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PESTE DES PETITS RUMINANTS (PPR) IN SHEEP AND GOATS

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HELD AT IITA, IBADAN, NIGERIA
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Editor's note

These proceedings are intended to draw attention to the main points made in the workshop presentations and discussions, and to clarify the conclusions and recommendations of the meeting.

Each presentation, other than those recorded directly from texts provided by speakers, is summarized along with discussions. Rapporteurs were appointed at the beginning of the workshop and are credited at the beginning of each topic.

I would like to acknowledge in particular the contributions by the four rapporteurs, Drs Adu, Asagba, Nwajei and Amogu towards the proceedings of the workshop and of our interpreting mesdames Tilly Gaillard and Elisabeth Benamar. With regard to all local arrangements for the meeting, special appreciation goes to the Director General of IITA, Mrs Bernadette and Mr David Sewell of IITA, Dr Miguel Velez, and Mrs Barbara Stone and Elizabeth Getahun of ILCA.

Abstract

These proceedings are a summary of the current state of knowledge on the epidemiology, symptomatology, pathology and diagnosis of *peste des petits ruminants* (PPR) in West Africa. The physico-chemical and biological characters of the PPR virus and the role of other agents responsible for the pathogenesis of PPR are also discussed. The conclusions give clear recommendations to governments and research institutes as to the further direction of policy and research on the problem.

Key words

/MEETING REPORT//WEST AFRICA//NIGERIA//PESTE DES PETITS RUMINANTS/
/SHEEP//GOATS/-/GEOGRAPHICAL DISTRIBUTION//ECONOMIC ASPECTS/
/EPIDEMIOLOGY//SYMPTOMS//DIAGNOSIS//ANIMAL PATHOLOGY//IMMUNOLOGY/
/VACCINES//DISEASE CONTROL//PATHOGENECITY//VIRUSES/

Preface

The International Workshop on *Peste des Petits Ruminants* (PPR) was held at the International Institute of Tropical Agriculture (IITA) at Ibadan in Nigeria from 24 to 26 September 1980. The workshop was jointly organized and sponsored by the National Veterinary Research Institute (NVRI) of Nigeria, the Federal Department of Agriculture (FDA; Nigeria) and the International Livestock Centre for Africa (ILCA).

The workshop focused on the problem of *peste des petits ruminants* (PPR) in West Africa. Different sessions of the workshop studied the history and economic importance of the disease, its symptomatology, pathology and diagnosis, the factors responsible for the pathogenesis of PPR, the characters of the PPR virus, the use of tissue culture rinderpest vaccine (TCRV) in the control of PPR and the planning of future research. The workshop produced conclusions as to the nature of the PPR virus and its economic importance and gave clear recommendations to governments and research institutes for the future direction of policy and research on the problem.

The workshop was attended by 46 scientists from West Africa, USA, UK and France representing national and international institutions.

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Welcome address

Dr A.G. Lamorde, Director, National Veterinary Research Institute,
Vom, Nigeria; Chairman of the workshop

It gives me great pleasure to welcome you all to this Workshop on *Peste des Petits Ruminants* (PPR) in Goats and Sheep. This meeting is unique in the sense that it is aimed at discussing and finding solutions to a common problem affecting goat and sheep production in West Africa.

Here in Nigeria sheep and goats (estimated at 8.5 and 25 million head respectively) contribute nearly as much meat for human consumption as cattle. In addition, these animals are valuable owing to the contribution of their skins not only to the foreign exchange earnings of the country, but also as a source of raw materials for our expanding local and modern tanneries. The Red Sokoto goat and the Brown Kano goat, for example, attract two of the highest ratings for skins on the world market.

Although Beaton working at Vom mentioned PPR in experimental goats purchased from local markets as early as 1930, it was French scientists who described the disease a decade later and called it *peste des petits ruminants* (PPR) because of its similarity to cattle rinderpest. About 50 to 80% of goats (especially young ones) that come up with this disease die, while those that survive fall victim to parasitic and bacterial complications. Pregnant goats abort during the course of the disease. In short, the direct economic loss caused by PPR in Nigeria alone is over one million naira every year.

The National Veterinary Research Institute at Vom has been conducting research into the problem of PPR in collaboration with other agencies. Tissue culture rinderpest vaccine (TCRV) was found to afford protection against PPR for a period of up to one

year. However, there seems to be a certain problem in its general use in some subhumid zones of the country. Since it is difficult to differentiate between healthy animals and those incubating the disease, vaccination of the latter can occasionally trigger off the disease. This poses a serious setback in planning a strategy for the control and eradication of PPR.

Since PPR is known to exist in virtually all the countries that come under the umbrella of the Economic Community of West Africa States (ECOWAS), it is desirable for all the scientists in these countries to cooperate in combatting the disease. This also holds true for other diseases such as contagious bovine pleuropneumonia (CBPP), rabies, streptothricosis etc. It is only through such cooperation that the member countries will be able to maintain the health of their livestock populations and prevent the re-introduction of the disease from the neighbouring countries.

It is therefore gratifying to have internationally reputed scientists in a gathering like this to deliberate on the burning issu of PPR and its prophylaxis. The experts here are expected to discuss and offer suggestions on the ways of controlling the disease as well as on the necessary experiments that need to be carried out to achieve the goal.

Once again I would like to welcome you all to this workshop and wish you successful deliberations and a happy stay in Ibadan.

Thank you.

Opening address

Mr Emmanuel Aguma, His Excellency the Minister of State, Federal Ministry of Agriculture, Lagos, Nigeria

It gives me great pleasure to welcome both our guests from various parts of the world as well as Nigerian participants who have assembled to take part in this workshop jointly sponsored by the National Veterinary Research Institute (NVRI), the International Livestock Centre for Africa (ILCA), and the Federal Department of Agriculture (FDA). My pleasure derives particularly from the fact that one of my responsibilities in the Federal Ministry of Agriculture is to look into projects and programmes for the development of livestock in the country. My interest in this workshop that is beginning today is therefore understandable.

I will not bore you with a long opening address because it is desirable that you settle down to work without delay in view of the limited time you have for the workshop. The other reason, however, is that there is nothing that a novice (which I am in the field of veterinary science) can give to impress such a qualified assembly of experts as this.

Sheep and goats contribute significantly to rural incomes and the protein consumption in this country. They are found in practically every rural household in most states of the Federation of Nigeria, and they provide about 40% of the quantity of meat consumed in the country. However, there are indications that in Nigeria as well as in other West African countries the population of sheep and goats has remained static over the years. This country experiences a mortality rate in small ruminants of between 15 and 20% annually, and yet there have been no systematic disease control

programmes to promote the development of sheep and goat production systems.

The present administration is determined to put a stop to the declining food (including livestock) production level per caput and to ensure the adequate nutrition of, and equitable distribution of income among, the rural population. To achieve these goals, the Federal Government is making concerted efforts to improve the production and productivity of cattle by developing grazing areas and to expand poultry production by improving disease control, feed availability and quality, and input supply. Very modest efforts are being made in the area of sheep and goat breeding. The Federal Ministry of Agriculture has set up a pilot sheep meat production centre at Katsina, which now has a population of over 2000 local sheep maintained on improved pastures. The purpose is to develop an elite breeding flock through selection in order to assist in the improvement of smallholder flocks and establish a production system that can be transferred to the private sector. It is anticipated that such breeding units will be established in other ecological zones of the country as well.

My attention has also been drawn to ILCA's work on small ruminants in the humid areas around Ikenne and Fashola. We are watching with been interest the progress of the programme, in the hope that adaptable improved packages would be developed to strengthen our national efforts.

In respect of goat production, much has yet to be done, particularly in the southern parts of the country where isolated flocks of disease-ridden goats roam the village streets, each animal fending for itself as best it can. The Nigerian Animal Production Research Institute (NAPRI) is currently carrying out investigations on the breeding of these animals. Hopefully, the result of the Institute's efforts will further help the production of goats for meat in the future.

It is needless to state that the central theme of this workshop is very relevant to the Nigerian situation. PPR was first identified in Nigeria in 1965 and since then a lot of research work has been done in this country and elsewhere. Despite this,

I am informed that effective control continues to elude our veterinarians and that the disease still constitutes the major constraint to developing sheep and goat production in the country. It is therefore gratifying that most of the eminent scientists from Europe, America and West Africa who have worked in this field are gathered today here to deliberate on the thorny issues of PPR diagnosis, epidemiology and control.

My ministry endorses this international approach to solving animal disease problems. Nigeria actively participated in the Joint Project 15 for the eradication of rinderpest and the Joint Project 28 for the eradication of contagious bovine pleuropneumonia. Nigeria anxiously looks forward to action for the eradication of *peste des petits ruminants* in this country.

It is my firm belief that this workshop will put forward proposals for a regional control strategy. It is also my hope that international organizations like the Office International des Epizooties (OIE) and the Food and Agriculture Organization of the United Nations (FAO) will join hands with the Inter-African Bureau for Animal Resources (IBAR) and various West African governments to implement programmes of action that may emerge from this workshop.

Ladies and Gentlemen, it is my pleasure to declare this workshop open and to wish you successful deliberations and also a pleasant stay in Ibadan.

Vote of thanks

Dr K.B. David-West, Director, Federal Ministry of Agriculture, Federal Livestock Department, Lagos, Nigeria

Dr David-West extended on behalf of all those attending the workshop a sincere vote of thanks to His Excellency the Minister, Mr Emmanuel Aguma, for his interest, encouragement and good wishes for the success of the meeting.

Welcome to IITA

Dr E.H. Hartmans, Director General, International Institute of Tropical Agriculture, Ibadan, Nigeria

Dr Hartmans extended a warm welcome on behalf of the staff of IITA to the distinguished visitors and participants attending the workshop. He expressed his personal interest in, and support for, the workshop and its deliberations on the problem of PPR in sheep and goats in West Africa and stressed the need for a continued and cooperative effort between research organisations concerned with crop production and those with animal production, in which the development of more effective and integrated systems of management are of increasing importance at both the traditional and the more intensive levels of farming.

Welcome letter

(Received from Dr E. Blajan, Director General, OIE, Paris, in response to the invitation from Dr David-West to attend the workshop-Ed.).

I had wished to be able to participate personally in this International Workshop on *Peste des Petits Ruminants*, to which I have been invited by Dr. K.B. David-West, Director of the Federal Livestock Department in Nigeria.

This workshop unhappily coincides with the regional conference of the OIE for Europe, being held at Budapest from 23-26 September. To my great regret I am therefore unable to attend your workshop, for which however I wish you the greatest success.

In choosing *peste des petits ruminants* as the subject for an international workshop the organizers have made an excellent choice, reflecting the world-wide concern for "rehabilitating" production from those species which are particularly well adapted to difficult zones, and which could thus help to solve the problem of increasing the supply of animal protein.

The considerable importance of *peste des petits ruminants* as a factor limiting goat production in West Africa has been recognized for several years by the OIE, which devoted a part of the proceedings of its 40th General Session in May 1972 to this subject.

As I recall, on this occasion the committee of the OIE recommended:

1. That surveys should be carried out in those African countries where the infection is not yet indicated in order to prove or disprove the existence of the disease in their territories; particular attention should be given to the possibility of infection of small ruminants by rinderpest strains.

2. That where the disease exists and it is a cause of mortality in small ruminant populations, especially on the West African coast, vaccination should be systematically undertaken with TCRV.

3. That research should be continued and extended on the nature of the PPR virus, the antigenic relations with other paramyxoviruses and the susceptibility of different African breeds of small ruminants to the wild strains of PPR virus.

I would like to take this opportunity to inform colleagues attending your international workshop that the 4th Conference of the Regional Commission of the OIE for Africa is being held in Rabat on 7-10 October. Among the topics which have been chosen for discussion is "the importance of small ruminants in the animal production programmes of African countries".

Finally I would like once again to convey my best wishes for the success of the International Workshop on *Peste des Petits Ruminants*, and to send my greetings to Dr A.G. Lamorde, Director of the National Veterinary Research Institute and the Chairman of the workshop, to Dr K.B. David-West, Director of the Federal Livestock Department, and to all the participants.

TOPIC I

HISTORY, EPIDEMIOLOGY AND ECONOMIC SIGNIFICANCE OF PPR IN WEST AFRICA AND NIGERIA IN PARTICULAR

Chairman: Dr G.R. Scott
Rapporteur: Dr F.D. Adu

History, epidemiology and economic significance of PPR in West Africa and Nigeria in particular

Speaker: Dr P. Bourdin, Institut National d'Elevage et de Recherche Vétérinaire, Dakar, Senegal

PPR is an endemic disease in West Africa, especially in the tropical areas. It was first described in 1942 by Gargadennec and Lalanne in the Ivory Coast and confirmed in Dahomey (Benin) by Cathou in 1944. Subsequently it was recognized as occurring in the small ruminant ("lagoon") breeds (dwarf breeds) in the southern regions of Nigeria, Togo and Benin, where small ruminants were highly susceptible to the disease.

In Senegal, a country with a Sahelian climate, PPR was observed and described in 1955 by Mornet and colleagues. Later it occurred in an enzootic state, with flare-ups of an epizootic nature in some years. The highly acute form, most frequently seen in goats, is rare in Sahelian sheep. In a goat population, the highest incidence occurs in young animals between 1 and 24 months of age with losses of the order of 50%. PPR has doubtless existed in Senegal for a considerable time past, but the term ovine pasteurellosis has covered up the presence of the more "primitive" aetiological agent of the PPR virus. The principal foci of infection are found in the northern and central parts of the country. Outbreaks occur during the rainy season, extending southwards and westwards due to the movement of transhumant flocks to the agricultural regions in the dry season. The chilling of temperatures at night favours the onset of the disease.

Clinically, PPR occurs in the acute form in goats. The virus is easily isolated in tissue culture during the hyperthermic phase. In Sahelian sheep it occurs in a benign form and is complicated by secondary conditions of pleuropneumonia due to the

multiplication of secondary invaders. Various bacteria can be isolated from pleural exudates or from the lung parenchyma of sheep, the majority of which belong to the genus *Pasteurella*. Mention should also be made of the large number of mycoplasma strains isolated by Dourtre in 1979 from the lung parenchyma of sick goats.

The economic importance of PPR is difficult to assess due to the lack of precise statistical records. One can only assert that, for the present, it is the most destructive viral disease amongst small ruminant flocks.

Workshop Editor's note: A further speaker who had been invited to participate in this topic, Dr P.C. Lefèvre of the Institut d'Elevage et de Médecine Vétérinaire Tropicale (IEMVT), was unavoidably unable to attend the workshop, but sent for distribution among the participants a most welcome and illustrated contribution on PPR entitled: "La peste des petits ruminants (synthèse et bibliographiques)." IEMVT, 1980. See appendix.

Highlights from ILCA's Humid Zone Programme

Speaker: Professor D.H. Hill, Head of Training and Senior Scientist, Special Studies, ILCA, Addis Ababa, Ethiopia

The humid zone of West Africa extends over an area of about 2 million km^2 and has a human population of over 100 million. Rainfall in the zone averages between 1250 and 1800 mm per annum, temperatures range from 27° to 32°C and relative humidity from 80 to 90%. It is a region of forests and derived savanna perennially inhabited by the riverine tsetse fly - which is vector of animal trypanosomiasis - limiting livestock production for the most part to what are generally called the trypanotolerant domestic animal species. The small "dwarf" breeds of goats and sheep (Djallonke) in the area surveyed by ILCA, including Ivory Coast, Togo, Ghana and Nigeria, are estimated at over 10 million in number of which 8 million are found in Nigeria. They are considered an integral part of the local economy; but with increasing urbanization of humid zone communities, the escalation of human population and the expansion of areas devoted to cash crops, the area available for subsistence production has been reduced and the threat of declining food production, particularly animal protein, has been intensified.

The aim of ILCA's current humid zone research programme is to assist national efforts to augment local supplies of animal protein and to increase the income of farmers in the region through improvements in existing production systems.

The following salient problems have been highlighted from ILCA's studies to date:

- The initial ILCA survey under village conditions in Nigeria came up with kidding and lambing rates of 150-160% and 141% respectively. In the same study survival rate to 6 months for kids

was 76% and for lambs 87%, weight gains to 6 months for kids averaged 27 g and for lambs 62 g per day. The productivity index for goats was 0.179 and for sheep 0.187 - 0.316 per kg live body weight of dam. Improvement by breeding and selection among local stock can be considered. Crossbreeding with imported stock, on the other hand, brings with it the associated problem of increased susceptibility of the crossbreds to local diseases.

- A major constraint to improved production of indigenous stock would appear to be the interaction of high disease incidence with low nutritional levels. ILCA village studies have indicated that supplementary feeding of sheep and goats with household scraps reduced mortality by 32% and 27% respectively. Among the diseases, *peste des petits ruminants* (PPR) is still considered a major constraint, notwithstanding the proven value of tissue culture rinderpest vaccine (TCRV) in the field, especially for the entrepreneur bringing in new and susceptible breeding or fattening stock. Of particular importance is also the high rate of infection (over 50% observed cases in rural flocks) by mange mite (*Sarcoptes scabiei*) causing severe deterioration in health and sometimes death.

- PPR continues to be one of the main - though certainly not the only - disease problems. Data from ILCA study areas indicate that of the 10-32% of annual losses in sheep and the 15-30% in goats, 15% occur before weaning. Mortality continues to be higher in the forest than in the savanna zone, with heavy losses from PPR in the 4- to 8-month age group. The need, therefore, is to approach PPR control from at least two major angles:

1. Control at the village level
2. Control at the more intensive, entrepreneur level

The farmers involved in the ILCA study believe that their stock could be doubled without undue strain on feed resources. On the other hand, labour would be a problem for the entrepreneurs.

Workshop Editor's note: During the ensuing discussion Dr Sansi emphasized the need for some form of "package deal" that could be used to cope with both husbandry and the health situation of flocks managed under traditional as well as under the more entrepreneurial systems.

Epidemiology of PPR in the humid forest and the derived savanna zones

Speaker: Dr B.A. Opasina, Humid Zone Programme, ILCA, Ibadan, Nigeria

Introduction

The International Livestock Centre for Africa (ILCA) carried out a preliminary small ruminant survey in 12 villages in the humid forest and in the derived savanna zones of Oyo State of Nigeria between October 1978 and March 1980. During the period, 355 dwarf goats and 156 dwarf sheep in the humid forest zone, and 299 dwarf goats and 102 dwarf sheep in the derived savanna zone were neck-tagged and monitored for productivity and disease incidence.

In addition, between April 1979 and March 1980, 88 dwarf goats and 78 dwarf sheep were purchased form local markets and individual owners and quarantined at the ILCA/Ikene experimental station in the humid forest zone. Similarly, 100 goats and 86 sheep were quarantined at ILCA's Fashola experimental station in the derived savanna zone during the same period.

Results

Under the village traditional system of management, a total of 4 outbreaks, 2 in the humid forest and the other 2 in the derived savanna zone, were observed. Goats were the only species affected. The first outbreak occurred in 4 villages within a radius of one kilometre in the humid forest zone in November 1978. Goats showing varying degrees of clinical signs of PPR, pyrexia, serous or mucopurulent oculonasal discharge, coughing, erosion of the gum and/or tongue, pneumonia and diarrhoea, were first observed in one of the villages during the second week of November. Within three weeks, the disease had spread to the other three neighbouring villages. The

spread from village to village might have been effected by human and animal contact. The sharp decline in the rainfall and relative humidity, reduced wind velocity and a rise in the ambient temperature, that is a sudden change of weather in November, might have contributed in no small measure to the outbreak.

The morbidity and mortality patterns during the first outbreak are shown in Table 1. Morbidity and mortality were 42.4% and 36.8% respectively. Goats in the 4- to 8-month age group showed the highest morbidity and mortality (76.2% and 62.8% respectively).

Table 1. *Age and sex distribution of PPR in dwarf goats at Eletu, Elesu, Butubutu and Bamishinde villages in the humid forest zone of Nigeria (November - December 1978 outbreak).*

	Age in months							
	0-4	4-8	8-12	12-24	24+	M	F	Grand total
Total population	24	21	23	27	49	27	117	144
Morbidity	12	16	11	7	15	20	41	61
Mortality	11	13	9	7	13	17	36	53
% Morbidity	50	76.2	47.8	25.9	30.6	74.1	35.0	42.4
% Mortality	45.8	62.8	39.1	25.9	26.5	62.9	30.8	36.8

The second outbreak occurred in New Eruwa village in the derived savanna zone late in January 1979, affecting a small group of 10 animals of various ages. Morbidity and mortality were 13.7% and 5.6% respectively. The highest morbidity (20%) and mortality (14%) occurred among the 4- to 8-month age group (Table 2). This is a comparatively large village extending for about one kilometre on its longest, and 3/4 km on its shortest side. Total rainfall from January to December 1979 for the area was 1520 mm. Sudden rainstorms (about 32 mm of precipitation) in late January and early February, which is in fact the dry season, and high ambient temperatures might have contributed to the outbreak.

Table 2. *Age and sex distribution of PPR in West African Dwarf goats at New Erwa in the derived savanna zone of Nigeria (January - March 1979 outbreak).*

	Age in months							
	0-4	4-8	8-12	12-24	24+	M	F	Grand total
Total population	89	50	25	34	87	121	164	285
Morbidity	4	10	8	12	5	8	31	39
Mortality	2	7	2	3	2	5	11	16
% Morbidity	4.5	20	32	35.5	5.7	6.7	18.9	13.7
% Mortality	2.2	14	8	8.8	2.3	4.1	6.7	5.6

The spread of the disease during the outbreak is illustrated in Fig. 1. A natural barrier created by 1/4 km of vegetation might have prevented its spread towards the other side of the village. Infection started from one household and spread geometrically to other households within the village.

In late November 1979, the third outbreak was observed in another village (Olukosi) in the derived savanna zone. Morbidity and mortality were 37.1% and 23.1% respectively. Goats in the 4- to 8-month age group suffered the highest morbidity and mortality (Table 3). Infection subsided during the first week of January 1980, that is about 5 weeks after the onset. Olukosi is a small village of 35 closely built households, which probably accounted for the relatively high morbidity and mortality.

Figure 1. *Illustration showing the spread of PPR at New Eruwa in the derived savanna of the south-west humid zone of Nigeria (January - March 1979).*

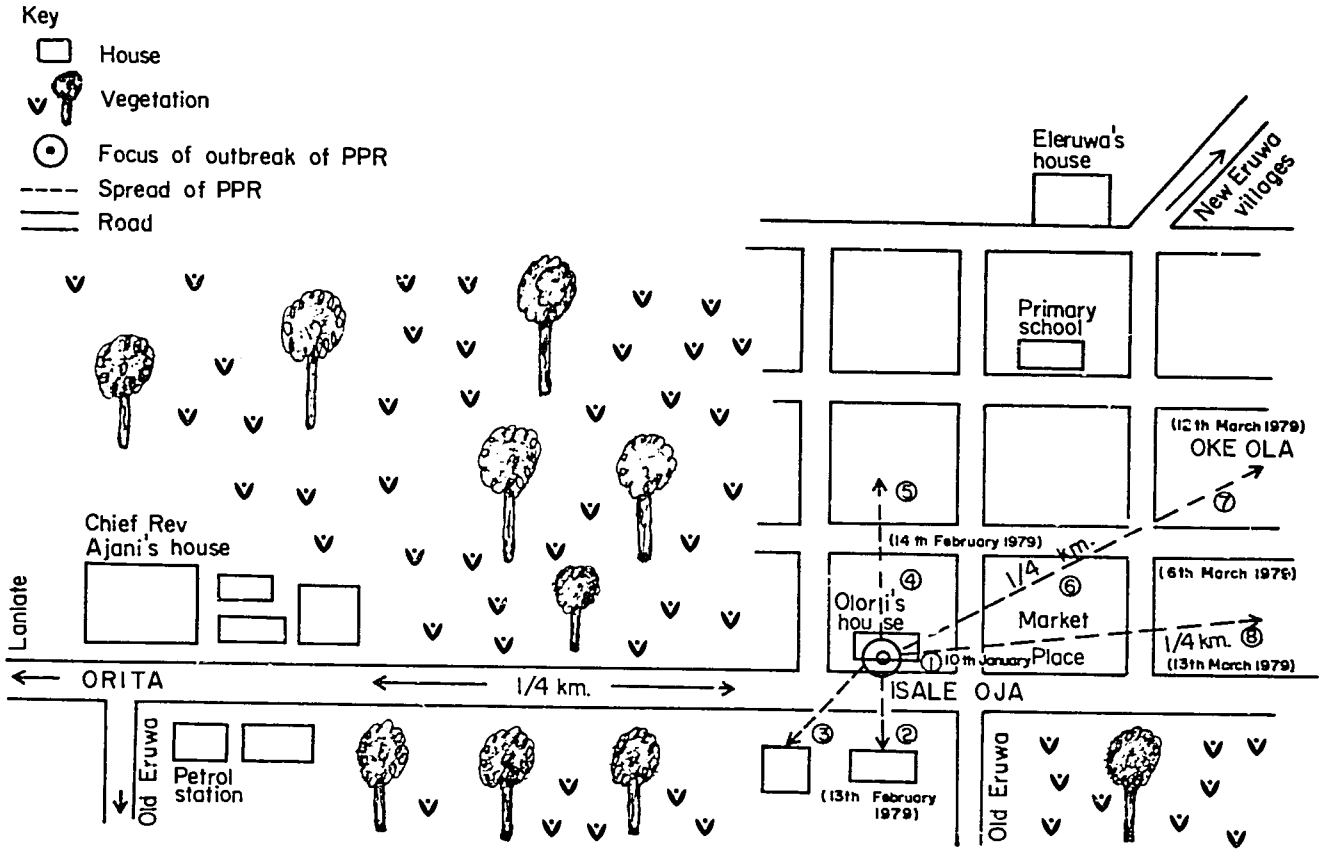

Table 3. *Age and sex distribution of PPR in West African Dwarf goats at Olukosi in the derived savanna zone of Nigeria (November 1979 - January 1980 outbreak).*

	Age in months							Grand total
	0-4	4-8	8-12	12-24	24+	M	F	
Total population	20	9	30	13	25	23	74	97
Morbidity	2	5	15	5	9	10	26	36
Mortality	1	5	10	2	5	8	15	23
% Morbidity	10	56.1	50.0	38.5	36.0	43.5	35.1	37.1
% Mortality	5	56.1	33.3	15.4	20.0	34.8	20.3	23.1

The fourth outbreak occurred in January 1980 in one of the villages in the humid forest zone. This is a very small village of 8 households, and there was also an outbreak there in November 1978. The morbidity and mortality were 50% and 20% respectively. Goats, again in the 4- to 8-month age group, suffered the highest morbidity and mortality. All the goats involved had been kept in the village at the time of the November 1978 outbreak. They were either newly bought-in animals or kids born 6 months after a previous outbreak. The source of the outbreak was considered to be a newly purchased doe.

Intercurrent diseases like sarcoptic mange infestation caused by *Sarcoptes scabiei* appeared to be one of the factors aggravating the severity of PPR among goats under village conditions. Table 4 shows the pattern of sarcoptic mange infestation in goats between October 1978 and September 1979, in the humid forest and the derived savanna zones. Morbidity was highest in goats 4 to 8 months old. The disease caused constant irritation and loss of condition in the animals.

Table 4. *Age and sex distribution of sarcoptic mange infestation in dwarf goats (Oct. 1978 to Sept. 1979).*

	Age in months							Grand total
	0-4	4-8	8-12	12-24	24+	M	F	
Derived savanna	35	70	30	19	25	34	135	169
Humid forest	10	37	10	10	9	12	64	76
Grand total	45	107	40	29	34	46	199	245

It will be observed that all the four PPR outbreaks occurred during the dry season (November-March): two outbreaks in the early dry season (November) and the other two in the latter third of the season (January).

PPR in small ruminants under quarantine

A number of dwarf goats and sheep under quarantine at ILCA experimental stations came down with PPR during the rainy season, i.e. between late April and September 1979. Clinical manifestations of the disease usually surfaced on the fourth day of the quarantine and subsided by the twelfth day. The PPR incidence in quarantine was probably due to the fact that one or more of the animals were incubating the virus before they were bought.

In the humid forest zone experimental station, total morbidity and mortality rates of 6.4% and 3.6% were observed in sheep, while in goats morbidity was 26.1% and mortality 11.5%. In the derived savanna zone experimental station, sheep morbidity was 31.4% and mortality 3.5%, while in goats a 17% morbidity and 9% mortality were observed.

Economics of PPR outbreaks in ILCA study areas

In the four village outbreaks from October 1978 to March 1980, a total of 94 goats died of PPR, representing a total liveweight loss of 808.3 kg (Table 5A). The price per kilogram liveweight in both zones was ₦ 2.00 (US$ 3.20). The total monetary loss was ₦ 1,616.60 (US$ 2,586.60). Of the 94 dead animals 23 were breeding does.

Forty clinical cases in goats and 32 cases in sheep were encountered under quarantine in the two experimental stations. The total goat and sheep mortalities were 18 and 6 respectively. The cost of treating the clinical cases against secondary bacterial infection was ₦ 57.00 (US$ 91.20). The total local market price for the lost goats and sheep was ₦ 980.00 (US$ 1,568.00). The total monetary loss was therefore ₦ 1,047.00 (US$ 1,687.20). This did not include the costs of attendants, housing and supplementary feeding during the quarantine period (Table 5B).

Table 5. *Economics of PPR outbreaks and incidence in the ILCA study areas (October 1978 to March 1980).*

A. Villages (survey)

	Mortality	Total liveweights of goats (kg)	Total monetary loss (₦ 2 per kg liveweight) (₦)*
New Eruwa		147.4	294.8
Olukosi	23	208.5	417.0
Eletu & Co.	53	425.4	850.8
Butubutu	2	27.0	54.0
Grand total	94	808.3	1,616.6

* ₦ 1 = US$ 1.60 (1981 exchange rate).

B. Stations (quarantine)

(i) Goats

	Total clinical cases	Mortality	Cost of treatment of clinical cases (₦)*	Market price of animals (before death) (₦)*	Total monetary loss (₦)*
Ikenne	23	9	18.4	360	378.4
Fashola	17	9	13.6	360	373.6
Total	40	18	32.0	720	752.0

(ii) Sheep

Ikenne	5	3	4.0	135	139
Fashola	27	3	21.0	125	156
Total	32	6	25.0	260	295

*₦ 1 = US$ 1.60 (1981 exchange rate).

Epidemiology of PPR in Nigeria

Speaker: Dr Z. Mathew, Epidemiology Unit, Federal Livestock Department, Kaduna, Nigeria

The disease, *peste des petits ruminants* (PPR), also referred to as the pneumoenteritis complex or *Kata* of sheep and goats, is a viral disease characterized by stomatitis, enteritis and pneumonia. PPR may be translated as "rinderpest of small ruminants". Whitney et al (1967) described *Kata* as a disease of goats which clinically resembled PPR. Rowland et al (1970 and 1971) found that *Kata* and PPR are similar both clinically and pathologically and that PPR shows cross-protection against rinderpest. Cattle and pigs do not suffer clinically from this disease, although they develop serum antibodies and withstand challenge with the virulent rinderpest virus. PPR is the fourth member of the *Morbillivirus* group, which includes the viruses of measles, distemper and rinderpest. PPR can be diagnosed by the agar gel diffusion test and by crossover counterimmuno-electrophoresis, as will be discussed later. Tissue culture rinderpest vaccine (TCRV) protects sheep and goats against PPR.

According to the analysis carried out by the Federal Livestock Department (FLD) of the reported outbreaks of PPR/*Kata* in Nigeria (for example, in the Oyo and Ondo States during 1977-78), 191 outbreaks of PPR occurred mainly in goats. Oyo State reported the highest incidence, a total of 104 outbreaks. Of the total number of 2701 animals affected, 115 died. This shows a mortality rate of only 4.2%. During April 1978 to March 1979, 114 outbreaks (87 from Oyo and 27 from Ondo State) were reported, with 59 deaths out of a total of 5373 goats involved in the outbreaks.

In 1979-80, 122 outbreaks were reported involving 1459 goats and resulting in 65 deaths. Again, Oyo State reported the

highest incidence (85 outbreaks), followed by Ondo State with 35 outbreaks. An analysis of the reported outbreaks in Nigeria over the last 3 years shows a high incidence (between 100 and 700 cases) of the disease although overall mortality was low, in contrast to the belief in the field that it is between 20 and 30%. The disease was reported throughout the year and, in the absence of a national control policy, appears to be endemic in such States as Oyo and Ondo. Imo State reported one outbreak and Kaduna State two outbreaks in 1977-78. Cross Rivers State reported two outbreaks for the 1979-1980 period. In some states vaccination of sheep and goats with TCRV to control PPR has already begun, and no adverse effects have so far been reported to the Department.

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Brief notes on history, epizootiology and the economic importance of PPR in Nigeria

Speaker: Dr O.A. Durojaiye, Department of Veterinary Microbiology and Parasitology, Faculty of Veterinary Medicine, University of Ibadan, Ibadan, Nigeria

Introduction

Definition. PPR is a contagious rinderpest-like disease of small ruminants characterized by fever, oculonasal discharges, diarrhoea, ulcerations of the buccal cavity and pneumonia.

Geographical distribution. The disease is endemic in most parts of West Africa. Outbreaks have been reported in Togo, Benin, Ivory Coast, Ghana, Nigeria and Senegal. It is likely that PPR occurs in other West African countries but the disease has not yet been officially reported. In most cases, the humid southern parts of the countries experience more severe outbreaks than the northern regions.

History

PPR was described in francophone West Africa as early as 1942 (Mornet et al, 1956).

Gilbert and Monnier (1962) first reported the isolation of the viral aetiological agent in Senegal.

In Nigeria, Beaton (1930) described a "rinderpest-like" condition in Nigerian goats.

Whitney et al (1967) described the disease clinically in affected goats in Nigeria.

Rowland et al (1970, 1971) described the disease and reported clinical, pathological and serological identity between the agents of PPR and *Kata*.

Nduaka and Ihemelandu (1973) described the disease in eastern Nigeria as the "stomatitis-pneumoenteritis complex".

Mann et al (1974) described the experimental transmission of the disease in Ibadan.

Taylor and Abegunde (1979) reported the isolation of the virus of PPR in northern Nigeria.

Epizootiology

Breeds of affected sheep and goats. Both the northern and southern breeds of sheep and goats are susceptible. However, field observations indicate that the West African dwarf (WAD) breed of goats found in the humid south are more susceptible than the northern breeds. It has been observed that when the northern breeds are transported down to the south, their degree of susceptibility compares well with the WAD. Both sheep and goats can be affected during outbreaks, but goats are far more susceptible than sheep.

Age of affected sheep and goats. Generally, younger sheep and goats are more susceptible than adult ones, although sheep and goats of all ages can be affected during an outbreak. Our studies in southern Nigeria indicate that sheep and goats as young as 3 - 4 weeks and as old as 5 years or above could be affected.

Sex of affected sheep and goats. Our observations of many field outbreaks do not suggest any difference in susceptibility between sexes.

Susceptibility of other species of animals. In Nigeria, the natural disease is known only in sheep and goats. Results of experimental transmission indicate that cattle and pigs do not suffer from the clinical disease. Hamdy and Dardini reported the susceptibility of deer to PPR. Attempts are being made to study the probable role of wildlife in the epizootiology of PPR in Nigeria.

Seasonal occurrence. PPR outbreaks occur throughout the year. However, most outbreaks occur during the rainy season, particularly in its earlier part.

Morbidity. Depending on many factors, morbidity in goats and sheep could vary between 50% and 100% but it could be lower. In one outbreak in Oyo State, sheep morbidity was 60% as compared to 100% in goats in the same village.

Mortality. Mortality is also variable. We have observed that in goats the mortality rate could vary between 10% and 100%. In outbreaks involving sheep that were studied, mortality was usually nil.

Virus excretion. The virus is excreted in body excretions such as oculonasal discharges and faeces. Virus excretion commences prior to the manifestation of the clinical signs.

Transmission. The virus is naturally transmitted through the mouth and the respiratory tract. (Contaminated feed and water can serve as sources of virus). The spread of the disease is by contact between infected sheep and goats excreting virus and susceptible sheep and goats. Experimentally, the virus has been transmitted nasally, orally, subcutaneously and intratracheally. The survival of the virus in tissue of dead goats is being investigated. On-going studies indicate that the virus can survive at -20°C for over 15 months.

Economic importance

It is difficult at present to evaluate the actual losses incurred at the national level due to PPR. This is mainly because no records are available on the actual national incidence of the disease. The information on Oyo and Ondo States could however be useful in assessing the role of PPR in these two states.

Despite the non-availability of comprehensive statistical data, our observations of field outbreaks in western Nigeria indicate

that PPR could be important economically. In some cases whole flocks have been wiped out. FLD records show that in the 1977/78 period 2701 goats were affected in 191 outbreaks resulting in the death of 115 animals. In Oyo State the price of an average goat (Sahelian breed) is about ₦ 30 while a WAD goat costs about ₦ 40 - 50. (US$ 48.00 and US$ 64.00-80.00 respectively - Ed.).

A case of PPR in eastern Africa

Speaker: Dr W.P. Taylor, Animal Virus Research Institute, Pirbright, Woking, Surrey, United Kingdom

Previous reports on PPR have limited the disease to a certain geographical range, so far confined exclusively to West Africa, but there is no reason why it should not spread to eastern Africa.

In November 1972 two isolates of a rinderpest-related virus (Sennar & Mielig) were obtained from goats in the heavily irrigated Nile region of the Sudan. These isolates were inoculated into experimental animals (all British breeds) at the Animal Virus Research Institute at Pirbright in the United Kingdom. Out of four goats inoculated with either Sennar or Mieling virus, two died. Thermal responses ranged between 40-41°C (104.2 - 106.0°F). In four sheep similarly inoculated, temperatures ranged between 39.5 - 40°C (103.2 - 104.2°F). All sheep showed typical PPR symptoms, but in a milder form than that seen in the goats. Symptoms in the goats were typical of PPR and included depression, nasal discharge, ulceration of the buccal cavity and diarrhoea. Of four cattle inoculated with either Sennar or Mielig virus, none showed any temperature reaction or clinical symptoms of PPR, but all four cattle survived when later challenged with the virulent Kabete 'O' strain of rinderpest serum and showed an SN_{50} antibody end-point 3.04 with PPR serum and 1.95 with rinderpest antiserum, while the SN_{50} antibody end-point with TCRV was 0 with PPR serum and 3.70 with the rinderpest antibody.

The conclusion that can be drawn from the above is that PPR exists not only in West Africa, but also in eastern Africa.

Discussion

Dr Bonniwell: What is the evidence that animals imported from outside the ILCA study areas will not go down with PPR, since you seem to attribute a lot of significance to the weather.

Dr Opasina: It would be difficult for me to say how animals from outside the areas which I have studied might behave. I have just enumerated what I noticed during my study, and from my observations the major outbreaks were during both the rainy season and during the dry harmattan period of the year. I have observed the same weather trend during these outbreaks.

Dr Obi: Outbreaks are usually at the beginning of the harmattan (January-February) and at the beginning of the rainy season, that is April to June. These outbreaks may have been predisposed by the sudden change in weather, which is very marked during these periods. Moreover, outbreaks appear to occur usually in 3- to 5-year cycles which suggests that a period of about 4 years is needed to build up a susceptible flock.

Dr Provost: A serological study was carried out on 2000 serum samples in Tchad and 37% had rinderpest antibody. In a country where rinderpest had been eradicated some time ago, it is surprising how such a high percentage of animals could still carry rinderpest antibody.

Clinical PPR is not known in Tchad but recently the IEMVT carried out a study in West Africa and found that PPR in Sahelian breeds started to be a problem, with more outbreaks being reported each year. It therefore seems that PPR is a disease that has been present, but not recognized as such.

PPR has now been reported from southern Arabia, also from India and Pakistan. But the question remains whether we are dealing with PPR or with rinderpest.

Dr Dardiri: We have all been talking about the epidemiology and symptoms of the disease, but nobody seems to have said anything about the diagnosis of the disease, and again nobody has really given a definition of a disease outbreak.

Dr Bourdin: I am going to restrict my comments to Senegal. We have seen that when the disease appears in the rainy season, it is carried forward into the dry season to the agricultural regions. There is a marked increase in the incidence of the disease in the cold season, then it subsides. We wonder how the virus is sustained in the animals? As late as 15 days after vaccination with

TCRV the virus was recovered by autopsy from an animal's lymph nodes, or, in a PPR-infected animal, from nasal swabs taken before the advent (day 5) of febrile symptoms. The virus is self-sustaining in the animal during the period that the clinical disease subsides. Diagnosis is by virus isolation. It is grown in the kidney cells of sheep, while rinderpest virus will not develop in those cells.

Again, as far as outbreaks are concerned, I am limiting myself to the Sahelian region. An outbreak of the disease may be defined in general as occurring in several animals at the same time and as being associated especially in those villages where the animals drink from the same water source.

Dr Sansi: Were the goats under quarantine vaccinated and also dewormed? Is PPR the same as *Kata?* Some animals showing clinical symptoms at the veterinary laboratories in Vom died and postmortem findings revealed that these animals were anaemic. Factors such as helminthiasis and bacterial infections may aggravate the course of PPR. I would also like to know from Dr Opasina what were his postmortem findings.

Dr Opasina: The process of acquiring animals for ILCA stations is a long one. Animals are purchased from the market in batches of 12 or more until there are enough in number to start routine quarantine procedures. During quarantine, animals are examined for worm burdens, ectoparasites and blood parasites. They are then dipped, after which, at day 3, they are dewormed and at day 4 treated with sulphonamide as a prophylactic measure for 5 days. All are vaccinated with TCRV 7 days after the last animal has been brought into quarantine, on the assumption that any animal incubating the disease at the time of purchase would show clinical symptoms by day 7. Animals are given hyperimmune serum (if available) at day 1. Postmortem findings usually included pneumonia, hepatisation of the lungs, especially in the cardiac lobes, and zebra markings in the caecum. Samples are usually sent for histopathological and virological examination to the University of Ibadan and to Vom.

Dr Ihemelandu: We have all been hearing of reports on PPR outbreaks in the west, while nothing has so far been said about reports from the eastern parts of Nigeria, where the disease is very much in evidence. Village goats are kept in small numbers and once the number is increased there will be disease outbreaks, usually occurring during the rainy season and the harmattan. Dr Matthew's projection is misleading and should not be presented

as what is generally happening in Nigeria. The disease has occurred in other parts of the country but it has been referred to by different names.

Dr Ezebuiro: I support the view of Dr Ihemelandu. In some villages where the owners "protect" their animals (i.e. evacuate the area in the dry season and return in the rainy season), there are no outbreaks. I would like to know from Dr Taylor whether experimental PPR infections are usually complicated by secondary infections.

Dr Ihemelandu: Despite the movement referred to, if it is a closed flock or herd, there will be no outbreaks, but if animals are added from outside, there may be an outbreak.

Dr Taylor: PPR was first noticed in Vom (when the speaker was working there-Ed.) in experimental goats purchased for other purposes. Mortality was between 70 - 80%. It is not yet known whether the different isolates of PPR vary in virulence.

Dr Durojaiye: Two experimental transmissions have been studied. There were no clinical symptoms of PPR from tissue culture derived virus, but when tissues were used, clinical symptoms were produced. There were also no typical postmortem patterns following experimental transmission with the virus.

Dr Ezebuiro: Although virus was not isolated in the Vom cases, clinical symptoms of PPR and a positive line with rinderpest antiserum were produced, indicating that it may have been PPR.

Dr Dardiri: Factors aggravating PPR symptoms include age of the animal, season etc. Experimental disease caused by PPR in American goats is characterized by oculonasal discharge, pneumonia and enteritis.

Dr Plowright: Question to Dr Durojaiye. Were any serological tests done, and was there any search for coccidia?

Dr Durojaiye: Yes.

Dr Ikede: It is only when experimental animals are purchased from local markets that they come down with PPR. Dr Ezebuiro may be wrong to say that the Vom outbreak was not actually PPR.

TOPIC II

SYMPTOMATOLOGY, PATHOLOGY AND DIAGNOSIS OF PPR

Chairman: Dr G.R. Scott
Rapporteur: Dr F.D. Adu

SYMPTOMATOLOGY

Speaker: Dr T.U. Obi, Department of Veterinary Medicine, University of Ibadan, Ibadan, Nigeria

The symptoms of PPR described here are based on our observations in Oyo State of Nigeria from 1976 to 1980. These include diarrhoea, ocular discharge, staring coat, foaming at the mouth and mouth sores. Farmers at this point usually suspect poisoning.

The first prodromal stage of the disease is characterized by a sudden onset of fever (102-106.5°F; 39-41°C). The fever may last 4 to 5 days. Twenty-four hours after the onset of fever, diarrhoea begins. At this point there is also congestion at the commissures of the mouth. Twenty-four hours later, the diarrhoea becomes profuse and bloody. The diarrhoea at this stage coincides with discrete necrotic spots in the oral mucosa. The gums around the incisors become hyperaemic. The necrotic spots later coalesce. There is mucopurulent nasal discharge and other symptoms of pneumonia, with increased respiratory rate (120 rpm), extended head, and difficult mouthbreathing. Oral lesions become more extensive and the lips oedematous. There is also conjunctivitis coupled with ocular discharge. Labial scabs develop in those animals that survive the acute stage of the disease. Atypical symptoms are also sometimes observed. These include abortions, nervous symptoms, vulvovaginitis, mastitis and erosion of the scrotal skin.

DIAGNOSIS

Speaker: Dr D.R. Nawathe, Virology Division, National Veterinary Research Institute, Vom, Nigeria

The disease has been given different names by different authors. This has been due to different diagnostic procedures discussed in the following paper (by Drs D.R. Nawathe, K.A. Majiyagbe and A. Abegunde of the National Veterinary Research Institute at Vom - Ed.):

Some observations on the diagnosis of *peste des petits ruminants* in Nigeria

Peste des petits ruminants (PPR) or *Kata* is an endemic disease in goats and sheep of considerable economic significance. The control and eradication of this disease are envisaged during the coming 5-year development plan. Diagnosis of this disease is therefore imperative to planned prophylaxis, and it will form the basis of assessment of the efficacy of measures taken.

In the past, due to the lack of adequate diagnostic facilities, coupled with the diversity of symptoms and the epidemiological picture, the disease was described by its clinical symptoms and considered different from PPR in francophone West Africa (Whitney et al, 1967; Johnson and Ritchie, 1968: Isoun and Mann, 1972; Nduaka and Ihemelandu, 1973). Rowland et al (1971) established histological and immunological relationships between *Kata* and PPR, while Hamdy et al (1976), Taylor and Abegunde (1979) and Abegunde et al (1980) isolated the PPR virus in different parts of Nigeria. It has been established that PPR is prevalent throughout the country and that differences in symptoms and the epidemiological picture are due to the degree of susceptibility of breeds, husbandry practices and environment.

The relative merits of the diagnostic procedures followed at the Institute is the subject of discussion of this paper.

Our observations are based on the materials submitted for diagnosis from outbreaks in and around Vom and a few others occurring in different parts of the country throughout 1975 to 1980.

Tests for the detection of PPR antigen

Agar gel precipitation test (AGPT)

The AGPT (White, 1959; Scott, 1967; Provost and Joubert, 1973) is useful for quick, field diagnosis. It is group specific and would require the support of other tests in the event of a return of rinderpest to the country.

We found one line of precipitation with rabbit hyperimmune serum, but two lines appear if immune serum from goats is used. Scrapings from mucous membranes of the respiratory and alimentary tracts and lymphoid tissues from field cases as well as experimentally inoculated goats gave lines of precipitation. Taylor and Abegunde (1979) suggested that AGPT can be used to study the epidemiological pattern of the disease. The results of the AGPT correspond to those of the immuno-electro-osmo-precipitation test (IEOPT), but not with the virus recovery rate.

For the hyperimmunization of rabbits additional doses of lapinized rinderpest virus are required, as suggested by Mohamed et al (1977). Procedures using tissue culture rinderpest vaccine (TCRV) mentioned by Bourdin and Doutre (1976) give false positive reactions, and this is confirmed by Bourdin (1977) as well.

Immuno-electro-osmo-precipitation test (IEOPT)

The IEOPT was developed using the technique described by Pan et al (1972) and Majiyagbe (1979). Like AGPT it is group specific, but it is 8 to 16 times more sensitive, with the result that it can be employed for the detection of antigen as well as antibody. It gives results in an hour or two, the test can be performed wherever there is electricity, and it does not require sterile conditions for manipulation. It is thus considered a most useful test for routine diagnosis, and for the study of epidemiology as well as monitoring the efficacy of control programmes. It will be later discussed in greater detail by Dr Majiyagbe.

Complement fixation test (CFT)

Like AGPT, CFT is also a group specific test and would need the support of other tests to differentiate PPR from rinderpest. Many times lymph node suspensions turn out to be anticomplementary, which complicates the interpretation of results. Here again, fresh specimens are preferred to those preserved for longer periods.

Immunofluorescent test (IFT)

A direct IFT with goat conjugated immune serum was tried without success. Even the negative lymph node smears showed fluorescence of some degree. Probably the conjugate supplied had gone bad. Indirect IFT is being developed instead.

Animal inoculation

Since the disease is endemic, the susceptibility of animals and superimposition of infection from outside have got to be ascertained. Provost (1975) failed to produce disease in Sahelian goats with a virulent PPR virus from the Republic of Benin. Virus isolated in cell cultures infrequently produces typical symptoms (Benazett, 1973). Moreover, animal inoculation for diagnosis of PPR is expensive and cumbersome. Coccidiosis and haemonchosis are so common on the plateau that many experimental animals die from these as well as from non-specific bacterial pneumonia. It could be difficult to explain the results of animal inoculation without supporting serology tests (Whitney et al, 1967; Mann and Isoun, 1974).

Allergic test

A few goats that recovered from PPR infection were inoculated intradermally with heat inactivated TCRV. A swelling appeared on the fourth day of the size of a gooseberry and disappeared by the eighth day. Histological examination of the biopsy specimen revealed accumulation of macrophages and lymphocytes, as one would expect in an allergic reaction indicative of cell-mediated immunity. After standardization the test may turn out to be a useful tool to detect immune animals in a flock.

Virus isolation and identification

Fresh samples are preferred to preserved ones. The virus recovery rate from samples positive to AGPT is much lower. Some preserved specimens continued to give lines of precipitation, but failed to yield virus even in highly susceptible embryonic goat and lamb kidney cells. The use of Vero cells from the American Type Culture Collection for primary isolation of the virus was not successful, however virus could be adapted to Vero cells subsequently. This point was already mentioned by Taylor and Abegunde (1979).

Cross-neutralization tests in cell cultures for virus identification and cross-protection tests in animals to prove Koch's postulates and establish relationship with prototype virus are the best procedures, but too time consuming and expensive to be employed for routine diagnosis.

Tests for detection of PPR antibody

Indirect agar gel precipitation test

The indirect agar gel precipitation test described by Yedloutschnig and Stone (1974) was tried without success.

Immuno-electro-osmo-precipitation test (IEOPT)

The IEOPT and the serum neutralization test (SNT) agree up to 80%, but unlike SNT the IEOPT is group specific. Paired serum samples collected at acute and convalescent stages are required to diagnose the outbreak as PPR. IEOPT can also be used for titration of serum antibody.

Serum neutralization test (SNT)

The SNT in cell cultures is by far the most specific test for PPR and shows little or no cross-reaction with rinderpest. It is the test of choice to measure the serum antibody. Until recently, SNT for rinderpest was being used for PPR (Bourdin, 1977) but so far it has not been possible to use SNT in microtitre systems.

Measles hemagglutination inhibition test

This test was not done but from the results reported by Rowland et al (1977), it appears that it is much less sensitive than SNT.

Complement fixation test (CFT)

The CFT was used by Nduaka and Ihemelandu (1975) to assess the immune response in goats vaccinated with chloroform-treated infected tissues. The test is group specific and because of the routine use of SNT, it is not employed in this laboratory for this purpose.

Enzyme-linked immuno-sorbent assay (ELISA)

The ELISA test has so far not been tried for PPR in this laboratory.

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Diagnosis of PPR infection using the immuno-electro-osmophoresis (IEOP) technique

by

Dr K.A. Majiyagbe, Dr D.R. Nawathe and Dr A. Abegunde,
National Veterinary Research Institute, Vom

Speaker: Dr K.A. Majiyagbe, National Veterinary Research Institute, Vom, Nigeria

The significance of PPR as a major economic disease of sheep and goats in West Africa has been established over the last 5 years (Hamdy et al, 1976) and whatever control measure is designed, the importance of an accurate diagnosis of infection in new as well as in already known endemic areas to the success of such a control measure cannot be taken for granted. A previous speaker (Nawathe et al, 1980) presented a detailed review of currently used diagnostic protocols, as well as of others still under development. This presentation deals with the application of the IEOP technique in PPR diagnosis and the results we have obtained.

IEOP technique - protocol test antigens

Test antigens: The diagnostic material was extracted from the lymph nodes (preferably mesenteric) of suspected animals. The extract was obtained either by squeezing fluid out of the lymph nodes, or in the same way as described by Scott and Brown (1961) for rinderpest fluid antigen. Similarly, antigens were obtained from tissue cultures using appropriately adapted virus strains (Vom Nig 75/81 or Eruwa '79). Control antigens were used from normal lymph nodes or from uninfected tissue culture cells.

Test sera. These consisted of materials obtained from the field either during suspected outbreaks or from abattoir animals. In addition, known positive and negative PPR sera from the Institute's

flocks were also included in the test. As a further control, hyperimmune and convalescent bovine sera were also used.

Gels and buffers: The gels used were ion agar no. 2, purified agar and agarose (Sigma Medium EEO type II). The tank buffer was a barbital sodium acetate - HCl, pH 8.6, 0.1 M(4) and 2 g sodium azide per litre. The gels were made into 1% (w/v) solutions using as a solvent a 1:4 dilution of the tank buffer.

Immuno-electro-osmophoresis: The protocol used was essentially as previously described (Pan et al, 1972; Majiyagbe, 1979), with some minor modifications. A standard horizontal commercial electrophoresis tank with power supply capable of delivering constant voltage was used. Pre-cleaned and appropriately labelled 76x26 mm microscope slides were pre-coated with agar film (labelling of glass is done with a diamond-tipped glass marker usually on the right-hand side of the slide before pre-coating).

Eight ml of gel were layered on the pre-coated slide supported on a levelling table capable of holding as many as 15 slides. Seven parallel rows of wells were cut along the major axis of the slide. The paired wells along the major axis were 5 mm apart. Each well was 3 mm in diameter while the wells in each pair were 5 mm apart. The appropriate test or hyperimmune sera were added to the wells near the anode side while the antigen was near the cathode side, which corresponded to the slide marking.

Each set of tests included appropriate negative and positive controls. The filled slides were placed on a support inside the tank containing the cooled buffer. The gels were connected with the buffer by means of a strip of filter paper or a white drill cotton piece. The run was performed at room temperature for 60 minutes at a constant voltage of 10 volts per 1 cm of gel (current reading is usually stable at 10 milliamps when a single slide is being run, or at 35 to 40 milliamps when the full complement of five slides is being run). The slides were examined for precipitation lines and results recorded immediately after the run. Then they were placed in

trays or petri-dishes containing phosphate buffered saline (PBS) (Ca^{2+}, Mg^{2+} free) pH 7.6 for about 2 hours, read again and the results compared with the previous ones.

If staining is required, the slides are left overnight in PBS, then in distilled water for 3 hours before being dried. Drying is achieved by covering the slide with a lint-free filter paper soaked with distilled water, placing it on a rack or in a petri-dish and allowing it to dry overnight at room temperature.

The thin sheet of dried gel was stained for 5 minutes in 0.5% napthalene black 12B and subsequently cleared in a 7.5% glacial acetic acid - 50% alcohol destainer. The slides were allowed to dry at room temperature and stored as permanent records.

PPR serum neutralization test (SNT): Some of the serum samples found positive for PPR antibody by the IEOP test were checked by SNT to establish correlation between IEOP and SNT.

Results

A number of variables were examined to determine the optimum test condition, namely gel type and composition, run time, and effect of PBS or 2% NaCl on the stability of precipitation lines and stains.

Sigma agarose type II gave the best result in terms of clarity and sharpness of the precipitin lines formed; with the other two agar media tried, lines were fuzzy and the area between the wells cloudy. With the agarose it was possible to use a 0.9% agarose solution and still have a gel layer of sufficient strength for the test.

With a distance of 5 mm between the paired wells, and at 10 volts per 1 cm of gel, a precipitin arc was visible after 30 minutes and after even 15 minutes the distance was reduced to 3 mm. With some sera cloudiness developed around the precipitin lines, which sometimes makes very weak reactions difficult to see, or precipitin arcs were formed very close to the serum well when lymph node antigen was used. To remove these non-specific reactions, it was decided to soak the slide in either 1% or 2% NaCl or PBS.

The 1% NaCl or 1% PBS were found to be suitable media for this purpose. Some very weak lines were lost in 2% NaCl with the goat and sheep serum test system, and PBS was selected in preference to 1% NaCl because it is easier to obtain in our laboratory.

Two types of stains - 0.5% naphthalene black 12B or Coomassee blue - were found suitable. We have not used Coomassee blue regularly because it is scarce but whenever it was available, it was the stain of choice because it is more sensitive and gives clearer background.

Since field samples came in irregularly, most of the test evaluations were carried out with lymph node samples derived from our experimental animals and the results correlated well with agar gel tests or virus isolation, when they were attempted from known samples. For the detection of antibody in serum samples, standardized antigen preparations from PPR-virus infected Vero cells or goat/lamb kidney cultures were used. The antigen filters in a checkerboard titration were 1:16 and 1:32 respectively (Table 1).

Table 1. *Result of checkerboard titration of PPRV tissue culture antigen.*

Serum dilution	Vero-PPRV antigen					CRC-PPRV antigen					
	1:2	1:4	1:8	1:16	1:32	1:2	1:4	1:8	1:16	1:32	1:64
Neat	+	+	±	+	-	+	+	+	+	+	-
1:2	+	+	+	+	-	+	+	+	+	+	-
1:4	+	+	+	+	-	+	+	+	+	+	-
1:8	±	±	+	+	-	+	+	+	+	+	-
1:16	-	-	±	-	-	±	±	+	+	+	±

+ = positive (precipitin line formed)
- = negative (no precipitin line formed)
± = doubtful reaction

Serum samples taken from our own experimental flock during the Ibadan and Makurdi oubreaks were screened by the IEOP test, and the results are shown in Table 2.

Table 2. *Results of serum samples screened by IEOP.*

Serum source	Total no.	No. positive	% positive
Vom	35	25	71.4
ILCA, Ibadan	14	8	57.1
Eruwa	45	17	37.7
Makurdi	35	24	68.6

Table 3 shows correlation between the results of IEOP and SNT for PPRV antibody detection and indicates that the IEOP is a far more simple and reliable test than the SNT. While IEOP detects minute traces of antibodies, the SNT will not.

Table 3. *Correlation between IEOP and SNT for PPR antibody in serum samples.*

No. serum tested	Number of correlations	
	IEOP	SNT[c/]
10	10[a/]	10
12	12[b/]	10
20	20	15

a/ 1:4 dilution, Vero standard PPR antigen used.

b/ 1:8 dilution, FGK standard PPR antigen used (FGK = foetal goat kidney cell culture).

c/ Done with foetal lamb kidney culture using 10^{-2} virus dilution.

Conclusion

Our results show that the IEOP test is a useful and reliable tool for the diagnosis of PPR. It is highly adaptable for use in epidemiologic work as well as in monitoring the efficacy of control programmes, should these be started. This test is not specific for PPR alone as it will detect antigen and antibody due to rinderpest virus. However, based on clinical and pathological findings, a diagnosis of PPR can be made in conjunction with the result of the IEOP test without recourse to a cross-neutralization test *in vitro*. Whenever a PPR specific diagnostic reagent is developed, the IEOP test is the test of choice.

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PATHOLOGY

Speaker: Professor B.O. Ikede, Department of Pathology, Faculty of Veterinary Medicine, University of Ibadan, Ibadan, Nigeria

Editor's note: Professor Ikede supported his presentation on the histopathology of natural cases of PPR with slides to explain certain characteristic lesions and changes occurring during the disease. The slides demonstrated the following:

Oral cavity. Serious loss of the epithelia and numerous giant cells can be seen here, as well as ulceration in the oral mucosa.

Intestine. This slide shows the commonly found zebra markings in the gut lining. Degenerative changes can also be seen. There are inclusion bodies in the cytoplasm of the lymphatic cells, as well as necrotic and inflammatory cells in the glands.

Lungs. A considerable number of giant cells in the alveoli of the lungs, also necrosis due to *Pasteurella* spp. The giant cells contain numerous intercellular inclusion bodies.

Nasal passage. Extensive proliferation and ulceration of nasal cells can be clearly seen, with intracytoplasmic inclusion bodies in the cells. There is hyperplastic and interstitial pneumonia and congestion of the nasal passage. There is also epithelization which makes the whole resemble fat cells. Small giant cells can also be seen in the nasal passage.

Liver. Here a few lesions, including hyperaemia and necrosis can be seen.

The pathology of PPR (caprine pneumoenteritis)

Speaker: Dr M. Uzoukwu, University of Nigeria, Nsukka, Nigeria

Introduction

Caprine pneumoenteritis (PPR, *Kata)* has been described in all the regions of this country. Although the clinicopathologic manifestations seem to vary from region to region (Durtnell and Eid, 1973; Rowland et al, 1969; Uzoukwu, 1977), the latest evidence from cross-neutralization, cross-protection and gel diffusion tests (Nawathe, personal communication) proves conclusively that the aetiologic virus is the same for all the described syndromes. Since little or no difference was observed between the natural and the experimental disease, this presentation will discuss the pathological basis for its various manifestations.

Pathology

As the name "pneumoenteritis" implies, the systems most severely affected are the alimentary and the pulmonary systems. Severe damage of either of the two systems can result in the death of the animal. At the risk of saying the obvious one could suggest that death is a consequence of the combined effects of the pathology in the two systems, although it is my opinion that diarrhoea is the critical factor.

Clinically, diarrhoea is a marked symptom. Gross post-mortem evidence of this is the usually marked enteritis which is often haemorrhagic. Histologically, the villi are shortened, their tips are necrotic or desquamated and the capillaries are dilated and engorged. The submucosa is usually oedematous. These changes would appear to be the direct and acute effect of the PPR virus on the

intestine. It is most likely that the submucosal oedema is a sign of the severe inflammatory response in the tissue, manifested by dilation of the capillaries rather than a lymphoedema due to occlusive lymphangitis which can be seen in rinderpest. Thus it is obvious that the virus induces a severe and persistent irritation of the intestine resulting in the marked diarrhoea observed clinically.

The cases observed by us (Uzoukwu, 1977) have been associated with hepatic necrosis, both the centrilobular and the midzonal. At the periphery of the lesion, hyperaemia and mononuclear cell infiltration are prominent. Since the midzonal hepatic necrosis is said to be unusual in domestic animals, this lesions may in fact be an important finding which, we suggest, is a direct effect of the infective virus on the hepatocytes. The widespread damage to the liver tissue could obviously interfere with hepatic functions and thus contribute to the death of the patient.

Toxic degeneration of the proximal tubular epithelium of the kidneys and albuminous cast formation are frequently associated with infectious diseases. The damage to the kidneys may have a complex aetiology, which combines whatever direct effect the virus may have on the renal parenchyma, the dehydration caused by the profuse diarrhoea, and the hypoxia resulting from the pulmonary lesions.

Our experience agrees with findings reported by Provost (1973), especially in the absence of inclusion bodies. Of similar significance is the notable absence of vacuolar degeneration, vesiculation and pustulation in the occasionally observed labio-gingivitis. These, in our opinion, clearly differentiate the condition of contagious echthyma ("Orf").

Conclusion

Neither the clinical nor the pathological manifestations of this disease are pathognomonic. However, for the host involved - goat or sheep - the simultaneous occurrence of diarrhoea and pneumonia is highly suggestive. Gross and histopathological lesions provide partial confirmation of infection. In less acute cases it may be

difficult to differentiate between the lesions produced in various parenchymatous organs by the virus and the severe dehydration which is caused by it. We believe that further work needs to be done to determine the role of secondary bacteria in the production of the clinical syndrome and pathological lesions, as well as on the pathogenesis of the lesions.

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Discussion

Dr Taylor: I congratulate Dr Majiyagbe for a tremendous breakthrough. I would like to see his method used in Britain.

Dr Hamdy: I also congratulate Dr Majiyagbe. Using his method, we may obtain a monospecific antigen that will be used in the final diagnosis of PPR and the rinderpest virus.

Dr Wosu: Has anybody done the RBC spectrum of the HA test for PPR?

Dr Durojaiye: There was no agglutination property from sheep and goats but others will be tested later.

Dr Provost: I did not get agglutination with PPR but one may get a very weak agglutination with the rinderpest virus using monkey red cells (measles) in the indirect method. This suggests that haemagglutinin does exist in RV. (Note: Johnson and Richie published a paper in 1968 on HA antigen and on PPRV and found that they were negative).

Dr Ihemelandu: Have the francophone participants found any symptoms of PPR different from those described here so far?

Dr Bourdin: In the Sahelian countries, symptoms of PPR were observed mainly in goats. The animals usually suffer from primary infection of the upper respiratory tract, rhinotracheitis and nasal discharges. They also have fever. Other symptoms already described here have also been noticed. There is a high rate of abortion in the female, which is very important.

Dr Evbuoma: What tissue should be used for the agar gel precipitation test?

Drs Nawathe and Durojaiye: The mesenteric lymph nodes, but other lymph nodes from the lungs and bronchus can be used as well.

Dr Mathew: I suggest that RBC at different pH levels be used for the HA test. The heamo-adsorption test should also be tried.

Dr Bonniwell: We find large intestine lymph nodes more positive than the mesenteric lymph nodes.

Dr Okoye: 99% of goat diseases with diarrhoea are considered to be PPR. There is always an accompanying corneal opacity.

Dr Sansi: In relation to histopathology and pathology, differential pathology should be noted here. I disagree with Professor Ikede regarding the formation of inclusion bodies. Giant cells are non-specific. Similar reactions can be seen in viral diarrhoea. For diagnosis, most of the histopathological changes seen are due to complications. I am of the opinion that before PPR is confirmed, virus isolation should be done. I congratulate Dr Majiyagbe on his work.

Dr Provost: Mine is a semantic question. One always has the right to say that *Kata* is the same as PPR. However, it has been shown that a goat that recovers from *Kata* always reacts to the PPR virulent virus. I believe that in *Kata* there are a lot of things that are hidden, although it includes PPR. This hidden "something" can be parainfluenza-3 which, when it attacks goats, produces similar symptoms, as possibly also will mycoplasma and other organisms. *Kata* should therefore perhaps not be used as a synomym for PPR?

Dr Adu: Dr Provost's comment is a very interesting one. If *Kata* is not synonymous with PPR, where then do we draw the line between PPR and *Kata?*

Dr Bourdin: I will answer that question by telling an anecdote about PPR. We isolated PPR virus in Porto Novo, and just about at the same time Dr Rowland isolated *Kata* virus in Ibadan. Dr Rowland sent his *Kata* sample to me for identification and comparison. I found the two isolates to be exactly the same thing in all respects. I then wrote back to Dr Rowland that "your virus is the same as my virus". Therefore, *Kata* is the same as PPR.

Dr Wosu: Has any biochemical work been done on PPR?

Dr Taylor: Some work was done in Vom with 3 typically purified PPR isolates. There may as well be a "hush-hush" on what we call *Kata.* Bacterial and other complications contribute, but PPR is a highly pathogenic virus on its own.

Dr Abegunde: All isolates tend towards PPR despite the fact that a lot of other materials complicate the picture. The major issue in PPR is whether other complicating materials are significant or not.

Dr Dardiri: My question is directed at Dr Majiyagbe. When is the best time to obtain materials for the IEOP test?

Dr Majiyagbe: The best time to get samples is when there is maximum pyrexia, but if we have a dead animal, the lymph nodes from the mesentery can be used. If it is virus isolation, one can get the virus from tissue culture and use the supernatant fluid. The convalescent serum can be used as antiserum.

TOPIC III

ROLE OF OTHER AGENTS AND FACTORS RESPONSIBLE FOR THE PATHOGENESIS OF PPR

Chairman: Professor W. Plowright
Rapporteur: Dr B.N.C. Nwajei

Role of other agents and factors responsible for the pathogenesis of PPR

Speaker: Professor M.O. Ojo, Department of Microbiology, Faculty of Veterinary Medicine, University of Ibadan, Ibadan, Nigeria

According to reports from Ondo State in Nigeria, cases of *Kata* and pneumonia occur within 48 hours of the onset of the rains. In most of the samples from field cases with a history of diarrhoea which were submitted for laboratory diagnosis, *Escherichia coli*, *Staphylococcus pyogenes*, *Salmonella* spp., and *Pasteurella haemolytica* were isolated. It was also observed that about 80% of the cases where *E. coli* was isolated showed the presence of enterotoxin.

Attention has been drawn to the similarity of the role of *Staphylococcus pyogenes* in both human measles and PPR. Mycoplasmata have been found to complicate the pneumonia picture in goats, in which *Mycoplasma mycoides* var. *mycoides* plays a significant role. Much work needs to be done on the role of *Pasteurella haemolytica*, especially since we do know that there are several serotypes. It is also known that these serotypes vary from flock to flock and from one season to another. More work is required to differentiate between contagious caprine pleuropneumonia (CCP) and pneumonias associated with these other organisms. Coccidiosis has also to be investigated with regard to its involvement in PPR outbreaks.

PPR and other intestinal factors in goats at Vom, Nigeria

Speaker: Dr O. Onoviran, National Veterinary Research Institute, Vom, Nigeria

We at the laboratories at Vom have attempted to determine the pathogenicity of some of the mycoplasmas isolated from some contagious caprine pleuropneumonia (CCP) cases. A group of supposedly healthy goats were bought from the local market for this purpose. All died about 5 days later showing clinical signs of PPR and postmortem examinations revealed typical gross lesions of PPR. In addition *Pasteurella haemolytica*, *Pasteurella* spp., *Mycoplasma* spp., *Staphylococcus aureus*, *Corynebacterium pyogenes*, *Klebsiella* spp., *Proteus* spp. and *E. coli* were isolated from the small and large intestines.

The next batch of goats brought in were given prior treatment with antibiotics and anthelmintics. No clinical manifestations of the disease were later evident although PPR antibody was demonstrated in their sera by the agar gel precipitation method. As a routine procedure for the subsequent purchase of goats temperatures were taken prior to purchase and those animals with temperatures above 38.5°C (101°F) were not bought. It is therefore considered that if other factors such as parasites and bacteria are eliminated, mortality, as reportedly due to PPR, would be drastically reduced in any field outbreak of the disease.

Discussion

Dr Taylor: At the time I was working with goats at Vom there were many deaths of unspecific aetiology that occurred during attempts to carry out an experiment. The animals showed pneumonia although no bacteriological examinations were carried out.

Dr Oluokun: What was the reason for the high incidence of PPR and pneumonia during the months of March and April, as reported in Ondo State by Professor Ojo?

Professor Ojo: Rains are usually heavy and start early in Ondo State because it is situated well inside the heavier rainfall region of the country.

Dr Obi: I think that environment plays a role in PPR infections although at the moment this is difficult to prove experimentally. Adenovirus could also be involved in PPR infection.

Dr Gibbs: Some adenoviruses have been isolated from PPR cases in goats, but these have not yet been put back into either goats or sheep. Yet in a survey carried out in northern Nigeria by Dr Taylor a significant incidence of adenovirus was observed.

Dr Provost: Questions have also been raised about the possible role of the parainfluenza-3 virus in PPR. This virus is completely different and hence cannot be said to play any part in PPR infection.

Dr Oluokun: What role does nutrition play with regard to infection in experimental animals?

(Summary - Ed.): It was generally considered that isolating and confining the animals already constituted a stress factor and that mere provision of adequate nutrition was not in itself enough. Experimental goats should be left to roam, though not too freely, and graze in an enclosure.

Dr Musa Momoh: My experience with PPR while I was working in Ondo State shows that the sequence in the clinical picture was first stomatitis, then foaming at the mouth, then some days later, diarrhoea developed. *E. coli* has a part to play in this infection, especially in the more pathogenic strains.

Professor Ojo: The role of *E. coli*, which is normally present in the gastrointestinal tract, depends on whether or not the strains produce enterotoxins.

TOPIC IV

PHYSICO-CHEMICAL AND BIOLOGICAL CHARACTERS OF THE PPR VIRUS

Chairman: Professor W. Plowright
Rapporteur: Dr B.N.C. Nwajei

Physico-chemical and biological characters of the PPR virus

Speaker: Dr F.M. Hamdy, Plum Island Animal Disease Centre, Greenport, New York, USA

Before I begin my presentation I would like to extend a warm vote of thanks to the organisers of the workshop, NVRI, FLD and ILCA.

PPR is a member of the *Morbillivirus* genus of the family Paramyxoviridae. Other members of the genus are measles, canine distemper and rinderpest. The PPR virus was the last member to be identified, and therefore this virus has not yet been as well characterized in terms of physical, chemical, biological and molecular aspects as the other members.

It might be useful to touch on the general characteristics of the *Morbillivirus* genus, as numerous studies have been carried out with measles, canine distemper and rinderpest viruses at the molecular level. We expect to have common properties among all the members and we also expect to experience as few differences in the molecular details as there were in the biological details.

Like other morbilliviruses, the PPR virus contains a helical nucleocapsid which is membrane bound. The chemical structure of the morbillivirus consists of a non-segmented single-stranded RNA molecule with a molecular weight of approximately 6×10^6, six major polypeptides, two of which are glycosylated, and lipid derived from its host cell. It is polymorphic in shape as shown by the electron micrograph. The proteins are designated:

	Approx. mol. wt.
L	
G or H	
L	200,000
G or H	70,000
P	80,000
N_p	60,000
F	65,000
M	35,000
Cellular actin A	60,000
N_s	15-18,000

The functions of these proteins are not completely clear, but by analogy with the paramyxoviruses that have been studied, it is generally accepted that the H-polypeptide associated with haemagglutination of the measles virus has a role in absorption to susceptible cells in relation to cell receptors in the other morbilliviruses. It is glycosylated protein in measles, canine destemper and rinderpest. The F-polypeptide is glycosylated and is associated with cell fusion. It is made up of two polypeptides designated F_1 and F_2 which are attached by a disulphide bond. It has been demonstrated in paramyxoviruses that cleavage of a similar protein into F_1 and F_2 influences virus virulence.

The L-polypeptide is an integral part of the nucleocapsid and has a very high molecular weight of 200,000.

The P-polypeptide is a nucleocapsid-associated protein and considered to be the viral RNA polymerase.

The N_p polypeptide is a nucleocapsid protein involved in virus replication.

The M-polypeptide is associated with the internal portion of the virion envelope and appears to have very strong interactions with the nucleocapsid - thus designated M for matrix protein. Its function involves nucleocapsid recognition and alignment at the plasma membrane. In addition to the structural polypeptides, a putative,

non-structural protein with a rapid turnover in pulse-chase experiments has been observed.

On infection, the viral envelope fuses with the cell membrane interfacing in the extracellular space and only the nucleocapsids enter the cytoplasm. This substructure must contain all the virus components necessary to initiate replication. The F protein is accepted to play a part in cell fusion. It is also believed to contribute to the cytopathic effect (CPE) observed in virus infection. The nucleocapsids of the morbilliviruses have not been shown to be infectious as, for example, in the rhabdoviruses.

Although the viral RNA contains the informational base sequence which must be recognized by the viral RNA polymerase, it is the nucleocapsid, not free RNA, which is the active template structure for the RNA transcription in virus replication. The important implication is that a device exists for a base recognition by the viral polymerase despite the continued association of capsid proteins with the RNA.

As in other morbilliviruses, nucleocapsids have been visualized in the cytoplasm and also in the nucleus in PPR virus infection, but the role of the nucleus is not clearly explained.

The viral nucleocapsids are transported to the cell surface, and the latter develops modified segments of the plasma membrane containing the viral envelope proteins, where the virus matures by budding from the plasma membrane in a heteromorphic fashion.

Immunologic identification is the same, regardless of the strain. This finding/conclusion is based on the neutralization kinetics shown in the graph demonstrated here (demonstration slide, not in text- Ed.).

Studies on PPR at the Plum Island Animal Disease Centre

Speaker: Dr A.H. Dardiri, Plum Island Animal Disease Centre, Greenport, New York, USA

The following is a summary of the results of work done at Plum Island, involving four studies on *peste des petits ruminants* (PPR).

1. *Response of American goats and cattle to the PPR virus*

The exposure of American goats to the PPR virus resulted in an acute to subacute disease with approximately 25% mortality. The disease features were similar to those of rinderpest. Cattle immunized with PPRV resisted challenge with the rinderpest virus. Although PPR-infected goats transmitted the virus to contact cattle, they exhibited no signs of the disease and resisted challenge with the rinderpest virus. The PPR virus demonstrated a serological relationship to rinderpest, measles and canine distemper by the complement fixation test.

2. *Immunologic relationship between the rinderpest and PPR viruses*

The CFT and cross-protection tests showed the rinderpest and *peste des petits ruminants* viruses to be immunologically related. The two viruses, however, were differentiated by virus neutralization tests. The localization of PPR viral antigens in virus-infected cells treated with homologous antibody was demonstrated by ferritin tagging and electron microscopy around the virions, virus particles at the budding stages, and in some regions of the plasma membrane of infected cells. Little, if any, tagging was seen in specimens treated with rinderpest antiserum. The possible origin of the common antigens between rinderpest and PPR viruses was discussed.

3. *Response of white-tailed deer to infection with the PPR virus*

White-tailed deer *(Odocoileus virginianus)* were infected experimentally with two strains of the PPR virus. The response varied from fatal consequence to sub-clinical infection. The clinical signs and gross lesions were similar to those in goats. Virus was recovered from all the infected deer, and the survivors developed specific antibodies demonstrated by CFT. The survivors also resisted challenge with the virulent rinderpest virus that was lethal to control animals.

4. *Aetiology of the stomatitis-pneumoenteritis complex (SPC) in Nigerian dwarf goats*

The experiment was necessary to demonstrate similarity or differences between PPR and SPC. The causative agent of the stomatitis-pneumoenteritis complex was isolated in domesticated goats and Vero cell culture. It was identified immunologically and morphologically as identical with the PPR virus. There were cross-reactions between the SPC virus isolates and the rinderpest virus by immunodiffusion and complement fixation tests, but no cross-neutralization. Goats which recovered from the stomatitis-pneumoenteritis complex were protected against the challenge with rinderpest virus that was lethal to control goats. Ultrastructural morphology revealed intracytoplasmic and intranuclear inclusions made up of random arrays of fibrillar strands. Pleomorphic particles budded from the plasma membrane of infected cells and enveloped virions were seen extracellularly. Specific ferritin tagging was demonstrated in the SPC virus-infected cells treated with homologous and PPR viral antibody systems but little, if any, tagging appeared in the heterologous rinderpest system.

Relationship between the PPR virus and other members of the *Morbillivirus* genus

Speaker: Dr E.P.J. Gibbs, Department of Preventive Medecine,
College of Veterinary Medecine, University of Florida,
Gainesville, Florida, USA

Dr Gibbs gave a brief résumé of his work with colleagues at the Animal Virus Research Institute, Pirbright and the National Veterinary Research Institute, Vom, Nigeria on the classification of the *peste des petits ruminants* virus as the fourth member of the genus *Morbillivirus* (see other papers - Ed.).

It had previously been reported (Mornet et al, 1956) that PPR virus (PPRV) was not pathogenic for cattle, that it conferred immunity against rinderpest and could be neutralized by rinderpest antiserum. Since rinderpest virus - although principally a pathogen of cattle - can produce disease in sheep and goats, these workers at the time concluded that PPR virus was a variant of the rinderpest virus that was adapted to small ruminants and had lost virulence for cattle. However, more recent work, details of which were published while the study being reported here was in progress, indicated that the PPR and rinderpest viruses can be differentiated by cross-neutralization tests *in vitro* (Hamdy et al, 1976). No published information was available on the relationship of the PPR virus to measles and the distemper virus.

It was considered important to establish the relationship of the PPR virus to the other morbilliviruses, especially rinderpest, for two reasons. First, the virtual eradication of rinderpest from cattle in West Africa by the international use of rinderpest vaccine (Joint Project 15 of OAU/STRC) raises the question of whether PPRV infection of small ruminants represents a reservoir of rinderpest for cattle should vaccination be discontinued. Secondly, a vaccine is required to control PPR in West Africa, as small ruminants are

an important source of animal protein in areas where most cattle cannot be raised satisfactorily because of trypanosomiasis.

The study described here was on the biological and physico-chemical characteristics and serological comparisons of the PPR virus with the other morbilliviruses. Electron micrographs of the virus showed the virus to be pleomorphic and to consist of an outer envelope surrounding a coiled nucleocapsid. There was considerable variation in particle size (surface projections: length 15.8 nm, period 5.6 nm and width 18.6 nm). In sheep and goat cell mononuclear layers, the CPE consisted of large 'clock-faced' syncytia, the nuclei of which sometimes contained intranuclear Cowdry type A inclusions.

The biological and physico-chemical characteristics, and serological relationships of the PPR virus are given in Tables 1 and 2.

Table 1. *Biological and physico-chemical characteristics of PPR virus compared with those of other Paramyxoviridae and unrelated viruses associated with similar infections of sheep and goats.*

Virus	Morphology	Resistance to lipid solvents	Type of nucleic acid in genome	CPE in tissue culture		
				syncytia	intra-cytoplasmic inclusions	intra-nuclear inclusions
PPR	pleomorphic	sensitive	RNA	+[1]	+	+
Rinderpest	pleomorphic	sensitive	RNA	+	+	+
Canine distemper	pleomorphic	sensitive	RNA	+	+	+
Measles	pleomorphic	sensitive	RNA			
Other viruses associated with sheep and goat infections						
Parainfluenza-3	pleomorphic	sensitive	RNA	+	+	+
Respiratory syncytia	pleomorphic	sensitive	RNA	+	+	-
Bluetongue	icosahedral	resistant	RNA	-	-	-
Foot-and mouth disease	icosahedral	resistant	RNA	-	-	-
Adeno	icosahedral	resistant	DNA	-	-	+

[1] +=Positive; -= negative.

Table 2. *Serological relationships of PPR virus (PPR Nig 75/1) with vaccine strains of canine distemper (Onderstepoort), measles (Schwarz) and rinderpest (RBOK)*[1] *viruses.*

Antiserum[2]	Virus			
	canine distemper	measles	rinderpest	PPR
Canine distemper	8 200[3]	12	4	12
Measles	6	128	12	8
Rinderpest	32	32	256	12
PPR	24	12	4	1 536

[1]Rinderpest RBOK and RBT/1 viruses gave very similar results. Data for RBT/1 therefore are omitted.

[2]Serum from natural host.

[3]Reciprocal of 50% endpoint dilution of serum against 100 $TCID_{50}$ virus.

It was observed in path genicity and cross-protection experiments that subcutaneous inoculation of sheep and goats with the PPR virus usually produced clinical disease and excretion of virus, with infection spreading to in-contact sheep and goats. PPR virus was not pathogenic for cattle and protected them from challenge with the virulent rinderpest virus. Neigher replication nor excretion of PPR virus in cattle could be demonstrated, although serum antibody was produced. Transmission of infection from inoculated cattle to in-contact cattle did not occur.

Sheep and goats vaccinated with the attenuated RBOK strain of rinderpest virus did not develop clinical disease when infected with PPR virus. The Schwarz vaccine strain of measles virus did not protect sheep and goats from developing PPR. A degree of cross-protection apparently exists between the viruses of canine distemper and PPR, since only one animal in the group vaccinated with canine distemper virus showed mild clinical lesions of PPR.

However, PPR virus replicates in sheep and goats given rinderpest, canine distemper and measles vaccines, virus being detected in nasal swabs collected from each group 7 days after challenge (Table 3). To investigate whether goats and sheep that had recovered from clinical PPR could also excrete PPR virus if re-exposed to infection, the animals in the control group in the same trial were subsequently challenged 21 days after initial infection. PPR virus could not be recovered from nasal secretions collected 7 days later.

Table 3. *Pathogenesis and cross-protection between PPR and other morbilliviruses.*

Vaccine	Pyrexia	Clinical signs	Virus excretion
Canine distemper virus	-	+	
Measles virus	+	+	+
Rinderpest virus	-	-	+
Control[1]	-	-	-

[1]Recovered from clinical PPR and subsequently challenged 21 days after initial infection.

In conclusion, Dr Gibbs said that in this particular study it was shown that rinderpest vaccine protects sheep and goats from clinical PPR. Rinderpest vaccine is produced in large quantities in West Africa, and some of this vaccine has already been used empirically to protect sheep and goats from PPR. Although there has been no critical evaluation of its effectiveness, the 'Office International d'Epizooties' has endorsed its use (OIE, 1976/77). However, the observation reported here that sheep and goats that have been vaccinated with rinderpest vaccine can develop subclinical PPR indicates that, under field conditions, animals vaccinated with rinderpest vaccine possibly may perpetuate PPR in sheep and goat populations even though they are themselves protected from developing

the clinical disease. Whether a specific vaccine for protection of sheep and goats against PPR is required will depend upon the results of field trials using rinderpest vaccine.

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Discussion

Dr Nawathe: Was the virus used by Dr Dardiri live or inactivated?

Dr Dardiri: It was formalin inactivated virus.

Dr Mathew: Have the PPRV and RV* been accepted by the International Committee on Virus Nomenclature as separate entities, that is as different viruses?

Dr Hamdy: They have. If we accept the idea of square roots of homologous and heterologous titres, they are two different viruses, and although there is a kind of relationship between them, they cannot be regarded as the same virus.

Dr Bourdin: Experiments were made in which heterologous virus was put into a heterologous tissue. Then, when injected into cattle, this virus can multiply and provide protection.

Prof. Plowright: PPRV is accepted by the International Committee on Virus Nomenclature as the fourth member of the *Morbillivirus* genus.

Dr Scott: Were any nervous signs seen in PPR cases in places other than in Africa?

Dr Obi: The nervous signs I observed may have been due to electrolyte imbalance, and also due to a severe dehydration following diarrhoea. However, nervous signs have sometimes been reported in rinderpest.

Dr Bourdin: When animals imported from France were inoculated with infected lymph nodes, virus excretion was observed up to the 12th day after the inoculation.

Dr Evbuoma: Could wild animals be a reservoir of infection? In other words, are they susceptible?

Dr Dardiri: They are susceptible but the survivors withstood the challenge.

Dr Sansi: How long is the latent period following inoculation with the formalized PPR virus, i.e. how long does it take for protection/immunity to develop?

Dr Dardiri: What is the duration of this conferred immunity? Three weeks post inoculation/vaccination the animals withstood challenge with the virulent PPRV.

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*Rinderpest virus

Dr Wosu: How old were the vaccinated animals? It seems important because of the possibility of interference by maternal antibodies.

Dr Dardiri: The animals we used were approximately 7 months old. Immunity lasted for about 2-3 months. The animals were screened for PPR antibody prior to the experiment.

TOPIC V

NATURE OF IMMUNITY TO PPR

Chairman: Dr A. Provost
Rapporteur: Dr M.O. Asagba

The use of TCRV and PPRV to confer immunity to PPR

Speaker: Dr W.P. Taylor, Animal Virus Research Institute, Pirbright, Woking, Surrey, United Kingdom

The role of non-specific agents like *E. coli* and *Pasteurella* species, all of which could add to the clinical picture and mortality in PPR, had been referred to on the previous day of the workshop. To combat PPR there should be both vaccination and treatment, the policy being to aim at the primary agent, the PPR virus (PPRV), by producing a cheap and effective vaccine. I would like you to consider the following:

(i) Production of a homologous vaccine by attenuation of the PPRV. Research in Senegal has already provided a lead in this direction.

(ii) To make use of the normal relationship between PPRV and the rinderpest virus (RV). At the National Veterinary Research Institute (NVRI), Vom, tissue culture rinderpest vaccine (TCRV) was used to vaccinate experimental goats. No field trials were attempted with this vaccine. The amount of TCRV required to immunize a goat and that required to immunize a cow were found to be the same. Hence, the recommended minimum amount for cattle was adopted as the international dose for immunizing goats.

Clean, susceptible goats were vaccinated with TCRV and at various periods of 3, 6, 9 and 12 months, the animals were challenged with virulent PPRV. The vaccinated goats showed completed immunity to PPR and there was no transmission of the virus to susceptible goats co-housed with animals undergoing challenge.

Serologically, rinderpest neutralizing antibodies lasted at least 12 months in unchallenged animals while only a very low level of cross-neutralizing antibody was detected with PPRV. Anti-PPRV neutralizing antibodies were, however, rapidly detected following PPR challenge, a situation that could have resulted due to replication of the challenge virus.

Field experience might show that the homologous vaccine produces an unacceptably low level of immunity. Adequate trials should therefore be conducted before TCRV is replaced by a homologous PPRV vaccine that might be expensive and time-consuming to produce.

Vaccination of sheep and goats with TCRV in Benin and Senegal

Speaker: Dr P. Bourdin, Institut National d'Elevage et de Recherche Vétérinaire, Dakar, Senegal

In Senegal and in the Republic of Benin, experiments were conducted on the use of TCRV in the field to protect goats against PPR. The vaccine strain of the virus used was the classical rinderpest virus obtained from Prof. W. Plowright, which had been passed 65 times in bovine kidney cell culture. The virus titer was $10^{7.0}$ $TCID_{50}$/ml and it was administered subcutaneously to dwarf goats in Senegal and to the Sahelian breed of sheep and goats in Benin. The animals were bought in the market and screened for PPR antibodies. The homologous PPR vaccine was not used in the experiments.

In Benin many of the sheep and goats succumbed to PPR even before vaccination. In Senegal, no clinical signs of PPR were observed in animals vaccinated with TCRV. Of the 75 dwarf goats vaccinated with TCRV and challenged with virulent PPRV, only 2 died. PPR was widespread among goats in Senegal in 1976. By 1977, after the vaccination with TCRV, there was not a single PPR case among 5000 goats.

Five hundred sheep and goats were screened for the presence of rinderpest antibodies after vaccination with TCRV. High levels of antibodies were detected even one year post vaccination in both sheep and goats. Experiments were carried out to find the effect of PPRV in goats vaccinated with TCRV. The results showed that the PPR virus replicated on the 3rd and 15th day of the experiment. In Benin, the results were disappointing because the animals were incubating the disease.

Editor's note: Bourdin, P., Reoche, M. and Paurent, A. (1970) published a detailed 'Report on TCRV for the prophylaxis of PPR in small ruminants in Dahomey (Benin)' in Rev. Elev. Méd. Vét. Pays Trop. 23: 295-300. Flock owners accepted free vaccination but refused to have their animals vaccinated when the vaccine was to be paid for.

Discussion

Dr Nawathe:	Were pregnant goats used?
Dr Bourdin:	In the villages all kinds of goats were used; some were pregnant but no untoward incidents were reported following vaccination.
Prof. Plowright:	Was any work done on the decline of antibodies in kids and lambs?
Drs Taylor and Bourdin:	None.
Dr Provost:	This probably occurs at 1-2 months of age.
Dr Evbuoma:	Did Dr Taylor try the attenuated homologous vaccine on goats in Vom?
Dr Taylor:	No attempt to attenuate the vaccine was made.
Dr Wosu:	Why did you use 1-year old goats?
Dr Bourdin:	We found this represented the general age group for the vaccinated village population.
Dr Onoviran:	Not many field trials have so far been conducted with TCRV and more need to be conducted.
Dr Gibbs:	Will Dr Bourdin use prevaccinated animals?
Dr Bourdin:	Annual vaccination is recommended in well controlled areas.

Dr Okoye: Field results with TCRV are erratic and at times useless. If animals are given antibiotics and trypanocides, the results might be promising.

Dr Majiyagbe: It is a quite different thing to vaccinate animals in the field. In the south animals may be incubating the disease and if they are vaccinated, other problems may interfere with an effective immune response. With animals vaccinated at the age of 4 to 6 months, immunity should be checked after 3-4 days when pyrexia and cell mediated immune responses are observed. TCRV vaccination works but the problem is with animals already incubating the disease.

Dr Sansi: Dr Majiyagbe is right. The application of vaccine in the field is not well understood. The first requirement is that the animals are healthy. It is also necessary to improve the conditions under which vaccination is done (the use of non-sterile syringes is disgusting). When many field assistants are needed, as for example in poultry vaccination, detailed instructions should be passed on to them to avoid accidents. TCRV has been used against PPR in several countries, especially in the francophone African countries. During the Benin conference in 1976 a good report was presented on the use of TCRV against PPR. Its conclusion was that TCRV is not erratic, if correctly used, in producing immunity to PPR.

Dr Provost: Dr Sansi is right. There are accidents during vaccination. The desired characteristics of a vaccine are: a) innocuity; b) effectiveness in the field. Live TCRV should be used to achieve the best effectiveness. The vaccine must be diluted with the right diluent, using a molar solution of magnesium sulphate at room temperature.

Dr Evbuoma: At the National Animal Production Research Institute (NAPRI) tissue culture rinderpest vaccine was used for goats and found to be useful. I am surprised that Vom delegates are discouraging its use. (Objection from audience: "No") - I apologize. No PPR has been observed ever since TCRV has been used.

In 1978 NAPRI obtained some goats from Ibadan. They were quarantined and 10 days later, 35 were lost due to PPR infection. Since then, the policy has been to vaccinate goats against PPR with TCRV. No sheep were so far vaccinated. TCRV works in goats.

Dr Aliyu: Our health programme for sheep and goats involves vaccination with TCRV. One hundred and fifty thousand animals were so far vaccinated. No losses were recorded under proper management and hygienic conditions. The conclusion: TCRV protects against PPR.

TOPIC VI

PROBLEMS CONNECTED WITH THE VACCINATION OF SHEEP AND GOATS WITH TISSUE CULTURE RINDERPEST VACCINE

Chairman: Dr A. Prevost
Rapporteur: Dr M.O. Asagba

Problems connected with TCRV vaccination of sheep and goats

Speaker: Dr A. Abegunde, National Veterinary Research Institute, Vom, Nigeria

It has been accepted at this meeting that small ruminants are important because they provide valuable protein and skins, they are easy to maintain and do not compete with humans for cereal grain. In the past, these animals received little attention but the situation changed after the last drought in Nigeria, when a more specific study was directed towards them.

The topic today refers more specifically to goats. In this context three major issues are being proposed for discussion:

1. The relationships between *Kata, peste des petits ruminants* (PPR) and the stomatitis-pneumoenteritis complex (SPC).
2. The use of TCRV in goats.
3. What are the possible alternatives to the protection against PPR?

1. *Kata* - do we have it or not? Some speakers sa what we have is PPR. In SPC outbreaks in eastern Nigeria, the PPR virus was isolated; furthermore, in western Nigeria, only the PPR virus was isolated from outbreaks of a similar disease. The presence of the PPR virus was also reported in other parts of Nigeria, although previous workers could not at the time with certainty identify the agent. The clinical picture of *Kata* was thought by some to be different from that of PPR, but the symptoms of the affected animals in the humid zone were in addition complicated by secondary bacterial

infections. However, where virus isolation had been attempted, only the PPR virus was isolated from cases reported either as *Kata* or as SPC.

2. Experiments on the use of TCRV have been carried out, both in the laboratory and in the field, in Plateau, Kaduna, Benue and other States in the north as well as in several States in the south. In the northern States no side effects have been observed whereas in the south, some side effects, especially abortions in goats, were reported. Many animals that aborted developed a good virus titre and resisted challenge with the virulent PPR virus. Labial encrustations were reported to develop in some purchased goats vaccinated with TCRV. However, labial encrustations are not considered specific to the natural PPR infection, nor as a sequel to TCRV vaccination. The condition may be seen as a reaction to various stress factors such as transportation, humidity and change of feed and environment.

In Nigeria, most market goats harbour disease and many, when housed, develop PPR. It was also observed (by ILCA scientists) that if animals are not screened prior to vaccination, they may come down with PPR. In Vom, side reactions to TCRV vaccination have not been a serious problem. This vaccine is extensively used in other West African countries as well, and very few side effects have so far been reported.

3. Tissue culture rinderpest vaccine is currently used because both the TCR virus and the PPR virus are related and there is cross-immunity between them. The two possible alternatives are:

3.1 Homologous PPR vaccine

However, inactivated PPR vaccine is disadvantageous in that it is a dead vaccine and immunity following its use is short-lived.

3.2 Attenuated PPR vaccine

No useful results from its use were achieved in Senegal. In Vom attempts to produce such a vaccine are in progress. Fifty-eight

serial passages have been obtained in Vero cells and the vaccine has been tested in goats, with no thermal or clinical reactions occurring. The virus replicated in goats appears in the nostrils and in circulating leucocytes. The search for this vaccine is complicated and there is no guarantee that it will become available soon. Therefore, the use of TCRV, which is available at Vom, is advocated. At the same time the search for a PPR vaccine should be continued.

4. Considerations on the use of TCRV in goats

- Its efficacy is doubted by some.
- The immunity following its use is not long enough.
- Much manpower is needed to assemble village goats for vaccination.
- Early vaccination is necessary.
- A nation-wide policy on the use of TCRV in goats should be adopted until such a time that homologous PPR vaccine has been developed. The National Veterinary Research Institute at Vom has been advocating such a policy since 1978.

Results of TCRV vaccination in Nigeria

Speaker: Dr T.U. Obi, Department of Veterinary Medicine, University of Ibadan, Ibadan, Nigeria

Between 1979 and 1980 several experiments were conducted on the use of TCRV to protect sheep and goats against PPR. First, 255 goats and 95 sheep were vaccinated with TCRV 4[illegible] and 146 from Vom. Later on another batch of 400 goats and 95 sheep was vaccinated. Before that vaccination serum samples were taken. At 21 days post vaccination, more serum samples were taken, as well as faecal samples.

Results

In some animals a rise in temperature was observed for one day, but the majority of the animals were afebrile. Only one goat aborted, and no clinical PPR was observed. On one farm two PPR cases were reported but the animals survived. No conclusion could be drawn from an experiment involving four goats exposed to PPR-infected animals.

The problems in vaccinating with TCRV, as already pointed out, are:

a) Animals may be incubating PPR at the time of vaccination. Furthermore, death usually occurs in animals that have been infected with trypanosomiasis prior to the vaccination.

b) The incidence of the disease is higher during the harmattan, i.e. during the dry season, and in April, May and June, i.e. before the height of the rainy season. If vaccination is carried out during these periods of higher risk, the results are usually poor.

c) Tissue culture rinderpest vaccine is supplied in 100-dose ampoules. Village goats are kept in small groups, usually a few

kilometers apart, and therefore they can not be brought for vaccination at the same time. But if the reconstituted vaccine is not used all at once, the left-over becomes a waste.

d) The shelf-life of the vaccine is 9 to 12 months, and if due to transportation problems it is not brought to the field early enough, its potency could deteriorate.

At Owerri, in eastern Nigeria, it was found that TCRV sometimes [illegible]ects, sometimes not. At present goats are given the equivalent of the cattle dose, but previously it was only half of the dose, which could have affected the efficacy of the vaccine.

Procedures for vaccination with TCRV

Speaker: Dr E.C. Ihemelandu, University of Nigeria, Nsukka, Nigeria

The problem lies in the use of TCRV. In 1972 a publication appeared on the use of TCRV to protect goats against PPR, and the Chief Veterinary Officer in what was then the Eastern Region of Nigeria requested that it be tried.

Under certain conditions, TCRV does protect. However, when goats were vaccinated against PPR and later taken to the market, it was observed that they shed the virus. The Government's advice, therefore, was that thermometers should be taken to the field in order to detect animals incubating the disease. The assembly of animals at one place usually resulted in outbreaks. The solution then was to prepare sheep hyperimmune serum. When temperatures rose to 105°F (40.5°C) hyperimmune serum was given during the first 10 days, followed by TCRV 7 days later.

Many veterinarians in eastern Nigeria doubted the efficacy of TCRV in the field, mainly because the animals to be vaccinated had to be healthy. We recommend the following procedure when buying in animals from the market: a) quarantine for 7 days. At first temperature rise, give hyperimmune serum; b) do serology to monitor the antibody level; c) then proceed to vaccinate with TCRV.

In an outbreak, there is no guarantee that the disease could be reversed. However, in cases where there is pneumonia and oral lesions, the use of the hyperimmune serum can bring about recovery. If diarrhoea sets in, on the other hand, animals may still die in spite of the treatment recommended.

The results of TCRV in Ghana

Speaker: Dr M. Bonniwell, Veterinary Diagnostic Clinic, Ministry of Agriculture, Animal Health Division, Kumasi, Ghana

Ghana's sheep population is larger than that of goats: in Ashanti Region, for example, the ratio is 4:1. The average flock consists of 20 to 25 animals. PPR is very severe in sheep, with a mortality rate sometimes reaching 100%. This, as can be seen, is in contrast to the situation in Nigeria. Furthermore, there have been only limited virus isolations to support clinical and post-mortem findings in Ghana. As many as 75 to 100% of the deaths occur in animals that have been in contact with new flocks, which usually happens during festivals when animals are moved from the north to the urban south. Among the most affected are 2- to 6-month old sheep.

Tissue culture rinderpest vaccine was first used in Ghana in 1977 and it is still being used. The two types of the vaccine are:

a) Kabete 'O' strain of TCRV obtained from Kenya. The vaccine has been passed 90 times in bovine kidney cells.

b) TCRV obtained from the Animal Virus Research Institute at Pirbright. It is used for healthy animals one month old and above.

Effectiveness of vaccination

Up to the present time 7000 sheep and 1500 goats have been vaccinated and so far no problems have been encountered with TCRV in the field. Ring vaccination is carried out around existing outbreaks.

Problems of vaccination

No vaccine reactions either local or systemic were observed in healthy flocks, but heavy mortality resulted following the vaccination of animals already infected with PPR. There were no abortions in

Ashanti but elsewhere abortions were reported. About 95% of the owners cooperated in the vaccination programme but in general, information about the flock size, incidence of the disease and mortalities among the animals were difficult to obtain, and dead or dying animals were usually not made available for postmortem examination.

PPR outbreaks over the last 3 years

There were 150 outbreaks over the last 3 years, six of these occurring in vaccinated flocks. The sick animals had fever and diarrhoea. On one farm with sheep and goats outbreaks occurred over a period of 3 months. One of the vaccinated sheep showed symptoms of fever, rhinitis and diarrhoea, but the animal did not die. The goats did not show any PPR symptoms. Among the other vaccinated small ruminants no deaths were reported. In another flock consisting of both vaccinated and non-vaccinated sheep and goats, five non-vaccinated sheep died but none of the non-vaccinated goats. Among the vaccinated animals nine showed PPR symptoms and two sheep died. No deaths occurred in the vaccinated goats. However, whether or not PPR was the actual cause of the sickness and death of the two sheep is not certain since adequate diagnostic procedures could not be carried out at the time.

Diagnosis

- Clinically and pathologically stomatitis is regarded to be a pathognomonic symptom.
- Agar gel diffusion and precipitation tests are used in the laboratory.

Conclusions

- It is practical to vaccinate sheep and goats in Ghana.
- Our experience shows that TCRV is safe for use in small ruminants.
- TCRV offers protection against PPR. The best results are obtained in ring vaccination as PPR does not spread among the vaccinates.
- TCRV comes under criticism if it is given to flocks already infected with PPR.

- The role of other agents in the pathogenesis of the disease still needs to be clarified.

Discussion

Dr Provost: When you last vaccinated, did you not see a decline in pulmonary diseases, e.g. in pasteurellosis?

Dr Bonniwell: Pneumonia was seen in goats in many cases. This could have been due to a previous PPR infection. The incidence of pasteurellosis should decline with vaccination.

Dr Aliyu: In our experience there has been no decline in lung diseases even after a mass vaccination of goats and sheep. In general we have observed that the incidence of diseases in small ruminants has not declined after vaccination. Animals are also dewormed after vaccination.

Dr Bourdin: During our vaccination experiments TCRV was used but no vaccine against pasteurellosis. An outbreak of pneumonia involving 4000 sheep occurred in northern Senegal.

What is the estimated cost of vaccination and what is the cost of vaccine per animal?

Dr Sansi: Tissue culture rinderpest vaccine used to be expensive, but at present 100 doses cost N 1.50,* which is very cheap.

Dr Akeredolu: Are there differences in the clinical manifestations of PPR in sheep and goats in Ghana?

*N 1.50 = US$ 2.40 at the 1980 exchange rate - Ed.

Dr Bonniwell: There are more sheep than goats in Ghana. Owners of sheep do not usually keep goats and *vice versa*. The disease is more severe in sheep than in goats. Young sheep are usually affected. The clinical signs in sheep are similar to those in goats except that in sheep the encrustation of the mouth is less.

Prof. Plowright: If 100-dose ampoules of TCRV cannot be used in the villages, then why not produce smaller ampoules? If we need to use hyperimmune serum, how is it produced? What is its assessed value? Could its cost of production, storage and administration be higher than for TCRV?

Dr Abegunde: We tried to produce hyperimmune serum but it was found uneconomical. To produce serum on a large scale and to store it costs more than to produce a vaccine for PPR.

Dr Majiyagbe: The National Veterinary Research Institute at Vom tried to find antibodies to PPR in goats and other animals vaccinated with TCRV. Using immuno-electrophoresis, two to five precipitin lines were seen when a TCRV antigen was used with the serum of animals vaccinated with TCRV. Convalescent serum from cattle would be more useful than the hyperimmune serum. Alternatively, develop a homologous vaccine. The best bet is to develop a homologous vaccine to avoid the presence of two agents.

Dr Oluokun: What can be done to reduce wastage of TCRV?

Dr Nawathe: Smaller-dose ampoules would be all right. However, the costs of freeze-drying must be considered. Our vaccine is cheap at 1 kobo* per dose in the 100-dose ampoule. If you can pay more, we will produce 25-dose ampoules. In East Africa, the cost of the vaccine is according to its titre: if the titre is high, the vaccine costs more; and if it is low, the cost is low. Were we engaged in commercial production, we would produce 10-dose ampoules. A problem also occurs with the expiry dates. I suggest that Dr Oluokun makes his request in writing.

Wherever the hyperimmune serum is used, it should be followed by TCRV to see any reactions.

Give the same cattle dose, then challenge with TCRV, and then collect serum. This is preferable to producing hyperimmune serum.

* 1N = 100 kobo

Dr Lamorde: 100-dose ampoules are produced at NVRI, Vom, to meet large-scale vaccination programmes. The cost of production is not a primary consideration.

Dr Okoye: In eastern Nigeria, vaccination of cattle and sheep is free.

Dr Dardiri: What might be the consensus of cattle owners on the use of TCRV as against PPRV?

Dr Majiyagbe: In northern Nigeria, most people have both cattle and sheep that move together. Goats graze alone, but collecting points for vaccination could easily be organized.

Dr Obi: The effects of the use of hyperimmune serum are costly, especially in sheep vaccinated with TCRV and later challenged with a virulent PPR virus. If you use hyperimmune serum and later vaccinate, do you get protection? Does hyperimmune serum interfere with vaccination?

Dr Provost: Would not the problem be solved if the virus were attenuated?

Dr Provost: In West Africa there is no need to vaccinate sheep and goats for rinderpest. One can control rinderpest by vaccinating cattle alone. If you also vaccinate sheep and goats, logistic problems will have to be faced.

Dr Dardiri: So far only the technical aspects of vaccination have been considered. Other aspects include:

a) Socio-economic conditions pertaining to stock owners and to the animals themselves.

b) Availability of vaccines, their costs, the movement of animals and herdsmen, and quarantine situations.

c) Diagnosis.

Dr Ihemelandu: To protect against PPR animals should be given hyperimmune serum and subsequently inoculated with the PPR virus, not with TCRV. In mass vaccination it is necessary to know which animals are incubating the disease and which do not because, as experience shows, those incubating it die. That is why until the laboratories are in a position to tell us which animals are incubating the disease, we shall continue to advocate the use of the hyperimmune serum prior to vaccination.

Dr Scott: Rinderpest outbreaks cannot be controlled by vaccination alone. Other measures include quarantine and advice to people not to buy animals from the market.

Dr Evbuoma: In cell-mediated immunity animals could be screened to find those incubating the disease.

Prof. Hill: I do not see that hyperimmune serum can have much use in the villages due to difficulties in, and the expense of, its production which were already mentioned. However, I agree with Dr Ihemelandu that it can be used for newly acquired animals under quarantine and where vaccinating animals already incubating the disease may trigger off an outbreak.

Prof. Ikeda: If no giant cell pneumonia is observed, then it is the enteric form of PPR. Coccidiosis does cause complications. The PPR virus causes primary damage, hence it can precipitate an invasion by other pathological agents. More studies are needed on the PPR virus both in sheep and goats: for example, on how it spreads the effects of dehydration in animals and the significance and effects of mouth and intestinal lesions.

Dr Opasina: Animals that are purchased in bulk should be quarantined. The village pratice is to keep 2 to 3 animals per person, which does not facilitate quarantine. When animals were quarantined in the ILCA stations for 7 days and then vaccinated, abortions and labial encrustations were observed but this may have happened because the animals were already incubating the natural disease. A viable strategy for vaccination still needs to be worked out. The best solution is to operate a comprehensive health programme, particularly in livestock investigation and breeding centres. However, such programmeshave not yet been devised and PPR is now a very unpleasant reality.

If animals die as a result of vaccination, village owners should be compensated. Should we also vaccinate against pasteurellosis, give prophylactic treatment for coccidiosis etc? And if PPR is conquered, will other disease agents rear their heads?

Dr Ezebuiro: Were any side effects observed at Pirbright in goats and sheep vaccinated with TCRV?

Dr Taylor: No.

Dr Ezebuiro: Probably every flock in Nigeria has animals that carry the PPR virus. Dr Bourdin in Benin found antibodies to PPR in 46 goats and 64 sheep. It is advisable to map out the incidence of the disease, as far as possible, before the vaccination. If the incidence is high, massive vaccination efforts might be spreading the disease. Therefore, careful field surveys are necessary before vaccination.

Dr Obi: Labial encrustations are usually associated with, or caused by, contagious pustular dermatitis. Are we sure that we can precipitate the onset of PPR with vaccination? Since assembling a larger number of animals poses a problem, smaller vaccine ampoules should be used to reduce wastage.

Dr Provost: The use of magnesium sulphate ($MgSO_4$) will prevent wastage.

Dr Evbuoma: How do we detect the PPR carrier status of animals?

Dr Abegunde: We have no definite answer for this.

Dr Wosu: What is the situation regarding the loss of passive immunity?

Dr Majiyagbe: There is no definite information on the loss of passive immunity. In Newcastle disease, for example, the level of maternal antibody is not high enough to protect the chick even by the 10th day after hatching. I would suggest a 2-week passive immunity in the case of PPR.

Dr Scott: I suggest it may last for a longer period.

Dr Evbuoma: In Kano, horses came down with the African horse sickness. Serum was collected from the sick animals and injected into other sick animals. Some horses were saved in that way. So if serum is taken from sick goats and injected into other sick goats, the latter could probably be saved.

Dr Ihemelandu: In our experience PPR does not occur in goats less than 2 months old.

Dr Obi: We have recorded a few cases of PPR in 2-month old goats. Many cases occur between 3 and 9 months of age. This suggests that maternal antibody circulates for the first 2 to 3 months after birth.

Prof. Plowright: Three things have to be considered: a) the use of the homologous or heterologous serum; b) the type of antibody and its rate of decline; c) the range of antibody titres.

Dr Orkpe: In Benue State, many outbreaks occurred during 1975 and 1976. Medicaments were usually administered and samples from the last outbreak were sent to Vom for diagnosis. During outbreaks deaths were usually heavy but quite low between the outbreaks. Vaccination was carried out, and the epidemiology of the disease, clinical signs, including oral erosions and diarrhoea, were recorded in the clinics. The disease was diagnosed as *Kata*.

Dr Ghaji: Can we record post-vaccination reactions during outbreaks?

Dr Provost: TCRV can at present be considered a good vaccine against PPR. Its innocuity in controlling rinderpest in small ruminants was proved in India as well. There could be problems with the use of the hyperimmune serum in the field, including logistic problems.

TOPIC VII

COMMENTS ON THE POLICY TO BE ADOPTED FOR THE CONTROL OF PPR AND PLANNING OF FURTHER RESEARCH

Chairman: Dr Amogu
Rapporteur: Dr K.A. Majiyagbe

Comments on the control of PPR

Speaker: Prof. W. Plowright, Department of Microbiology, Institute for Research on Animal Disease, Compton, Berkshire, United Kingdom

I endorse the statements of the previous two speakers and wish to emphasize the following points:

1. In the past, sheep and goats had been neglected by veterinarians. Any programme for the control of PPR offers great opportunity for observation, study and, in some cases, treatment of the other diseases of small ruminants. Without a comprehensive approach, however, the effects of a PPR control programme would be short-lived.

2. Elimination of PPR is the ultimate aim, and this implies effective international collaboration; otherwise, the reintroduction of the infection could occur at any time after the level of immunity in Nigeria had been allowed to fall.

3. I am convinced by the evidence presented at the workshop that TCRV is the only vaccine at present available to control PPR. It has proved to be highly effective in Ghana and Benin. A new "homologous" vaccine would take at least 2 to 3 years to develop and test adequately. That is too long a period to delay action.

Comments on the control of PPR

Speaker: Dr G.R. Scott, University of Edinburgh, Centre for Tropical Veterinary Medicine, Easter Bush, Roslin, Midlothian, Scotland, United Kingdom

Our deliberations have highlighted four major issues. First, *peste des petits ruminants* (PPR) is undoubtedly a disease of national and international importance in West Africa. Today only the recrudescence of rinderpest is of greater importance.

Secondly, action is urgently needed to stem the catastrophic losses resulting from PPR. Fortunately, as we have heard at this workshop, an efficient tool for limiting the losses already exists, namely, attenuated tissue culture rinderpest vaccine (TCRV).

Thirdly, the control of a disease of national importance can never be achieved through vaccination alone. Legislation is essential to enable the implemertation of sanitary and technical measures. To this end, PPR should be declared a notifiable disease in all West African countries.

Fourthly, there is a cliché that disease knows no boundaries. This certainly applies to *peste des petits ruminants* and it is essential for efficient control that international coordination be sought and established.

Discussion

Dr Bourdin: It is important to decide when to interfere with a broad vaccination programme. Should it be towards the end of the cold season? It is also necessary to avoid vaccination during a PPR outbreak.

Dr Ezebuiro: If we are to consider PPR as a notifiable disease, are we sure of the immune status, or are we dealing with something as ubiquitous as the "common cold"?

Dr Sansi: The administrative and other details involved should be considered when deciding whether a particular disease should be classified as notifiable.

Dr Ikede: It seems we are all in agreement that the stomatitis-pneumoenteritis complex, *Kata* and PPR are one and the same disease. There should be regular follow-up meetings to discuss current problems as well as progress in PPR research.

Dr Fabunmi: Research should also be conducted on the relationship between the helminth burden status of an animal and PPR.

Prof. Hill: The width and scope of the views put forward by the various speakers at this workshop testify to the fact that much thought has been given to the PPR problem. As regards the final conclusions reached, an analogy can be drawn to a hotel menu that is presented daily, much the same, but with the ingredients placed in a different order, which nonetheless comes up with the same recipe, in this case to vaccinate against PPR with TCRV, at least for the present. However, there still are certain problems in vaccinating newly established groups of animals intended for intensive rearing or other purposes, which will necessitate a substantial research input into this particular aspect of PPR control in the future.

Dr Provost: A 19-country study has been undertaken in West Africa by such organizations as EDF and ECOWAS, in which emphasis is being given to animal health problems. I have good reason to hope that EDF might look favourably on funding a regional vaccination programme against PPR and that, although costs might be high, exceeding perhaps one billion French francs in the first year, it would be possible to build up a PPR resistant flock of sheep and goats in the region. Research should start with epidemiological studies, which EDF might consider supporting on a regional basis.

Summary: Conclusions and recommendations

The International Workshop on *Peste des Petits Ruminants* (PPR) was convened to examine the increasing economic importance of losses from PPR in sheep and goats in Nigeria and other West African countries, to develop the strategies to be adopted to combat the disease, and to make recommendations to West African Governments accordingly.

A summary of the conclusions and recommendations prepared by the workshop drafting committee was approved as follows:

Conclusions

Kata and the stomatitis - pneumoenteritis complex are synonymous with PPR and indicate infection by the *peste des petits ruminants* (plague of small ruminants) virus. *Peste des petits ruminants* is considered a more appropriate name than any other to represent the disease.

PPR is widespread in sheep and goats in many West African countries in the humid zone and is of particular economic importance in that zone.

Recommendations

1. The possibility of declaring PPR a notifiable disease should be investigated.
2. Where sheep and goat populations are affected by this disease, systematic vaccination should be undertaken with tissue culture rinderpest vaccine (TCRV).
3. In view of the economic importance of sheep and goats, efforts should be made to study and control other disease problems in these animals.

4. While TCRV offers the best presently available prospect for the control of PPR, this workshop considers it imperative that there should be a continued research into the diagnosis, epidemiology and immunology of the disease; for this purpose an effective laboratory network should be established.

5. This workshop recognizes that the implementation of these recommendations will require the necessary funding and recommends that the Governments concerned should provide funds or, where necessary, seek financial assistance from international agencies.

6. This workshop also urges the Federal Government of Nigeria to seek the cooperation of other West African countries for the effective coordination of PPR control at the international level.

Appendix

The following papers (published elsewhere) were tabled by speakers and other participants for general information:

1. Gibbs, E.P.J. Taylor, W.P., Lawman, M.J.P. and Bryant, J. 1979. Classification of *peste des petits ruminants* virus as the fourth member of the genus *Morbillivirus*. *Intervirology* 11: 268-274.

2. Gibbs, E.P.J., Taylor, W.P. and Lawman, M.J.P. 1977. The isolation of adenoviruses from goats affected with *peste des petits ruminants* in Nigeria. *Research in Veterinary Science* 23: 331-335.

3. Gibbs, E.P.J. (s.d.). Serological and cross-protection studies with *peste des petits ruminants* virus. AVRI mimeograph, Pirbright, Woking, Surrey, UK.

4. Obi, T.U. and Gibbs, E.P.J. 1978. Orf in sheep and goats in Nigeria. *Trop. Anim. Health Prod.* 10:233-235.

5. Lefèvre, P.C. 1980. La peste des petits ruminants (Synthèse et bibliographiques). IEMVT.

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