species of some microbes (e.g., *Plasmodium, Mycobacterium, Yersinia*) are able to infect birds as well as mammals, so it is not automatically implausible that bird species died out in FC episodes in hyperdisease situations that principally affected mammals. There are also diseases carried by domestic fowl that can transfer to wild species (e.g., Newcastle disease, falcon herpesvirus disease; Hanson 1976; Maré and Graham 1976). This is of interest because remains of introduced species (chicken and guineafowl) were found in faunal collections from Tsiandroina and Ambolisatra by Goodman and Rakotozafy (this volume).

Why Did Most Late Quaternary Extinctions Occur outside Eurasia and Africa?

Pattern features 3, 6, and 7 (large mammals survived best in Africa; extinctions occurred without replacement; extinctions followed man's footsteps) are consistent with Martin's (1984b) inference that the Afroeurasian megafauna developed appropriate behavioral mechanisms to deal with the evolving human predator (fig. 7.1). It is Martin's contention that, as a result, this group did not experience extinction to nearly the same extent that behaviorally naive faunas did elsewhere in the world.

We suggest that the reason there were few extinctions in Africa and Eurasia could be genetically determined lack of susceptibility rather than any aspect of natural behavior. In those areas, hominids and other animals were exposed to, and exposed each other to, the same pool of diseases over millions of years. As a result, fewer species collapses would have occurred in Africa and Eurasia owing to "new" disease than in places where the fauna was genetically unprepared.

Both hyperdisease and blitzkrieg fail to explain certain anomalies of survival in the paleontological record. Proboscideans whose ranges straddled Asia and North America (e.g., *Mammuthus primigenius*) all died out, but under the terms of either model the Asian members should have survived. Remarkably, an isolated population of mammoths did survive on Wrangel Island until the mid-Holocene (fig. 7.1); unfortunately, it does not seem to be known when FC with humans might have occurred there (Vartanyan et al. 1993). It would be of great interest to know if there were similar holdouts on other islands in the Russian and Canadian Arctic.

Another apparent anomaly of survival is *Saiga tatarica,* an Old World bovid that crossed during the late Quaternary into North America, where it subsequently disappeared in the terminal Pleistocene (Kurtén and Anderson 1980). This is an anomaly not only because saiga and humans would be regarded as coadapted under both models, but also because the existing American megafauna is overwhelmingly composed of species that were either recently derived from Asian populations or always well distributed across Holarctica (Kurtén and Anderson 1980). If Eurasian geographic origin per se had any bearing on the likelihood of survival, then the loss of saiga populations in North America is difficult to explain.

As a complication for the hyperdisease hypothesis, it may be noted that species that have been associated over geologically long periods of time are not immune to outbreaks of "new" diseases. For example, the viruses causing "emerging" diseases in humans, such as Ebola virus and some other hemorrhagic fever viruses, must have jumped from their as-yet-unidentified natural hosts very recently—despite the fact that humans and the original hosts have presumably lived in the same areas of Africa for millennia. On the other hand, the ability of humans to penetrate deep tropical forest and sustain themselves there in large numbers is comparatively recent (Beran and Steele 1994). Quite possibly the natural reservoirs of Ebola and some other emerging diseases of humans are taxa that were not regularly encountered by humans in previous epochs—an analogue of "first contact."

Why Are Sites of Mass Mortality Ambiguous as to Cause of Death?

The most unusual of Martin's (1984b) pattern features is his eighth, which is that "the archaeology of extinction is obscure." Martin was referring to the fact that remains of now-extinct mammals are rarely encountered in human habitation sites in North America, where many terminal Pleistocene extinctions occurred, whereas they are common in sites of various ages in Eurasia and Africa, where complete extinctions were rare during the late Quaternary. He tried to convert this absence of remains of extinct animals in archaeological contexts from a liability for blitzkrieg into a virtue, using the following principle: "Given the rapid rate at which highly vulnerable and previously unhunted prey might be destroyed by the first invading human hunters, the chance of finding appreciable evidence of man's deadly passage is small" (Martin 1984b, p. 379; see also Mosimann and Martin 1975).

Martin's (1984b, p. 360) basic argument is that "lack of kill or processing sites . . . would be expected if man's impact were truly swift and devastating." However, it could be argued that the deposition of carcasses in a swift extinction event differs little from the deposition of rare earths or tephras in single-event horizons. Barring reworking, such items will be commonly encountered at, and only at, surfaces in existence at the moment in time when the items were being deposited, and, unsurprisingly, will be rare or absent at all others. Granted, catastrophic assemblages of bones will never be distributed as densely or continuously as a volcanic ash, and it may never be practicable to identify the "12,000 to 10,500-yr B.P. surface" in North America using such unpromising marker material. On the other hand, if a blitzkrieg actually occurred in North America, then we are speaking of the annihilation, in the space of a few centuries or so, of the entire standing crops of at least 71 mammal species (Kurtén and Anderson 1980; Graham and Lundelius 1984)—a biotic holocaust that we estimate must have consumed between 10^7 and 10^9 individual animals. It thus seems odd to us to assert that this interval of time is so narrow that it may never be sufficiently intersected, paleontologically or archaeologically, to permit the drawing of any meaningful conclusions. We think that sufficient evidence is already in hand, thanks in large measure to Martin's painstaking analyses (1984a, 1990), although he interprets the absence of evidence of numerous mass-kill sites differently. As Haynes (1991) has noted in connection with mammoth remains at sites in North America, the skeletons themselves do not

regularly display signs of butchery or any other indication that they were processed in any manner (e.g., dismemberment), even when artifacts are associated. Mammoth "hunters" may in fact have been mammoth scavengers who opportunistically collected meat from animals already dead. The presence of spirally fractured long bones may well imply human processing, but nonanthropogenic breakage by other animals or by geomorphological processes often cannot be excluded (Binford 1981; Dunbar et al. 1990). In any case, this kind of evidence is ill suited to the problem of establishing that humans actually did the killing, which is the issue under discussion. At present, the question of whether the majority of proboscidean catastrophic assemblages are die-offs or kill-offs seems to us to be beyond resolution using categories of evidence currently available.

In the case of Madagascar, it is probably true that not enough sites have been excavated within the 1500–500 yr B.P. envelope to make useful comparisons with scenarios for North America (cf. Dewar 1984; MacPhee 1986). Butchered hippo bones from Lamboharana and a few other sites in the southwestern part of the island provide a minimum date for the start of human-faunal interaction at circa 2000 yr B.P. (MacPhee and Burney 1991). A few other examples of modified bones and teeth are known (MacPhee and Raholimavo 1988; H. James and D. Burney pers. comm.), but these have not been dated.

Why Did Extinction Rates in Affected Places Level Off after First-Contact Times?

In all FC contexts, the period of rapid loss eventually ended, and we need to know why. Martin's (1984b) attributes 4 and 5 (extinctions could be sudden; regional extinctions were diachronous) imply that large losses eventually stopped but do not specify why this point is critical. In the blitzkrieg scenario, by the beginning of the Holocene human groups had either a reduced capacity to cause extinction because so many losses had already occurred, or else a reduced interest in excessive hunting because they were increasingly turning to other means of subsistence (see McDonald 1984; Martin 1984b). But the facts demand a more compelling answer: how could the first American Indians with their small numbers and limited toolkits cause dozens of extinctions in a few centuries, whereas their descendants failed to prompt a single one in the subsequent 10,000 years? Since aboriginal hunting continued in parts of the continent into European times, increasing sedentism (Rick 1980; Martin 1984b) is not a satisfactory explanation. Denevan (1992, p. 373) provocatively states that Amazon rain forests "are largely anthropogenic in form and composition." This may be too extreme a view for general adoption, but if it is allowed that Indians were modifying environments to suit themselves throughout the Holocene, then why did this activity fail to prompt any extinctions? Aboriginal habitat modification has certainly been argued in extinction scenarios for Madagascar (Dewar 1984) and Meganesia (Merrilees 1984; Flannery 1994a,b). If the argument is meritorious and of general application, it is hard to understand how it was that Holocene Indians did no harm but their Pleistocene forebears caused unprecedented despoliations.

Hyperdisease removes the apparent paradox by offering the alternative explanation that, after catastrophic FC losses, what was left of the original fauna consisted of nonsusceptible and less-susceptible taxa that

had managed to pull through disease-induced bottlenecks. According to this argument, moose, elk, musk-oxen, white-tailed deer, and other surviving megafauna must represent either resistant species or bottleneck survivors, and it would be interesting to know which is which. The advantage of this alternative view is that it avoids the need to assume that the objectives and styles of aboriginal hunting were radically different before and after 10,000 yr B.P., the presumed end point of the megafaunal extinctions in the Americas. It also permits the conclusion that aboriginal Americans may *never* have produced extinctions solely as a result of unrestricted hunting.

There is another aspect to this question that relates to the significance of modern-era extinctions for understanding the extinction process. Overkill explanations assume that, past a certain point of reduction, populations (and therefore species) slide ineluctably toward extinction. This is a necessary precondition, since under no conceivable real-life scenario could paleo-Indians or paleo-Malagasy have slaughtered every last mammoth or *Megaladapis*. If overhunting directly causes extinction after a certain proportion of losses are incurred (which we may figuratively call "Martin's limit"), then it should be possible to point to other cases, peppered through historical times, in which Martin's limit has been overstepped. Although the collapse of Steller's sea cow (Nowak 1991) and several other taxa can be mentioned in this context, there are very few others in which it is clear that hunting pressure—and nothing else—was to blame for depressing population size below Martin's limit. Indeed, there are substantive counterexamples that might be cited, in which massive and rapid population declines have not resulted in species elimination. During historical times, bison, pronghorn, koalas, and right whales have been hunted down to incredibly small residual populations (e.g., by 1890, *Bison* in the United States had been reduced to <0.00001% of the population estimated to have existed in 1820; Nowak 1991). Notwithstanding the vital conservation efforts that have been mounted on their behalf, that these species have managed to rebound at all is nothing short of marvelous. Yet, given such examples of great resiliency in the face of extreme levels of hunting pressure, is it plausible that small groups of prehistoric hunters in the Americas, Australia, Madagascar, and elsewhere were able to depress hundreds of megafaunal populations permanently beyond any hope of recovery? The usual answer to this question is to point out that overhunting was not the only deleterious effect of human practices in "new" lands, and that the FC extinctions were probably multifactorial. But this point should apply with equal force to "old" lands as well: Africa and Eurasia experienced human impacts throughout the late Quaternary, yet no large-scale megafaunal extinctions occurred there during prehistoric times. Has some paramount factor involved in many of these extinctions been overlooked?

Modeling Hyperdisease

Preliminaries

At present, the hyperdisease hypothesis is an abstraction awaiting marriage with suitable empirical data. Efforts to collect the correct kinds of data are now under way. Although this is not an appropriate place to undertake an extended discussion of proposed procedures to search for ancient pathogens, a brief overview is warranted.

Our immediate goal is to attempt to amplify nucleic acid sequences of ancient

pathogens from multiple, dated samples of selected late Quaternary taxa from North America (e.g., *Mammuthus,* various sloths, extinct species of *Bison*). Use of "ancient" DNA is now relatively commonplace in phylogenetic, population genetic, forensic, and biogeographical studies (e.g., papers in Herrmann and Hummel 1994). However, there are several technical and evidentiary challenges to be overcome in pursuing this investigation (Salo et al. 1994).

Paleontological specimens normally have no documented history of handling or storage. Contamination by modern pathogenic organisms certainly exists as a potential complication, although this problem can be partly overcome by searching for suitable samples deep within intact specimens (marrow cavities of bones or remnant soft tissues of permafrost mummies; Höss et al. 1994). This problem can also be partly controlled for by testing for contamination in samples from periods before there was any possibility of human arrival.

To test the hyperdisease hypothesis meaningfully, it must first be demonstrated that a specific pathogen of potentially lethal effect was broadly disseminated in terminal populations of target taxa. Adding to the inherent difficulty of this task is the fact that the genetic nature of the hypothesized disease organism is, at present, completely unknown. One starting point is to select pathogens that meet certain criteria (some of which are discussed in the rest of this section) and for which sequence data are available in gene banks.

Positive identification of a specific pathogen—even an unarguably lethal one—in a single sample of an extinct target species would be unpersuasive. Multiple positives are needed, from samples drawn from all parts of the ranges of the extinct species, with the added requirement that such samples be of the same or similar age. Multiple coeval positives would still not constitute persuasive evidence of extinction by disease unless it were also true that the samples corresponded in age to the last known appearance of the species in the fossil record (i.e., terminal populations). As a control, specimens radiometrically determined to be of much earlier age than the terminal populations must be sampled and analyzed in the same manner. This procedure would also help to guard against false positives.

Absence of resolvable pathogens in either target or control samples would be ambiguous: pathogens could be present, but the primers chosen may be ineffective in resolving them, or the pathogen's genetic material could be too degraded to permit detection (a particular problem with respect to viruses that replicate through RNA pathways). Obviously, ambiguous results are meaningless as evidence (absence of evidence cannot be interpreted as evidence of presence).

Finally, assuming that an identifiable pathogen can be found in coeval samples drawn from target terminal populations, its presence would have to be sought in other taxa believed to have become extinct at roughly the same time as the targets, under similar control conditions. Otherwise, there would be no basis for asserting the existence of a common cause. Obviously, the hunt will not be easy!

Until the problem of agency is better constrained, the notion of "hyperdisease" is best explored through a set of conditions that have to be met by the disease-provoking entity. At present we cannot exclude the possibility that, during the human diaspora, several discrete pathogens may have created hyperdisease conditions at different times and places. Accordingly, we shall simply refer to the etiologic agent of hyperdisease as HP, the hyperdisease pathogen(s).

We begin by examining the argument that either the HP still exists or it disappeared along with its last victims. If the HP still exists, in extant species it could be (1) currently nonpathogenic; (2) moderately pathogenic, but causing a disease less lethal than hyperdisease; or (3) highly lethal, but no longer sustainable as a hyperdisease in available hosts. These states might occur variously in different species that survived an FC episode of disease-induced extinction. If the HP still causes a related disease in some survivor species, we may be able to gain some clues about its past behavior in FC episodes. The alternative—that the pathogen responsible for hyperdisease is itself extinct—is possible but considered unlikely. Even if the specific genotype of the HP has been lost, there ought to be extant relatives that can be used as models to characterize it.

Classically, an outbreak of a lethal disease in a population follows a bell-shaped growth-decay curve: the number of new infections rises steeply at the beginning of the outbreak, levels off after a period, then declines as the number of new cases drops toward zero. Would an outbreak of hyperdisease have followed a similar course? In general, two factors control the shape of any mortality curve: the attack rate (AR) and the mortality rate (MR). AR is defined as the number of cases of the disease in question divided by the total number of individuals exposed or at risk. For known diseases, an estimate of AR is based on the transmissibility of the pathogen (i.e., the relative ease within which it establishes itself at portals of entry into the body), the prevalence of carriers, and other controlling influences. Highly transmissible diseases, such as measles, exhibit ARs that approach 1.0. MR is the number of deaths divided by the number of cases. An MR of 1.0 means that all infected individuals ultimately die as a result of the induced disease. Death may occur from the pathological process itself, or some individuals may recover, and even become immune, but fail to survive because of debilitation.

It is reasonable to hypothesize that, in hyperdisease contexts, both AR and MR must be extremely high, i.e., ~1.0. Figure 7.2 models an idealized hyperdisease epizootic occurring in one species, in which both AR and MR have values of 1.0. The first outbreak begins in population A at FC_A, marking the time of first contact with humans. Infection, disease, and death ensue within a confined period. In the simplest possible scenario, the pathogen causing the outbreak would be transported by its carrier to successive populations B at FC_B, . . . , i at FC_i, until all populations are affected. Local extirpations occur as the last individuals in each population die. Complete extinction of the species occurs when the last individuals in the ith population succumb. Since the curves for the separate populations are essentially the same at this level of analysis, the form of the ith curve thus summarizes the extinction trajectory of the entire species.

Although MRs of 1.0 are plausible on the basis of existing knowledge of disease-causing organisms, most microbes do not achieve ARs of 1.0. When MR = 1.0 but AR < 1.0, the mortality graph will be skewed. This situation is illustrated in fig. 7.3A, in which the single curve represents effects at the level of individual populations as well as at the level of the entire species. In each population, accumulation of cases would rise precipitously following FC, as in fig. 7.2. However, because of lowered AR, some individuals escape infection at first outbreak. If these individuals are subsequently infected, owing to continuous reintroduction

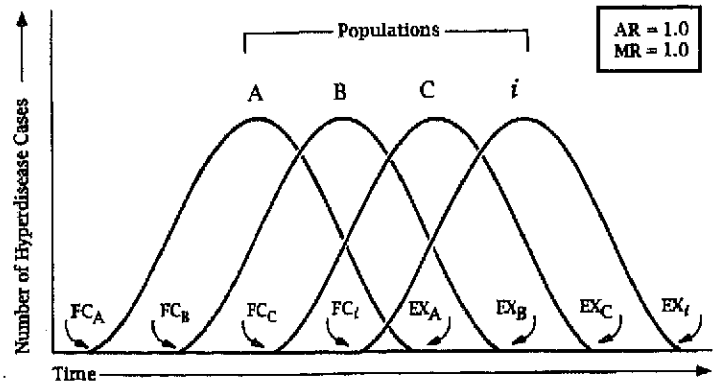

Figure 7.2. Hypothetical epizootic of hyperdisease in populations of an imaginary naive species, initiated by introduction of an HP with maximal possible attack and mortality rates (AR = 1.0, MR = 1.0). FC_A marks the time at which population A first encounters the HP via its carrier(s); the HP spreads throughout the population as a result of continuous exposure to the carrier, to newly infected individuals, or to both simultaneously. All individuals in population A become infected because AR = 1.0 and all will succumb because MR = 1.0; extirpation occurs at EX_A. In this most straightforward of scenarios, the inapparent carrier then successively introduces the HP in populations B through *i* until all groups are infected and extirpated. Species-level extinction occurs with the death of the last individual in the *i*th population.

of the hyperdisease pathogen by the inapparent carrier, then eventually all will die. However, the slope of the mortality graph will be less steep in this case compared to that in fig. 7.2, because the pathogen is infecting an ever-shrinking population. Extinction still occurs but takes longer.

Figure 7.3B models an epizootic in which AR < 1.0 and MR < 1.0. The curve resembles the one seen in fig. 7.3A, in that all individuals in the species are eventually infected. However, in this scenario, not all animals die; at the end of the epizootic phase, there are survivors who have become immune to the HP following spontaneous recovery. Stochastic processes will largely govern what happens to these survivors and the species they represent. There simply may be too few of them to form a recovery population, or they may be too widely dispersed to enable matings sufficient for species persistence. Furthermore, the disease may continue to claim victims even after the conclusion of the epizootic phase. For example, it may become endemic, attacking susceptible newborns or juveniles that lack immunity (suggested by the hatched area in fig. 7.3B). If attrition of young animals is great enough, or if reintroductions from carriers continue to occur, whatever population fragments remain will be highly susceptible to extirpation, and, if this happens universally, a species-level extinction will ensue.

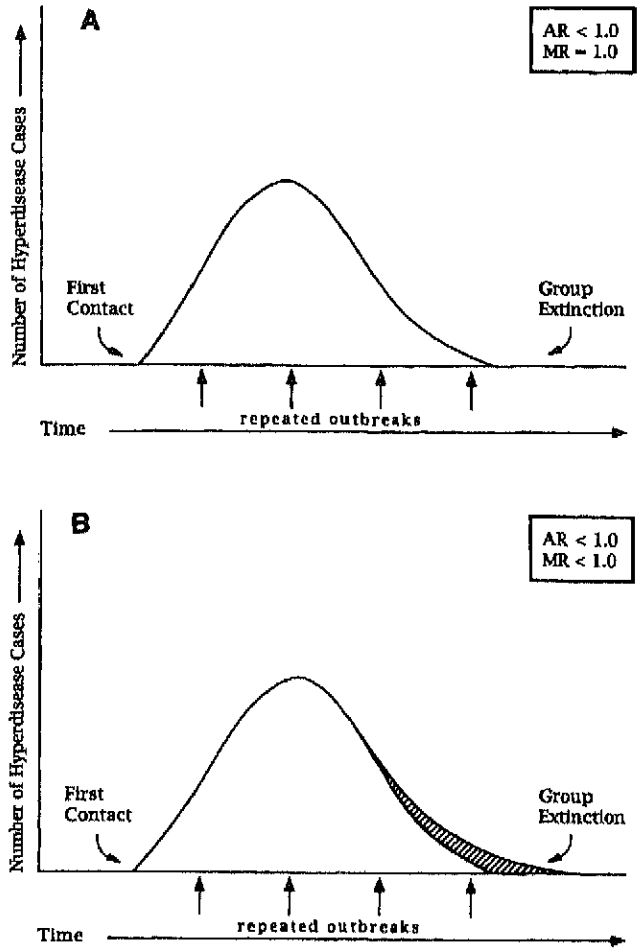

Figure 7.3. (A) Extirpation of a single population of an imaginary naive species by an HP when MR = 1.0 but AR < 1.0. As in fig. 7.2, the HP is transmitted population-wide by inapparent carrier(s) and newly-infected individuals (or both sources simultaneously). In this model epizootic, less than 100% of individuals are infected upon FC (AR < 1.0), but all eventually become infected and die (MR = 1.0) as the carrier reexposes the group during repeated contacts. The curve trails to the right because the HP is attacking an ever-shrinking population. (B) Extirpation of a single population by an HP when both MR and AR < 1.0. As in fig. 7.3A, first exposure results in the outbreak of disease in a portion of the group. As before, the HP is reintroduced by the carrier and by newly infected individuals (arrows), and the decay side of the curve changes in slope to reflect infection of an ever-shrinking population. However, since MR < 1.0, there will be survivors. The survivors will be immune and will resist reinfection and disease, even if multiply exposed to the HP by the carrier. These resistant individuals form a residual group that could experience an endemic period (hatched area) of hyperdisease after the initial epizootic. New cases gradually decrease toward asymptotic value; extirpation, if it occurs, will be due to factors additional to the hyperdisease (e.g., the population declines below the minimum viable size).

Four Hyperdisease Criteria

Because we do not know the identity or disease potential of the HP in extinct species, our only avenue at present is to explore some plausible recognition criteria for hyperdisease (table 7.3) by examining traits of known diseases of extant species (table 7.4). Modern diseases vary greatly in the degree to which they meet hyperdisease criteria, and this diversity is purposely reflected in the 20 entities chosen for table 7.4. (Summaries of the etiology, epidemiology, and pathology of these entities can be found in the individual papers cited and in standard texts such as Acha and Szyfres 1980, Fields and Knipe 1990, and Salyers and Whitt 1994.)

In increasing order of exclusivity, hyperdisease as we define it demands the following conditions:

1. *A reservoir species that presents a stable carrier state for the hyperdisease pathogen.* The first criterion that an HP must meet is that it has a stable carrier state—that is, either it is free-living at some point in the life cycle or it occurs as a persistent infection in certain species in a form transmissible by shedding (Lerche et al. 1987; Fields and Knipe 1990). All the pathogens listed in table 7.4 have one or more stable carrier states, except the two that are free-living. In the context of hyperdisease, the carrier state should also be the HP's vector species, unless the HP is free-living, as are some mycotic infectious agents. Presumably, the vector species would not itself be significantly victimized by the HP, because this would destroy the biological carrier needed as a reservoir. In FC contexts, the HP must have been able to persist in relatively small, mobile populations of humans or some synanthropic species, from which it could have emerged to strike other, susceptible species.

TABLE 7.3. Four Criteria of Hyperdisease

To create hyperdisease conditions, a candidate pathogen should have or exhibit:

1. A reservoir species in which a stable carrier state for the pathogen occurs.
2. A high potential for causing infections in susceptible species, affecting all age groups.
3. A capacity for causing hyperlethality in "new" hosts, with mortality rates >75%.
4. The ability to infect multiple species without seriously threatening human groups.

The concept of "inapparent carriers" provides a basis for further insights. An inapparent carrier is one that hosts a known pathogen, but without obvious ill effect. For example, Lerche et al. (1987) found that a type D retrovirus causing simian AIDS was about 90% lethal in a closed group of rhesus macaques (*Macaca mulatta*). The virus was evidently introduced into the *M. mulatta* group when it was housed with a number of bonnet macaques (*M. radiata*)—a classic example of the accidental emergence of a new disease in a captive population. Of great interest was the discovery of a carrier female in the rhesus group. Epidemiological data showed that this female had been infected for at least nine years. She did not present immunosuppressive disorder at any time, but for five years her saliva contained retrovirus capable of infecting other animals and causing an illness similar to acquired immunodeficiency syndrome (AIDS) (Marx et al. 1984; Marx 1988). The virus also persisted in the bonnet group. The germane point here is that the D retrovirus was able to emerge from a reservoir, cross a species barrier (albeit a minor one), and thereafter induce significant mortality in a new host. Had it been able to cause even higher mor-

TABLE 7.4. Extant Pathogenic Entities and Hyperdisease Criteria[a]

Disease	Pathogen	1. Stable carrier	2. Epizootic potential	3. Mortality >75%	4. Affects multiple species, but lacks epidemic potential in *Homo*
Protozoan					
Malaria	*Plasmodium* sp.	A	X		
Chagas' disease	*Trypanosoma cruzi*	A, M	X	X	
Mycotic and chlamydial					
Histoplasmosis	*Histoplasma capsulatum*	FL			
Ringworm (favus)	*Trichophyton mentagrophytes*	FL	X		
Avian chlamydiosis	*Chlamydia psittaci*	B, M			
Bacterial					
Plague	*Yersinia pestis*	M	X	X*	
Tuberculosis	*Mycobacterium* spp.	M	X		
Cholera	*Vibrio cholerae*	M	X		
Lyme disease	*Borrelia burgdorfi*	A			
Enteropathies	*Escherichia coli* (various serotypes)	M			
Leptospirosis	*Leptospira interrogans*	M			X
Tularemia	*Francisella tularensis*	A, M	X	X	
Viral					
Simian immunodeficiency disease	Simian immunodeficiency virus/simian D retrovirus	M	X	X	
Simian hemorrhagic fever	Simian hemorrhagic virus	M	X	X	
Ebola disease	Ebola virus (Filoviridae)	U	X	X	
Herpes simplex	Herpes simplex virus	M	X	X	
Herpes B	*Herpesvirus simiae*	M	X	X	
Encephalomyocarditis	Encephalomyocarditis virus	M	X	X*	
Lassa fever	Lassa virus (Arenaviridae)	M	X	X	
Myxomatosis	*Myxoma* virus	A, M	X	X	
Rabies	*Lyssavirus*	M	X	X	X

[a]A, arthropod; B, bird; FL, free-living (soil or water); M, mammal; U, unknown; X, entity meets the specified hyperdisease criterion; *, borderline. Note: Two different retroviruses can cause simian AIDS (Lerche et al. 1987; Marx et al. 1984; Marx 1988).

tality, or infect additional species, the D retrovirus outbreak would have met some hyperdisease criteria.

2. *A high potential for causing infections in susceptible species, affecting all age groups.* As previously noted, in order for a given pathogen to have hyperdisease potential, it must be able to induce infections in all or nearly all exposed individuals (figs. 7.2 and 7.3). A stable reservoir ensures that a pathogen has the opportunity to strike multiple times at susceptible species, until all or nearly all individuals in a group are infected. To achieve such high attack rates, the likeliest portals of entry for the HP would be the mucous membranes of the respiratory tract or the conjunctiva. There are other constraints that seem to us to be very important, such as the ability to infect all age groups. AIDS is an example of a disease that does not have this kind of epidemic potential because it is usually sexually transmitted and therefore is not efficiently spread among juveniles or other non–sexually active individuals within a population. Also failing this test are diseases that do not spread except from the original vector or source (e.g., histoplasmosis, avian chlamydiosis). These agents maintain themselves by constant reintroduction, but this is not an efficient mechanism for causing epizootics in multiple species. Chagas' disease (Acha and Szyfres 1980) remains on the list of possible candidates because the reduviid bug that is the ordinary vector can maintain its life cycle once it is in contact with humans without the intervening stage of another animal vector (such as *Neotoma* or *Procyon*). Whether an arbovirus can be transmitted efficiently enough to cause extinctions, however, is open to question. By definition sporadic, self-limiting infections cannot result in hyperdisease.

3. *A hyperlethal effect in "new" hosts, with mortality rates >75%.* Hyperlethality is not a defined entity, so we arbitrarily define mortality rates of 75% and higher as a marker of hyperdisease conditions. However, rates as low as 50%—still catastrophic in any epidemiological situation—might be considered to be borderline hyperlethal, depending on what other factors are in play.

It is helpful to look at highly lethal diseases of extant species to model hyperdisease conditions. Of the 20 diseases listed in table 7.4, 10 may be considered hyperlethal or borderline hyperlethal. Encephalomyocarditis is borderline hyperlethal in pigs, killing 75% of sucklings but much lower numbers of adults. Mortality from Chagas' disease may approach hyperlethality where it occurs in the New World, but like HIV-1 it is relatively slow to kill. Modern diseases that can be considered both highly mortal and rapidly lethal are fewer in number. In our list, they include viral infections due to Ebola in humans, herpes simplex in owl monkeys (*Aotus*), herpes B in humans, simian hemorrhagic fever in macaques (*Macaca*), and plague and rabies in a large variety of mammals. The causative agents of these diseases spread rapidly in some species and are hyperlethal when newly introduced.

If the HP still exists, its distribution should now be worldwide as a natural consequence of its spread during the human diaspora. Disease entities that are thought to have originated outside Africa and Eurasia obviously cannot qualify as HPs if the hyperdisease version of the coevolutionary hypothesis is accepted. Similarly, diseases of any origin that had a broad distribution prior to the appearance of humans in "new" lands are also disqualified, since their initial spread cannot be linked to human colonization. Among the diseases in

exterminated by an unknown epizootic in the 1870s (but see Fuller 1987, who doubts that any cause can be unequivocally ascribed in this case).

As we have already noted, whether or not the hyperdisease hypothesis can be falsified is ultimately an empirical question, and the data necessary for meaningful testing are not yet available. Here we anticipate some of the objections that are sure to be raised to the hyperdisease model, and we also propose the directions to take in trying to resolve them. There will undoubtedly be other criticisms that we have not thought of, and we welcome future opportunities to respond to them.

It Is Unlikely That an Epizootic Could Propagate through Dozens of Species in a Continent-Sized Area

Islands are probably the likeliest places to experience catastrophic losses due to disease. Not only would island mammals be susceptible by virtue of their long biological isolation, but in addition an epizootic occurring in a confined area like an island could potentially infect many or most of the individuals in a species at the same time.

With continental extinctions, however, hyperdisease encounters a plausibility problem. North American mammals that died out in the terminal Pleistocene were surely a behaviorally and trophically varied group. If "new" diseases played any role in their extinction, the pathogens responsible must have challenged a wide spectrum of species. Furthermore, if small size conferred no benefit in terms of resistance to exotic disease, then the corollary seems practically unavoidable—that hyperdisease epizootics would have infected far more species than the ones that actually died out, implying a host range of almost unimaginably large proportions. One avenue that might lead to some insights would be to search for a biological signature in living taxa that might imply one or more bouts of hyperdisease in their recent history. There are some candidates. Extremely low levels of heterozygosity in some carnivores have been used as a proxy indicator of bottlenecks and extreme population reduction in earlier times. Lack of significant genetic variation in African populations of the cheetah (*Acinonyx jubatus*) is possibly consistent with the proposition that living cheetahs derive from a very small number of recent ancestors (O'Brien et al. 1983, 1987; O'Brien 1994). Obviously, severe bottlenecks could have many potential causes (Merola 1994). Extant species that have been extensively hunted, for example, ought to display reduced heterozygosity compared to the ancestral populations. This characteristic has been provisionally demonstrated for *Bison bison* by K. Chambers (pers. comm.), who compared mitochondrial DNA amplified from 300-year-old bison to that of the living U.S. wildherd, which is derived from a late-nineteenth-century population of <100 individuals. Unusually high frequencies of skeletal anomalies have been thought to be evidence of possible inbreeding in late Pleistocene *Bison*, occasioned by catastrophic reduction in their numbers (McDonald 1981; for comparable anomaly frequencies in Rancho La Brea dire wolves, see Duckler et al. 1994). Whether these anomalies can be correlated with specific disease processes that killed many of their owners remains to be determined.

Although there are interpretative problems with assuming that low heterozygosity necessarily implies drastic population reduction in the past (Ward et al. 1992), it would be of interest to know how large mammals compare to smaller ones for

heterozygosity measures. Reduced heterozygosity in large species is consistent with overhunting as well as hyperdisease, but overhunting would not explain reduced variability in certain small mammals (although other, primarily ecological, factors might; Nevo et al. 1984).

Neither hyperdisease nor blitzkrieg can account very well for the last act of any extinction event, the point at which survivability drops to zero for affected species. Modeling of the extinction point is still in its infancy, as Caughley (1994) points out in the different but relevant context of conservation studies. Theoretical evaluations of minimum viable population (MVP) size posit that 50–500 sexually functional individuals may be enough to ensure population survival without undue inbreeding (Franklin 1980). Real populations would have to be several times that size to deal with even minor stochastic perturbations (Caughley 1994), although there are several examples of dramatic comebacks from populations of a few tens of individuals (e.g., northern elephant seal, *Mirounga angustirostris;* Hedrick 1995). Unless one believes that paleo-Indians habitually mopped up all pockets of remaining megafauna as the blitz "front" advanced (as required by the model of Mosimann and Martin [1975]), population remnants sufficient to form MVPs should have regularly survived in inaccessible areas. Recovery of American *Bison* in the late nineteenth century, mentioned earlier, confirms the notion that population sizes have to be low indeed to effect complete extinction. Unless late Pleistocene megafauna in North America had some unusual range limitations or population structures, it is hard to see how human activities alone could have caused complete extirpation of all individuals.

There Is No Mechanism by Which Diseases of the Prescribed Level of Lethality Could Maintain Themselves in Nature

In our model, an HP has to maintain itself in reservoir species in which it causes little or no disease; otherwise it will not survive. But an obvious related requirement is that host density must be great enough to ensure that new infections occur regularly among susceptible individuals. If a disease follows a very rapid, fatal course, the host population will be reduced in a short time to widely dispersed and/or relatively or completely immune individuals.

High host density is permanent in herding species and can periodically occur during the rut or its equivalent in species that do not herd. The hardest cases to explain are species that probably did not form aggregations of more than a few individuals at any time in the annual cycle. Accounting for the disappearance of species that can be plausibly regarded as carnivores and scavengers, for example, is not made any less difficult by observing that individuals might have made themselves susceptible by going after sick or dead prey, because the problem of transmittal to other members of their species still remains. Here it may be noted again that, if there were reservoirs capable of introducing and reintroducing the hyperdisease agent, epizootics could surge across the range of susceptible taxa year after year until population sizes were reduced below viable levels.

It Is Undemonstrated That Disease Can Cause Complete Extinction of Species

It is reasonable to assume that any severe threat to a species, deterministic or stochas-

tic (Schaffer 1987), could ultimately lead to its complete extinction. Because there are many diseases that cause significant mortality in the populations they affect, there should be no objection to the argument that disease is, in and of itself, an example of a severe potential threat. The real issue is whether even the worst outbreaks of lethal disease on record show anything approaching the kind of virulence required to obliterate entire species. We shall concentrate on viruses in the examples considered in this section.

Induced myxomatosis epizootics in the European rabbit, *Oryctolagus cuniculus,* are perhaps the best example on record of the effects rendered by near-hyperdisease conditions on a naive species (Fenner and Ratcliffe 1965; Fenner and Myers 1978). *Myxoma,* a member of the Poxviridae, is naturally hosted by the tapeti, *Sylvilagus brasiliensis,* a South American lagomorph. Prior to its use as a pest control measure in Australia and elsewhere, this virus did not occur outside certain parts of the New World. Although the age of this virus-host relationship is not known, it is probably of appreciable antiquity since related but different viruses are harbored by other New World lagomorphs (Fenner and Ratcliffe 1965). In *S. brasiliensis* myxoma infection usually causes only a benign fibroma; virus and mammal are, in effect, highly coadapted. By contrast, in O. *cuniculus* myxoma virus causes highly lethal disease that kills either directly by general systemic effects or indirectly by immunosuppression so that death occurs as a consequence of secondary bacterial and viral infections (Parer et al. 1994). In Australia, myxomatosis epizootics in rabbit populations attained mortality rates of 99–100% in the early 1950s (Fenner and Ratcliffe 1965). The fact that the same organism might in one species cause only mild infection and in another cause a lethal disease is a common pattern, especially among "new" or "emergent" diseases. Virulence and lethality are not intrinsic properties of disease-provoking organisms; they are measurements applied to those infected, to gauge the severity of the disease's effect (Salyers and Whitt 1994).

Despite the virulence and lethality of these epizootics in immunologically naive populations of *Oryctolagus,* myxomatosis has not caused losses above the level of local extirpation in Australia. One reason is that its chief method of transmission appears to be mechanical infection, by biting arthropods; when arthropod populations diminish seasonally, the rate of infection in rabbits declines as well. This finding suggests that the virus has limited capacity to distribute by aerosolization or similar means. Another reason is that the highly lethal original strain was rapidly supplanted by less lethal mutants, and rabbits that survived were thereafter immune. By surviving less lethal strains, newly resistant rabbits became proportionately more common (Fenner and Ratcliffe 1965). Another effort at rabbit control is currently being made in Australia using a rabbit calicivirus disease from China that kills within 36 hours, but the experiment has already been compromised by accidental releases of the pathogen (Holden 1995).

There is a traditional notion that natural selection cannot favor the emergence and maintenance of hypervirulent, hyperlethal pathogens (see discussion by Morse 1994). A more insightful view on the evolution of host-parasite relationships is presented by Ewald (1994), who argued that, under the right circumstances, a pathogen might in fact opt for unusually great virulence, as long as its vector or method of infection is

so pervasive in the environment that it can continue to spread even while it is causing substantial mortality (the "burning bridges" strategy). One example of this process at work in human populations might be the "Spanish" influenza pandemic of 1918–1919, which may have been derived from a swine influenza A virus and which caused 26 million deaths (Slemons and Brugh 1994). Other appropriate examples pointing up the importance of host incompetency in defining virulence and lethality would be measles, smallpox, and typhoid epidemics among New World aboriginals at FC (Cliff et al. 1993), although recent work indicates that the virulence of these epidemics may have been exaggerated (Larsen 1994; Snow 1995).

It appears from these points that, for a hyperdisease to cause complete extinction, the pathogen would have to occur only in highly lethal, aggressive strains and spread throughout populations comprising the species before attenuated viruses became common. This subject leads naturally to a second issue, which is the intertransferability of pathogens among several potential hosts. The hyperdisease model is plausible as an explanation of complete extinction when only one or a few closely related species are under consideration. As already noted, for hyperdisease to explain the data for large-scale losses such as those that took place in North America about 11,000 yr B.P., we must infer an additional element—multiple simultaneous or closely sequential panzootics (fig. 7.2) occurring among a wide array of "new" hosts. In such cases the schedule of infection would have to have been tight enough to permit the hyperdisease pathogens to ensure their survival by "burning" through one host or group of hosts after another.

Providing credible mechanisms for multiple panzootics is a heavy requirement that we cannot model more precisely at this time without having some idea of what the pathogen(s) might have been. *Myxoma* itself would not be an appropriate candidate because it seems unable to sustain infection in hosts other than lagomorphs, although the family Poxviridae includes a number of disease-provoking entities (cowpox, buffalopox, monkeypox, fowlpox) that can infect a fair range of species (Fenner 1994). A more appropriate model might be the rinderpest outbreaks in Africa at the end of the nineteenth century. The disease was accidentally introduced in the 1890s through the importation of infected cattle from Asia, where it is endemic (Gray 1919). From a small focus the disease spread throughout eastern and southern Africa, decimating many species of wild artiodactyls as well as domestic stock (Scott 1976). Yet as virulent as these rinderpest epizootics were, no species was completely exterminated (Kingdon 1982). Rinderpest is essentially limited to ungulates (Scott 1976), but the morbilliviruses as a group induce a wide array of diseases in mammals. Examples include measles, affecting humans; distempers, affecting canids and pinnipeds; and equine morbillivirus disease, a recently discovered emerging disease of horses (K. Murray et al. 1995). All of these are, or are suspected to be, airborne contagions that spread when there is direct contact between sick and healthy animals.

On the other hand, there is no need to postulate a completely unknown microbe as the cause of extinction in terminal Pleistocene North America. Under the terms of our argument, any zoonosis habitually carried by humans, their commensals, or pests might have had the potential to become a hyperdisease by jumping to the right new hosts. Indeed, in addition to the other examples of exotic disease already considered,

there is a parallel of sorts to our hypothesized FC devastations in the spread of disease within mixed colonies of laboratory animals. In such colonies, species that do not occur together in the wild are often kept in the same facilities, a situation that enhances the potential for spreading "new" pathogens. Mortality approaching 100% has been encountered in some cases (e.g., simian hemorrhagic virus in species of *Macaca;* Peters et al. 1994). Other recently described emerging diseases are known to have produced massive lethality in their new hosts (e.g., canine parvoviruses causing distemper and enteritis in other carnivores; Parrish 1994; Roelke-Parker et al. 1996).

The constraint presented by experience with myxomatosis is that, to be effective as an agent of extinction, a pathogen either must be relatively invariable genetically, so that less lethal mutants cannot quickly become dominant, or it must induce and maintain lethality levels of nearly 100% from the start of the epizootic, so that populations of the species disappear before the microbe mutates into a more benign form. The first alternative is not necessarily implausible: as previously noted, the rabies virus is an ancient pathogen that can attack a great variety of mammals, and it has maintained high virulence (Beran 1994).

It Is Undemonstrated That Wild Species Can Be Infected by Human-Hosted Pathogens

Since transmissions of pathogens from people to animals are of much less importance to human health than transmissions in the reverse direction, the problem of human-hosted pathogens passing to other species is underinvestigated. Many pathogens that cause human diseases probably originated, in the phylogenetic sense, from pathogens originally harbored in other mammals, and some, like HIV-1 and HIV-2, may have a complex host history. Measles is a human disease, but the pathogen that causes it is morphologically and molecularly similar to viruses that cause diseases in other mammals. Cliff et al. (1993) point to a number of other human diseases that probably have a host ancestry in a domestic animal: respiratory tuberculosis (from cattle), leprosy (from water buffalo), and diphtheria (from cattle). Tuberculosis and fungal infections have been transmitted to pinnipeds by humans (King 1983), and influenza A strains seemingly pass back and forth with ease between humans and pigs (Slemons and Brugh 1994). In short, it is plausible that disease agents have regularly passed from humans (and animals culturally or economically associated with humans) to other species, even if the number of actual examples that can be presently identified is small.

Why Do Humans Have to Be the Carriers?

Although humans may not have entered the New World until approximately 12,500 yr B.P., for most of the Tertiary and Quaternary there was a continuous, bidirectional stream of biotic traffic across Beringea. Migratory birds, for one, could have moved without impediment between continents even when the landbridge was impassible to most terrestrial animals. If it was possible for an extinction-inducing hyperdisease to develop at any time in the past 40,000 years, why was it not carried to North America much earlier by nonhuman vectors?

The same question, *mutatis mutandis,* could be asked of extinctions in Madagascar, Australia, and elsewhere. A possible answer is that humans, or species that

associate with humans, were the only sustained vectors of hyperdisease. Warner (1968) addressed a similar issue in his discussion of disease as a cause of massive population depletion and extinction among endemic Hawaiian birds. He posited that avian malaria, from North America, was a major cause of species loss in Hawaii, especially among the endemic honeycreepers (tribe Drepanidini). Since these islands are on flyways that have doubtless existed for millennia, North American migrants harboring plasmodia must have come in regularly, but provoked no species-threatening diseases among local endemics. Warner (1968) suggests that panzootics among endemic birds in Hawaii did not occur until the American mosquito *Culex quinquefasciatus* (=*C. pipiens fatigans*) was accidentally introduced into Maui in 1826 by a ship. Prior to that, malarias in migrants were dead-end infections because there was no vector to transmit sporozoites to local hosts. (For a re-evaluation of Warner's hypothesis, see Van Riper and Van Riper [1986]).

Although Antarctica has lacked land mammals since the early Tertiary (Savage and Russell 1983), its baregrounds and offshore islets have doubtless provided breeding areas and haulouts for a great variety of birds and pinnipeds over many millennia. What happened to the FC extinction in Antarctica? There is no evidence that one occurred when Antarctica and the sub-Antarctic islands were first visited by humans during the nineteenth and early twentieth centuries. Two possible reasons for the lack of recent extinctions can be offered, but we concede that neither is very satisfactory. The first concerns the restricted and apparently highly resilient nature of Antarctic faunal elements. The mammals are whales and pinnipeds, and it is simply a fact that, despite remarkable levels of exploitation, marine mammals have suffered virtually no extinctions at the species level during the modern era—a point that is as hard to explain from an overkill perspective as it is from the hyperdisease position.

It might be argued that some whale species have been insulated to a degree from the full force of human impacts because they travel enormous distances during their annual cycles and are therefore not under threat of continuous predation. However, at least from a historical perspective, their migratory behavior seems to have been as much a curse as a boon, at least for sought-after species with highly predictable routings. Yet even in the case of the gray whale (*Eschrichtius robustus*), to which this point applies a fortiori, only the population that lived in the Atlantic appears to have been completely extirpated, and that loss happened before the seventeenth century (Nowak 1991). Obviously there could not have been a *terrestrial* extinction event in the Antarctic because no obligate terrestrial mammals occur in that region. (We regard the Falkland or Malvinas Islands as high-latitude South American rather than sub-Antarctic islands, but the WCMC survey [Groombridge 1992, table 16.6] lists them under the heading "Antarctica" and cites the extinct Falklands dogs, *Dusicyon australis*, as the sole example of a terrestrial casualty among Antarctic vertebrates.)

A second possible explanation for the lack of an FC event is that the Antarctic environment is famously inimical to all exotics, including pathogens. We note that this point may also have some cogency for the North American post-Wisconsinan extinction, in that the onset of faunal losses at high latitudes evidently began after periglacial environments began to retreat rapidly poleward, removing whatever *cordon sanitaire* cold conditions had previously provided.

If Disease Can Cause Complete Extinctions, Why Is There No Conclusive Record of Species Disappearing from This Cause in Modern Times?

Part of the problem is to know what to look for. How does a species "look" that is dying out from virulent disease, in contrast to one that is dying out from habitat alteration, competition from exotics, or human exploitation? Increasing rarity of a formerly widespread species—which is the observation on which most threat assessments are ultimately based—is an effect of whatever is causing declining numbers; it is not a cause. Unless a species has some economic, esthetic, or other cultural importance, the chances are that few people would notice the occurrence of a loss so long as the existence of the epizootic was not obvious. After the passage of time, the absence of a particular taxon might be noted, but by then it is usually impossible to establish definitively what caused the loss (e.g., the extinction of the Labrador duck, *Camptorhynchus labradorius;* Fuller 1987). For purposes of discussion we shall cite three examples in which disease has been mentioned as a possible factor in the complete extinction of modern species. There are many other examples of disease contributing to population depletion (Young 1994), but as these have not (or have not yet) resulted in complete extinction of affected species they are not counted here.

Drepanid losses in Hawaii. In addition to laboratory experiments with avian malaria, Warner (1968) adduced historical evidence strongly suggesting that introduced diseases have seriously affected endemic Hawaiian birds. For example, he showed from historical records that, at the time of European discovery of Hawaii, the native honeycreepers were widely distributed at all elevations. Today surviving species are limited to mountainous areas. He argued that "abandonment" of intact lowland forest by all surviving species and extinction of species having strong altitudinal migration patterns indicated that the lowlands had become untenable for these birds, perhaps because of disease introduced in the 1820s. Abandonment could not have been due to disturbance of the environment by Europeans, since that had not yet occurred, although this theory does not account for impacts occasioned by native Hawaiians.

Declines and extinctions among native Australian mammals around 1900. Anecdotal reports assert that disease may have been responsible for declines among native Australian mammals around the turn of the century. None of them is compelling (Burbidge and McKenzie 1989). Typical of them is Shortridge's (1909, p. 818) brief note regarding the loss of many ground-dwelling marsupials in Western Australia since 1880: "The entire disappearance of so many species, over such large tracts of country, is generally considered to be due to some epidemic or disease, which I have been told appeared to be a kind of marasmus, perhaps brought into the country by introduced mammals." Doubts about the observational basis of this explanation are raised by Shortridge's reference to the disease as a "marasmus" (i.e., a wasting, a term usually applied to cases in which flesh is lost without apparent fever or disease). Somewhat later, Wood Jones (1923–1925) stated that the disappearance of the eastern quoll (*Dasyurus viverrinus*) from the mainland of Australia evidently resulted from a disease of unknown nature. No evidence to support this statement was offered, although it has

been dutifully repeated by later authorities (e.g., Rounsevell 1983).

Thylacines (*Thylacinus cynocephalus*) were common enough to be considered a pest in mid-nineteenth-century Tasmania, and both private and government bounties were instituted for their destruction. Although several thousand pelts were redeemed for bounties before 1909, it was not until 1905 that thylacines seemed to become suddenly rare throughout their remaining range. Rounsevell (1983, p. 83) stated that "despite this deliberate destruction, it is difficult to account for the rapid decline of the species between 1900 and 1920 only in terms of hunting pressure. It is possible that the same epidemic which, at the turn of the century, appears to have decimated the Tasmanian Devil and, on the mainland, the Eastern Quoll, swept the Thylacine into oblivion." By the time the rapid decline was finally noted, it was evidently already too late to do anything about it: the last documented thylacine, a zoo animal, died in 1936 (Rounsevell 1983).

Christmas Island Rats. Christmas Island may have been visited by humans on occasion in the distant past, but true occupation and inland penetration did not occur until the 1880s (Andrews 1900). The island supported two distinctive endemic murids, the nocturnal/crepuscular climber *Rattus macleari* and the semifossorial *Rattus nativitatis*. *R. macleari* in particular was described by Andrews (1900, pp. 33ff) as being "extremely common," "completely devoid of fear," and "a great nuisance" in 1897, the year of his visit. He went on to say that dogs were used to keep the rats in check and that "near the settlement they are certainly already less numerous than elsewhere." Less than a decade later the rats were nowhere to be seen; perhaps, Andrews (1909) surmised, they had died out in an epizootic of trypanosomiasis transmitted by ship rats. Five or six years previously, he found out, individuals of *R. macleari* could be seen crawling about paths in the daytime, in evident distress and "apparently in a dying condition." The trypanosome, if there was one, remains unknown.

Other Points

Some final points relevant to this discussion may be briefly listed. First, the literature is of limited assistance. The largest FC extinctions occurred in places where, at the time the losses were occurring, human society was preliterate (e.g., the Americas, New Zealand, Madagascar). There is therefore no direct evidence except from oral traditions about now-extinct animals (e.g., the tradition of the *lalomena* in Madagascar; Godfrey 1986). Such reports might conceivably incorporate information about cause of loss, but their historicity is uncertain (see Anderson 1989; Simons et al. 1995).

Second, FC extinctions that occurred during the European age of exploration mostly took place on small islands, where the entire elimination process typically went to completion in a matter of a few years to a few decades. Often the animals dying out were of no economic or other interest to human visitors or inhabitants, and it is scarcely surprising that their loss would go unremarked. Even when biologically informed observers were on the scene, there was rarely any effort applied to trying to understand why endemic species were being lost.

Third, as noted previously, under the terms of the hyperdisease model, there are only two kinds of taxa left after the FC extinction process has run its course in a "new" land: species that were nonsusceptible to

begin with, and those founder populations that became facultatively nonsusceptible through intense, disease-mediated selection. Virtually all existing vertebrate species have been exposed to humans and their pathogens for a period of not less than one century. In a real sense, they now stand in relation to *Homo sapiens* as only African and Eurasian species did in the Paleolithic: they have adapted, as best they could, to the human presence.

Summary and Conclusions

Extinction occurs when the last individuals of a genetically distinct metapopulation disappear (May et al. 1995). What causes such losses is often unknown, despite the usual confident assertions to the contrary that are made in the relevant literature. Disease is regarded as a minor threat to biodiversity at present (Groombridge 1992), and this may be an accurate assessment for the modern world. Whether disease forced numerous extinctions in the past, however, is open to question and investigation. We have developed the hyperdisease hypothesis as an avenue into this question. Our chief concepts, observations, and conclusions may be summarized as follows:

1. Hyperdisease—hypervirulent, hyperlethal disease—can occur when exotic pathogens are introduced into immunologically naive species. According to our conception of hyperdisease, diseases that qualify as hyperdiseases must (1) have reservoirs in species that are not susceptible to hyperdisease, whence they can be introduced and reintroduced into susceptible populations opportunistically; (2) represent completely novel challenges to their new hosts, who are physiologically and immunologically ill equipped to deal with such pathogens; (3) cause severe systemic disease or debilitation and kill before immune responses can develop to control the infection; (4) spread in groups of all ages by contact that is independent of (or is certainly not limited to) reproductive behavior; and (5) produce severe epizootics in numerous species quasi-simultaneously without causing serious epidemics in migrating or settled human populations. We hypothesize that hyperdiseases have broken out repeatedly during the 40,000-year human diaspora, as humans or their camp followers introduced pathogens that were catastrophically lethal to species having no prior experience with them. How multiple simultaneous epizootics can occur and be sustained to the point of extinction of the species affected remains to be elucidated.
2. On general theoretical grounds, hyperdisease is at least as plausible as its chief competitor, blitzkrieg, as an explanation for the primary features of several late Quaternary extinctions. Hyperdisease explains (1) why the general extinction rate drops off after the FC episode of disease introduction, panzootics, and mass losses; (2) why larger mammals are more susceptible to extinction than smaller ones; (3) how an extinction episode can pass with dramatic rapidity through an area without regard to local topography, plant cover, or the distribution of habitat favorable to human occupation; and (4) why it need not be automatically inferred that hunting or other cultural practices of aboriginal residents in diverse places were responsible for species losses.
3. Hyperdisease is possibly less plausible than blitzkrieg in requiring that, for extinctions to ensue at scale, (1) extinc-

tion schedules per species had to have been very rapid, and (2) multiple simultaneous panzootics had to have occurred among diverse taxa.

4. Both hyperdisease and blitzkrieg require that the effective agent (disease or overhunting) depress populations of species so rapidly that they have no possibility of recovery anywhere in their original range. In a normative world, it seems improbable that any single factor could produce this effect. But extinction happens.
5. In situations in which no single cause for a phenomenon can be conclusively favored (Pappas and Swain 1978), there is a natural tendency to assume that many causes must be responsible. This is a relatively weak approach epistemologically, because in many instances the adoption of a multifactorial explanation is really an admission of defeat rather than a breakthrough in understanding. This is especially true when each cofactor selected seems to explain one part of a phenomenon but lacks a logical and testable connection with any other factor. Yet despite the arguments advanced here, it is conceivable that hyperdisease could have been a unique cause of extinction only in sharply confined contexts like small islands (e.g., an epizootic possibly causing eradication of native rodents on Christmas Island). On continents and larger islands, where species' density distributions would have been less constrained, hyperdisease may have been only contributory or secondary to the main extinction process. In the case of Madagascar, for example, Dewar (1984) suggests that cultural practices short of outright overkill might have prompted or accelerated some subfossil losses, whereas Burney (1993a) notes that increasing aridity in the southern part of the island could have had a cascade of deleterious effects, including the forcing of population crashes. These are all possibilities awaiting confirmation. The proper way to adjudicate their influence (including that of hyperdisease), however, is to search for independent evidence of each.
6. The hyperdisease hypothesis is potentially testable by searching for candidate pathogens in multiple specimens of extinct taxa, using bone and other tissues from which ancient but nonendogenous genetic material might be extracted. In order to optimize the likelihood of success in the present exploratory phase of research, searches for evidence of relevant pathogens should be conducted using paleontologically common, geographically widespread taxa whose time of "last appearance" can be narrowly constrained from densely sampled radiocarbon chronologies.
7. Recovery of the same pathogen from multiple same-age specimens of several taxa from a given area would not constitute a demonstration of the hyperdisease hypothesis. Nor would it be a trivial result, and further testing using other extinct taxa would clearly be warranted. Demonstration that introduced disease was a factor in provoking extinctions during the past 40,000 years would prompt a reevaluation of the human role in initiating such catastrophes.

A real question was: Had we finally run into the Andromeda strain, like the deadly virus in the novel? It turned out not to be the case. In general, [Ebola] virus is not transmitted by aerosol from man to man. But it certainly has

that potential and one wonders what might happen if and when the day comes that an agent of this degree of lethality obtains the right kind of receptors, or whatever is needed, to replicate in the respiratory tract and be transmitted. This would be, I think, the most lethal of influenzas.

—Karl M. Johnson (1994, p. 56), recalling the 1976 outbreak of Ebola in Zaire.

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