Domino Effect Invoked in Ice Age Extinctions

Large herbivores have such a dramatic effect on vegetational patterns that their removal can affect the lives of smaller species; could this be a model for ice age extinctions?

DURING the latter part of the Pleistocene (ice age) period, which lasted from 2 million to 10,000 years ago, fully 50% of large mammalian genera became extinct worldwide—the woolly mammoth and its relative the mastodon being notable among these doomed species. Some researchers invoke climatic change as the prime culprit in this dramatic die-off, while others finger human predation, the overkill hypothesis. Norman Owen-Smith, of the University of the Witwatersrand, Johannesburg, now offers a third explanation, which he calls the keystone herbivore hypothesis.

This hypothesis is not a single explanation of the late Pleistocene massacre, but a combination of explanations: once the extinction process has been initiated by the loss of the very large herbivores—those weighing more than 1000 kilograms—the hypothesis seeks to explain the disappearance of other, smaller species. Simply stated, it proposes that the normal feeding habits of very large herbivores, or megaherbivores, have a tremendous impact on the vegetational environment; often, this impact opens up habitats in which smaller herbivores can thrive; if the very large herbivores are removed for whatever reason, vegetation cover will close up, thus elimating the smaller herbivores too.

"Protagonists of climatic change have yet to explain satisfactorily why no increase in extinction rate occurred at previous glacial-interglacial transitions," says Owen-Smith. "The human predation hypothesis fails to account for the extinctions of mammalian and avian species that were not obvious prey species of human hunters." He therefore decided to approach the problem as "a neontologist familiar with the ecology of African large herbivores."

The pattern of extinctions is dramatic: the bigger the species, the more likely it would succumb. "All species exceeding 1000 kilograms in adult body mass—that is, all megaherbivores—disappeared from the Americas, Europe, and Australia, together with three-quarters of herbivore genera with weights typically in the range 100 to 1000 kilograms," notes Owen-Smith. Some 41% of species weighing between 5 and 100 kilograms vanished, whereas less than 2% of creatures under 5 kilograms became extinct.

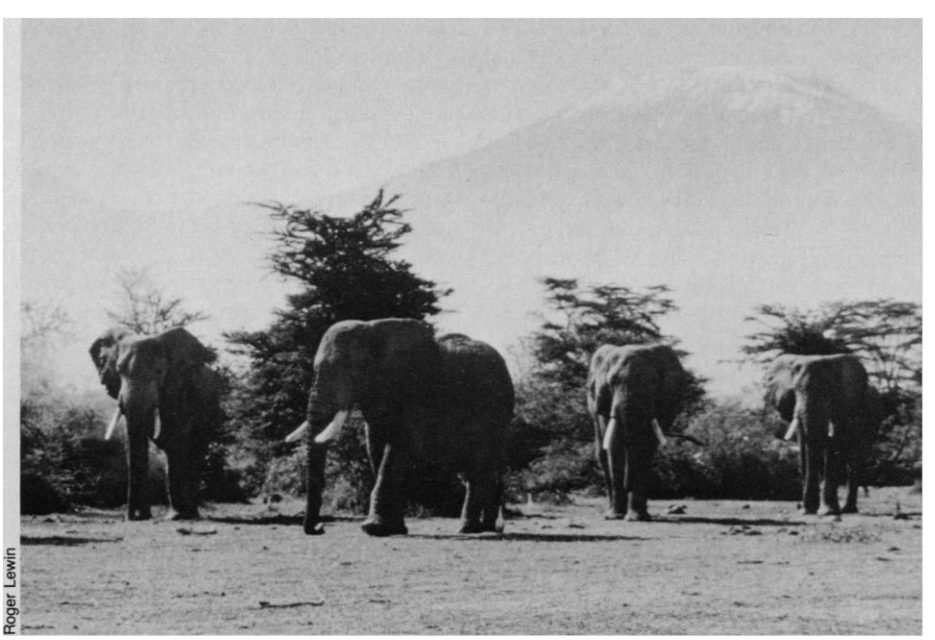
The fossil record is of course punctuated with extinction episodes, often apparently associated with climatic change. "However, these were different from the extinction episode at the end of the Pleistocene in that all size classes of mammal were affected equally," says Owen-Smith. Moreover, these earlier extinctions were frequently followed by radiations of new species that replaced the old, sometimes by migration from other locations, sometimes by speciation. "In contrast, the late Pleistocene extinctions occurred without replacement."

Owen-Smith also points out that many extant and extinct megaherbivores had broad geographic distribution and climatic tolerance. Why, then, "should [they] have been so much more vulnerable to the habitat changes of the late Pleistocene than smaller spcies of herbivore?"

Human predation therefore looks a good prospect, especially as the timing of extinctions in different parts of the world coincide more or less tightly with the arrival of modern *Homo sapiens*. In the Americas, for instance, some 70 species of large mammals became extinct around 11,000 years ago, which coincides with human expansion (and possibly arrival) in the continents. Extinctions in Australia occurred about 25,000 years ago, which postdates human arrival by perhaps 10,000 years. As might be expected, the long coexistence of human hunters and their potential prey in both Africa and Europe has left a much less clear cut pattern of late Pleistocene extinction.

If, as seems likely, predation played some part in these extinction, it cannot be the complete explanation, suggests Owen-Smith, because many species of birds and mammals that would not have been vulnerable to human hunting also became extinct. Hence his search for an additional factor.

Megaherbivores are relatively invulnerable to nonhuman predators, and therefore their populations frequently come close to or even exceed for a while the carrying capacity of the local habitat. Substantial impact on the habitat is therefore inevitable. "By felling or damaging trees, elephants can transform wooded savanna to open grassy savanna or shrubby regrowth, and create openings in forests," explains Owen-Smith. "Grazers like white rhinos and hippos convert medium-tall grasslands to short grass lawns, suppressing fires and thereby making grasslands vulnerable to invasion by woody plants."



Roger Lewin

Modern megaherbivores. *Elephants exert a considerable impact on the environment, thus affecting the potential niches of other species around them.*

The overall effect of these kinds of environmental changes are generally favorable, and include an increase in productivity and the acceleration of nutrient recycling. "The disturbing effects of megaherbivores on vegetation can promote higher rates of production of more nutritious forage than occurs in their absence," says Owen-Smith, "and these habitat changes may benefit other mammalian herbivores with similar but more selective feeding habits."

The elimination of megaherbivores—by whatever agency—presumably would be followed by a reversal of these environmental impacts: specifically, open forest glades close up; shrubland becomes forested; grassland mosaics become uniform tall grassland; and so on. Transformations of this sort, which, says Owen-Smith, "seem to be just the kinds of changes in vegetation documented by the fossil pollen record," would severely restrict the habitat of smaller hebivores. "The restriction of associated herbivores to relatively isolated pockets would have made these herbivore species more susceptible to chance events, and may have blocked migratory routes they needed to follow shifting vegetation zones."

Such dramatic effects may be difficult to envisage, admits Owen-Smith. Nevertheless, he cites the example of Hluhluwe Game Reserve in Natal where, "following 100 years or so without elephants, three species of antelope have become locally extinct, while open country grazers such as wildebeest and waterbuck have been reduced to low numbers."

The keystone herbivore hypothesis links two events: the initial elimination of megaherbivores, probably by human hunting; and the consequent loss of medium-sized herbivores as a result of vegetational change. The hypothesis may be tested, notes Owen-Smith, by examining the nature of vegetational change during the late Pleistocene: is it the result of climatic change alone, or does the loss of megaherbivores contribute significantly? In addition, the timing of extinctions should provide a clue: if, for instance, the medium-sized herbivores became extinct before the larger species, then the hypothesis cannot be correct.

In any case, once the large herbivores became extinct, the removal of their environmental perturbation would surely have had an impact. This, urges Owen-Smith, "needs to be taken into account, in addition to climatic shifts, in explaining the vegetation transformation that occurred at the end of the Pleistocene." ■ **ROGER LEWIN**

ADDITIONAL READING

N. Owen-Smith, "Pleistocene extinctions: The pivotal role of megaherbivores," *Paleobiology* **13**, 351 (1987).

Animals Yield Clues to Huntington's Disease

Findings that quinolinic acid produces brain lesions in animals similar to those in Huntington's brains suggest a possible strategy for preventing the disease

THE excitation of certain receptors in the brain can, it seems, be a two-edged sword. Although the receptors are part of the normal machinery for receiving incoming nerve signals, overstimulation of the receptors may damage, or even kill, the neurons on which they are located. Such overstimulation may contribute to the nerve cell degeneration occurring in several brain diseases—of which Huntington's disease is a case in point.

Investigators have identified quinolinic acid, a chemical that occurs naturally in the brain, as an "excitotoxin" that may produce the brain degeneration of Huntington's disease by activating a receptor for the excitatory neurotransmitter glutamic acid. They have shown, for example, that treatment of experimental animals with the chemical produces brain lesions similar to those occurring in Huntington's patients.

If the proposal that quinolinic acid causes Huntington's disease is correct, then the way might be open to preventing the development of the condition by inhibiting the chemical's activity at the receptor. In fact, according to results presented at the recent meeting of the Society for Neuroscience,* a drug called MK-801 can reduce the brain damage caused in rats by quinolinic acid and is a possible candidate for human trials.

Although it is far from certain that the animal results are applicable to the human situation, a preventive treatment for Huntington's disease would be highly welcome. The condition is caused by a dominant gene, which means that a patient's children have a 50% chance of inheriting the gene and developing the disease themselves. The symptoms do not usually become apparent until the fourth or fifth decade of life, but, once the mental and physical decline that are characteristic of the disease begin, they progress inexorably, if slowly, until the patient dies.

Within the past few years, molecular biologists have devised a test for identifying those members of Huntington's families that carry the gene. In the current absence of a preventive treatment, however, an individual who learns that he is likely to develop the disease is put under a terrible burden as he anticipates the onset of symptoms.

Researchers began to focus on excitotoxins as a possible cause of Huntington's disease about 10 years ago. The striatum is the area of the brain in which the principal neuronal losses of Huntington's disease occur. In 1976, Robert Schwarcz and Joseph Coyle of Johns Hopkins University School of Medicine noted that when kainic acid, an analog of glutamic acid, is injected into rat brains, it kills neurons originating in the striatum, but not those coming into the striatum from other parts of the brain. "That is what you see in Huntington's disease," explains M. Flint Beal of Harvard's Massachusetts General Hospital, "but Huntington's disease is more complicated."

Beal, Joseph Martin, also of Massachusetts General Hospital, and their colleagues have found that degeneration does not strike all types of striatal neurons equally in the brains of Huntington's patients. Nerve cells that make the neurotransmitters γ-aminobutyric acid (GABA) and substance P show the greatest losses. Neurons that make somatostatin and neuropeptide Y are relatively spared, as are cells that make the neurotransmitter acetylcholine.

Although the brain lesions caused by kainic acid bear some resemblance to those of Huntington's disease, the chemical does not occur naturally in brain and is therefore unlikely to cause the disease. Schwarcz, who is now at the Maryland Psychiatric Research Center in Baltimore, and his colleagues went on to show that quinolinic acid, which is present in brain and is another analog of glutamic acid, also kills striatal neurons.

Moreover, according to Beal, Martin, and their colleagues, nerve-cell killing by quinolinic acid is more specific than that by kainic acid and more similar to what is happening in Huntington's disease. Kainic acid kills all types of neurons, but Beal says, "To our surprise, we found that we did get a relative sparing of the cholinergic and somatostatin-producing neurons with quinolinic acid."

These results were challenged this sum-

*The 17th Annual Meeting of the Society for Neuroscience was held in New Orleans on 16 to 21 November.