Reports from the Field

Anthrax, Spared Calf-Mortality, and Opposite Effects on Two Wildebeest Populations; One With Human Disturbance and One Without

Robert Gainer

P.O. Box 1058, Hanna, Alberta, Canada T0J 1P0.

Corresponding author (email: gainer.bob@gmail.com.)

I made a retrospective comparison of studies of two populations of blue wildebeest (*Connachaetes taurinus*) that had undergone significant long-term anthrax (*Bacillus anthracis*) epizootics. The two studies, one made before and one after human development, revealed similar direct effects on population components but dissimilar effects on population outcomes.

At the time of the first study in 1970 (Gainer 1987), the Selous Game Reserve, Tanzania, was an enormous wildlife sanctuary (55,000 km^2) that was isolated from human disturbance. It was situated in an even larger area–virtually all of the interior of south-eastern Tanzania–with minimal human settlements and no association with domestic livestock. Regions of the Reserve were many hundreds of kilometers of woodland savannah (Rodgers 1980). At the time of the second study in 1990 (Gasaway et al. 1996), Etosha National Park, Namibia, had been reduced in size to about 25,000 km^2 and was bordered by human settlements, agriculture, and domestic livestock. It was not isolated from human disturbance.

The age-class graphs of both studies' mortality rates, constructed from field categories of age, skull collections, and pregnancy rates, were essentially horizontal at about 25% a year. This is unusual because wildebeest populations normally have a “typical” ungulate mortality-rate graph which is described as U-shaped (Caughley 1977) that represents high calf and old-age mortality rates of about 75% per year in concert with low (but proportionally much larger) middle-age rates of about 10% per year.

Both studies recognized that the disease had a predilection for animals older than the calf-age class, as most studies do (Hugh-Jones and De Vos 2002). In addition, during the hot rainy season, the short-grass regions would become lush leading to a concentration of plains ungulates and their predators. Tabanidae (Tsetsie flies, *Glossina* spp.), known to transmit anthrax as well as to reduce animals' resistance to the disease (Kolonin 1971), were almost unbearable. The wildebeest would calve during the middle of the anthrax season. Several species of carnivore were observed feeding on carcasses later determined to have anthrax, but there seemed to be very little predation of calves that even humans on foot could catch. The implication was that scavenging had replaced the normal heavy predation of calves and this, in conjunction with anthrax not infecting young of the year, was the reason for the abnormally low calf mortality-rate. The other implication was that the disease had virtually eliminated the old-age category, the reason for it not having a high mortality-rate.

The combination of the carnivores gorging themselves on anthrax carcasses of older animals instead of the normal, easy-prey calves, the older animals having an estimated 11% additional mortality at this time from the disease, the body weight of older carcasses being more than ten-fold and their numbers three-fold those of the calves represented at least a 300% increase in food quantity obtained from wildebeest carcasses alone (in addition other prey species were afflicted). This volume would have more than offset the loss of meat that the higher survival of calves would have represented. That is, the mortality actually increased the populations' sizes when several calves lived (that normally would have died from predation) for every older animal that died of anthrax. In addition, the resultant populations' young age structures were resilient and productive.

In the Selous Game Reserve, two nearby areas of short grasslands were studied. One had the vast majority of the anthrax carcasses from which over 90% of the skulls that were collected were obtained. This anthrax area represented 41% of the total study area and by the end of the rainy season, it contained less than half the density of wildebeest of the non-anthrax area. Despite this, it showed the most severe signs of deteriorated range conditions and environmental stress. From a comparison of the quantity of skulls collected between the two areas, it was determined that almost half (approximately 44%) stemmed from the disease. The implication from having almost all the disease occurring in the one area with overgrazing and environmental stress was that it was part of a natural, density-dependent self-regulation of the population (Sinclair 1989). Both this 2 yr study and the 10 yr study of the Selous Game Reserve (Rodgers, 1980) found the population of wildebeest to be increasing to a level at or above it's resource ceiling. An increase in population size would increase both the stress on the environment and on the wildebeest population, and consequently an increase in the number of older animals dying of the disease. Such compensatory, non-additive mortality is an example of a natural balanced, self-perpetuating host-parasite relationship (Sinclair 1989).

The Etosha population of wildebeest was in many ways similar to that of the Selous's. The population size, the mortality rate from anthrax, and the effect of the disease on the age structure was essentially the same, but the population had been decreasing for the previous 25 years and was well below its resource ceiling. The authors concluded that the disease was not beneficial to the population, sharing the usual agriculture outlook, that it was an additive, and bottom-down forcing. They considered this to be one of the main reasons why the population was disappearing. The fact that in Etosha, the reduced environmental stress from a relatively low population size was not a bottom-up forcing meant that the wildebeest population was not regulated in a natural, density-dependent manner. The fact that it did not result in a reduced anthrax-mortality rate suggests that the population did not have a balanced self-perpetuating host-parasite relationship, both features seen in the Selous. This implies that there are other factors involved in the occurrence of the disease in Etosha that are related to human disturbance. These top-down forcings, that interfered with the disease' normal relationship with its host, were responsible for the population being below its resource ceiling.

In summary, both these studies illustrate the actual and theoretical effects on a prey population if predation pressure is reduced significantly on the young of the year. Anthrax mortality of blue wildebeests provided more than enough meat to compensate for the carnivores to switch from predating the extremely vulnerable calves to scavenging carcasses it had created. In the natural Selous setting this appeared to be a positive, bottom-up forcing and resulted in a counter-intuitive situation whereby a mortality caused a population to increase in size to attain or surpass its resource ceiling. The opposite seemed to prevail in the Etosha setting with human disturbance. This illustrates that studying disease in a different way, that of the effect on the population (a population biologist's perspective) rather than assuming that all major epidemics are “bad” (a traditional veterinary perspective), provides additional insight.

LITERATURE CITED

Gasaway WC, Gasaway KT, Berry HH. 1996. Persistent low densities of plains ungulates in Etosha National Park Namibia: testing the food regulation hypothesis. *Can J Zool* 74:1556-1572.

Gainer RS. 1987. Epizootiology of anthrax and Nyasa wildebeest in the Selous Game Reserve, Tanzania. *J Wildl Dis* 23:175-178.

Rodgers WA. 1980. *The ecology of grazing herbivores in the Miombo woodland of South East Tanzania*. PhD thesis. Department of Biology, University of Nairobi, Nairobi, Kenya. 808 pp.

Caughley G. 1977. *Analysis of Vertebrate Populations*. Wiley and Sons, Toronto, Canada. 234 pp.

Hugh-Jones ME, De Vos V. 2002. Anthrax and wildlife. *Rev Sci Tech OIE* 21:359-383.

Kolonin GV. 1971. Evolution of anthrax in the USSR II: History of the spreading of the disease and the formation of nozogeographic areas. *Zh Mikrobiol Epidemiol Immunobiol* 48:118-122.

Sinclair ARE. 1989. Population regulation in animals. In: *Ecological Concepts*, JM Cherrett, editor. Blackwell Scientific Publications, Oxford, UK. pp.197-241.