

SHORT COMMUNICATION

EXPERIMENTAL TRANSMISSION OF DUGBE (A NAIROBI DISEASE GROUP) VIRUS TO WEST AFRICAN DWARF SHEEP

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ABSTRACT

West African Dwarf sheep were inoculated with a low mouse brain passage of Dugbe (Nairobi sheep disease group virus). Mild febrile reaction and low level of neutralising antibody were demonstrated in experimental animals.

INTRODUCTION

Dugbe virus, a tick-borne virus was first isolated by Causey *et. al.* (1971) from *Amblyoma variegatum* ticks. Further virus surveillance has yielded many more isolates from cattle, other species of ticks and *culicoides*. (Causey *et. al.* 1971, Kemp *et. al.* 1971). Serological studies showed that Dugbe is related to Ganjam a Nairobi sheep disease (NSD) group virus by complement fixation test (CFT) (Davies and Casals 1971).

Morphological and morphogenetic studies have also revealed similarities of Dugbe to Ganjam (David-West *et. al.* 1974, Murphy *et. al.* 1973). Although Dugbe virus has been repeatedly isolated in the country its veterinary importance is unknown. In the present studies, three West African Dwarf sheep were infected with Dugbe virus in an attempt to determine its pathogenicity for this breed.

MATERIALS AND METHODS

Virus: The prototype virus Ib-AR 1792 isolated from *Amblyoma variegatum* ticks collected at Dugbe market in 1965 was used in transmission experiments. The virus had undergone three passages in suckling mouse brain. Stock virus titre was $6.0 \log_{10}/0.02 \text{ ml}$

Animals: For West African dwarf sheep aged 5 – 9 months which were tested and found negative for serum neutralising antibody to Dugbe virus were used. They were given anthelmintics, bathed in acaricide solution and kept in screened quarters before infection. Three animals were inoculated subcutaneously with 1.5×10^5 suckling mouse LD₅₀ of a third mouse brain passages of the virus. The infected animals were examined daily for signs of illness and rise in rectal temperature. Daily blood samples were collected for viraemia studies and total leucocyte count. Test for viraemia was carried out by intracerebral (i.e.) inoculation of whole blood into 2 day old mice. Sera were collected on days 7 and 21 post infection (p.i.) and tested for antibody by neutralisation test (NT).

Neutralisation test: Neutralisation test was performed on suckling mice using the standard method of constant serum – varying virus technique. Neutralisation indices (NT) were expressed as differences between \log_{10} suckling mouse LD₅₀ of test and normal control sera.

RESULTS

None of the infected animals developed viraemia nor overt clinical disease 10 days p.i. Two sheep exhibited a rise in rectal temperature – 104.00°F and 104.00°F respectively on days 1 and 2 p.i. There was no change in total leucocyte count; but low level of neutralising anti-

body was found in convalescent sera. Table shows the antibody response in sheep inoculated with Dugbe virus. Although no N antibody was detectable in sera collected from infected animals on day 7 p.i. convalescent sera obtained two weeks later contained low titre of N antibody.

TABLE

Neutralising antibody response in Sheep infected with Dugbe virus

Animal No.	Clinical Response	Antibody Titre in Log ₁₀	
		Acute Phase Day 7	Convalescent Day 21
1	Febrile	0	0.8
2	Febrile	0	0.8
3.	Non-Febrile	0	0.4
4	Control	0	0

DISCUSSION

Natural and experimental infection of sheep with Nairobi sheep disease virus in East Africa produced severe and fatal disease (Weinbren *et. al.* 1958, Mugeru and Chema 1967). In the present studies the West African dwarf sheep were not susceptible to overt disease following inoculation with Dugbe virus. The low level of neutralising antibody found in all the experimental sheep indicated that although these animals were infected, the immune response was poor. Previous studies had demonstrated poor antibody response in experimentally infected cattle (Causey, 1965)^a.

Although the available data is scanty, the mild manifestation of Dugbe virus infection in the West African dwarf sheep indicated that this breed might be resistant to infection by Nairobi sheep disease group of viruses. However, the effect of stress factors such as starvation and intercurrent disease in the modification of the course of natural infection is not known. The high prevalence of this virus in Nigeria and its ability to produce severe natural human disease (Causey, 1965)^b suggest that this virus might be of great public health importance.

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