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The role of sheep and goats in the epizootiology of foot-and-mouth disease in Kenya

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SUMMARY

The pathogenicity of two bovine field strains of virus for indigenous goats was examined in the laboratory. The goats failed to develop clinical disease or become virus carriers although the majority showed a definite immune response. A field survey in a foot-and-mouth disease enzootic area showed that the indigenous sheep and goat populations were frequently exposed to infection as evidenced by a high proportion of sero-positive animals but the incidence of virus carriers was very low in goats and no carriers were detected in sheep.

INTRODUCTION

There are some 9 million sheep and goats in Kenya and a large proportion are of indigenous breeds found in areas where foot-and-mouth disease is enzootic. Their exact role in the epizootiology of the disease has not been studied and they are not included in vaccination control programmes, which are confined to cattle, although all species are subject to the normal quarantine procedures. Sheep and goats may be infected experimentally (Dellers & Hyde, 1964; Geering 1967; Burrows, 1968; McVicar & Sutmoller, 1968, 1972) and natural infection has been described by Zaikin (1959) and Littlejohn (1970). The disease has been reported as frequently occurring in sheep in certain regions of Iran (Hedjazi, Ansari & Nadalian, 1972) particularly associated with the appearance of the exotic SAT 1 and A_{22} strains of virus. In addition to being susceptible to infection, sheep and goats may also become virus carriers after exposure. Burrows (1968) showed that cross-bred Southdown sheep can remain carriers for up to 5 months after exposure and McVicar & Sutmoller (1968, 1972) found that about 50% of a group of sheep and goats exposed to an infected steer were carriers 4 weeks later. Sellers & Parker (1969) showed that sheep can be readily infected by airborne virus and since the resulting disease is frequently mild or inapparent they might remain an undetected source of further infection for other species.

There are no confirmed reports of overt disease in indigenous sheep and goats in Kenya. Should they be involved as reservoirs or amplifiers of virus this would be indicated by a significant incidence of carrier animals and of those showing positive

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serology. This report gives the results of a comparative study on the incidence of virus carrier and sero-positive animals in an enzootic area of the country in the cattle, sheep and goats. In addition, the course of the disease in indigenous goats following experimental exposure is described.

METHODS

Laboratory pathogenesis studies with goats

Animals

Indigenous goats 1–3 years old were used. All were negative for FMD antibodies as judged by a micro cell metabolic inhibition test (Anderson, Doughty & Anderson, 1974).

Virus exposure

Experiment 1. A type O virus originally isolated during a field outbreak of foot-and-mouth disease in cattle and which had subsequently been passaged a further 7 times in cattle was used. It was titrated in 2 susceptible steers (Henderson, 1949) and a suspension containing $10^{4\cdot3}$ bovine ID 50 per ml prepared. A 0·5 ml. volume of this suspension was inoculated into the coronary band of the left fore foot of each of 14 goats. Sixteen days after exposure these goats were moved to a clean loose-box in the same isolation unit and 4 susceptible uninfected goats introduced.

Experiment 2. A susceptible steer was infected by intradermolingual inoculation of a bovine field strain of type SAT 2 virus. When tongue vesicles had developed, 10 susceptible goats were introduced. Two days later, when the steer had generalized lesions in the form of vesicles on three feet, the goats were moved to a clean loose-box in the same isolation unit.

Collection of samples

At intervals after exposure the following antemortem samples were collected for attempted virus isolation:

(i) Oesophageal-pharyngeal (O-P) scrapings using a probang as described by Burrows (1968).

(ii) Nasal swabs.

(iii) Whole blood in 0·2% w/v ethylenediaminetetra-acetic acid (EDTA) for plasma.

(iv) Faeces.

The O–P samples, nasal swabs and approximately 1 gm. faeces were collected into 5 ml. phosphate buffered saline (PBS) pH 7·6 containing 0·62% w/v gelatin, 200 units/ml. penicillin, 200 units/ml. polymixin B sulphate, 150 μg./ml. neomycin sulphate and 100 units/ml. mycostatin.

In the first experiment, animals were autopsied at intervals after exposure and 10% w/v suspensions of retro-pharyngeal, bronchial and mesenteric lymph nodes, lung and spleen made in PBS for attempted virus isolation.

Isolation of virus

Samples were examined for the presence of virus in primary bovine thyroid cell monolayers (Snowdon, 1966) in test-tubes, 0·2 ml. of each sample being inoculated on each of 5 monolayers, incubated at 37° C. for 30 min., and 2 ml. of maintenance medium added. Tubes were examined for 3 days and after the development of cytopathic effect (CPE) the presence of virus was confirmed by typing in a micro-complement-fixation test. The amount of virus in each positive sample was titrated in primary bovine thyroid cells.

Serum antibody assay

This was carried out in a micro metabolic inhibition test (Anderson, *et al.* 1974) and titres expressed as the logarithm of the reciprocal serum dilution neutralizing 100 TCID 50 virus in the test. Antibody titres of 1·35 or greater were considered positive.

Field survey

O–P samples for virus isolation and sera for antibody assay were collected from 350 Masai fat-tailed sheep and 407 goats at widely separated places in the Masailand area of Kenya where FMD is enzootic. As a comparison samples were also collected from 676 Zebu cattle in the area, the O-P samples being collected as described by Sutmoller & Gaggero (1965). It is customary in this area for the cattle, sheep and goats to be enclosed at night in close contact, with much opportunity for the spread of virus from one species to another.

RESULTS

Laboratory exposure

Experiment 1

No clinical lesions developed in any of the animals inoculated.

The results of virus isolation from the ante-mortem samples collected at intervals after exposure are shown in Table 1. A viraemia, indicative of virus multiplication, was present in all but one of the goats 2 days after exposure and in the majority of the viraemic goats (9/14) at least 100 TCID 50 of virus/ml of plasma was present. The viraemia declined rapidly. Virus was recovered from the throat in nearly all the goats up to the 7th day but thereafter virus could only be recovered from one animal up to the 21st day after exposure. Unfortunately, this animal was autopsied on the 21st day and as virus was still present in quite large amounts it would probably have remained a carrier for some time longer.

Virus was present in the nasal secretions on the 2nd day after exposure in most of the animals but thereafter the number of positive animals and the amount of virus being excreted decreased and all were negative on the 21st day. A small amount of virus was found in the faeces of 2 goats on the 2nd day after exposure.

The post-mortem samples showed a similar distribution pattern (Table 2). On the 2nd day after exposure, when a viraemia was present, all the tissues collected contained virus, the largest amount occurring in the spleen. On the 5th day small

Table 1. *Results of virus isolation from ante-mortem samples in Experiment* 1 *at intervals after exposure*

Days after exposure to virus	Plasma		Oeosphageal/ pharyngeal sample		Nasal secretions		Faeces	
	Proportion virus positive	Mean titre (log_{10}/ml)	Proportion virus positive	Mean titre (log_{10}/ml)	Proportion virus positive	Mean titre (log_{10}/ml)	Proportion virus positive	Mean titre (log_{10}/ml)
2	13/14	3·2 (trace–5·4)	13/14	2·5 (trace–4·0)	13/14	2·3 (trace–5·4)	2/14	Trace
5	Nil	—	12/13	1·8 (trace–2·0)	9/13	1·5 (trace–2·8)	Nil	—
7	Nil	—	12/12	1·8 (trace–3·0)	5/12	1·4 (trace–2·4)	—	—
14	Nil	—	1/11	Trace	—	Nil	Nil	—
21	Nil	—	1/10	2·6	Nil			
29	—	—	Nil	—	Nil	—	—	—
40	—	—	Nil	—	Nil	—	—	—
56	—	—	Nil	—	Nil	—	—	—

Table 2. *Results of virus isolation from tissues at intervals after exposure*

Tissue	Days after exposure: Virus isolation and titre (log_{10}/ml.)					
	2	5	7	14	21	29
Retro-pharyngeal lymph node	1·4	Trace	Nil	Nil	Nil	Nil
Bronchial lymph node	Trace	Trace	Nil	Trace	Nil	Nil
Mesenteric lymph node	Trace	Nil	Nil	Nil	Nil	Nil
Lung	1·4	Nil	Nil	Nil	Nil	Nil
Spleen	3·0	Nil	Nil	Nil	Nil	Nil

amounts of virus could be detected in the bronchial lymph node and the retro-pharyngeal lymphoid tissue. Thereafter, virus was only isolated from the bronchial lymph node on the 14th day after exposure. The goats that were autopsied on the 7th and 14th day had positive O-P samples but attempted virus isolation from a suspension of retro-pharyngeal lymphoid tissue failed. The 4 susceptible goats that were housed with the virus-inoculated goats on the 16th day after inoculation did not develop viraemia or become virus carriers.

Experiment 2

Similar ante-mortem samples were collected on days 1, 3, 6, 7, 12 and 15 after removal of the infected steer. None of the 10 goats developed a viraemia and only one became a virus carrier (No. G.34). No clinical lesions were detected. Virus was isolated from the O-P samples from the positive goat for 6 days, the highest titre ($10^{2·6}$/ml.) being present on the 3rd day. Small amounts of virus were present in the nasal secretions for 3 days. No virus was isolated from this animal after the 6th day.

The antibody responses in the two experiments are shown in Fig. 1. There was

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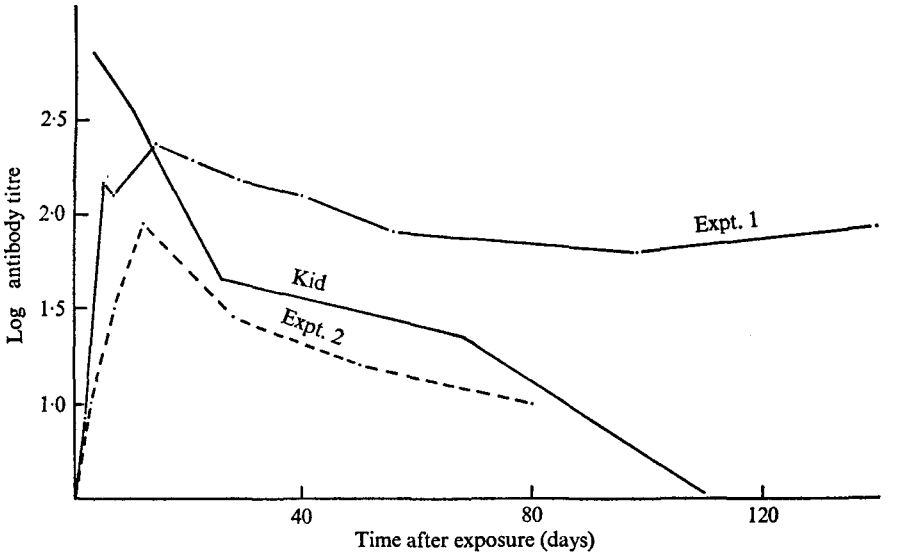

Fig. 1. Immune response in goats following exposure to FMD virus.

Table 3. *Virus carrier incidence in cattle, sheep and goats in an endemic area*

	Sampling point 1		2		3		4		5	
	No. of samples	Carrier incidence	No. of samples	Carrier incidence	No. of samples	Carrier incidence	No. of samples	Carrier incidence	No. of samples	Carrier incidence
attle	144	Nil	134	O:0·75% A:1·5%	100	O:2%	98	Nil	200	O:1% A:2·5%
heep	50	Nil	56	Nil	44	Nil	146	Nil	54	Nil
oats	40	Nil	69	O:1·5%	50	Nil	136	Nil	51	Nil
linical outbreaks in cattle in previous 18 months	Type A		Types A and SAT2		Types O and A		Types O and A		Types O, A and SAT 1	

an immune response in all the goats in Experiment 1 characterized by a rapid rise in antibody concentration which reached a peak 14 days after exposure and declined slowly. There was still a large serum antibody concentration 140 days after exposure. A kid was born to one animal on the 30th day after exposure when her antibody titre was 2·7. It had a definite passive immunity at birth with the antibody concentration declining steadily over 90 days. The antibody half-life was about 10 days. In Experiment 2 the antibody response was more varied. Only three animals, including the carrier animal No. G.34, showed an immune response and their mean titres are shown in Fig. 1. They were lower than those found in Experiment 1 and declined more rapidly.

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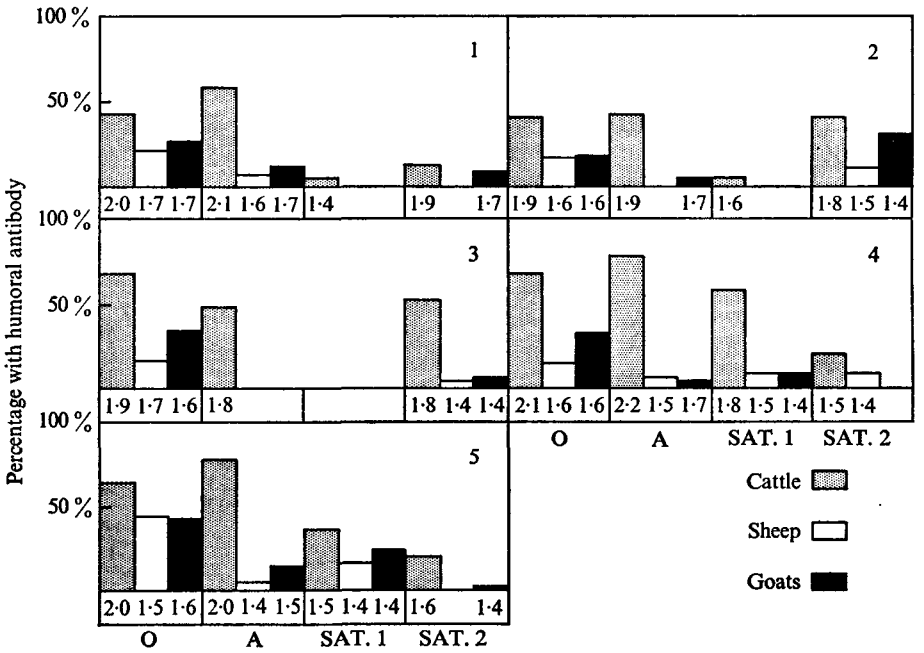

Fig. 2. Proportion of cattle, sheep and goats with humoral antibody at each sampling point (serotype and group mean titre).

Field survey

O-P and serum samples were collected from cattle, sheep and goats in five widely separated places in an area where clinical outbreaks of types O, A, SAT 1 and SAT 2 occur frequently in cattle. The numbers of each species sampled, the clinical outbreaks that have occurred at each sampling point in the previous 18 months and the incidence of virus carriers found are shown in Table 3. Virus carriers were found in cattle at three of the sampling points and in goats at one sampling point. No carriers were found in sheep samples. Where carrier virus was found in the goats it was also found in the cattle. Only a trace (1/5 bovine thyroid monolayers showing CPE) of virus was present in the goat O-P samples. The immunological types of carrier virus isolated did not always correspond to the types of confirmed clinical FMD in the area. This is because the disease is frequently mild in the indigenous Zebu cattle and often goes undetected.

In addition to these random surveys O-P samples were collected from 61 goats which were closely confined at night with cattle that had confirmed type O infection. No carriers were found in the goats although 89 % had significant type O antibody titres. In another confirmed SAT 2 outbreak in cattle no virus was isolated from the sheep and goats at the same place although 56 % had significant antibody titres. In both places the sheep and goats had obviously been exposed to virus sufficient to stimulate an immune response but the virus had not persisted in the throat.

The antibody concentrations found in the three species at the five sampling

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points are shown in Fig. 2. They are given as the percentage of the sample with antibody titres of 1·35 or greater and the geometric mean titre of each positive group is also indicated. In all the places the percentage of cattle with significant antibody titres was higher than for sheep and goats and the mean titre of the positive animals was also greater. In general there were fewer sero-positive sheep than goats.

DISCUSSION

In the laboratory exposure experiments, clinical lesions failed to develop in the indigenous goats used even though many developed viraemia. This is in contrast to the findings of McVicar & Sutmoller (1968, 1972), who found that mouth lesions and less frequently foot lesions developed in the majority of goats exposed. However, this difference is probably due to the use of different breeds and also to the use of different strains of virus. In this study bovine field strains were used as they are the ones to which the sheep and goat populations are most frequently exposed. After inoculation of virus, the majority of the goats developed viraemia but did not remain long-term virus carriers. Only 1 animal out of 14 was still a carrier 21 days after exposure. When exposed to an infected steer none of the goats developed a viraemia and only 1 out of 10 became a carrier and only for 6 days. All the group infected by inoculation and 30% of the group exposed to constant infection developed specific antibodies. There was, therefore, good evidence of virus multiplication but there was no persistence of virus in the tissues examined or development of a significant carrier state.

The field survey showed an almost complete absence of virus carriers in both the sheep and goat populations even where there was close contact with clinically infected cattle. That they had been exposed to virus was evident from the large proportion of animals with specific antibody titres, although the mean titres were lower than those found in cattle.

The results of this study indicate that the indigenous sheep and goat populations probably do not contribute to any great extent to the persistence or spread of FMD virus and there is no indication at present that these species should be included in vaccination programmes. Should a variant appear which readily infects sheep, such as was observed in Kenya by Geering (1967) where a type O virus caused severe disease associated with a high lamb mortality in exotic sheep, then local control measures could be instituted.

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REFERENCES

Anderson, E. C., Doughty, W. J. & Anderson, J. (1974). The effect of repeated vaccination in an enzootic foot-and-mouth disease area on the incidence of virus carrier cattle. *Journal of Hygiene* **73**, 229–36.

Burrows, R. (1968). The persistence of foot-and-mouth disease virus in sheep. *Journal of Hygiene* **66**, 633–40.

Dellers, R. W. & Hyde, J. L. (1964). Response of sheep to experimental infection with foot-and-mouth disease virus. *American Journal of Veterinary Research*. **25**, 469–89.

Geering, W. A. (1967). Foot-and-mouth disease in sheep. *Australian Veterinary Journal* **43**, 485–9.

Hedjazi, M., Ansari, H. & Nadalian, M. Gh. (1972). Clinical study of some foot-and-mouth disease outbreaks in sheep and goats in Iran. *Revue. Med. Vet.* **123**, 1085–8.

Henderson, W. M. (1949). The quantitative study of foot-and-mouth disease virus. *Report Series, Agricultural Research Council, London*. No. 8. H.M. Stationery Office.

Littlejohn, A. I. (1970). Foot-and-mouth disease in sheep. *State Veterinary Journal* **25**, 97–115.

McVicar, J. W. & Sutmoller, P. (1968). Sheep and goats as foot-and-mouth disease virus carriers. *U.S. Livestock Sanitary Association Proceedings* **72**, 400–6.

McVicar, J. W. & Sutmoller, P. (1972). Experimental foot-and-mouth disease in sheep and goats: an epizootiological model. *Archiv für die gesamte Virusforschung* **38**, 85–96.

Sellers, R. F. & Parker, J. (1969). Airborne excretion of foot-and-mouth disease virus. *Journal of Hygiene* **67**, 671–7.

Snowdon, W. A. (1966). Growth of foot-and-mouth disease virus in monolayer cultures of calf thyroid cells. *Nature, London* **210**, 1079–80.

Sutmoller, P. & Gaggero, A. (1965). Foot-and-mouth disease virus carriers. *Veterinary Record* **77**, 986–9.

Zaikin, D. G. (1959). Clinical picture of foot-and-mouth disease in sheep under pasture maintenance conditions. *Veterinariya* **36**, 32–4