

CLINICAL AND ANTIGENIC RELATIONSHIP BETWEEN ISOLATES OF SHEEP AND GOAT POX VIRUSES

R. P. KITCHING and W. P. TAYLOR

Animal Virus Research Institute, Pirbright, Surrey GU24 0NF, UK

SUMMARY

Isolates of sheep pox and goat pox from Nigeria, Sudan, Kenya, Yemen Arab Republic, Turkey, Pakistan and India were inoculated into British breeds of sheep and goats. Although the isolates displayed a host preference the gross clinical pathology of the disease produced by the different isolates was indistinguishable. The Yemen, Nigeria and India isolates could not be distinguished using homologous and heterologous antisera in neutralisation tests. Animals that had recovered from infection with one isolate were resistant to challenge with any of the other isolates and a single vaccine for use against sheep pox and goat pox is described. The classification of the malignant pox diseases of sheep and goats is discussed.

INTRODUCTION

Sheep pox is defined as a malignant pox of sheep characterised by fever and generalised pocks; goat pox is a malignant pox of goats similarly characterised. Sheep-and-goat pox as described in Kenya (Davies, 1976) embraces both definitions. Sheep pox virus, goat pox virus and the virus of bovine lumpy skin disease (Neethling virus) make up the capripox group of pox viruses. These viruses cannot be distinguished serologically (Davies and Otema, 1981) and the clinical signs they produce have many features in common (Burdin, 1959; Green, 1959; Plowright, Macleod and Ferris, 1959).

A number of research workers have reported that it is possible to infect goats with sheep pox virus and sheep with goat pox virus (Sen, 1968; Sharma and Dhanda, 1971; Al-Bana, 1978) and that on recovery the goats or sheep resist challenge with virulent goat pox virus or sheep pox virus respectively. This present study examines the relationship between a variety of isolates of sheep pox virus and goat pox virus collected from different countries within the endemic area in terms of clinical disease and their ability to induce cross-protection.

MATERIALS AND METHODS

All viruses were grown and maintained in secondary lamb testis (LT) cells using Glasgow modified Eagle's medium (GMEM) supplemented with 2% ox serum (OS). Isolates of field virus were made from pox lesions taken during post-mortem examinations. Samples were prepared by grinding with sterile sand and GMEM to give a 10% (w/v) suspension. Antibiotics were included at the following rates: sodium penicillin 10^3 iu per ml, streptomycin sulphate 1 mg per ml, mycostatin 10^2 iu per ml and neomycin 200 iu per ml. After clarification supernatant fluid was inoculated on to confluent LT monolayers in 25 cm² plastic Falcon flasks. At the same time the sediment was examined with a transmission electron microscope for particles with pox virus morphology. Infected cells developed a characteristic cpe consisting of retraction of cell membrane, rounding of cells and margination of nuclear chromatin. Commencing between four and

nine days post-inoculation these effects increased in extent over the next six days and, when maximal, cultures were harvested to -20°C . Stock virus was prepared from the thawed supernate by low speed clarification and stored at -70°C . All passages were made in LT cells using similar methods. The designation, source and passage level of the virus isolates used are given in Table I. In addition to field isolates some were obtained from other laboratories and were used as supplied.

Stock virus was titrated in $0.5 \log_{10}$ steps in microtitre plates, inoculating $50 \mu\text{l}$ of virus together with a suspension of 2×10^4 LT cells in $100 \mu\text{l}$ of GMEM containing 6% OS. Eight-well replicates were used and the sealed plates incubated at 37°C for nine days. Those wells showing cpe were considered positive and the virus titre was calculated after Kärber (1931). When received the Pakistan isolate did not produce a cpe on LT cells while the India isolate was so contaminated with fungus that titration was impossible; all remaining isolates could be titrated without difficulty.

Twenty-two adult Soay sheep, eight yearling Blackface, three yearling Border Leicester cross, six yearling and two four-month-old Dorset Horn cross, three yearling Suffolk cross and one yearling Southdown cross sheep were used together with 13 British White goats of between six months and four years old. Animals were housed in high security isolation units and separate boxes were used for each experiment. All sheep were sheared before the inoculation of virus. In addition an area of approximately 100 cm^2 was close-clipped over the last four ribs on each side; a similar area was prepared on the goats. Virus was inoculated using a single intradermal injection of 0.2 ml into the centre of the clipped area on the right side with a 26 g needle. Two animals received the same volume of inoculum intravenously and in two experiments susceptible contacts were left in the presence of inoculated animals. Animals were clinically examined daily, their temperatures recorded and the diameter of primary skin lesions and subsequent secondary lesions measured. Details of the virus dose, route of inoculation and clinical reaction can be seen in Table II. Post-mortem examination was carried out on those animals that died or were humanely destroyed *in extremis*. Tissue samples were preserved in 10% formol-saline for histological examination.

TABLE I
Sheep and goat pox viruses used in comparative studies

Virus	Year of isolation	Passage level ¹	Source	Reference
Nigeria sheep pox	1977	LT/3	Sheep skin	Asagba and Nawathe, 1980
Yemen goat pox	1983	LT/1	Goat lung	
Sudan sheep pox ²	1983	LT/2	Sheep skin	Davies, 1976
Sudan goat pox ²	1983	LT/2	Goat skin	
Pakistan sheep pox/P5	Unknown	Unknown	Unknown	Davies, 1976
India goat pox	1983	LT/1	Goat skin	
Kenya sheep-and-goat pox/0240	1976	LT/5	Sheep	
Turkey sheep pox	1983	LT/1 LK/3	Sheep skin	

¹ LT, lamb testis; LK, lamb kidney.

² The Sudan isolates were collected from one flock in which both sheep and goats were affected.

TABLE II
Clinical reaction in sheep and goats infected with capripox isolates

Isolate	Animals inoculated	Virus dose (log ₁₀ TCID ₅₀ per animal)	Primary reaction	Pyrexia ¹	Secondary papules	Enlarged lymph node	Rhinitis	Conjunctivitis	Scab ³ formation	Death/Euthanasia	Days to:	
											14-16	14-21
Nigeria sheep pox	12 sheep	0.5-3.5	3-5	6-9	7-11	9	7-11	9-11	14-16	14-21		
	2 goats	3-5	2-3	8 ²	—	—	—	—	8	—		
	10 Soay	3.5-4.3	2	4-6	7-9	—	6	6	—	8-14		
Yemen goat pox	3 Soay	Contact	—	16-30	not recorded	—	not recorded	not recorded	—	25-36		
	2 sheep ⁴	4.3	5	6	8	10	11	13	—	14-15		
	2 goats	4.3	3	7-8	6	8	7	11	—	14		
Pakistan sheep pox	3 Soay	not recorded	1	6-7	8-10	10	—	—	14	—		
Sudan sheep pox	2 sheep	2.2	3	10	7 ²	8 ²	10 ²	10 ²	12	20 ²		
	2 goats	2.2	3	6	7	8	10	10	—	14-17		
	2 sheep	1.9	3	7-10	11 ²	12 ²	—	—	12	—		
Sudan goat pox	2 goats	1.9	3	6	7	8	10	10	—	16-17		
	1 sheep	Contact	—	21	25	not recorded	25	—	31	—		
	1 goat	Contact	—	18	19	not recorded	23	23	9	31		
India goat pox	2 sheep	not recorded	3	7-8	—	8	—	—	—	—		
	2 goats	not recorded	1-2	6-7	7-8	7	10	10	—	14-17		
	2 sheep	3.8	3	7	8	not recorded	9	9	—	12-15		
Kenya sheep- and-goat pox	6 Soay	1.1-5.1	2-10	—	—	—	—	—	10	—		
	2 goats	3.1	4	—	—	—	—	—	—	—		

¹ Rectal temperature above 40°C.

² One animal only.

³ Animals that recovered.

⁴ Four-month old animals.

— Not seen.

Sheep and goats that had recovered from infection with one of the virus isolates were challenge inoculated intradermally with 0.2 ml of one of the other isolates into the clipped area on the left flank. Two of the recovered animals were used as controls and inoculated with 0.2 ml of uninfected tissue culture fluid prepared in a similar manner to the virus isolates but from an uninfected LT culture.

Four additional adult Soay sheep and four additional yearling British White goats were used as uninfected controls in the experiment to assess the immunity of the sheep and goats that had recovered from the Kenya isolate and seven additional Lowland cross sheep that had recovered from an aerosol infection experiment with the Yemen isolate were used in the challenge experiments. The interval between first virus inoculation and challenge inoculation varied between 22 and 74 days.

Whole blood was collected daily into heparin (5 units/ml final concentration) from 12 sheep and two goats inoculated with the Nigeria isolate. Swabs were taken from the conjunctiva and nasal cavity of six of these animals. Pre- and post-inoculation serum samples were collected from all animals and stored at -20°C until used. Buffy coat cells were removed from the heparinised blood samples, washed and inoculated on to LT monolayers in 25 cm² plastic Falcon flasks. The swabs were placed in Universal bottles containing 5 ml of M25 phosphate buffer with antibiotics. The bottles were agitated for 30 min at 4°C , the swabs removed and 1 ml of the fluid inoculated on to LT cells in 25 cm² plastic Falcon flasks. All cells were examined daily for evidence of cpe.

The sera collected from three sheep that had recovered from infection with the Nigeria isolate, the Yemen isolate and the India isolate were inactivated at 56°C for 30 min and two-fold serial dilutions of each serum were prepared from 1/10 up to 1/1280. Each dilution in 50 μl was added to four complete rows on three microtitre plates. Half log₁₀ serial dilutions of suspensions of the Nigeria, Yemen and India virus isolates were prepared and 50 μl added to the microtitre plates in columns so that each virus was titrated against fixed dilutions of homologous and the two heterologous antisera (Westaway, 1965). The plates were covered and incubated at 37°C for 2 h and LT cells were added at the ratio of 2×10^4 cells per well in 50 μl . Each virus isolate was separately titrated as described. Plates were examined for cpe after nine days. The neutralising activity of each antiserum against the three viruses was calculated for each serum dilution and regression lines calculated. The lines were defined by the equation $Y=A+BX$ where A is the log₁₀ decrease in virus titre produced by undiluted serum and B is the "neutralisation slope" (Westaway, 1965). The area defined below each line was calculated from the equation: $\text{area} = -\frac{1}{2}A^2/B$.

RESULTS

The clinical signs produced by the different isolates were very similar (Table II) and do not require separate descriptions. Following experimental inoculation a reaction developed at the inoculation site within one to five days. Goats reacted more quickly than sheep and animals receiving a higher virus inoculum reacted earlier. The rise in rectal temperature to over 40°C occurred within 10 days of inoculation in all animals independent of the titre of inoculum.

Secondary skin papules appeared two to five days after the development of fever. The papules on the goats were preceded by red macules easily seen on the white skin. Invariably they first appeared on the hairless skin under the tail.

Macules were less obvious on the sheep and their appearance was as frequent on the woolled areas as on the hairless areas of the groin, axilla and tail. The diameter of the secondary skin papules was between 0.5 and 1.0 cm except when they coalesced as occurred on many of those animals which died. On these animals papules covered the whole body and head but only rarely were the lower limbs affected. Soon after the development of secondary papules rhinitis and conjunctivitis were seen and there was gross enlargement of superficial lymph nodes especially the prescapular. The eyelids were swollen and eventually completely covered the eyeball. The discharge from the eyes and nose became mucopurulent as the papules on the conjunctiva and external nares ulcerated. The mucous membranes of the eyes, nose, lips, anus, vulva or prepuce became necrotic. The papules on the skin of the animals that survived changed directly into scabs with no vesicular or pustular stage. Those animals that died did not develop scabs; their papules were usually less obviously separated from surrounding skin and tended to be flatter. Their colour deepened from bright red to dark purple particularly apparent on the white skin of the goats.

Animals continued to eat in spite of a high fever until they were unable to because of oral lesions. Terminally the animals that died developed respiratory embarrassment expiration being particularly laboured. They became weak, disorientated and eventually unable to stand; occasionally keratitis was seen. The scabs on the surviving animals were shed over a period of three to six weeks leaving a raw granulating area smaller than the original lesion. Secondary scabs developed which were also eventually shed leaving small scars.

The Nigeria sheep pox virus was particularly virulent for Soay sheep whether given by intradermal or intravenous injection. All affected animals died between eight and 14 days of inoculation. The animals appeared depressed while secondary papules were not numerous and tended to disperse prior to death. The body lymph nodes were not grossly enlarged. Conventional breeds of sheep were more resistant to this isolate and three survived infection. The clinical lesions in these sheep were more severe, however, and the enlargement of the superficial lymph nodes was very apparent. In goats the Nigeria sheep pox virus produced a transient fever and no more than a local lesion at the site of inoculation. In contrast the India goat pox virus was highly virulent for goats but mild in sheep where it gave pyrexia but no secondary lesions. The Yemen goat pox virus was equally lethal for sheep and goats while the Sudan isolate was more lethal for goats whether it was derived from a sheep or a goat. Of the isolates examined the Kenya sheep-and-goat virus was the mildest producing only a local lesion in both sheep and goats.

Papules were present on the skin and mucous membranes of the conjunctiva, nares, anus and vulva or prepuce of all the animals that died. There was no distinction between the isolates as to where the secondary papules appeared on the skin. On some animals the papules were more numerous on the woolled or haired areas, on others on the hairless areas of the groin or axilla. The papules on the mucous membranes were typically ulcerated and surrounded by necrotic tissue. On post-mortem the skin papules in many instances appeared to have dispersed or possibly the papules had coalesced. The skin of the body was always grossly thickened and on the goats had a parchment-like texture. All the body lymph nodes were grossly enlarged. The retropharyngeal lymph nodes in some cases were sufficiently enlarged to cause respiratory obstruction by pressure on the pharynx.

TABLE III
Distribution of post-mortem lesions in sheep and goats infected with capripox isolates

Isolate	Animal	Number of animals with lesions on															
		Sk ¹	Ton	Ph	Tr	Oes	Lung	Ru	Ab	Ret	Turb	Caec	LI	Liv	Kid	GB	Cornea
Nigeria	Sheep	9/9	3/7	2/7	1/7	1/7	5/7	1/7	2/7	0/7	0/7	0/7	0/7	1/7	0/2	0/7	2/7
	Soay	8/9	1/9	2/9	0/9	0/9	9/9	2/9	2/9	0/9	0/9	0/9	0/9	0/9	0/9	0/9	0/9
Yemen	Sheep	2/2	2/2	2/2	0/2	0/2	2/2	2/2	0/2	0/2	0/2	0/2	0/2	1/2	1/2	0/2	0/2
	Goats	2/2	1/2	0/2	0/2	0/2	2/2	0/2	2/2	0/2	0/2	0/2	0/2	0/2	2/2	0/2	0/2
Sudan	Sheep	1/1	1/1	1/1	0/1	0/1	1/1	1/1	1/1	0/1	0/1	0/1	0/1	0/1	1/1	1/1	0/1
	Goats	5/5	2/5	2/5	0/5	1/5	5/5	0/5	4/5	0/5	1/5	0/5	0/5	3/5	0/5	0/5	0/5
India	Goats	2/2	0/2	0/2	0/2	0/2	2/2	2/2	1/2	0/2	1/2	0/2	0/2	0/2	0/2	0/2	0/2
	Sheep	2/2	0/2	0/2	1/2	0/2	2/2	1/2	1/2	1/2	0/2	1/2	1/2	0/2	0/2	0/2	1/2

¹Sk, skin; Ton, tongue; Ph, pharynx; Tr, trachea; Oes, oesophagus; Ru, rumen; Ab, abomasum; Ret, reticulum; Turb, turbinate; Caec, caecum; LI, large intestine; Liv, Liver; Kid, kidney; GB, gall bladder.

Papules were present on the buccal mucosa of the lips and in some animals on the hard and soft palate, tongue, pharynx and turbinate mucosa (Table III). These papules were usually pale in colour, 2 to 3 mm in diameter and slightly raised above the surrounding tissue. Only in two animals were papules found on the tracheal or oesophageal mucosae. With only two exceptions the lungs of all animals that died had nodules throughout all lobes although their appearance and size varied. Some were pale others haemorrhagic and diameters were between 2 mm and 5 cm; in the more severely affected animals nodules coalesced. Papules were occasionally seen in the wall of the rumen but more commonly on the abomasal mucosa where they were frequently ulcerated affecting up to 90% of the surface. Three animals that died from the Yemen isolate and the sheep that died from the Sudan isolate had a small number of pale areas approximately 2 mm in diameter on the cortex of their kidneys. Five animals had similar pale areas on the surface of their livers which appeared grossly similar to the papules seen on other organs. It was not possible to distinguish between the isolates of sheep and goat pox according to the lesions they caused in the experimental animals.

Four groups of three crossbred sheep and two goats inoculated with the Nigeria sheep pox isolate were examined for viraemia and excretion of virus (Table IV). The viraemia was first seen on day 3 post-inoculation in sheep (Nos. 1-3) receiving the highest dose of virus, on days 4 and 5 in the next group and on day 5 or 7 in the remaining animals (Table III). The viraemia lasted between 10 and 12 days in those animals that recovered except sheep 7 which was apparently free of blood-borne virus two days prior to death; all other animals were viraemic up to and including the day of death. The goats were inoculated with the same dose of virus as the first group of sheep but no virus was recovered from their blood. The recovery of virus on conjunctival and nasal swabs coincided with the development of papules on the external mucous membranes.

Results of challenge experiments are shown in Table VI. Sheep recovered from Nigeria sheep pox failed to react to challenge with Yemen goat pox. Although a small papule developed at the inoculation site on the goats and a

TABLE IV
Viraemia and virus excretion in sheep following graded doses of Nigeria sheep pox virus

Sheep No.	Virus dose $\log_{10}\text{TCID}_{50}$	Viraemia		First day virus present in		
		First day	Last day	Conjunctiva	Nares	Day died
1	3.5	3	18	11	14	18
2		3	17	8	9	17
3		3	14	9	8	14
4	2.5	4	14		NE	14
5		5	16		NE	Survived
6		4	16		NE	16
7	1.5	5	17	11	11	20
8		7	16	14	11	Survived
9		5	18	11	9	18
10	0.5	5	18		NE	18
11		5	15		NE	15
12		7	17		NE	Survived

NE not examined.

TABLE V
Results of the dose-response analysis of the virus-antiserum mixtures

Antiserum		Nigeria	Virus Yemen	India
Nigeria	A	4.00	4.88	4.65
	B	-0.84	-1.21	-1.14
	Coefficient of correlation	-0.94	-0.98	-0.99
	Area	9.52	9.84	9.46
Yemen	A	4.09	5.04	4.50
	B	-0.91	-1.32	-1.20
	Coefficient of correlation	-0.91	-0.97	-0.99
	Area	9.20	9.62	8.44
India	A	4.67	5.24	3.92
	B	-1.28	-1.32	-0.82
	Coefficient of correlation	-0.97	-0.95	-0.97
	Area	8.48	10.38	9.37

Regression line equation $Y=A+BX$, where A is the \log_{10} decrease in virus titre produced by undiluted serum and B is the neutralisation slope (Westaway, 1965). The area is calculated from the formula: $\text{area} = -\frac{1}{2} A^2/B$.

transient fever of 40°C was recorded on day 3 the papules had disappeared by day 5. Sheep that had recovered from either the Sudan sheep or goat isolate developed a soft oedematous papule 1.5 cm in diameter and raised 0.25 cm within 24 h of inoculation with the Nigeria sheep pox virus. No fever was recorded and the local reaction quickly subsided and had completely disappeared by day 4. When challenged with the Indian goat pox isolate sheep recovered from the Sudan goat isolate failed to react. Sheep recovered from the Pakistan isolate developed a papule 0.2 cm in diameter two days after challenge with the India isolate. This quickly reduced in size and no fever was recorded. The day following challenge with Nigeria sheep pox virus sheep recovered from the India goat pox virus developed a small (0.2 cm diameter) painless, oedematous papule at the inoculation site. These increased in size to 0.4 cm in diameter on the second day but then started to reduce in size until by day 5 they could no longer be palpated. No secondary lesions were seen and the temperatures of both animals remained normal.

A reaction developed at the inoculation site within 24 h on all six Soay sheep that had been infected with the Kenya isolate and challenged with the Nigeria

TABLE VI
Results of cross challenge experiments with sheep and goat pox virus isolates

Challenge virus	Isolates used in primary infection					
	Nigeria	Yemen	Sudan	Pakistan	India	Kenya
Nigeria		R 2 Sh	R 2 Sh		R 2 Sh	R 6 Sh
Yemen	R 2 Sh 2 G	R 1 Sh				
India		R 2 Sh	R 1 Sh	R 2 Sh		R 2 G
Sudan		R 2 Sh				

R, resistant; Sh, sheep; G, goat.

isolate. By day 3 the reaction site had increased in size to between 1 and 1.5 cm diameter. This reaction had disappeared by day 7 except in two animals in which the papules persisted. None of the animals developed a fever or secondary papules. Similarly two goats challenged with the India isolate following inoculation of the Kenya isolate each developed an oedematous red papule 2.5 cm in diameter within 24 h at the inoculation site. By the following day these had become darker but had not increased in size. On day 4 the papules were harder and reduced to 1 cm in diameter; they had completely dispersed by day 7 without the formation of a scab. At no time did either animal develop a fever.

The dose-response line was linear for all the combinations of virus and antisera. An analysis of variance of the area defined by these lines could find no significant difference between them and a composite graph combining the results of the nine neutralisation tests produced a straight line with a coefficient of correlation of 0.99 (Table V).

DISCUSSION

The appearance of the clinical signs and post-mortem lesions produced by the different isolates in British sheep and goats is consistent with previous descriptions of sheep pox and goat pox (Al-Bana, 1978). The similar development and clinical appearance of the diseases produced by the different isolates suggests that they are very closely related even though the isolates originated at the opposite extremes of the endemic area. The assumption that strains of sheep pox and goat pox are geographically isolated is in the majority of the affected areas probably unjustified. The well-established trekking routes which cross Africa, the Middle and Far East provide an ideal method for the spread of the diseases. Thus strains of sheep pox virus and goat pox virus would tend to circulate particularly around those centres in Africa and the Middle East where trekking routes merge.

A second assumption – that except in Kenya sheep pox is distinct from goat pox – is also probably unjustified. The isolates all produced lesions in sheep and the five isolates that were inoculated into goats also produced lesions in the goats. The Yemen and Kenya isolates were both equally virulent for sheep and goats although the remaining isolates had a clear host preference. It is possible that strains of virus become locally adapted to a particular host either sheep or goats with the result that the ability to infect both becomes reduced the longer they remain in a single species. Viewed in this light the distinction between sheep pox virus and goat pox virus is hard to maintain.

Sheep pox and goat pox are contagious diseases although the usual portal of entry of the virus is not known. Bennett, Horgan and Mansur Ali Haseeb (1944) concluded that the most effective way of infecting an animal was by intradermal inoculation. Lumpy skin disease (Neethling virus), also a member of the capripox group of viruses, is transmitted allegedly by biting insects which inoculate the virus intradermally or subcutaneously. Davies (1976) described the spread of an isolate of Kenya sheep-and-goat pox from infected animals to animals in nearby boxes and thought that aerosol spread was possible as occurs with smallpox in humans and rabbitpox both caused by orthopox viruses. The almost invariable appearance of lung lesions in severely affected animals and the presence of virus in conjunctival and nasal secretions lends support to this hypothesis. The excretion of virus in the conjunctival and nasal secretions coincides with the appearance of papules on the external mucous membranes. Virus is also present

in the skin papules (Plowright *et al.*, 1959) and these lesions appear to be irritant as affected animals rubbed their heads and bodies on convenient corners in the isolation boxes or on other animals. Virus could in this way contaminate areas where the animals had been or be passed directly to susceptible animals. The disease did not, however, always immediately affect all susceptible animals present in the box. Four Soay sheep were kept in contact with five others inoculated with the Nigeria isolate. Two of these developed the disease soon after the death of the five inoculated sheep while a third developed the disease following the death of these two. The fourth contact animal did not contract the disease and reacted as a fully susceptible animal when challenged with the virus 71 days after the start of the experiment. Thus although the disease is certainly contagious transmission can be slow.

If as has been shown sheep pox virus is not distinct from goat pox virus and if the strains of virus are not geographically isolated sheep and goat pox as it appears in India must be similar in many respects to the disease as it appears in West Africa. A real possibility must therefore exist for the production of a vaccine which would be effective in protecting sheep and goats throughout the entire endemic area. Animals which have recovered from one isolate have in those challenge experiments carried out been immune to challenge with one of the other isolates. Goats which reacted only locally at the site of inoculation with the Nigeria isolate were immune to challenge with the lethal Yemen isolate. The Nigeria isolate could thus be considered a possible vaccine to protect goats against Yemen goat pox. The virulence of this isolate in sheep, however, would make it unsuitable. The Kenya sheep-and-goat isolate produced only a local reaction in sheep and goats. This produced sufficient immunity to protect the Soay sheep against the Nigeria isolate which otherwise caused 100% mortality. The two goats were similarly protected against challenge with the India isolate which caused 100% mortality in unprotected animals. The local reaction produced at the inoculation site by the challenge virus, within 48 h when it occurred, suggests a delayed type hypersensitivity reaction. This reaction usually soon dispersed although in two sheep it persisted.

The cross protection shown by the different isolates indicates a close antigenic relationship and is consistent with the serological results reported by Davies and Otema (1981) and the results of the cross neutralisation tests reported here which could not distinguish between Yemen, India and Nigeria isolates. It also supports the hypothesis that goat pox is not distinct from sheep pox. The three diseases – sheep pox, goat pox and sheep-and-goat pox – are caused by closely related strains of a capripox virus which probably become locally adapted to the host in which it finds itself whether sheep or goat. There can now be little justification for the continuance of the existing nomenclature and a single term for the malignant pox disease affecting sheep and goats should be used thereby avoiding the considerable confusion which surrounds the classification of the disease. It is proposed that it be called capripox irrespective of the species involved.

An isolate such as the one from Kenya, which protects sheep and goats against challenge with virulent virus while only causing a local reaction, would provide a vaccine suitable for use in goats and sheep. At present a vaccine based on isolate 0240 of Kenya sheep and goat pox is being used in Kenya and has been used on a small scale in India (Davies, pers. comm.). It is intended to further investigate attenuated strains of 0240 Kenya sheep-and-goat pox to test their suitability for use throughout the endemic area.

ACKNOWLEDGEMENTS

We wish to thank Drs J. McGrane, S. Kenyon, R. Patnaik, E. Siddiqui, M. Kurtkaya, G. Davies and P. Gibbs for post-mortem material or virus isolates from the Yemen Arab Republic, Sudan, India, Pakistan, Turkey, Kenya and Nigeria respectively. Thanks are also due to Mr J. Hammond for technical assistance. The work on sheep and goat pox at the Animal Virus Research Institute is financed by a grant from the ODA.

Accepted for publication June 1984

REFERENCES

- Al-Bana, A. S. (1978). PhD thesis, Cornell University.
 Bennett, S. C. J., Horgan, E. S. & Mansur Ali Haseeb (1944). *Journal of Comparative Pathology*, **54**, 131-160.
 Burdin, M. L. (1959). *Bulletin of Epizootic Diseases of Africa*, **7**, 27-36.
 Davies, F. G. (1976). *Journal of Hygiene, Cambridge*, **76**, 163-170.
 Davies, F. G. & Otema, C. (1981). *Research in Veterinary Science*, **31**, 253-255.
 Green, H. F. (1959). *Bulletin of Epizootic Diseases of Africa*, **7**, 63-74.
 Kärber, G. (1931). *Archiv der Pathologie und Pharmakologie*, **162**, 480-493.
 Plowright, W., Macleod, W. G. & Ferris, R. D. (1959). *Journal of Comparative Pathology*, **69**, 400-413.
 Sen, K. C. (1968). *Indian Journal of Medical Research*, **56**, 1153-1156.
 Sharma, S. N. & Dhanda, M. R. (1971). *Indian Journal of Animal Science*, **41**, 267-272.
 Westaway, E. G. (1965). *Virology*, **26**, 528-537.

RELATIONS CLINIQUES ET ANTIGENIQUES ENTRE LES ISOLATS DE
VIRUS DE VARIOLE OVINE ET CAPRINE

Résumé—Des isolats de clavelée et de variole caprine provenant de la Nigeria, de Soudan, du Kenya, de la République Arabe du Yemen, de la Turquie, du Pakistan et de l'Inde ont été inoculés à des races ovines et caprines britanniques. Bien qu'on ait observé une préférence d'hôte des isolats, l'ensemble de la pathologie clinique de la maladie produite par les divers isolats n'a pu être différencié. Les isolats du Yemen, de la Nigéria et d'Inde n'ont pu être différenciés à l'aide d'antisérums homologues et hétérologues dans les tests de neutralisation. Les animaux qui avaient guéri d'une infection causée par un isolat étaient résistants à l'épreuve d'infection causée par l'un quelconque des autres isolats et un seul vaccin contre la clavelée et la variole caprine est décrit.

La classification des varioles malignes du mouton et de la chèvre est discutée.

RELACION CLINICA Y ANTIGENICA ENTRE CAPAS DEL VIRUS DE LA VIRUELA
AISLADAS DE OVEJAS Y CABRAS

Resumen—Se inocularon cepas del virus de la viruela provenientes de ovejas y cabras de Nigéria, Sudan, Kenia, República Arabe de Yemen, Turquía, Pakistan e India, en ovejas y cabras británicas. Aunque las cepas mostraron preferencia por el huésped, la patología clínica de la enfermedad producida por ellas fue indistinguible. Las cepas de Yemen, Nigeria e India no se pudieron diferenciar, utilizando antisueros homólogos y heterólogos en pruebas de neutralización. Animales recuperados de la infección con una cepa, fueron resistentes a la descarga de cualquiera de las otras cepas aisladas. Se discute el uso de una solva vacuna contra la viruela de ovejas y cabras. También se discute la clasificación de las enfermedades malignas en ovejas y cabras, producidas por virus del grupo Pox.