Wildlife Disease Research and Economic Development

Proceedings of a workshop held in Kabete, Kenya, 8 and 9 September 1980

Editors: Lars Karstad, Barry Nestel, and Michael Graham
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Queries about Rinderpest in African Wild Animals

A. Provost

For many years, veterinarians have blamed wild ungulates as being active participants in the rinderpest scene. There is no need to cite references and experimental work on this subject.

In this context, it is puzzling that the veterinary authorities of the African continent embarked in the 1960s in a pan-African rinderpest campaign, known under the name JP15. If wild artiodactyls were a reservoir of virus, there was no hope of eradicating rinderpest from Africa because the prospect of vaccinating the wild fauna was (and still is) rather remote. Despite this evidence, the vaccination campaign has been conducted with success. At present, rinderpest has almost disappeared from Africa, with the exception of the following countries: in West Africa, Mauritania and Mali; and in East Africa, Ethiopia, from where infection spreads from time to time to eastern Sudan and northern Somalia (although the situation is not quite clear in the latter country). Some countries still continue to vaccinate the young stock, others do not vaccinate at all (Guinea, Sierra Leone, Ghana, Togo, Benin, Central African Republic, and Chad). Nevertheless, with the exception of the states already mentioned, no records of rinderpest have been made since 1972 in coastal states and since 1974 in Nigeria. In East Africa, Tanzania, Uganda, and Kenya are reputed to be free of the disease, apart from the scientifically interesting but potentially disastrous isolation of rinderpest virus from an eland found dead in 1974 in the vicinity of Kinna, Kenya, north of Meru National Park.

The disappearance of any clinical evidence of rinderpest from domestic ungulates (cattle, sheep, and goats) has been paralleled by its spontaneous disappearance from wild ungulates. Also, rather interestingly, the few serological surveys undertaken in wildlife have evidenced a sharp decline in terms of numbers positive, with the exception of 4 out of 26 buffalo sera from the Narok district, in southwestern Kenya, which were found positive (FAO 1978).

The discrepancy between this result and the epidemiological evidence is worth further comment. There are only two possible explanations.

Natural Occurrence of a Low-Virulent (Hypovirulent) Strain of Rinderpest Virus

Such strains do exist naturally. It has long been recorded that the passage of a fully virulent rinderpest isolate through a wild species (antelope, giraffe, wild buffalo, wild pig) at first decreases its pathogenicity for cattle and wild animals, although further passages in susceptible domestic calves allow recovery of full virulence. The Masai are aware of this phenomenon and deliberately mingle their cattle with sick buffalo to get "vaccinated" (Scott 1970).

Strains of rinderpest virus already "hypovirulent" for cattle have been isolated on numerous occasions from wildlife in western and eastern Africa. One of the tragic issues is that these seemingly attenuated strains can regain their virulence when they are passaged in cattle.

If such a strain prevails in the buffalo population of Kenya, which could explain the positive results with the buffalo sera, a genuine danger exists, and it may be prophesied that a rinderpest outbreak in cattle will occur because it is unlikely that all weaned calves are vaccinated.

Natural Contact of Buffalo with PPR Virus

The disease "Peste des petits ruminants" is prevalent not only in western and central Africa, but also in the Arabian peninsula, and possibly in India. Surprisingly enough, it has not been recorded from...
eastern Africa although I have a strong feeling that it does exist in Sudan where it may have been confused with rinderpest. In the laboratory, confusion is possible because these two "brother" viruses have almost identical behaviours in tissue culture, gel diffusion, and other serological tests. The difference lies in the pathogenicity for cattle: PPR virus is apathogenic for cattle although infective (with subsequent immunity to rinderpest); rinderpest is pathogenic. Another, more subtle difference, is the disparity of magnitude in serum-neutralization tests when they are performed with either the homologous or the heterologous virus: sheep and goat sera react with higher titres when tested with PPR virus than when tested with rinderpest virus and reversely, cattle sera (from rinderpest immune animals) have lower titres to PPR virus than to rinderpest virus.

Now comes a crucial question: In nature, is PPR virus able to infect other ungulates spontaneously, exclusive of any clinically recognizable reaction? I think that this question cannot be answered, possibly because it has never been raised.

I have a feeling this question may be answered by looking at very recent findings in Oman. In that country, where rinderpest is unknown in human memory, it has been found that small ruminants have genuine PPR antibodies but also that unvaccinated cattle have higher antibody titres to PPR than to rinderpest virus.

Is it possible that this is the case in Kenya for buffalo? The approach is then simple and lies in the performance of comparative titrations of PPR and rinderpest antibodies on the same sera.

I hope that the results of the survey are positive, because it would be less alarming, and certainly more scientifically fruitful, to assume the possible occurrence of PPR infection in the buffalo population rather than the existence of a hypovirulent strain of rinderpest virus. I would also urge that the eland isolate from 1974 be reexamined in the light of these comments.


The survey was undertaken before a virgin outbreak of rinderpest flared up in the country, introduced from a neighbouring state.