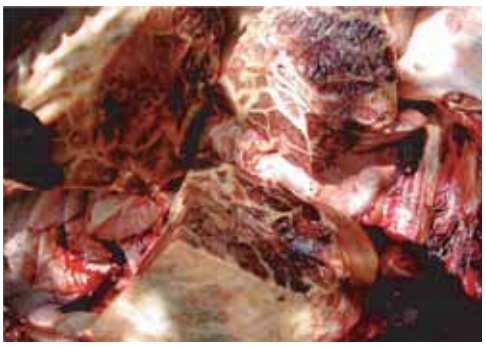
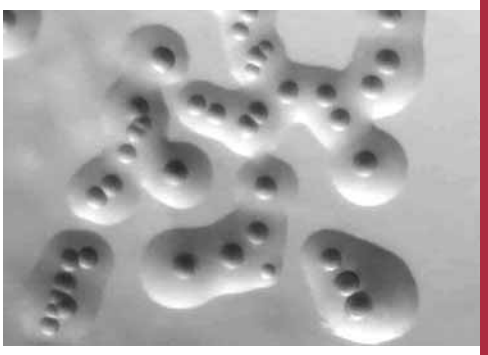
3 ISSN 1810-0732

FAO ANIMAL PRODUCTION AND HEALTH

proceedings

TOWARDS SUSTAINABLE CBPP CONTROL PROGRAMMES FOR AFRICA

FAO-OIE-AU/IBAR-IAEA Consultative Group on
Contagious Bovine Pleuropneumonia
Third meeting, Rome, 12-–14 November 2008

3
FAO ANIMAL PRODUCTION AND HEALTH
proceedings

TOWARDS SUSTAINABLE CBPP CONTROL PROGRAMMES FOR AFRICA

FAO-OIE-AU/IBAR-IAEA Consultative Group on
Contagious Bovine Pleuropneumonia
Third meeting, Rome 12–14 November 2003

FOOD AND AGRICULTURE ORGANIZATION OF THE UNITED NATIONS
Rome, 2004

FOREWORD

The Contagious Bovine Pleuropneumonia (CBPP) Consultative Group meeting is an officially recognized gathering of the relevant scientific community with recognized expertise and knowledge of CBPP. The meetings are the joint undertakings of the Food and Agriculture Organization (FAO) in collaboration with the *Office International des Epizooties* (OIE), African Union/Interafrican Bureau for Animal Resources (AU/IBAR) and the joint Division of the International Atomic Energy Agency (IAEA). Regular meetings of an *ad hoc* Expert Panel, as it was formerly known, were held in the sixties where experience with the performance of CBPP diagnostic techniques, various control measures and strategies, were exchanged and debated on. Recommendations that arose from these meetings were then disseminated across the world, especially to countries where the disease is of particular significance. Apparently, the last gathering of the Ad Hoc group was in 1970 when the eradication of CBPP from Australia was complete and the situation in Africa seemed to be under control.Dr Alain Provost chaired this meeting.

In recent years, there has been an upsurge in the incidence of CBPP in Africa to an alarming level reminiscent of that of the early 1960s. This situation prompted a similar meeting of an expert panel, now called the CBPP Consultative Group that met in Rome in October 1998, to update the current knowledge of CBPP in recognition of the fact that CBPP had become the major cattle disease in Africa. The second meeting of this group was held in October 2000 and it reported a worsening CBPP situation. The group considered scientific and technological advances and tools necessary to aid in the control of CBPP against the background of rural poverty and an increasing global demand for meat, milk and other animal products. Efforts were directed towards designing effective and realistic strategies that could lead to control of CBPP in Africa.

In 2003 the Meeting of CBPP Consultative Group involved field veterinarians, laboratory diagnosticians, researchers, policy makers and international partner institutions that drew its expertise from national diagnostic laboratories in African countries and internationally from reference laboratories and individuals. This report provides an account of the presentations made at the Third CBPP Consultative Group Meeting, the recommendations made, summaries of discussions and contributed reports from each presenter. Progress made with diagnostic tests that may aid surveillance and research advances that may be useful for epidemiology and vaccine development are reported in this proceedings.

CONTENTS

FAO-OIE-AU/IBAR-IAEA
Consultative Group Meeting on CBPP in Africa
Towards sustainable CBPP control programmes for Africa
(Rome, 12 – 14 November 2003)

Opening Address

Ms. F. Guerrieri,

Chief, Emergency Operations, TCEO

Mr. Chairman, Ladies and Gentlemen,

It is indeed an honour and pleasure to be asked by the Animal Health Service (AGAH) of Food and Agriculture Organization of the United Nations (FAO) to give the opening address at the Third Consultative Group Meeting on contagious bovine pleuropneumonia (CBPP). It is my fervent hope that this gathering of eminent scientists, field officers, laboratory diagnosticians and representatives of the international community will put their ideas together and provide practical solutions that address the theme chosen for this meeting; "*Towards sustainable CBPP control programmes for Africa".*

According to *Office International des épizooties* (OIE) reports, CBPP is essentially confined to Africa. The effect of the disease on beef, milk and crop production through the use of plough oxen is devastating with particular implications for food security and poverty levels within countries affected by CBPP. The onerous responsibilities of ensuring sustainable control strategies for the disease so as to support and improve the livelihoods of many in Africa who depend on cattle farming for sustenance, relies to a large extent on the outcome and follow up actions emanating from this meeting.

We in the Emergency Operations Service (TCEO) of FAO have responsibility for ensuring that projects and activities categorised as emergencies are designed to meet urgent and immediate needs arising from unexpected calamities including animal disease outbreaks, which affect or are expected to affect food and agricultural outputs of countries. The emergency interventions from TCEO are directed essentially towards resumed productivity or the containment for the decline in productivity. It is in this connection that the synergy between the activities of EMPRES – Livestock in progressive control of transboundary animal diseases and that of TCEO are to be viewed or assessed. Some of us are aware of the difficulties some countries face in convincing national treasuries to finance veterinary/livestock services in the absence of epidemic diseases such as CBPP. Secondly, we also recognize the difficulties for national veterinary services to convince governments to finance effective prevention and progressive control of livestock epidemic diseases against a backdrop of competing immediate needs of social, health, education and other agricultural systems on national treasuries.

Despite these recognitions, sustained livestock production and trade in livestock products are practically impossible in the presence of epidemic diseases such as CBPP. This particular disease as you all know, has contributed to great economic losses and thus to increased poverty levels in many parts of Africa. Those of us in FAO look to a deep analysis and critical assessment of various control options at this meeting. Your recommendations on how to sustain control strategies for CBPP that could lead to the reduction of poverty will be eagerly expected.

At this juncture, let me digress from the focus of my address to pay a brief tribute to a colleague who has served the FAO Animal Health Service for almost 12 years as Chief of that Service and is due to retire from FAO in two weeks time. He is – Dr. Yves Cheneau. This meeting being perhaps one of his last official international engagements in AGAH as a staff member, I take this opportunity to acknowledge his high sense of duty, dedication and professionalism and to wish him well in retirement.

Ladies and Gentlemen, the task before you is daunting especially given the tract record of CBPP control in Africa and the current situation of further spread of the disease especially in Southern Africa. However with the calibre of technical personnel present at this meeting, I am confident that innovative ideas on sustainable strategies for CBPP control will be forthcoming.

I declare the meeting duly open and wish you fruitful deliberations.

Thank you.

Posthumous Presentation of FAO Silver Medal

by

Dr Y. Cheneau, Chief-Animal Health Service, AGAH

to

ALAIN PROVOST

A Tribute from the Animal Health Service of the Food and Agriculture Organization (FAO) of the United Nations (UN)

This silver medal from the Food and Agriculture Organization (FAO) is presented posthumously to Dr Alain Provost, a distinguished international expert in tropical animal diseases.

Dr Alain Provost's research work on contagious bovine pleuropneumonia (CBPP) will forever remain part of the foundation of fundamental research in the control of CBPP from the world particularly, his scientific contributions in the development of CBPP vaccines.

Dr Provost has on many occasions served as FAO's International Expert on CBPP disease to many countries in Africa. Author of numerous publications on CBPP and other tropical diseases such as rinderpest, Rift valley fever, heartwater and others, he maintained very useful collaborative research relationships with his scientific colleagues and peers.

This medal is presented to Alain Provost in recognition of his distinguished services to FAO in particular and to humanity in general.

Principal Objectives of the Consultation and Expected Outputs

Dr. J. Lubroth,

Senior Officer, EMPRES

The Consultative Group Meeting for CBPP is unique in character in that it brings together field veterinarians, laboratory diagnosticians, researchers, policy makers and international partner institutions such as the AU-IBAR, OIE and the FAO/IAEA Joint Division. The meeting attempts to synthesise scientific (technical experience and ideas) coupled with practical field experience in the hope of coming to a common consensus on the way forward in the protection of cattle for the progressive control of contagious bovine pleuropneumonia (CBPP). That being the case, the responsibility on this group is tremendous considering the present epidemic situation of CBPP in Africa. The cross fertilization of ideas, technical exchanges, forceful interpretation of the way forward in CBPP control have at times led to sterile debates and arguments that have unfortunately, led to less than productive meeting outcomes. Today, CBPP has invaded parts of countries in Africa where the disease has not been reported for over half a century. We must, as a technical group, make a difference for the better by:

- Developing and suggesting novel workable strategies for the control of CBPP in the light of new technical information some of which will be presented at this meeting. Something new that is workable and takes cognisance of present day realities in economic trends in animal disease control are essential outputs expected from his meeting.

- Ensuring that, in the face of challenges to national veterinary services in down sizing, restructuring and dwindling resources, the capacities for early warning and early response to CBPP incursions are not lost. Concepts such as tackling control of transboundary animal diseases including CBPP at source should be thoroughly explored in defining disease control strategies for CBPP in the future.

- Ensuring that experience of what has worked or not worked in the past should form an important component for suggesting sustainable strategies for CBPP control.

- Promoting CBPP control on the basis of improving national cattle production and also in reducing the risk of spread of the disease to neighbouring countries based on critical analysis of particular risk factors responsible for the spread of the disease. The current outbreaks of CBPP in the Caprivi strip of Namibia where the disease was last reported in 1939, and in Eritrea are worrisome because of severe implications for potential spread of CBPP to Botswana, Zimbabwe and other SADC countries currently free from the disease.

- Finally, it is essential to re-enforce the need to work with cattle producing communities in finding effective mechanisms for CBPP disease containment because of implications for food security and improvements in livelihoods.

FAO-OIE-AU/IBAR-IAEA

Consultative Group Meeting on CBPP in Africa

Towards sustainable CBPP control programmes for Africa

(Rome, 12 – 14 November 2003)

Summary of Recommendations

CBPP Control Strategies

- Strategic control of CBPP should be progressive and based on impact assessments and cost benefit analyses done with appropriate methods including participatory techniques to cover regional, national and zonal levels.
- PANVAC and the production of CBPP vaccine should be internationally accredited.
- Research into antibiotic treatments, vaccines and their targeted delivery and diagnostic tools should continue.
- Pilot projects to assess the effectiveness of antibiotic treatments and elective vaccination should be conducted.
- CBPP should be a model for the improvement of veterinary services and public/private partnerships.
- Mathematical modelling should be used in CBPP research.
- Adequate funding should be available to control CBPP in sub-Saharan Africa.

Tools for CBPP Control – Vaccines

- PANVAC should assume a central role in the research and improvement of vaccine production, formulation and its proper reconstitution.
- AU/IBAR should financially empower PANVAC to enable compliance to OIE and other manufacturing principles for vaccine production and certification.
- Research into the improvement of vaccines should continue and include the possibility of differentiation between vaccinated and non-vaccinated animals.
- OIE and FAO should be sent a list of CBPP vaccine producers and performances as established by PANVAC.

Tools for CBPP Control – Use of Antibiotics and Diagnostic Tests

Daignostic Tests

- The prevalence of CBPP should be established by serological surveillance.
- Serological, clinical and pathological investigations should be performed to confirm the absence of CBPP.

- New outbreaks should be confirmed by the isolation and identification of *Mycoplasma mycoides* subspecies *mycoides* small colony variant (*Mmm*SC), because currently available serological tests are inadequate for individual diagnosis.
- Research must be conducted to establish the effects of antibiotic treatment and multiple vaccinations on CBPP.
- Robust penside tests must be developed including those based on the detection of CPS antigens.
- Standardised reagents and quality control sera should be introduced by FAO/IAEA into Africa for the CFT.
- Immunoblotting test should be considered during the critical phases of CBPP control programmes.
- Serological tests that differentiate vaccinated from non-vaccinated animals should be developed.
- The general quality assurance scheme for CBPP diagnostic reagents should be devised by FAO/IAEA.

Antibiotics

- Pilot trials: PACE and FAO should instigate pilot trials to assess the effectiveness in the field in Africa of antibiotics and chemotherapeutic agents against CBPP.
- Studies on Microbial Sensitivity: The VLA should be requested to carry out *in vitro* studies to establish the MIC of relevant antibiotics to African *Mmm* SC strains.
- Studies on the Safety and Impact of Antibiotics on the Consumer: Systems to monitor antibiotic residues in meat and recommendations for antibiotic use in animal productions systems should be followed.

Sustainability of FAO Technical Support for Contagious Bovine Pleuropneumonia Control

Dr. W. Amanfu,

Animal Health Officer (Bacterial and Zoonotic diseases), AGAH

Introduction

A precise definition of the term sustainability is difficult to obtain. The closest that seems to address the issue of sustainability and relates to FAO's strategic interventions to control animal diseases such as contagious bovine pleuropneumonia (CBPP) is that of Gro Harlem Brundtland in which she states "Sustainable development is development that meets the needs of the present without compromising the ability of future generations to meet their own needs. This definition contains two key concepts, namely:

- The concept of 'needs', in particular the essential needs of the world's poor to which overriding priority should be given; and,
- Limitations imposed by the state of technology and social organization on the environment's ability to meet present and future needs" (1).

Applying Brundtland's definition to the control of animal diseases, it could be argued that the greater burden of sustainability of FAO technical assistance projects in CBPP and other animal disease control lies in a partnership approach involving governments, the donor community and FAO in which the needs are addressed together with the application of the appropriate technology to solve animal disease outbreak problems.

CBPP has been a major cause of cattle mortality and production losses in many parts of Africa. Being an OIE List A* disease and with implications for rapid spread between herds and across international borders, CBPP has engaged the attention and resources of the FAO for many years in attempts to curb the spread of the disease and limit its devastating economic effects, especially at the village or community level. The institution of FAO Emergency Prevention System for Plant Pests and Diseases (EMPRES) programme in 1994 by its Director General, Jacques Diouf, provided additional impetus for the progressive control of CBPP among six other priority animal diseases. The main objective of the animal diseases component of EMPRES is to assist member nations of the FAO in the progressive control of the major epidemic diseases of livestock through facilitating effective implementation of national and regional control strategies (early warning and early reaction systems and enabling research) within an environment of international co-ordination and cooperation. Since its inception, EMPRES precepts have been consistently applied in attempts to curb the outbreaks of the disease in Africa. This paper assesses the current epidemiological situation of CBPP in countries that have benefited from FAO technical assistance in the control of the disease and to draw lessons from the current situation of the disease in those countries that could be crucial in the evolution of sustainable control strategies for the future.

*The OIE plans to change the animal disease list system to a single list in the near future.

General Epidemiological Observations

CBPP is caused by *Mycoplasma mycoides* subspecies *mycoides* SC (small colony, variant) (*Mmm* SC). The disease is present in West, Central, East and parts of Southern Africa but not North Africa (4). From a historical perspective, CBPP was a disease of Europe and Asia. A comprehensive historical account of the spread of CBPP in view of the economic significance of the disease in Europe and Africa in the 19th century has been provided by Windsor (5). With the near eradication of rinderpest in Africa (except the Somali ecosystem), CBPP has become the most significant epidemic disease of cattle in Africa with 22 countries reporting outbreaks of the disease in 2003 (4). The disease was present in the Iberian Peninsula [(Portugal-1999, declared free in 05/2003 at the 70th session of the OIE, Spain-1994) and Italy (1993)] during the past decade. The presence of the disease in Asia has not been clearly defined although Myanmar (1995) and other countries Bangladesh (1997), China (1996), Qatar (1997), Kuwait (1991) reported the disease for the last time in the years indicated against their names (4). India, as recently as October 2003 (4), declared herself provisionally free from CBPP (with vaccination). The disease has never been reported in South America.

CBPP is spread by direct contact between infected and susceptible cattle. Introduction of the disease into susceptible cattle populations results in widespread mortality. In the chronic stage, the disease is insidious in nature with variable clinical course that makes epidemiological study of CBPP based on clinical manifestation alone, difficult. Molecular epidemiological studies conducted by Lorenzon *et al*. (3) on 44 strains of *Mmm*SC obtained from wide geographical sources, demonstrated three distinct lineages of *Mmm*SC circulating in Africa. This tool, termed multilocus sequencing analysis, could be useful in distinguishing between different types of *Mmm* SC, especially in countries carrying out control/eradication programmes and requiring tools to trace the origin of remaining or re-emerging CBPP foci.

FAO's Technical Cooperation Programme

The Technical Cooperation Programme (TCP) of FAO was launched in 1976 as an essential tool to make FAO's specialized competence more readily available to member countries for the solution of their most pressing development problems in the agriculture, fisheries and forestry sectors. Through TCPs, FAO allocates limited, but identifiable resources to fulfil one of its key constitutional functions, i.e. to provide such technical assistance as governments may request. It is an integral part of the Organization's Regular Programme, financed from its assessed budget. In particular, TCP is the instrument that enables FAO to respond rapidly to urgent needs for technical and emergency assistance in member countries and to contribute to their capacity building. The main features of TCP are its extemporised and urgent character; its flexibility in responding to new technical issues and problems, clear focus, limited project intervention with short duration, low costs, and practical orientation, and as a catalytic role for in-country or region uptake. By design and in practice, TCP meets unforeseen needs, fills critical gaps, complements other forms of assistance and promotes resource availability for technical cooperation in the above fields. Requests for technical assistance under the programme may be presented by governments of member countries that qualify for development assistance under the UN system and by intergovernmental organizations of which such countries are members, and are recognized as such by the UN system and FAO. The EMPRES programme of FAO's Animal Health Service has been active in assisting countries to meet animal disease emergencies such as outbreaks of CBPP and other transboundary animal diseases through the instrumentality of TCPs.

With reference to request by member countries for assistance in the control of CBPP, the following CBPP specific projects have been undertaken from 1990-2003:

i) TCP/RAF/6611, Regional Project (East Africa) Prevention of transboundary spread of CBPP from Southern Tanzania to neighbouring countries. Duration 24 months 1996–1998. FAO Contribution US$ 381,743;
ii) TCP/RAF/0172, Regional Project (West Africa) Coordinated programme to strengthen capacity for epidemio-surveillance of CBPP. Duration 24 months 2001–2003. FAO Contribution US$ 387,000;
iii) TCP/RAF/2809, Regional Project (SADC) Control of FMD and other transboundary animal diseases in Southern Africa. Duration 18 months. FAO Contribution US$ 351,000;
iv) TCP/BOT/4452E, Surveillance for the control of CBPP. Duration 12 Months extended for further 12 months. 1995–1997. FAO Contribution: US$180,000;
v) TCP/BOT/6712E, (Phase II). Duration 12 Months. FAO Contribution US$ 79,000;
vi) TCP/BDI/8821, *Campagne de prophylaxie contre la péripneumonie contagieuse bovine et surveillance epidemiologique de la peste bovine*. Duration 24 months; 1998–2000. FAO contribution, US$310,000;
vii) TCP/ANG/8992, *Surveillance et Contrôle de la péripneumonie contagieuse et d'autres maladies transfrontalières*. Duration 24 Months; 1999–2001. Total FAO Contribution US$ 291,190;
viii) TCP/URT/0058, Contagious bovine pleuropneumonia Emergency control. 1990–1991. Duration 12 months. Contribution US$ 275,000;
ix) TCP/MAU/6611 *Renforcement des capacités de diagnostic et de surveillance épidémiologique de la péripneumonie contagieuse bovine.* Duration 24 Months 1996–1998. FAO Contribution US$ 198,500;
x) TCP/MLW/4552, Protection from transboundary spread of CBPP. 1995–1997. Duration 24 months. FAO Contribution, US$ 191,200;
xi) TCP/ZAM/6714, Emergency control of CBPP in Western Zambia. Duration 24 Months, 1997–1999. FAO Contribution US$370,000;
xii) TCP/ZAM/0169, Emergency control of transboundary animal diseases (CBPP and ASF). Duration 12 Months 2002-On-going. FAO Contribution US$297,000 UNHCR Contribution-US$68,000;
xiii) TCP/SUD/2908, Surveillance for CBPP and CCPP in the Sudan (Advance Allocation). Duration 3 months. July–September, 2003. FAO Contribution US$11,000.

Although the cumulative financial outlay for all these regional and country specific CBPP control programmes appear miniscule (US$ 3,322,633) in relation to the magnitude of the problem, financial provisions made in these projects and activities envisaged for project implementation, fulfilled critical needs and gaps in the overall strategy for the control of CBPP. The principal elements of prime consideration in the control of CBPP through the provisions of TCPs are the prime pillars of EMPRES that is; early warning, early reaction, contingency planning, enabling research and coordination. These elements are incorporated in the design of projects to ensure effective resolution of outbreaks.

Surveillance

Provision of laboratory equipment, diagnostic reagents, laboratory media and other consumables help to augment technical capacity to diagnose the disease and provide tentative

confirmatory evidence of CBPP outbreaks before final confirmation by a FAO/OIE designated reference laboratory. Such activity serves as a basis for early warning/early reaction and the drawing up of contingency plans for effective CBPP control. The supply of GIS equipment and the use of the FAO-developed software system and database, TAD*info,* to assist in geo-referencing of outbreaks and provide a basis for spatial and temporal analysis of outbreak trends for the adoption of counter epizootic measures. Disease recognition through the provision of manuals, publications, CD-ROMs, and videos, are integral parts of awareness creation and critical components of disease surveillance systems and disease management.

Capacity building

Most countries have a complement of competent trained specialists in the basic fields of CBPP control, laboratory diagnosis, data management, extension and in some cases, vaccine production. This provides a sound platform on which to build. In addition to assistance for establishing emergency preparedness in the control of CBPP, there is primarily, a need for technical assistance to transfer laboratory and surveillance technology – much of which has been developed by FAO and the Joint Division FAO/IAEA in Vienna, to member countries affected by the disease. Interaction and technical support from consultants, in country training workshops, study tours, development of country specific or regional strategies for the control of CBPP by FAO staff, consultants (international and partnership programmes) have been instrumental in capacity building for effective control of CBPP. Livestock dominates the livelihood activities and strategies of pastoralists. Therefore within the pastoral communities, capacity building in animal disease control is recognized as a key component in the development of overall strategy to control animal diseases such as CBPP. Such recognition is designed into TCPs to ensure long-term sustainability of control strategies

Summary of status of FAO projects related to CBPP control

Two regional projects for **East** and **West Africa** have been closed. Key elements of these projects in CBPP control were regional cooperation and coordination of control strategies for the disease, regional referral laboratories strengthened and stakeholder awareness in disease recognition conducted. Although CBPP is prevalent in the East African region, **Malawi** has been able to maintain its CBPP-free status due to improved surveillance for the disease especially along its northern border with Tanzania through the provisions of TCP/RAF/6611. The regional project for CBPP in **West Africa** (TCP/RAF/0172**)** strengthened laboratories through the supply of inputs. CBPP disease reporting from the region has improved and there is better collaboration in disease information sharing and stakeholder recognition of the disease. The sustainability of these completed regional projects will depend among other things, on continued cooperation and transparency in animal disease information sharing. The regional project for SADC countries is still on going. One of the major outputs of the project has been a workshop with Chief Veterinary Officers of SADC countries in Pretoria, South Africa at which regional strategies to control CBPP in affected SADC member countries and strategies to prevent the entry of disease into free areas and thereby jeopardize the livestock and allied industries were drawn up. This project proposal has now been submitted for possible donor funding. The TCP project in **Botswana** approved in 1995 was very instrumental in launching a coordinated national surveillance system for CBPP in Botswana which served as the technical basis for a decision to slaughter 320, 000 cattle to rid the country of the disease. Through the establishment of laboratory diagnostic capability, staff training and government commitment to the support of the veterinary sector, Botswana has sustained the key elements of this project

and has obtained CBPP disease free status from the OIE. The country still carries out bi-annual serological and clinical surveillance during their foot-and-mouth disease vaccination campaigns to ensure continued freedom from CBPP. The assistance to **Angola** was primarily on improvements in CBPP surveillance capabilities. Since the project ended in 2001, there has been limited follow up activities. With the end of the civil war, it is expected that CBPP surveillance activities will be stepped up to serve as a basis for targeted control of the disease and minimise the risk of transmission of CBPP to neighbouring Zambia. **Burundi** benefited from the improvements in disease surveillance and provision of vaccines for the control of CBPP. Since the project ended in 2000, there has been little follow up action. **Mauritania** benefited from improvements in laboratory capabilities in the diagnosis of the disease. The country also participated in the regional project on strengthening the epidemio-surveillance of CBPP in West Africa. **Zambia** has benefited from two TCPs back to back on the control of CBPP in the western province. The most recent TCP is in conjunction with additional support from the UNHCR in constructing holding pens for refugee cattle and vaccinating them against anthrax, haemorrhagic septicaemia and CBPP. In addition, the FAO TCP facilitated the improvement in diagnostic capabilities for CBPP. Unfortunately, due to several logistic and technical factors, the disease could not be contained in the Western Province to the extent that it in 2002, the disease was detected in North-western Province. Serological surveillance capabilities established by the project facilitated the testing of cattle in North-western province by the complement fixation test (CFT) and the competitive enzyme linked immunosorbent assay (cELISA) which showed that the disease was more widespread than thought. The disease was detected again in February 2003 in Kashima - Mufumbwe district close to the copper belt province. CBPP appears widespread in Zambia and threatens her immediate neighbours of Namibia, Zimbabwe and Botswana. The current outbreak of CBPP in East Caprivi Province of **Namibia** is thought to have originated from south western Zambia. Government commitment of resources and streamlining of the veterinary services in Zambia, appear crucial to the containment of the disease and reduction of risk of transmission of the disease to neighbouring countries that are free of the disease.

Sustainability issues

Analysis of issues discussed and entry points for technical assistance by FAO clearly show that there is a wide range in the sustainable components of various programmes and project activities. What are clearly evident as missing are long term strategic plans and objectives that seek to address the control of CBPP at the end of FAO technical assistance. Technical capacity and sustainability in general, are affected by:

- Staff retrenchment as a consequence of adoption and implementation of structural adjustment policies. The loss of very capable and experienced workforce at this stage is an important component of the loss of disease control initiatives in particular localities where epidemic diseases such as CBPP are predominant. Loss of local animal disease prevalence history in rural areas where record keeping is not a specific feature of activities is a critical factor in sustainability;

- Poor resource capacity to allocate financial, logistic and human resources to the development and maintenance of animal disease control infrastructure, is a key factor in the deterioration of veterinary services and with it, outbreaks of epidemic diseases such as CBPP. The contribution of livestock to the sustenance of rural livelihoods to a very large extent has to be fully appreciated by governments so as to commit the necessary resources for its long term sustenance;

- Cost recovery has affected the level of patronage of CBPP vaccination campaigns;

- Experienced field and laboratory staff that are often the frontline staff for CBPP and other livestock epidemic disease control suffer the ravaging effects of the HIV/AIDS pandemic. The effects of the HIV/AIDS pandemic for the livestock sector especially in animal health and the inter-linkages underlying these effects remain poorly understood. However results of a Namibian study (2) provide additional information on the impact of HIV/AIDS on the livestock sector.

Summary

Technical assistance from FAO apart from the attributes previously described is meant to be catalytic in eliciting synergy with member countries and the donor community so that the needs of the present are sustained for the future in long term strategic plans for animal disease control. Therefore, one of the major outputs expected from this consultation is to evolve mechanisms that couple technical assistance to long-term strategic objectives and goals geared towards the control of CBPP in Africa.

References

1. Brundtland GH (1987): Our Common Future; Report of the World Commission on Environment and Development. Oxford University Press.

2 FAO 2000:Http://www.fao.org/sd/WPdirect/WPan0046.htm

3 Lorenzon, S., Arzul, I., Peyraud, A., Hendrikx, F. & Thiaucourt, F., 2003. Molecular epidemiology of contagious bovine pleuropneumonia by multilocus sequence analysis of *Mycoplasma mycoides* subspecies *mycoides* biotype SC strains. Veterinary Microbiology, 93, 319-333.

4. Office International des Epizooties (www.oie.int).

5. Windsor, R.S., 2000: The eradication of contagious bovine pleuropneumonia from South western Africa: A plan for action. Annals of the New York Academy of Science, 916, 326-332.

Summaries of Presentations and Discussions

CBPP Control Strategies

Summary

According to the assessment made by the Epidemiological Unit of PACE, CBPP is endemic in many parts of Africa. However, the lack of data made this assessment and accurate zoning of disease distribution difficult. There was a pressing need for prevalence data to confirm zoning. The reasons for the persistence of CBPP were attributed to animal movement within and between countries. Other reasons were, the absence of adequate diagnostic tests, the lack of use of diagnostic tests because of diminished financial support and a downturn in the use and quality of CBPP vaccine. Therefore, efforts to build diagnostic capacity were required. A regional approach that takes into account differences in epidemiological situations, the provision of training, and veterinary surveillance for the disease at borders was proposed. A draft project proposal was summarised where surveillance, quarantine and serological screening were advocated for free zones. These together with ring vaccination with branding of vaccinated cattle were promoted for newly affected areas. The use of harmonised mass vaccination and antibiotic treatment for 5 years were proposed for enzootic zones. All efforts should be made to minimize the likelihood of the reintroduction of CBPP into free areas by the establishment of buffer and surveillance zones.

The recent resurgence of CBPP in Africa was attributed to the lack of funding. Animal losses in Africa due to this disease were estimated to be about US$ 2 billion. The threats to CBPP-free countries from affected areas within the SADC members were emphasised. The effects of CBPP infections were felt at several levels i.e. at household, national and regional levels but losses were likely to affect food sources and draft power leading to significant hardships at the household level. In Botswana, this sector suffered 80% of the losses and therefore efforts to keep this area free of incursions of CBPP were strongly reiterated. The endemic epidemiological clusters of CBPP in Angola and Tanzania were noted. Illegal cattle movement, ineffective vaccination campaigns, and the lack of contingency plans were identified as the immediate challenges to effective CBPP control. Plans for ‘the way forward’ included the development of a phased strategy where emergency and recovery phases that were designed for the containment of disease were explained. A 16-year guideline/plan for the progression from control of the disease from primary endemic foci infection to freedom from disease was discussed.

A participatory modelling study that was based in Northern Tanzania, and Sudan, was conducted by PACE/CAPE to gather information about the dynamics of cattle movement within the local communities. The complex social interactions that involved animal movement and the transmission of CBPP in a herd were modelled by using field data, and information available in the literature, respectively. Simulations from these models predicted that CBPP could persist indefinitely in small, interlinked herds of 50 head, or in single herds of 300 head. These data supported the requirement for quarantine to interrupt the transmission cycle. However, this was deemed to be unrealistic in pastoral communities. Simulation of mass vaccination showed control but not elimination of disease and thus would not achieve eradication of CBPP. Elective vaccination was proposed because the model predicted a short-term benefit to the owner, but liberalisation in the availability of the vaccine would be required in this case. Effective treatment, according to the model, was of more benefit than vaccination because it reduced persistence of

disease in herds. Near eradication was predicted by combined programmes of effective vaccination and treatment.

A longitudinal serological study conducted in the Ethiopian highlands was used in mathematical model work on the spread of CBPP disease. Of the 71 herds that were followed for about one and a half years, 35 were infected with CBPP. Fifteen of them were classified as newly infected and used in a serological and clinical incidence study. Cumulative risks of seroconversion over 8 and 16 months were calculated to be 26 % and 34 %. In these herds, the average serological, clinical, and mortality incidences were 34%, 39% and 13%. These were lower than those reported in the literature (70%, 30-70%, 10-80%, respectively), but there were no obvious explanation for this finding. They could have been *Mmm*SC strain or cattle breed related..Although 50% of the herds were treated with single injections of oxytetracycline, no effect from this was seen or could be shown statistically. The possibilities of underdosage and reduced antibiotic quality were proposed for these observations. Further studies were required to resolve these issues. Two disease transmission models were proposed, one that included the possibilities of interactions from animals with sequestra and another, simpler one that excluded these. Simulations from these provided estimations of transmissibility when periods of susceptibility or latency were varied, but many more simulations were necessary before firm conclusions could be made. More data were necessary to confirm mathematical situations e.g. the transmission from chronic carriers, and effects of antibiotic treatment.

Discussion

In drawing up strategic control policies based on epidemiological data, or contingency plans, mathematical models for transmission and disease, situation based on experimental or field data, the common fact and a stumbling block was the lack of reliable data. Considering the method of livestock production in Africa i.e. mainly transhumance, it was difficult to set up surveillance to gather epidemiological data. This reality and the fact that veterinary structures have been disrupted due to budgetary constraints made the setting and implementation of control strategies difficult.

Conventional and new approaches for CBPP control were discussed. It was obvious that movement control at the level that was practiced in the 60s was not possible today, and the control of CBPP may be more costly than the losses from the disease itself. Longitudinal studies and computational modelling, could be carried out in other countries and would account for the different livestock situations in different countries. These required accurate data to be useful. The observed seasonality in the incidence of CBPP in come herds in Kenya herds was surprising; owners described this seasonality which may have been due to mixing or weather patterns, but there was no supportive data.

The effectiveness of antibiotic therapy was questioned because no clear basis for its use was demonstrated. There were conflicting cases made for the consequences for the use of any antibiotics, but the potential impact was presented and the urgent need for more field research was highlighted. Antibiotics that are bacteriostatic cannot eradicate the disease but they may have a significant effect in the reduction of infection or the transmission of infection. Thus mathematical models including these factors could be used to test the potential benefits of therapy. Absolute quarantine as a way of transmission control was possible but not practical and models would be able to predict the epidemiological outcomes as the infection rates varied.

Heated debate ensued on proposals for the buse of elective vaccination. As CBPP is a notifiable disease and its prophylactic measures are obligatory, caution was expressed in the use of elective vaccination as a CBPP disease control option. However, at the moment prophylaxis was not regularly practiced so if the owner's choice was to choose a programme that benefited him then control option could be considered.

TOOLS FOR CBPP CONTROL – VACCINES

Summary

Toxins have never been described for *Mmm* SC nor have virulence factors, but potentially galactan, variable surface proteins, lipoproteins, transporter proteins and adhesions may modify virulence. These classes of proteins were major antigens and many are located on the surface of the organism and thus suitable targets for molecular manipulation towards the production of vaccine. The lipoprotein LppQ that was already used in a diagnostic assay exacerbated the disease when inoculated into cattle despite the fact that it is present in current vaccines. ABC transporters and associated systems for the export and import of molecules could influence virulence such as the glycerol uptake and metabolic system. In *Mmm* SC, especially in African/Australian strains, this system is capable of producing relatively large amounts of peroxidase, which, given the close cell to host cell association of mycoplasmas, results in the induction of apoptosis of the host cell. Several generic targets for vaccines and methods for their production were considered and discussed.

Two new preparations of dead vaccines were tested for their protective ability. One was saponised, whole-cell *Mmm*SC, and the other was purified LppQ ISCOM.. They were inoculated separately into cattle that were subsequently challenged with a local field strain of *Mmm*SC. Both preparations did not elicit specific antibody responses. After challenge, it was observed that there was disease severity as judged by the extent of lung lesions compared to experimentally infected controls that had not been vaccinated. The animals appeared to have been sensitised.

Improved vaccines could probably make the major contribution to CBPP control. Towards this goal, the immune responses of two vaccine preparations, T_1 44 and a saponised virulent field strain, were studied for their ability to elicit specific antibody and lymphoproliferative responses. Preliminary results showed that antibodies were elicited by both preparations and a single inoculation with the saponised strain produced responses similar to a booster with T_1 44. Various antigens produced varying degrees of proliferative responses from lymphocytes. Cell populations remained similar throughout the course of vaccination.

Does T_1 44 revert to virulence? Every now and then but with unpredictable frequency, some T_1 44 vaccinated cattle develop Wilhelm's reactions at the site of inoculation. These reactions are not caused by subsequent vaccinations with T_1 44 vaccines. The reactions range in severity and can be cured with antibiotic treatment. A study to assess if this phenomenon was due to differences in these *Mmm*SC organisms was undertaken. The vaccine T_1 44, a local field strain of *Mmm*SC and an *Mmm*SC organism named T_1 B, isolated from a vaccination reaction site, were re-inoculated into cattle that were monitored clinically. T_1 44 did not cause any local reactions, but field strains and T_1 B caused large local reactions and fever. T_1 B behaved like the local field strain. *Mmm*SC was also re-isolated from these lesions. Protein profiles of these organisms were compared using SDS-PAGE. Changes in the high molecular weight range between T_1 44 and T_1 B were observed. The significance of these differences was not known at the moment.

The apparent failure of the T1 44 vaccine in Botswana could have been due to incorrect vaccine seed strain, insufficient vaccine titre, or underdosage. Studies showed that the strain was correct, and further experiments were undertaken to study the dose and its protective effect. There were no significant differences between doses from 10^7 to 10^9 organisms; mortality rate in controls was about 30% compared to about 6% in vaccinated animals. The severity of lesions was scored. A reduction in that score in the vaccinated animals showed clear protection. Variations in individual animals were observed. T_1 SR was also tested and although statistical differences could not be shown, T1 44 appeared to be more protective according to the lesion scores. It was suggested that high titres were necessary to prolong the shelf life of vaccine.

With the current CBPP situation in Africa, the two options for CBPP control actions are either “accept it” i.e. live with the disease or “control/eradicate it”. To live with CBPP is politically unacceptable because the use of antibiotics would increase while production and income would decrease. To eradicate CBPP would involve losses through stamping out and movement restrictions. The only realistic option for Africa is vaccination and the two options are to develop new vaccines or to use existing ones. The development of new vaccines would be costly and require many years of research efforts.. Efficacy, production and political issues would have to be resolved before meaningful progress could be made. .The biggest issue however, is that of funding. Who would fund vaccine development/research? Therefore, the way forward is to improve existing vaccines by increasing their thermal stability, viability and immunogenicity. This is certainly possible because vaccines used in Australia were stable at 37°C. Improvements may be made by i) increasing the buffering capacity of media used in vaccine production by the inclusion of HEPES, ii) discontinuing the use of Magnesium Sulphate ($MgSO_4$) in the reconstitution buffer because it causes a pH change leading to decrease in viability of mycoplasma organisms and iii) use phosphate buffered saline as diluent. Inclusion of a pH indicator could help monitor shift in pH after vaccine reconstitution. These simple technical changes with proper funding for production and quality control, could improve the efficacy of current vaccines significantly.

Discussion

Current research must not to be abandoned because it could also lead to better vaccines and diagnostic tools Applied research to improve the stability of T1 44 vaccine were urgently needed. There are good opportunities to improve vaccine products and these may be made by simple modifications in the formulation including the reconstitution buffer, attenuation of strains by genetic modification and minimization of adverse reactions.

FAO has commissioned work using xerovac vaccine technology, in which trehalose is used to improve vaccine stability at higher temperatures, that may not require the need for cold chain storage.This work should be published and the work taken further. The simple but crucial observation of adverse effects of reconstitution with buffer containing MgSO4 sparked much discussion. Perhaps it would be relatively simple to change the buffer, but caution and further work to check the viability of organisms was recommended before these methods were standardized. This effect of $MgSO_4$ buffer was questioned. It was suggested that this diluent was added to measles and RP vaccine and afforded some thermal protection. However, the effect of MgSO4 on CBPP vaccine would also have to be investigated. Moreover, this solution was widely used as reconstitution buffer for other vaccines without adverse effects. It was also recommended because in the past almost anything was used and field ‘short cuts’ were

common. It was also argued that $MgSO_4$ did not inactivate the vaccine but drop in pH caused a rapid decrease in the viability of the organisms. Perhaps viability was not a problem for other vaccines used. Therefore, if the vaccine formulation was better buffered e.g. with HEPES, then the pH would be stable enough not to cause the loss in viability. A different reconstitution buffer such as phosphate buffered saline would also overcome this problem. A pH indicator in the buffer could also help to verify the correct pH after reconstitution. Such information suggesting practical changes could be disseminated very quickly and manufacturers could make the necessary adjustments to production equally fast, but pilot studies were required to show the effects of HEPES and $MgSO_4$ in field conditions of Africa. . In fact many of these ideas would require experimental validations and changes in the standard methods of production of existing vaccine would require retesting for efficacy etc. No funds are yet available for this activity from international donors.

Methods to improve vaccines by genetic modifications were also available, but the genes that lead to attenuation are not always virulence genes and may not be those essential for eliciting Wilhems reaction.

The apparent reversion of T_1 44 to T_1 B that consistently caused tissue reactions at the site of injection prompted much debate. How stable was this reversion? The reaction happened after the 2nd passage; T_1 B was still virulent after 2 *in vitro* passages. The pahogenicity of T_1 B was not known. One criticism was that T_1 B was not purified and so the inoculum may have contained wild type strains of MmmSC and therefore Koch's postulates were not fulfilled for this isolate. In fact this was a safety study and not a virulence study and only one batch of vaccine was used. Why was reversion occurring in animals but not *in vitro* e.g. during vaccine production? OIE guidelines stipulate that vaccine strains have to undergo two passages from the grandparent stock, thus in culture there is no pressure for much change. In animals, there is selection of more virulent strains at the expense of less virulent strains..

In the field, tissue reactions after vaccination with T_1 44 were seen in Kenya but none in Namibia, Cameroon and Chad. The unpredictable nature of the incidence of reactions could not be explained.

Assuming that vaccines could be improved, according to some models CBPP cannot be eradicated with vaccination alone, so even when a country wanted to eradicate CBPP, it could not do so.

TOOLS FOR CBPP CONTROL – USE OF ANTIBIOTICS AND DIAGNOSTIC TESTS

Summary

An optimistic view of the research trends driven by technological advances in molecular biology e.g. higher throughput capacity of sequencing was given. In fact the genome of the type strain of *Mmm*SC has been sequenced, but its origin and virulence are doubtful. No obvious virulence factors have been identified. The comparison of attenuated and virulent strains in terms of the production of protein i.e. proteomics, may establish virulence factors. The expected benefits from these types of studies on pathogen/host relationships and immuno-pathogenesis are, better vaccines and diagnostic tests. Other important areas of research were the description of transmission factors, usefulness of antibiotics, types and appropriateness of

surveillance systems and control strategies, and computational models (because there are no animal models for simulating CBPP disease).

Antibiotics are officially forbidden for use in CBPP but nevertheless still used often in the field. The *in vitro* activity of some of these is known but little information exists on their *in vivo* activities on *Mmm* SC. The activity of tetracycline that is used most often was studied in the field. Preliminary results showed that it reduced inflammation at the inoculation site but did not prevent infection. It reduced the severity of lung lesions but did not prevent them, and the pathogen was able to persist in the host. In the field where the quality and dosage of the antibiotic may not be optimal, these effects may not be sufficient for effective treatment of CBPP disease. Therefore, tetracycline has no place in eradication campaigns but may be of some benefit together with vaccination campaigns e.g. in the control of post-vaccination reactions.

The results of an FAO/IAEA Co-ordinated Research Project (CRP) on the "Monitoring of contagious bovine pleuropneumonia (CBPP) in Africa using enzyme immunoassays" showed that the complement fixation test (CFT) and a competitive ELISA for the detection of antibodies to *Mmm* SC were adequate tools for the monitoring and surveillance of CBPP. Although none of the validated diagnostic test was sufficient on its own, estimates of the sensitivity and specificity will allow the development of testing strategies which are suitable for the surveillance of CBPP and in the text detailed recommendations for a surveillance and testing strategy for different epidemiological situations are discussed. The inclusion of internal quality controls in the cELISA showed a high level of repeatability and reproducibility of the test which will ensure that test results produced by the laboratories are reliable and comparable. During the CRP, the CFT and the competitive ELISA were introduced into 11 African countries.

Portugal has successfully eradicated CBPP since its reintroduction in 1985. Strategies that led to a declaration of freedom from CBPP were as follows: accurate zonation, movement control, yearly serological surveillance from 1985 to 1994 that was increased to biannual testing between 1995 to 1997, abattoir surveillance and prompt follow up, culling of all serologically positive animals and eventual stamping out. During the first period, these measures firstly mapped the extent of the disease that was mainly in the north of the country and reduced the incidence of CBPP within these regions such that re-zonation encompassing smaller areas was feasible. The second period saw the further shrinking of these regions and a dramatic decrease in incidence. In this situation the inadequacies of CFT were unacceptable and a new confirmatory test, the immunoblotting test (IBT) was introduced to resolve the false positive results seen with the CFT. Since 1998 the CFT and IBT have been preformed serially on all sera for CBPP surveillance ensuring that an accurate diagnosis and assurance of freedom from CBPP was the prime target.

The impact of CBPP was assessed using participatory epidemiology techniques. Not only could these methods assess the relative incidences of diseases within the community, but they could also provide useful information on their importance to the owners in terms of lost production and real wealth in the absence of validated numerical data. These techniques could provide comparative impact assessments, had a proportional approach, random sampling was possible, could be standardized for valid comparisons with other populations and results could always be checked by conventional methods. Unlike conventional epidemiology that is commodity based and thus is an outsiders view, participatory epidemiology provided the insiders view that included private and sensitive information not accessible otherwise. Data gathered in Ethiopia were presented on the impact of several diseases including CBPP on cattle production.

Often, information on CBPP for research or teaching purposes is not readily available especially in many African countries. AVIS (Advanced Veterinary Information Systems) in partnership with TELOS-Aleff Ltd, the Institute of Animal Health (IAH), UK, FAO/OIE Collaborating Centre for CBPP,and the FAO have developed a web-based information system that strives to rectify this. The modular nature of the system and its user friendly interface and accurate information, offered by experts in the field were demonstrated.

CBPP is the second most important disease targeted for intervention within the PACE programme, but its inclusion in this list was questioned because of the lack of supportive data. The primary objective of PACE was to persuade regional integrated policies for surveillance to accumulate this data and control activities especially in endemic regions. To this end, several meetings, consultancies and draft policy documentation were carried out from 2001 to 2003. It was evident, that there were insufficient resources within PACE countries for the eradication of CBPP, estimated to cost about €300-450 million and mass vaccination needed extensive political support. Other factors that were deterrents to CBPP control were poor vaccine quality, the lack of an *in vitro* test for the differentiation of vaccinated from none vaccinated animals and the deterioration of veterinary services in some countries. The impact of CBPP was difficult to estimate, as the observed and reported mortality and morbidity data alone were not significant. The use of participatory epidemiology afforded some understanding of disease dynamics. Alternative control strategies included antibiotic therapies (although the choice of therapeutic agent was not clear), elective vaccination where the private sector (owner) decided the course of action. These measures would require the liberalization of the availability of CBPP vaccines, acceptance of antibiotic therapy, training of farmers and the acceptance of this concept by veterinary services.

Discussion

Discussions on the choice and use of antibiotics questioned the use of tetracycline. Tetracycline was chosen for these studies because it was the most common antibiotic used in the field by farmers. Although it did not prevent infection and it is basically bacteriostatic, it may have a place in therapy because it may provide time for the animal to develop immunity.

The activity of other antibiotics such as tylosin was questioned. It was used in southern Sudan and could be effective. Some *in vitro* work was done but it was impossible to extend this to the field. Broad *in vitro* studies needed to be done followed by focused field work. Research was also required on the likelihood of antibiotic resistance. Some work has been done in Muguga and there was some evidence of resistance to tetracycline in *Mmm* SC. Research was necessary to look for the type of resistance mechanism involved and if the resistance is transferable. Antibiotics that do not sterilize but may stop symptoms could produce animals that posed further threats by transmitting the disease thus confounding the issue. The influence of antibiotics on diagnosis was not known. However, the demand for treatment was high and farmers already treat animals, so the need for a good drug regimen was important. EU regulations for exporters of meat from Africa have to critically consider the issue of antibiotic residues.

.

COUNTRY SPECIFIC CONTROL STRATEGIES

Summary

In the late 60s there were great expectations for the control of CBPP in West Africa In the late 80s during the PARC project with mass vaccinationagainst rinderpest and CBPP, realtively few outbreaks of CBPP were encountered. The mid 90s saw a resurgence of CBPP prompting emergency activation of national and international programmes. Cattle production systems in these areas follow extensive pastoral nomadism and the spread of CBPP was assisted by uncontrolled transhumance across borders. A review of the current situation indicated that CBPP was widespread in West Africa and parts of Central Africa but the true picture of disease distribution was difficult to delineate because of imprecision in surveillance and reporting data. Guinea and Senegal have excellent surveillance systems. In some regions of Africa, laboratory capabilities were not equal or similar between countries and not balanced between peripheral and central laboratories. A review of the current control strategies showed inconsistencies in surveillance, notification and vaccination programmes between countries of the same region. Guinea and Nigeria currently have strong control measures for CBPP although the rate of vaccination decreased from 1999-2001. A phased control strategy was proposed, where building and refining epidemiological data collection, infrastructural dvelopment,, community involvement inj disease search and data collection; reduction of national risk by limitation of re-entry and uncontrolled movement of infected cattle; institutionalised surveillance, private and public sector involvement in CBPP disease control efforts; and regionally co-ordinated efforts, were defined. Strong political will and high commitment and tight co-ordination between sub-regions were identified as most important and critical factors in the success of this strategy for the control and eradication of CBPP in West and Central Africa.

Of the 16 million cattle in Nigeria, 90% were nomadically reared and 10% were intensively farmed. Transhumance is very important and CBPP in this population was most important. It caused direct and indirect losses and secondary social consequences. After virtual eradication in 1965, the disease has made a steady come-back due to civil strife or changes in socio-economic situations. There is considerable north to south movement of cattle in Noigeria and between 1995 to 2001, outbreaks increased from 8 to 31. A new policy and strategy for control were introduced comprising containment and control phases that included proper zoning, test and slaughter and vaccination with the aim of reducing CBPP incidence to 10%. Early detection, maintenance of a reporting chain, 5-year vaccination programme with minimum of 70% coverage, and liaison with neighbouring countries were the key elements of this plan.

In Angola, south of the 14th parallel is the most important endemic focus of CBPP in Southern Africa. Transhumance is a way of life and trade in livestock products, exchange of cattle for draft power, civil and military unrest all contributed to the increase and persistence of CBPP. Earlier, field vaccine, which was essentially pleural exudates, was used. This caused the spread of CBPP, but during 1970 to 1994 vaccines that were essentially $T_1 44$ were used albeit inconsistently due to interruptions by civil strife. Recently, 2002/2003, cattle movements have been mapped out and as part of a 5 year plan, a buffer zone between the 13th and 14 parallel has been established although there cannot be physical barriers (as it is in Namibia). Other activities such as rebuilding of laboratories destroyed during the civil war, establishment of general animal health networks, veterinary services and laboratory networks are being undertaken with the help of international donor funds. It may be possible to control CBPP from Angola given political will, a well-defined animal health policy, scientific efforts to improve the vaccine and International support.

In Namibia CBPP is endemic in the northern communal region. Historically, vaccination with T1 44, has been practiced in this region but between 1995 and 1999 there was a 10-fold increase in mortality due to CBPP. This may have been due to increased transhumance, or ineffective vaccination with T_1 SR, however, CBPP was kept in check by movement control. In 2003 CBPP returned to the Caprivi strip. There were 17 cases confined to the Kavango region, and an outbreak near the border with Botswana where 78 of 104 cattle were positive according to the CFT test and 80% had typical lesions. It is noteworthy that CFT was most useful test in this outbreak. Comprehensive stamping out was not possible in this case due to financial considerations but a vaccination programme was instituted.

CBPP spread from the East near Mali, to *Haut* Guinea and Guinea *Forestiere*. Mass vaccination with T_1 44 was started in 1987 followed by surveys.From 1995 onwards, zoning, legislation and regulation, active participation of stakeholders, animal identification and training of herdsmen and veterinary staff, abattoir surveillance and compensation improved this strategy. Dissemination of information was essential and done through national TV and radio, through workshops and distribution of handbooks. Financial support came from government and international bodies. The programme was well supported by livestock owners and private veterinarians carried out most of the vaccination in 2002. Peak vaccination coverage was in 1997 and has decreased a little because of civil unrest. These actions resulted in the decrease in outbreaks and slaughter.

In 1999 civil strife in Angola caused an influx of refugees and their cattle into the West of Zambia. Despite some stamping out efforts CBPP spread northwards prompting zonation and vaccination. These efforts were continued and supported by setting up testing facilities and training. At present abattoir surveillance, serological surveillance and sensitisation are being carried out. Vaccination in a large area to the West has been proposed.

Discussion

The significance of CBPP was questioned by some participants earlier during this meeting. Yet during this session it was obvious that 27 countries reported the disease because there was a problem. The lack of data in the public domain was explained by the unwillingness to publish due to political concerns or the sluggish attitude towards publication. In the Caprivi of Namibia 2000 animals may be infected and it is a big problem not only for that country but also for the surrounding disease-free countries. In Zambia the situation was worsening because of Angola, but the situation is changing and conducive to regional disease In the past individual country strategies have not made collective difference in this region.

There are many gaps in the scientific knowledge on CBPP. Virulence factors and the genes responsible have not been identified; these genes and those that lead to a protective response must also be identified. The genes that may confer virulence or protection are not the same as those responsible for attenuation of an organism. Research towards better vaccines is important. It is also important that diagnostic research continues to distinguish between vaccinated and non-vaccinated and infected animals. The serological tests, CFT and the successfully validated cELISA may perform differently in different countries, but are adequate for surveillance purposes. Sera collected for the Rinderpest campaign may be used to gather this data provided that relevant CBPP data is known. The PACE Epidemiology Unit should be able to provide clarification of this. The requirements for rapid, user-friendly diagnostic tools and better vaccines are still urgent and require continuing research.

The earlier proposal of elective vaccination prompted intense and lengthy debate. It was felt that elective vaccination may break down zonation and the precision of activities within them and interfere with surveillance and diagnostic activities. These criticisms were moderated by the suggestion that free vaccination was for endemic areas only and that the mixture of mass vaccination with elective vaccination could improve coverage.

Several strategies for the control and possible eradication of CBPP were presented that seemed contradictory. They reflected differences in livestock management or capacity of veterinary services the particular country or region. If policy makers in a region or locality acted synergistically, then control strategies would be poised for maximum effect. Synergism between the private and public sectors in complementary partnership would be desirable because it would be more efficient. Perhaps solutions to the problems in primary endemic areas could begin to appear. The presence of representatives from Namibia, Angola, Zambia at this meeting would provide opportunity for collaboration towards this. Therefore, despite the lack of data, but with the firm assurance from the meeting that CBPP is a significant problem, it was agreed that CBPP control should be driven ahead and proposals for control should not be postponed.

Closing Remarks

Dr. Y. Cheneau, Chief, Animal Health Service, AGAH

Mr. Chairman, Ladies and Gentlemen,

I apologise on behalf of Dr. Samuel Jutzi, Director of AGA, who could not be present at the close of this meeting due to prior pressing commitments.

Mr. Chairman, this has indeed been a highly satisfactory meeting over the last three days. New and relevant ideas were presented that have stimulated exceptional discussions and debate. We must not underestimate the impact of CBPP in Africa and realise that it is a problem despite the lack of supportive data in some instances. Data to substantiate this claim is scarce or lacking, or it may be inapplicable, but it is a problem of sufficient magnitude for the SADC countries to announce it in the popular press during the launch of an appeal for donor support in the control of CBPP and other transboundary animal diseases. We have to deal with this reality. The meeting has taken into consideration vaccination and antibiotic treatment, and progress in the basic research has been presented. I thank all the authors and presenters for their continued efforts. In this respect I am pleased to announce that this forum has been institutionalized and I hope that these meetings will continue and receive continuing financial support from FAO.

I wish to comment on two crucial issues in the control of CBPP. The first is the state of veterinary services in Africa, I am inclined to mention that the first and most important objective of PARC was to improve these services. We should do likewise for the control of CBPP because Veterinary services today are not up to strength. The second is the state of PANVAC. We know that it has not existed functionally for about two years. The operational presence of this vaccine quality control laboratory is of utmost importance to the effective production of CBPP and other vaccines used for animal disease control in Africa. CBPP vaccines must always be properly quality assured if they are to be effective in the field. All efforts must be directed to re-opening this laboratory and it must be maintained by AU/IBAR.

On behalf of FAO I thank you for the quality of your work at this meeting and promise that we will not be inactive in trying to promote CBPP control in Africa. In our deliberations and discussions, we have reached consensus and I am particularly pleased to see that we have not advocated the open and free use of vaccine in the field.

Ladies and Gentlemen, I declare the meeting closed, and although we are responsible for keeping you here for long hours, in the time remaining please enjoy Rome. My successor will welcome you in two years time to the next CBPP Consultative Meeting.

Thank you.

FAO-OIE-AU/IBAR-IAEA

Consultative Group Meeting on CBPP in Africa

Towards sustainable CBPP control programmes for Africa

(Rome, 12 – 14 November 2003)

Recommendations

Preamble

The continuing spread of CBPP disease, has confirmed the decreased capability of the control of the disease throughout Africa. The reasons for this include gaps in the basic understanding of the disease and the implementation of effective surveillance and control programmes. This prompted FAO together with the OIE, AU/IBAR and IAEA to convene a joint meeting of specialists to review the current situation with CBPP disease and to suggest actions for improvement of this situation. The meeting was held at FAO, Rome from 12 – 14 November 2003. Specialist working groups reflected on the current knowledge brought together here and deliberated on the needs for applied research and policy under the headings:

- CBPP control strategies;
- Tools for CBPP Control – Vaccines, and;
- Tools for CBPP Control – Use of Antibiotics and Diagnostic Tests.

The recommendations emanating from this meeting are as follows:

CBPP Control Strategies

Introduction

Whilst there is no doubt that CBPP is considered an important disease of cattle in Africa, there is scant data to accurately measure its extent and socio-economic impacts. The suppression of incidence of CBPP, especially in endemic zones, and the maintenance of disease free zones against disease incursions from neighbouring areas are the main aims of control efforts. To achieve these, given complex cross-border political and animal production systems, co-ordination of policy within countries and within the sub-region will be necessary. Concerted control strategies and actions be they vaccination, chemotherapy, or a combination may then be applied to full effects.

Considerations:

1. Cognizant of the fact that CBPP is widely regarded by veterinary policy makers to be a disease of strategic importance there is a need to verify the livelihoods impact of CBPP relative to other animal health issues.

2. Because CBPP is a strategic disease in many sub-regions for sub-Saharan Africa efforts directed toward defining more accurately the location and role of primary endemic areas in the persistence and spread of CBPP is vital.

3. Cost-beneficial application of CBPP vaccine is central to the progressive control of the disease. Targeted vaccine application in contrast to mass vaccination may be appropriate in some situations (as recommended in 2000).

4. Vaccination provides the basis for all feasible control strategies. It is therefore vital that only safe and effective vaccines are supplied to service providers. Furthermore, continued efforts to ensure the timely availability of thermostable vaccine needs to remain a priority.

5. Considering that modelling studies have indicated that strategic use of antibiotics may be beneficial their use needs to be considered.

Specific Recommendations

1. The strategic approach to CBPP should be based on progressive control leading ultimately to area-wide freedom from the infection. A long-term (10 to 15 year) programme encompassing the following should be applied:
   - Impact assessments of CBPP at regional, national and zonal levels need to be conducted to justify the anticipated expenditure required for progressive control of CBPP. Participatory approaches are among appropriate methods to achieve this end. These studies should be applied in all sub-regions (clusters of countries) of sub-Saharan Africa;
   - Cost-benefit analyses of the strategies in force in selected countries of the 3 sub-regions;
   - Depending on the epidemiological situation strategies need to be applied to free and epizootic regions as defined in the report of the CBPP Consultative Group meeting of 2000. For endemic regions targeted vaccination or other alternative strategies need to be investigated.

2. A mechanism to enable independent accreditation of CBPP vaccine quality for African countries needs to be established. Ideally, this should be based on the revival of PANVAC.

3. Research needs to be continued into:
   - Antibiotic treatment of clinical cases;
   - Improved vaccines and diagnostic tools;
   - Targeted application of vaccine as a strategy to improve progressive control of CBPP.

4. Pilot projects located in the field and directed towards improved integrated control of CBPP (including antibiotic treatment and liberalization of vaccine availability) need to be undertaken in carefully defined areas and the results made available to all interested parties.

5. CBPP control programmes could be used as a model on which to base improvement of veterinary services, especially in respect of surveillance, control and private/public sector collaboration.

6. Disease modelling is an appropriate tool for improved understanding of the epidemiology and impact of CBPP and its use should be encouraged.

7. Financial planning to ensure adequate financing of the progressive control of CBPP in sub-Saharan Africa.

Tools for CBPP Control – Vaccines

Introduction

The task of this working group was to consider progress on recommendations made at the two previous consultative group meetings on research of new and existing vaccines. In particular we looked at improvements in existing vaccines, input of PANVAC and the need for independent quality control; construction of vaccines that allow DIVA type differentiation of infected and vaccinated animals; and the set up of a database of vaccine producers, their capacity and the current need for vaccine doses in Africa.

Considerations and Specific Recommendations

The group recognized that most of the recommendations made at the last two meetings had been achieved. However the use of T1 44 and T1 SR vaccines needed to be reconsidered in the view of adverse reactions seen with the former in certain circumstances. Little progress had yet been made on the development of new vaccines. To date little was known of the molecular mechanisms of pathogenicity although some progress was made on virulence factors.

8. Concerning the improvement of existing vaccines and their use:
   - PANVAC is advised to investigate improvements in vaccine formulation including the use of diluents in relation to improved titres and thermal stability. This also includes testing of current vaccines for the stability of pH after reconstitution with currently used diluents;
   - Results of vaccine boosting experiments which are ongoing at KARI should be published within the year;
   - Results of experiments to investigate the use of trehalose in the freeze-drying medium to improve the thermotolerance of CBPP vaccines should be published.

9. Concerning the input of PANVAC:
   - Independent external quality control must be re-established in PANVAC;
   - All vaccines used at national level should be certified by PANVAC;
   - AU/IBAR should fully support the operational activities of PANVAC;
   - PANVAC should continue to strictly apply OIE guidelines on CBPP Vaccine manufacture.

10. Concerning the development of new vaccines:

- Encourage basic research to improve the understanding of pathogenicity and immune protection in CBPP. Data should be published promptly;
- Development and improvement of new vaccine strains must follow the basic rules of biological safety for recombinant vaccines;
- Future vaccines should include the capability for differentiation of vaccinated and infected animals.

11. Other:
    - List of CBPP vaccine producers and their capabilities as established by PANVAC should be sent to the OIE and FAO.

Tools for CBPP Control – Use of Antibiotics and Diagnostic Tests

Specific Recommendations: Diagnostic Tests

12. To establish the prevalence of infection in endemic areas cross-sectional serological surveys should be undertaken.

13. To confirm the absence of disease from an area clinical surveillance (including participatory techniques), abattoir/slaughter slab surveillance and serological surveillance must be undertaken.

14. To confirm new outbreaks isolation and identification of the infectious agent must be performed. None of the serological tests on its own is sufficient as a single diagnostic test but it may be useful if serum samples from several animals are collected and tested in the CFT and the cELISA to obtain a diagnosis on herd basis.

15. Detection of antibodies and duration of detection after infection, antibiotic treatment, vaccination and multiple vaccinations are important parameters and must be clearly defined. Insufficient information on the influence of antibiotic treatment and multiple vaccinations is a constraint that must be addressed.

16. For the confirmation of outbreaks and the early detection of circulating antigen penside tests are very useful. The existing tests need validation and if adequate should be transformed into robust tests to minimize operator bias and errors. More specific and sensitive tests based on the early fraction of the capsular polysaccharides (CPS) needs further assessment before it can be validated at the field level.

17. Quality assurance of the CFT is difficult. Standardized reagents and internal quality controls (high/low titre sera with a defined titre, borderline negative sera) should be introduced to limit the variation. The joint Division of FAO/IAEA, Vienna, should coordinate this activity.

18. The immunoblotting test is highly specific and should be introduced as a confirmatory test at critical phases of CBPP control programmes.

19. The differentiation between individual animals that are infected or had been vaccinated recently is important and serological tests for this purpose should be developed.

20. The CFT is more useful for the early diagnosis of infection; however, an ELISA that is capable of detecting animals at an early stage of infection would be highly desirable.

21. The quality assurance of diagnostic results is critical, and the joint Division of FAO/IAEA, Vienna should undertake its coordination.

Specific Recommendations: Antibiotics

I. Pilot trials

Introduction:

IBAR/PACE has recently commissioned studies of CBPP epidemiology that accessed indigenous knowledge of pastoral communities to construct mathematical models. Sufficient understanding has accrued from these studies to suggest that a new paradigm for CBPP control using antibiotics should be investigated. The prospective benefits are such that pilot trials should be established without delay.

Considerations and Specific Recommendations

The target populations, at least initially, are the pastoral communities of eastern, central and western Africa. The trials proposed need to be based on the use of antibiotics to treat acute cases and elective vaccination. Two scenarios in pastoral communities should be studied; in order of priority these are:

- Management of endemic disease;
  - with regard to the use of antibiotics as a therapeutic intervention;
  - with regard to vaccination and the possible influence of antibiotics on the immune response;
- Management of acute disease from recent introduction;
- In devising protocols to be followed, the antibiotics used will need to be selected carefully to ensure that:
  - Recently developed, and potentially more effective, mycoplasmacidal, chemotherapeutic agents are included;
  - Care is taken to avoid repercussions of the future use of chemotherapeutic agents for human.

22. PACE with FAO should embark on collaborative pilot trials in 2004 by establishing a virtual working group to draw up protocols and initiate field studies to be conducted in close collaboration with the national authorities in key countries. The collaborating partners should communicate with the pharmaceutical industry to obtain their inputs in protocol development and possible co-financing of studies. Thus, there should be three phases of the trials:
    (a) Preparatory phase: establishment of virtual working group – establish dialogue between partners and with the pharmaceutical industry; development of protocols, define logistics, source funding;
    (b) Study phase – overseen by PACE national programmes;
    (c) Analytical phase with final report produced after a workshop.

II. Studies on microbial sensitivity

Introduction

In order to facilitate the selection of candidate chemotherapeutic agents and to understand better the existing situation, there is a need to carry out MIC and MMC studies on current African strains of *Mycoplasma mycoides* subspecies *mycoides* SC.

Considerations and Specific Recommendation

The UK Veterinary Laboratories Agency has the relevant technologies and is provisionally interested to conduct this work within its existing mycoplasma research programme. The most important constraint which needs to be overcome is that VLA lacks the field strains required.

23. The Veterinary Laboratories Agency (VLA) management should be requested by FAO and AU-IBAR to conduct the study and the FAO/OIE World Reference Laboratory for CBPP be requested to make available to VLA, the required strains.

III. Studies on the Safety and Impact of Antibiotics on the Consumer

Introduction

The widespread use of antibiotics and their control are increasingly important for the safety of livestock products in developing countries.

Considerations and Specific Recommendation

Antibiotic residues in milk and meat products have been widely studied but no efficient systems to monitor and enforce their recommended use in developing countries are in place.

24. Monitoring systems for antibiotic residues and systems aimed at achieving compliance with the recommended use of antibiotics should be encouraged to minimize the impact of antibiotic residues on the consumer.

FAO-OIE-AU/IBAR-IAEA

Consultative Group Meeting on CBPP in Africa

Towards sustainable CBPP control programmes for Africa

(Rome, 12 – 14 November 2003)

List of Participants

Roger D. Ayling
Research Scientist
Veterinary Laboratories Agency (Weybridge)
Woodham Lane, New Haw
Addlestone, Surrey
KT15 3NB
U.K.
Phone: 0044 (0) 1932 357 616
Fax: 0044 (0) 1932 357 423
Email: r.d.ayling@vla.defra.gsi.gov.uk

Daouda Bangoura
Docteur Vétérinaire
Chef de Division des Services Vétérinaires
Direction Nationale Élévage
Republique de Guinée
Phone: 00244 11 29 14 68
Email: daoudabang@yahoo.fr
saf.dne@biasy.net

John B. Bashiruddin
Research Scientist (Meeting Secretary)
32 The Street
Tongham
Farnham
Surrey GU10 1DH
Phone 0044 1252 783 928
Email: john.bashiruddin@btopenworld.com

Folosu E. Fasanmi
Director
Livestock & Pest Control
Dept. of Livestock & Pest Control Services
Fed. Ministry of Agriculture & Rural development
New Secretariat, Area 11
P.M.B. No. 135
Garki, Abuja
Nigeria
Phone: 00234 9 3140 337
Fax: 00234 9 3140 336
Email: folusofasanmi@yahoo.ca

Joachim Frey
Professor
Institute of Veterinary Bacteriology
Laenggasstrasse 122
University of Bern
CH-3001 Bern
Switzerland
Phone: 0041 31 631 2414
Fax: 0041 31 631 2634
Email: joachim.frey@vbi.unibe.ch

Otto J. B. Hübschle
Head
Veterinary Laboratory Services
P. Bag 13187
Windhoek
Namibia
Phone: 00264 61 237 684
Fax: 00264 61 221 099
Email: o.huebschle@cvl.com.na

Matthieu Lesnoff
Agronomist
Livestock Production Modelling
ILRI
PO Box 5689
Addis Ababa
Ethiopia
Phone: 00251 1 463 215 ext 137
Fax: 00251 1 461 252
Email: m.lesnoff@cgiar.org

Moto Peter C. Mangani
Deputy Director
Research and Specialist Services
Box 50060
Lusaka
Zambia
Phone: 00260 1 252 608
Fax: 00260 1 252 608
Email: aphhq@zamnet.zm

Flora Mbithi
Veterinary Research Officer (KARI)
(Visiting Scientist at ILRI)
International Livestock Research Institute
PO Box 30709
Nairobi, 00100
Kenya
Phone: 00254 020 630 743
Fax: 00254 020 631 499
Email: fmbithi@cgiar.org

John Bernard March
Moredun Research Institute
Pentlands Science Park
Bush Loan
Penicuik
EH26 0PZ
Scotland, UK
Phone: 0044 131 445 5111
Fax: 0044 131 445 6235
Email: John.March@mri.sari.ac.uk

Frederick Lusonzi Musisi
Animal Production and Health Officer
Subregional Office for Southern and East Africa (SAFR)
FAO
6th (& 11th) Floor Old Mutual Centre
Cnr. Jason Moyo Avenue/Third Street
PO Box 3730
Harare
Zimbabwe
Phone: 00263 4 253 655/7
Fax: 00263 4 700 724
Email: fred.musisi@fao.org
fm141047@kla1.afsat.com

Robin Nicholas
Microbiologist
Veterinary Laboratories Agency (Weybridge)
Woodham Lane, New Haw
Addlestone, Surrey
KT15 3NB
U.K.
Phone: 0044 1932 357 379
Fax: 0044 1932 357 423
Email: r.a.j.nicholas@vla.defra.gov.uk

Attilio Pini
Veterinary Officer
Istituto Zooprofilattico
Abruzzo & Molise
64100 Teramo
Italy
Phone: 00390861 332 228
Fax: 00390861 332 251
Email: a.pini@izs.it

José Regalla
Head
Department of Bacteriology
Laboratorio Nacional de Investigçáo Veterinaria
Estrada de Benfica 701
1549-011 Lisboa
Portugal
Phone: 00351 21711 5339
Fax: 00351 21711 5236
Email: jose.regalla@lniv.min-agricultura.pt

Mark M. Rweyemamu
6 Robinsdale
Knaphill, Woking
Surrey GU21 2LQ
United Kingdom
Phone: 0044 1483 473774
Email: MarkRweyemamu@hotmail.com
mark.rweyemamu@btinternet.com
or
PO Box 9973
Dar es Salaam
Tanzania
Phone : 00255 22 2780 420

Boubacar M'Baye Seck
Veterinary Vaccines Specialist
FAO Consultant
BP 1317
Bamako
Mali
Phone: 00223 678 8271 or 00233 224 8994
Fax: 00223 224 9809
Email: boubacarmbaye@cefib.com
Bm_seck@yahoo.com

Ditutala Lucas Simão
General Director
Veterinary Research Institute
Ministerio da Agricultura e do Desenvolvimento Rural
Instituto de Investigaçao Veterinaria
Phone: 00244 2 372 873 or 00244 9231 9393
Fax: 00244 2 372 873
Email: iiv@snet.co.ao

Salome Wanyoike-Kairu
Senior Veterinary Officer
Central Veterinary Laboratory
PO Kangemi-00625
Ministry of Livestock and Fisheries Dev.
Kenya
Phone: 00254 020 632 231
Fax: 00254 020 631 273
Email:

Hezron Okwako Wesonga
Veterinary Research Officer
National Veterinary Research Center
Kenya Agric Research Institute
PO Box 32
Kikuyu
Kenya
Phone: 00254 66 32000
Fax: 00254 66 32450
Email: h_Wesonga@yahoo.com

Aboubakar Yaya
Vétérinaire
Laboratoire National Vétérinaire
B.P. 503 Garoua
Cameroun
Phone: 00237 227 1305 or 00237 999 9818
Fax: 00237 999 9875
Email: lanavet@iccnet.cm

AU-IBAR

Rene Bessin
Ag. Chief
Animal Health Section
PACE Coordinator
AU-IBAR
PO Box 30786
Nairobi
Kenya
Phone: 00254 2 338 544 or 00254 2 251 517
Fax: 00254 2 226 565
Email: rene.bessin@oau-ibar.org

Andy Catley
AU-IBAR
PO Box 30786
00100 Nairobi
Kenya
Phone: 00254 2 226 447
Fax: 00254 2 212 289
Email: andy.catley@oau-ibar.org

Bouna Alboury Diop
Regional Coordinator Western Central Africa
AU-IBAR-PACE
Bamako
Mali
Phone: 00223 224 6053
Fax: 00223 224 0578
Email: bouna.diop@pacereg.org

Bedjeh Kebkiba
PACE Epidemiologist
AU-IBAR
PO Box 30786
Nairobi
Kenya
Phone: 0025420 308 185 or 0025420 251 517
Fax: 0025420 226 565
Email: bidjeh.kebkiba@oau.ibar.org

Jeffrey Mariner
AU-IBAR/CAPE
PO Box 30786
00100 Nairobi
Kenya
Phone: 00 254 733 398 531 (mobile)
Fax: 00254 20 212 289
Email : jeffreymariner@yahoo.com

Jotham Musiime
Director a.i.
AU-IBAR
PO Box 30786
Nairobi
Kenya
Phone: 00254 20 252 906 or 00254 20 338 544
Fax: 00254 20 220 546 or 00254 20 226565
Email: jotham.musiime@oau-ibar.org

Felix Njeumi
Veterinarian – Epidemiologist
Zonal Veterinary Advisor
PACE-Somalia
PO Box 74916
Nairobi
Kenya
Phone: 00254 733 571 436
Fax: 00254 20 444 8563
Email: fnjeumi@hotmail.com

Gavin Thomson
Main Epidemiologist
PACE Programme
AU-IBAR
PO Box 30786
Nairobi
Kenya
Phone: 00254 733 610 045
Fax: 00254 20 226565
Email: gavin.thomson@oau-ibar.org

C.I.R.A.D.

Joseph Domenech
CIRAD-EMVT
jusqu'au 30 Nov 2003 Ex Directeur EMVT
Chef Service Santé Animale, AGAH à partir 1er Dec 2003

I.A.E.A.

Roland Geiger
Technical Officer
Joint FAO/IAEA Division
APHS
IAEA
PO Box 100
1400 Vienna, Austria
Phone: 0043 1 2600 26063
Fax: 0043 1 2607
Email: r.geiger@iaea.org
geigergeiger@iaea.org

O.I.E.

Francois Thiaucourt
CIRAD-EMVT
TA 30/G
34398 Montpellier Cedex 5
France
Phone: 0033 467 59 3723
Fax: 0033 467 59 3798
Email: thiaucourt@cirad.fr

FAO SECRETARIAT

Yves Cheneau
Chief, Animal Health Service, AGAH
Tel: 0039 06570 53531
Email: yves.cheneau@fao.org

Juan Lubroth
Senior Officer (Infectious Diseases/EMPRES), AGAH
Tel: 0039 06570 54184
Email: juan.lubroth@fao.org

David Ward
Senior Officer (Non-Infectious Diseases), AGAH
Tel: 0039 06570 56464
Email: david.ward@fao.org

Peter Roeder
Animal Health Officer (Infectious Diseases Group), AGAH
Tel: 0039 06570 54637
Email: peter.roeder@fao.org

William Amanfu
Animal Health Officer (Infectious Diseases Group), AGAH
Tel: 0039 06570 56493
Email: william.amanfu@fao.org

Ms Fairouz Larfaoui
Consultant (EMPRES-i), AGAH
Tel: 0039 06570 56435
Email: fairouz.larfaoui@fao.org

Ms Hanan Mohammed
Visiting Scientist (Parasitic Diseases Group), AGAH
Tel: 0039 06570 56750
Email: hanan.mohammed@fao.org

Ms Lucy Mensah
Bilingual Typist, (Infectious Diseases Group), AGAH
Tel: 0039 06570 52635
Email: lucy.mensah@fao.org

FAO, AGAH Fax: 0039 06570 53023
0039 06570 55749

AGENDA

FAO-OIE-AU/IBAR-IAEA

Consultative Group Meeting on CBPP in Africa

12 – 14 November 2003, FAO Headquarters, Rome, Italy

Chairman: **Y. Cheneau - Chief, Animal Health Service of FAO**

Secretariat: **FAO Animal Health Service, Rome**

Rapporteur: **John Bashiruddin, UK**

Theme: **Towards sustainable CBPP control programmes for Africa**

AGENDA

Wednesay, 12 November 2003: Austria Room, C 237 **Session Chair**		**Y. Cheneau, FAO, Chief Animal Health Service**
09:00 – 09:15	Opening Remarks	F. Guerrieri, Chief, TCEO Emergency Operations
09:15 – 09:25	Principal objectives of the Consultation and expected outputs	J. Lubroth, FAO
09:25 – 09:50	Sustainability of FAO technical support for CBPP control	W. Amanfu, FAO
09:50 – 10:20	Break for Coffee and Group Photograph	
Session Chair:	**CBPP Control Strategies**	**J. Domenech, Director CIRAD-EMVT**
10:20 – 10:40	Analysis of CBPP control strategies as proposed by PACE member countries	B. Kebkibah, AU-IBAR Nairobi, Kenya
10:40 – 11:00	CBPP epidemiological situation in SADC countries and strategies for control	F. Musisi, FAO-SAFR Harare, Zimbabwe

11:00 – 11:20	The dynamics of CBPP endemism and development of effective control strategies	J. Mariner, Consultant USA
11:20 – 11:40	Mathematical model for intra-herd transmission of CBPP	M. Lesnoff, CIRAD-EMVT Addis Ababa, Ethiopia
11:40 – 12:30	Discussions on control strategies	
12:30 – 14:00	Lunch break	
Session Chair:	**Tools for CBPP control – vaccines**	**J. Musiime, Director, AU/IBAR, Nairobi, Kenya**
14:00 – 14:20	Molecular mechanisms of virulence and antigencity of MmmSC: conclusions for prevention & control of CBPP	J. Frey, IVB Bern, Switzerland
14:20 – 14:40	An inactivated vaccine exacerbates the effects of CBPP	R.A.J. Nicholas, VLA Weybridge, U.K.
14:40 – 15:00	Studies on reversion to virulence by strain T1/44	H. Wesonga, KARI Nairobi, Kenya
1500 – 15:20	Studies on the dose effect of vaccinations with T1/44	A. Yaya, LANAVET Garoua, Cameroon
15:20 – 15:40	Break	
15:40 – 16:00	Improved formulations for existing CBPP vaccines	J. March, Moredun Research. Inst. Edinburgh, U.K.
16:00 – 17:00	Discussions on tools for CBPP control-vaccines	
17:30 – 19:00	Cocktails	Indonesia Room, 8th Floor C

Thursday, 13 November 2003: Philippine Room, C277/281

Tools for CBPP control – use of antibiotics and diagnostic tests

Session Chair:		**J. Frey, Berne**
08:30 – 08:50	CBPP: Research trends	F. Thiaucourt, CIRAD-EMVT Montpellier, France
08:50 – 09:10	Preliminary results on the efficacy of tetracycline treatments against CBPP	F. Thiaucourt, CIRAD-EMVT Montpellier, France
09:10 – 09:30	Surveillance and testing strategies for the diagnosis of CBPP-results of an FAO-IAEA coordinated research programme on the monitoring of CBPP in Africa	R. Geiger, IAEA Vienna, Austria

09:30 – 09:50	Diagnostic and epidemiological procedures for CBPP eradication programme in Portugal: strategic prototype for CBPP control in African countries	J. Regalla, LNIV Lisbon, Portugal
09:50 – 10:10	Use of Participatory techniques to measure the impact of CBPP relative to other major diseases	A. Catley, CAPE Nairobi, Kenya
10:10 – 10:30	Break	
10:30 – 10:50	CBPP Avis Presentation	M. Rweyemamu, AVIS London, UK
10:50 – 11:10	CBPP Policy Document of PACE	G. Thomson, PACE Nairobi, Kenya
11:10 – 11-30	Evaluation of immunogenicity and efficacy of vaccination	F. Mbithi, ILRI Nairobi, Kenya
11:30 – 12:30	Discussions on the use of antibiotics/diagnostic tests for CBPP control	
12:30 – 14:00	Lunch break	
Session Chair	**Country specific control strategies**	**W. Amanfu, FAO**
14:00 – 14:20	The situation of CBPP in west and central Africa and strategies for sustainable control	B. Seck, LCV Bamako, Mali
14:20 – 14:40	The status of CBPP in Nigeria with emphasis on control strategies	F. Fasanmi, Abuja, Nigeria
14:40 – 1500	CBPP in Angola: Prospects for control	J. Simão, Luanda, Angola
15:00 – 15:20	The situation of CBPP in view of the outbreak in the Caprivi region	O. Huebschele Windhoek, Namibia
15:20 – 15:40	Break	
15:40 – 16:00	Report from Guinea	D. Bangoura Conakry, Guinea
15:40 – 16:00	Report from Zambia	P. Mangani Lusaka, Zambia
16:00 – 17:00	Discussions on country specific strategies and avenues for harmonization of control strategies	

Friday, 14 November 2003: Philippine Room, C277/281

Session Chair:	**Working groups**	**W. Amanfu/J. Lubroth**
08:30 – 10:20	Working Groups	
10:20 – 10:40	Coffee Break	
10:40 – 12:30	Working Groups/ Reporting	
12:30 – 14:00	Lunch Break	
14:00 – 15:00	Drafting of recommendations	
15:00 – 15:20	Coffee Break	
Session Chair:		**Y. Cheneau, FAO**
15:20 – 16:00	Plenary, Finalizing Recommendations	
16:00	Report to Director of AGA and Closing	

FAO-OIE-AU/IBAR-IAEA

Consultative Group Meeting on CBPP in Africa

Towards sustainable CBPP control programmes for Africa

Group Photograph

FAO-OIE-AU/IBAR-IAEA

Consultative Group Meeting on CBPP in Africa

Towards sustainable CBPP control programmes for Africa

Individual Presentations

(Editor's Note : Papers are reproduced as submitted by the participants at the meeting)

Analyse des stratégies de lutte contre la péripneumonie contagieuse bovine (PPCB) dans les pays membres du PACE

Bidjeh Kebkiba,

Unité d'Epidémiologie du PACE (PEU), AU-IBAR, PO Box 30786, Nairobi, Kenya

Introduction

L'économie de la plupart des pays Africains reste essentiellement basée sur le secteur agricole qui occupe plus de 80% de la population, procure plus de 85% des recettes d'exportation et représente près de 40% du PIB national dont 12% pour le secteur d'élevage. Dans le PIB du secteur agricole, l'élevage représente en moyenne 35%. L'élevage contribue d'une manière considérable à l'amélioration de la sécurité alimentaire et à l'augmentation des revenus des paysans et des éleveurs.

Les maladies animales en général et la péri pneumonie contagieuse bovine (PPCB) en particulier, constituent des contraintes majeures au développement du secteur d'élevage et aux échanges commerciaux internationaux du bétail et des produits d'origine animale. De nos jours la PPCB demeure une contrainte majeure pour la production bovine dans la plupart des régions de l'Afrique sub-saharienne, même si un certain nombre de pays de l'Afrique australe en a été exemptée pendant de nombreuses décennies. Les pertes annuelles de deux milliards de dollars américains ont été attribuées à la maladie en Afrique, bien qu'on ne puisse pas garantir l'exactitude de ce chiffre (Masiga et al., 1998). En effet, après la peste bovine, la PPCB a été identifiée par un grand nombre de pays d'Afrique subsaharienne comme l'une des contraintes pathologiques majeures et prioritaires (Hendrikx, 2000). C'est ainsi que le Programme Panafricain de Contrôle des Epizooties (PACE) a inscrit la lutte contre la PPCB parmi un de ses quatre objectifs principaux.

Afin de mettre en place des stratégies de lutte contre la PPCB, les informations relatives aux données épidémiologiques et à l'impact de la maladie doivent continuer à être collectées à travers le système général de déclaration des maladies, des foyers, les investigations et la surveillance participative de la maladie de même que le diagnostic du laboratoire. C'est dans cette optique que les tentatives suivantes ont été initiées par la FAO, l'AIEA et l'UA/IBAR/PACE, dans le but de l'amélioration de la connaissance de l'épidémiologie de la PPCB et le développement des capacités et des stratégies fiables et pratiques pour son contrôle.

La FAO a financé un programme régional de coopération technique (PCT) appelé "Programme coordonné de renforcement des capacités en épidémiosurveillance et en contrôle de la PPCB" pour la période allant du mois d'avril 2001 à mars 2003. Couvrant 8 pays de l'Afrique de l'Ouest (Burkina Faso, Côte d'Ivoire, Ghana, Guinée, Mali, Mauritanie, Niger et Sénégal), ce projet a fourni une assistance technique aux programmes nationaux de contrôle de la PPCB notamment dans l'amélioration des structures et des systèmes de surveillance. Ledit projet avait pour objectif principal, l'amélioration des activités de contrôle de la PPCB et harmonisation des stratégies mises en œuvre dans ces différents pays. La dernière réunion de coordination de ce projet (Ouagadougou, le 23-24 octobre 2002) avait initié la définition d'une proposition d'un plan stratégique pour le contrôle coordonné et progressif de la PPCB dans la sous-région de l'Afrique occidental.

Un atelier relatif à la réunion finale de coordination de recherche sur le monitoring de la PPCB en Afrique a été organisé par l'AIEA à Bamako (Mali). L'objectif dudit atelier est de valider les outils de diagnostic de la PPCB: ELISA compétition (cELISA) et latex test d'agglutination (LAT) et de développer des stratégies appropriées des tests de diagnostic.

Depuis le démarrage du programme, la coordination du PACE a organisé trois ateliers relatifs aux stratégies de contrôle/lutte contre la PPCB (à Addis Ababa en novembre 2001, Accra en février 2003 et Nairobi en mai 2003). L'objectif des deux premiers ateliers était de développer en étroite collaboration avec les responsables des services vétérinaires, une politique, raisonnée et intégrée et des stratégies fiables, pratiques et applicables de lutte contre la PPCB dans les pays membres du PACE basée sur une approche régionale. Pour étayer les conclusions et recommandations de ces deux ateliers sur une base scientifique, un autre atelier fut organisé à Nairobi en mai 2003. Durant ce dernier, seuls les aspects techniques étaient soulignés à travers les récents travaux de modélisation et de simulation conduits au sud Soudan, en Tanzanie (Mariner, 2003) et en Ethiopie (Lesnoff, 2003).

Suite aux recommandations et plans d'action issus de ces différents ateliers, il a été demandé aux pays de préparer un projet document de politique nationale et de stratégies de lutte contre la PPCB basées sur les données épidémiologiques réactualisées et de l'envoyer à l' Unité d'épidémiologie du PACE (PEU). A partir de ces informations, le PEU devrait élaborer un avant-projet du programme régional de lutte contre la PPCB.

Le présent document fait la synthèse et l'analyse des projets de stratégies nationales de lutte contre la PPCB soumis par les pays PACE à l'Unité de Coordination du PACE.

Situation épidémiologique de la PPCB dans les pays membres du PACE

La PPCB est une maladie contagieuse causée par *Mycoplasma mycoides* subspecies *mycoides* small colony (MmmSC). Cette maladie connaît une recrudescence malgré les efforts déployés au niveau des pays. Historiquement, la PPCB était une maladie européenne, américaine et asiatique, mais elle a été éradiquée des Etats-Unis, du Canada et de la majeure partie de l'Europe au 19ème siècle par le biais de diagnostics cliniques, du contrôle des mouvements et de l'abattage sanitaire (Provost *et al.*, 1987). Bien que la PPCB ait été présente en Afrique sub-saharienne avant l'époque coloniale, elle a été importée de l'Europe par bateau dans presque toute l'Afrique australe au milieu du 19ème siècle, comme indiqué ci-dessus, et s'est propagée vers le nord jusqu'en Angola, où elle a persisté jusqu'à ce jour (Windsor, 2000). La complexité relative aux origines de l'infection en Afrique est confirmée par les récentes études épidémiologiques et moléculaires qui ont démontré l'existence de trois lignées distinctes de la PPCB (Lorenzon *et al*., 2003). Elle est répandue dans plusieurs pays d'Afrique à l'exception de ceux du nord et de l'Afrique Australe (OIE, 2003; 2002; 2001; 2000; 1999; FAO, 1998). Cependant, elle est souvent observée en Angola, Namibie et Zambie (Bessin 1998).

La maladie est transmise par contact direct entre les individus infectés et les individus susceptibles. Quand elle s'introduit pour la première fois au sein d'une population bovine sensible, la PPCB cause généralement une forte mortalité. C'est la raison pour laquelle elle est comprise dans la liste A des maladies de l'Office International des Epizooties (OIE, 2003). Dans des situations endémiques, la maladie connaît une évolution variable et est souvent insidieuse de nature. Les formes cliniques de la maladie sont : suraiguë, aiguë et chronique. Il y a aussi des cas d'infection inapparente lorsque la maladie clinique ne se déclare pas. Cette dernière

condition comprend les porteurs sains (des animaux apparemment sont guéris de la maladie) qui ont des séquestres encapsulés dans les poumons contenant des organismes vivants. La mesure de l'impact des porteurs sains sur la survie et à la transmission de l'infection est sujette à controverse et mérite d'être clarifiée. D'autre part, il faut signaler que la maladie est difficile à reproduire en laboratoire et l'étude de son épidémiologie est problématique dans des situations endémiques à cause de sa nature insidieuse.

Lors des ateliers tenus à Addis Ababa et à Accra en 2001et 2003 respectivement, il a été montré que la persistance et la propagation de la PPCB dans les pays PACE sont dues aux facteurs suivants:

- Le mouvement des animaux dû à la transhumance (à la recherche de pâturage et de points d'eau) et au commerce tant à l'intérieur des pays qu'aux frontières;
- Le faible taux de couverture vaccinale, conséquence de la mise en place du système de recouvrements des coûts de vaccination (pour certains pays) provoquant ainsi la réticence des éleveurs à présenter leurs animaux;
- Le faible taux de couverture immunitaire, conséquence de la qualité du vaccin utilisé et de la courte durée d'immunité induite par le vaccin;
- La non application des mesures de police sanitaire;
- La faible performance des laboratoires de diagnostic;
- L'insuffisance des données fiables sur l'utilisation des antibiotiques dans le traitement des animaux malades et leur rôle dans le maintien des porteurs sains;
- L'insuffisance des réunions/rencontres d'échange d'informations zoo-sanitaires et de coordination aux frontières;
- La faible implication des inspecteurs de viande dans la surveillance de la maladie;
- La détérioration des services vétérinaires publics due aux situations conflictuelles et l'insuffisance de l'appui financier du Gouvernement au secteur d'élevage pour la gestion de la santé animale.

Afrique de l'Ouest

La PPCB est endémique dans la plupart des pays d'Afrique de l'Ouest depuis des décennies. Le tableau 1 présentant la situation des foyers enregistrés au cours de ces huit dernières années dans les pays de la sous région montre que, à l'exception de la Guinée Bissau, de la Gambie et du Sénégal, cette maladie demeure endémique ou sporadique dans la plupart de ces pays. Ce tableau montre qu'un nombre important de foyers a été enregistré dans les pays de 1994 à 1998. A partir de 1999, on note une baisse et un nombre constant de foyers (en moyenne 75 foyers) pendant trois ans consécutifs. Toutefois, au vu de ce tableau on peut constater que la PPCB constitue et demeure une préoccupation majeure de cette partie du contient Africain.

Les informations relatives au nombre de cas de maladie et de morts enregistrés entre 1994 et 2002 sont présentées dans le tableau 2. Ces informations sont complètes dans 6 pays et restent fragmentaires pour les autres. Bien que le nombre de cas varie sensiblement d'une année à une autre, on peut constater que c'est en 1997 et 1998 que le plus grand nombre de foyers a été enregistré.

Tableau 1. Nombre de foyers de PPCB enregistrés dans la sous-région de 1994 à 2002 par pays

Pays/Année	1994	1995	1996	1997	1998	1999	2000	2001	2002
Benin	20	5	0	2	3	3	5	3	0
Burkina Faso	7	24	33	35	42	16	20	10	12
Côte d'Ivoire	6	12	11	10	8	11	7	8	5
Gambie	0	0	0	0	0	0	0	0	0
Ghana	3	1	5	49	51	23	21	4	26
Guinée Bissau	0	0	0	0	0	0	0	0	0
Guinée	71	50	30	36	11	6	0	1	1
Mali	21	32	12	15	9	15	12	15	5
Mauritanie	8	5	7	10	3	3	1	4	1
Niger	6	5	9	0	7	1	1	0	1
Nigeria	4	8	13	15	16	4	9	31	1
Sénégal	0	0	0	0	0	0	0	0	0
Togo	0	+							17
Total/année	**146**	**142**	**120**	**172**	**150**	**82**	**76**	**76**	**69**

Tableau 2. Nombre d'animaux malades et morts dans un foyer enregistrés de 1994 à 2002 par pays

Pays/Année	1994	1995	1996	1997	1998	1999	2000	2001	2002
Burkina Faso					168	944	389	518	53
					63	81	81	203	27
Côte d'Ivoire	97	170	87	302	184	160	152	203	398
	19	48	33	188	84	106	50	130	199
Ghana	38	12	572	122	655	75	51	166	251
	1	0	6	11	45	4	12	7	42
Guinée	122	335	71	719	61	24	0	42	
	?	103	11	341	57	19	0	30	
Mali		695	162	591	146	221	382	241	182
		294	52	230	55	100	202	78	34
Mauritanie	20	183	170	98	182	340	10	44	
	5	118 ?	109	67	133	67	7	7	
Niger	23	24							22
	35	13							6
Nigeria	498	258	518	2580	1793	181	1162	998	
	153	42	77	117	76	17	87	219	
Togo									1393
									3
Total/Année	**653**	**1677**	**1580**	**4412**	**3189**	**1945**	**2146**	**2212**	**1099**
	178	**618**	**288**	**954**	**513**	**394**	**439**	**674**	**311**

NB: première ligne correspond au nombre d'animaux maladies. Deuxièmes ligne correspond au nombre d'animaux morts

Le tableau 3 présente quelques paramètres épidémiologiques de la PPCB en Afrique de l'Ouest de 1994 à 2002. Les Taux de morbidité et de mortalité n'ont pu être calculés en raison de la non disponibilité et de la disparité des données relatives au nombre d'animaux sensibles ou exposés.

Tableau 3. Quelques paramètres de la PPCB dans la sous-région de 1994 à 2002

Année	Nombre de foyers	Animaux malades	Animaux morts
1994	146	633	178
1995	142	1677	618
1996	120	1580	288
1997	172	4412	954
1998	150	3189	513
1999	81	1945	394
2000	75	2146	439
2001	76	2212	674
2002	70	1099	311

L'analyse de différents rapports des pays montre la distribution des foyers de la PPCB dans la sous-région en deux zones distinctes: zones infectées (endémiques et nouvellement infectées ou épidémiques) et des zones dites libres/indemnes de la maladie pour la plupart des pays à l'exception du Nigeria où la maladie est endémique sur toute l'étendue du territoire.

Afrique du centre

Si la distribution géographique de la PPCB est bien établie en Afrique de l'Ouest, elle reste cependant confuse en Afrique centrale. En effet, les informations zoo-sanitaires sont souvent absentes ou irrégulières, comme on peut le constater dans le tableau 4. Cette situation ne permet pas d'apprécier les paramètres épidémiologiques de la maladie dans les pays de la sous région. Si les données confinées dans le tableau ci-après se confirment, il en découle que la maladie n'est pas largement répandue en Afrique centrale. Cependant, les quelques rares cas enregistrés montrent qu'elle apparaît de façon sporadique (à l'exception du Tchad). Selon les rapports des pays, la maladie est absente en Guinée Equatoriale, au Gabon, au Congo Démocratique et au Congo.

La PPCB n'a pas été observée en République Démocratique du Congo (RDC) depuis 1981. Mais suite aux échanges commerciaux du bétail entretenus avec les pays voisins (surtout l'Ouganda, District de Karamoja) où la maladie sévit sous forme endémique, elle a réapparu en RDC en 1989 et s'y est maintenue jusqu'en 1992. En deux ans, les zones d'Aru et Mahagi ont enregistré environ 1913 cas de mortalité, soit 9,2% de la population bovine de la région de l'Ituri.

Afrique de l'Est

Après une période d'absence, la PPCB est réapparue en Afrique de l'Est à partir de l'Ouganda et le Kenya. Petit à petit elle a progressé vers le sud et l'Ouest (Ituri, au nord de la RDC). Les données bibliographiques montrent que la PPCB a été introduite en 1990 dans le Nord de la Tanzanie et atteindrait le Sud de l'Ouganda vers le Rwanda en 1994 (Bessin, 1998).

De nos jours, tous les pays de l'Afrique de l'Est sont infectés par la maladie à l'exception de l'Erythrée dont les derniers foyers déclarés à l'OIE remontent à 1994 (tableau 5 et 6).

Bien que la PPCB soit une maladie endémique en Somalie (FAO, 1998), très peu d'informations sont disponibles en ce qui concerne sa prévalence et sa distribution géographique dans le pays. Cependant, les tentatives d'étude de prévalence ont été initiées en 2000 et 2002 à travers le programme PACE Somalie où 484 sérums ont été collectés (200 sérums et 284 en Somalie Centrale et la partie du sud respectivement). Les résultats de cette analyse ont montré que tous les sérums testés sont négatifs.

Tableau 4. Foyers de la PPCB enregistrés en Afrique centrale de 1995 à 2002

Pays/Année	**1995**	**1996**	**1997**	**1998**	**1999**	**2000**	**2001**	**2002**
Cameroun		1	2		5			
Gabon	0	0	0	0	0	0	0	2 (nc)
RCA							+	
Tchad	1	5	2 (nc)	2	4		+	3
Guinée Equat.	0	0	0	0	0	0	0	0
RDC	0	0	0	0	0	0	0	0
Congo	0	0	0	0	0	0	0	0
Total/Année	**1**	**6**	**7**	**2**	**9**	**0**	**+**	**5**

NB: **(**nc) - suspicion non confirmée par le laboratoire
(+) - maladie déclarée à l'OIE , mais absence de chiffres

Tableau 5. Foyers enregistrés en Afrique de l'Est de 1994 à 2002

Pays/Année	**1994**	**1995**	**1996**	**1997**	**1998**	**1999**	**2000**	**2001**	**2002**
Djibouti	?	?	?	?	?	?	?	?	?
Burundi	+	+	+	+	+				
Sudan	+	+	+	+	+	+	+	+	+
Kenya	19	12	11	8	7	9	14	18	18
Tanzanie		30	274	70	67	286	180		15
Erythrée	+	0	0	0	0	0	0	0	0
Ethiopie		48	96	43	187	94	56	27	32
Ouganda	44	37	32	42	15	18	13	30	54
Rwanda	**+**	**+**	**+**	**+**	**+**	?	?	?	?
Somali	?	?	?	?	?	?	?	?	?
Total/Année	**63**	**127**	**413**	**163**	**276**	**407**	**263**	**75**	**78**

NB: + - Foyers déclarés
? - informations non disponibles

D'une manière générale, comme en Afrique Centrale, les données épidémiologiques sont insuffisantes pour définir exactement le zonage classique de la maladie dans les pays d'Afrique de l'Est à l'exception du Kenya.

Impact de la vaccination sur l'incidence de la PPCB

Les informations épidémiologiques collectées et des données bibliographiques montrent que, les vaccins (T1 44, T1 SR) utilisés jusqu'à présent pour lutter contre la PPCB procurent une immunité de courte durée chez les animaux. Aussi, il existe une grande variabilité dans la réponse immunitaire chez les animaux vaccinés Hubshcle *et al*., 2002; FAO, 2000; 1998). Par ailleurs, l'inexistence d'une méthode efficace et fiable de détection des porteurs chroniques associée aux mouvements des animaux rend difficile l'application correcte des mesures de contrôle de cette maladie. Toutefois, l'analyse des rapports des pays montre que l'incidence de la maladie a considérablement baissé pendant la phase d'exécution du programme PARC dû à la vaccination massive et systématique des animaux avec le vaccin bivalent peste bovine-péripneumonie.

Afrique de l'Ouest

Les chiffres de vaccination contre la PPCB des huit dernières années sont représentés dans le tableau 7. Bien que les pays pratiquent annuellement la vaccination, l'analyse dudit tableau montre qu'elle n'est pas faite d'une manière massive et systématique, ce qui laisse évidemment des poches. Le faible taux de couverture vaccinale de 32,22% en moyenne en est une illustration.

La tentative d'illustration de l'impact de la vaccination sur l'incidence de la maladie durant les 12 dernières années est montrée par des rapports entre les foyers déclarés et le taux de couverture vaccinale à travers les tableaux 5, 6 (dressés par pays) et 7 (pour l'ensemble de la sous-région). L'analyse de ce trois tableaux ne permet pas de faire une relation directe entre le nombre de foyers déclarés dans l'année et le taux de couverture vaccinale. Ceci peut être expliqué probablement par le fait que les vaccinations sont faites par poches, et ne sont donc pas réalisées d'une manière systématique d'une part, et les informations sur les foyers de la maladie fournies aux instances internationales (OIE, FAO et UA/IBAR) par les pays sont fragmentaires et irrégulières, d'autre part.

Tableau 6. Chiffre de vaccination contre la PPCB de 1994 à 2001 par pays

Pays/Année	1994	1995	1996	1997	1998	1999	2000	2001
Burkina	491921	834186	1481672	1852898	1391387	1309043	1242857	1337527
Faso	4260900	4345900	4432900	4521500	4611900	4704000	4798000	4992000
Côte	0	732657	947000	1019282	1012125	1014480	874044	594485
d'Ivoire	0	832000	1020000	1082333	1101664	1084654	1409000	1409000
Ghana	11291	5800	6593	1846	0	835650	708190	2434
	1216677	1122730	1247861	1267826	1270000	1288000	1302000	1315951
Guinée	0	404571	414839	994369	902207	835650	708197	665706
	0	950497	757764	1467882	1241893	2368000	2679385	2679385
Mali	1231091	1517668	1962977	1899165	3306051	2849105	3321244	2971545
	5541689	5708000	5882000	5894000	6325764	6521100	6569946	6930000
Mauritanie	350000	281877	551557	802153	856115	841876	856598	700000
	1236749	1236302	1163622	1315005	?	1433000	1476000	1500000
Niger	635000	344000	631000	827596	678751	455252	516025	669333
	1968382	2050060	3085575	3138274	3202320	3282378	3382378	2260000
Nigeria	2804346	780291	1235544	522924	1263800	524327	644008	3200000
	?	?	?	?	?	?	?	?
Sénégal	1203278	1192728	1203278	1029268	1208809	1450695	1275001	1275000
	2693000	2800000	2870000	2898000	2898000	2898000	2961300	3227000
Total/	3572581	5031610	5847359	7624424	8499330	9592211	8628112	8216030
Année	15680640	17809187	19296100	20269815	18043341	23579132	23816322	23551649
Couverture Vac./Année	**22,80**	**28,25**	**30,30**	**37,60**	**47,10**	**40,68**	**36,22**	**34,88**

NB: La première ligne correspond au nombre d'animaux vaccinés
La deuxième ligne correspond au nombre d'animaux recensés

Tableau 7. Rapports entre les foyers enregistrés et la couverture vaccinale de 1990 à 1993

Pays/Année	1990	1991	1992	1993
Burkina Faso	9	3	10	8
	37,23	35,39	31,54	28,08
Côte d'Ivoire	18	16	9	12
	90,34	71,36	81,71	82,17
Ghana	14	2	0	2
	6,7	0,4	0,7	1,8
Guinée	38	82	49	60
	?	?	?	?
Mali	37	12	24	19
	?	?	?	?
Mauritanie			6	8
			?	?
Niger	1	11	11	7
	60,65	49,95	35,93	26,34
Nigeria	71	55	35	29
	?	?	?	?
Sénégal	0	0	0	0
	71,70	74,40	41,78	44,30

NB. première ligne correspond au nombre de foyers
Deuxième ligne correspond à la couverture vaccinale

Tableau 8. Rapports entre les foyers enregistrés et la couverture vaccinale de 1994 à 2001

Pays/Année	1994	1995	1996	1997	1998	1999	2000	2001
Burkina Faso	7	24	23	35	42	16	20	10
	11,55	19,19	33,42	40,98	30,17	27,82	25,90	22,16
Côte d'Ivoire	6	12	11	10	8	11	7	8
	0	88,10	92,84	94	92	94	91	91
Ghana	3	1	5	49	51	23	21	4
	0,9	0,5	0,52	0,14	0	64,88	54,39	0,18
Guinée	30	28	26	22	11	6	0	1
	?	42,56	54,74	67,74	72,64	66,16	57,24	24,85
Mali	21	32	12	15	9	12	12	15
	22,21	26,58	33,37	32,22	52,26	43,69	50,55	42,88
Mauritanie	8	5	7	10	3	3	1	4
	?	?	?	?	52,26	58,76	58,04	46,67
Niger	6	5	9	0	7	1	1	0
	32,26	16,78	30,45	26,97	31,86	20,95	25,78	29,61
Sénégal	0	0	0	0	0	0	0	0
	44,68	42,60	41,93	35,52	41,71	?	50,05	39,51

NB: La première ligne correspond au nombre de foyers
La deuxième ligne correspond à la couverture vaccinale

Tableau 9. Rapports entre les paramètres épidémiologiques et les chiffres de vaccination contre la PPCB dans la sous région de1994 à 2001

Année	Foyers	Animaux malades	Animaux morts	% couverture vaccinale
1994	126	633	173	22,78
1995	137	1478	495	28,25
1996	120	1417	186	30,30
1997	170	4437	1007	37,61
1998	147	3061	418	47,10
1999	78	1945	394	40,68
2000	70	2146	439	36,23
2001	73	2212	674	33,88

Afrique centrale

Bien que la PPCB soit rare ou absente dans certains pays de l'Afrique centrale, les campagnes de vaccination contre cette maladie sont quand même réalisées.

Au Gabon les campagnes de vaccination contre la PPCB utilisant le vaccin bivalent (peste bovine et PPCB) sont menées chaque année dans les ranches de 1980 jusqu'en 1997. Depuis l'arrêt la vaccination contre la peste bovine, la vaccination contre la PPCB a aussi cessé.

Depuis la déclaration officielle de la maladie en Ituri en 1992, les mesures de police sanitaire ont été instaurées. Ces mesures ont été renforcées par les campagnes de vaccination réalisées en 3 phases de 1993 à 1994. Pendant ces campagnes étendues sur toutes les zones d'élevage (Aru, Mahagi, Djugu et Irumu), 89,4% du cheptel bovin ont été vaccinés. Durant la troisème phase, les campagnes de vaccination ne se sont limitées qu'aux zones d'Aru et de Mahagi et ont atteint 49% de l'effectif du cheptel.

Au Tchad, la vaccination contre la PPCB se faisait à l'aide d'un vaccin bivalent (peste bovine et PPCB). Cette vaccination était gratuite et obligatoire jusqu'en 1993, date à laquelle l'état s'est désengagé en donnant le mandat sanitaire aux vétérinaires privés. La vaccination était devenue payante. Face à cette situation, les éleveurs étaient devenus réticents au paiement des frais de vaccination. Certains sélectionnent les animaux à vacciner d'autres fuient carrément la vaccination.

Afrique de l'Est

Comme indique le tableau 10, la persistance et la propagation de la PPCB en Afrique de l'Est peuvent être expliquées par le fait que les vaccinations ne sont pas réalisées d'une manière systématique. C'est seul le Burundi qui a obtenu un taux de vaccination de 75 % en moyenne pour les trois années consécutives de vaccination.

Tableau 10. Rapports entre les foyers déclarés dans l'année et le taux de couverture vaccinale

Pays/Année	1994	1995	1996	1997	1998	1999	2000
Burundi					ND	ND	ND
					93,5	62,3	69
Kenya	19	12	11	8	7	9	14
	12,72	31,12	17,8	9,28	6,25	5,8	11,8
Tanzanie		30	274	70	67	286	180
		75,0	47,3	62	40,1	50	43,8
Ethiopie		48	96	43	187	94	56
		ND	ND	ND	ND	ND	ND
Ouganda	44	37	32	42	15	18	13
	16	17	7	20	18	2	1

NB: La première ligne correspond aux foyers déclarés dans l'année
La deuxième ligne correspond au taux de vaccination réalisée dans l'année

Politique et stratégies actuelles de lutte contre la PPCB dans les pays PACE

Depuis son introduction en Afrique, la PPCB a fait l'objet de multiples programmes de lutte basés essentiellement sur:

- Les campagnes de vaccination de masse des animaux (appliquées par la majorité de. Pays de l'Afrique de l'Ouest et du Centre). Cette stratégie a connu quand même du succès dans cette partie du continent pendant la période de PARC dû peut être au fait que le vaccin utilisé entre temps était le vaccin bivalent (peste bovine, PPCB). La couverture vaccinale étant la même que celle de la peste bovine, avait comme impact, la réduction considérable des foyers de PPCB. Cependant, il faut noter que le taux moyen de couverture vaccinale global a sensiblement baissé au cours de ces trois dernières années (surtout depuis l'arrêt de la vaccination contre la peste bovine dans les différents pays). Les campagnes annuelles de vaccination consistent habituellement en une seule intervention. La mobilité des animaux, le coût élevé des campagnes de vaccination et les réactions post-vaccinales dues au vaccin T1/44 sont avancés comme étant les principales raisons de la faible couverture vaccinale et de la difficulté de procéder à une vaccination de rappel au cours d'une même année.
- L'application plus ou moins rigoureuse de la police sanitaire par certains pays. Cependant, l'application des textes réglementaires se heurte à certaines difficultés. On peut citer entre autre l'abattage sanitaire suivi d'indemnisation des éleveurs.
- Le « stamping out », une approche peu réalisable vu les situations financières des pays qui ne permettent pas l'indemnisation systématique des éleveurs. La persistance de la peste porcine africaine dans les pays côtiers de l'Afrique de l'Ouest en est un exemple palpant.
- La quarantaine associée à la vaccination est considérée comme mesure de lutte la plus appropriée pour certains pays.
- Dans les pays où la maladie n'a jamais été déclarée et la vaccination non pratiquée Guinée Bissau , Congo, etc.), la stratégie la plus appropriée adoptée par ces pays est la mise en quarantaine des animaux entrant dans les pays et l'abattage systématique de tous ceux suspects ou reconnus infectés.

- Le contrôle du mouvement du bétail, facteur limitant la propagation de la maladie semble être essentiel dans la lutte contre la PPCB. Pour rendre effectif ce contrôle, certaines mesures doivent être appliquées, à savoir:
  - L'enquête sur les origines, la destination et le devenir des animaux présentés sur les marchés
  - L'établissement et l'application rigoureuse des certificats de vaccination et de transhumance
  - L'implication des éleveurs au contrôle du mouvement de bétail à travers la formation des groupements d'éleveurs et leur sensibilisation sur le risque de propagation de la maladie au travers les mouvements incontrôlés des animaux
  - Le renforcement des postes vétérinaires au niveau des frontières.

Formulation de nouvelles stratégies de lutte contre la PPCB dans les pays PACE

L'analyse des rapports des pays fait ressortir les contraintes majeures limitant l'application des stratégies et l'amélioration du contrôle de la PPCB dans les pays PACE. Ces contraintes se résument aux facteurs suivants:

- L'insuffisance ou absence des systèmes nationaux de notification des maladies et d'information zoo sanitaire, en général, conduisant à une connaissance approximative des données épidémiologiques de la maladie et donc à des faiblesses dans la planification et la gestion des programmes de son contrôle;
- Difficultés d'application des mesures zoo sanitaires notamment le contrôle des mouvements, l'abattage sanitaire, l'indemnisation des éleveurs, etc. réglementés par la police sanitaire et la législation. Ceci est dû probablement à la nature extensive du système de production animale dominant sur de larges zones peu supervisées par les services vétérinaires et conduisant ainsi à des faiblesses dans la couverture sanitaire des troupeaux ainsi que dans le contrôle des mouvements du bétail;
- La faiblesse en ressources humaines et en structures techniques adéquates au niveau central pour une planification, une exécution, un suivi et une évaluation appropriés des programmes de contrôle des maladies en général;
- L'insuffisance des ressources matérielles et financières allouées à l'agriculture en général et au sous-secteur de l'élevage en particulier.

L'une des recommandations de l'atelier d'Accra est l'élaboration des stratégies de contrôle de la PPCB par les pays participant au Programme PACE. Elles devraient aboutir à la réduction de l'incidence de la PPCB dans les zones endémiques et la protection des zones où la maladie n'est pas déclarée. Ces stratégies proposées doivent reposer sur une approche régionale prenant en compte la situation épidémiologique, les conditions socio-économiques ainsi que les modes d'élevage dans les pays. Elles devront être accompagnées et soutenues par des activités d'information, d'éducation de base et de communication adéquates.

A cet effet, 14 pays sur 30 soit 46,6% ont envoyé leurs rapports relatifs à la politique et aux stratégies de contrôle de la PPCB à la date du 30 septembre 2003 à la Coordination du PACE. L'analyse de ces rapports montre les tendances ci-dessous énumérées:

Le contrôle de la PPCB, comme pour toute autre maladie épizootique passe absolument par le renforcement des capacités de surveillance épidémiologique au travers la formation des acteurs principaux (agents de terrain, personnel du laboratoire et les éleveurs….), la sensibilisation ou l'information des intervenants dans le système (propriétaires d'animaux,

bergers, autorités administratives et politiques locales…) et le renforcement des capacités de diagnostic. C'est ainsi que tous les pays ont fait mention de la surveillance épidémiologique dans tous les scénario de stratégies de lutte quelque soit leur statut sanitaire vis à vis de ladite maladie.

Dans les zones non infectées dites zones indemnes de PPCB, il est préconisé le renforcement de la surveillance aussi bien passive qu'active aux abattoirs, au niveau des marchés de bétail des axes de transhumance ou de commerce et aux frontières. Tous les animaux entrant dans ces zones font l'objet d'un dépistage par les tests sérologiques (c-ELISA et le test de fixation de complément) et, les séropositifs sont systématiquement abattus. La restriction des mouvements d'animaux entrant dans ces zones est de rigueur

Dans les zones endémiques, la vaccination massive des animaux est faite d'une manière systématique et annuelle et ce, pendant au moins cinq années consécutives comme se fut le cas de la peste bovine. Cette approche de lutte doit être harmonisée/ coordonnée avec les régions adjacentes et généralisée à l'ensemble des bovins de la région (action régionalisée). Dans les zones épidémiques (zones nouvellement infectées), à part les mesures appliquées dans les zones indemnes, en cas de confirmation de foyers, la vaccination sélective ou ciblée et en anneau associée au marquage obligatoire des animaux, la surveillance épidémiologique (sur le terrain et aux abattoirs et aires d'abattage) et le contrôle des mouvements des animaux tant à l'intérieur du pays qu'au travers les frontières sont appliqués. Une enquête sérologique est instaurée et les animaux réagissant positivement sont systématiquement abattus.

La stratégie de lutte contre la PPCB de la Guinée qui est considérée comme l'une des plus complètes de la région PACE reposent sur les éléments suivants:

- les campagnes annuelles de vaccination de masse;
- l'élimination par abattage de tous les bovins malades ou exposés au sein des foyers ou même dans des villages entiers selon le cas;
- l'établissement d'un cordon sanitaire divisant le pays en deux, avec une interdiction d'entrée pour les bovins provenant de la zone infectée sauf pour la boucherie et ceci par transport motorisé et sous escorte vétérinaire ;
- le marquage à l'oreille de tout bovin présenté sur un marché de la zone infectée;
- l'identification individuelle de chaque bovin par tatouage.

Pour les pays où la maladie n'est pas répandue sur toute l'étendue du territoire, voire localisée dans une zone bien définie séparée des autres zones par une barrière naturelle ou à travers le cordon sanitaire, la vaccination peut être réalisée de façon ciblée à base annuelle. Et, si le problème des porteurs sains après le traitement des cas cliniques est résolu, la vaccination associée aux traitements des animaux malades peut être envisageable comme l'une des stratégies de lutte contre la PPCB. A cet effet, seule la recherche portant sur l'efficacité de la chimiothérapie sur les *Mycoplama mycoides* subsp*. mycoides* (y compris le dosage) et la relation entre l'utilisation des antibiotiques dans le traitement des cas cliniques de la PPCB et le développement des séquestres dans les poumons des animaux traités à ces produits chimiques, pourrait clarifier la situation.

Conclusion

A la lumière des rapports des pays, le plan stratégique de contrôle de la PPCB au niveau national doit se faire par étapes en tenant compte de:

- L'histoire récente de la PPCB, de sa prévalence actuelle et de ses tendances d'évolution épidémiologique;
- L'état de préparation des systèmes de prestations de services vétérinaires à mener des programmes de surveillance et de contrôle de la PPCB;
- Contraintes ou facteurs limitant la mise en application rigoureuse de la législation et de la police sanitaire (mesures zoo sanitaires tels que: le système d'élevage, le contrôle des mouvements du bétail avec la mise en quarantaine des troupeaux infectés ou suspects, l'abattage sanitaire suivi des indemnisations des éleveurs, les facteurs sociaux socioéconomiques, etc.).

Etape 1. Actualisation des données épidémiologiques de la maladie afin de connaître sa distribution géographique (faire le zonage) dans les pays.

Pour améliorer la connaissance de l'épidémiologie de la maladie et ses impacts économiques afin d'aboutir à un système sous-régional ou régional efficace d'information zoo sanitaire et d'alerte précoce et de réaction rapide de la PPCB, l'étude de prévalence utilisant les outils de diagnostic existant, la surveillance épidémiologique de la maladie sur le terrain et les abattoirs impliquant les vétérinaires privés, les commerçants de bétail, les bouchers et les éleveurs, devraient être entreprises. Ces activités ne peuvent connaître succès qu'en améliorant les capacités des laboratoires vétérinaires dans le diagnostic de la PPCB et la création de conditions favorables pour la mise en place d'un laboratoire sous-régional de référence pour l'identification des souches de *Mycoplasma mycoides.*

Etape 2. Réduction de l'incidence et du risque de propagation de la maladie

La réduction de l'incidence et la limitation des risques d'expansion de la PPCB dans les pays endémiques/zones par la vaccination obligatoire associée à la mise en quarantaine des troupeaux affectés et le contrôle des mouvements du bétail. Ces activités doivent être suivies de la surveillance clinique sur le terrain et la recherche des lésions pulmonaires aux abattoirs. Malheureusement, la menace pesant sur les possibilités actuelles de lutte contre la PPCB reste l'inexistence de vaccins procurant une immunité de longue durée et des méthodes de détection des porteurs chroniques de la maladie.

Dans les pays ou zones dits indemnes de la PPCB, l'application des stratégies combinées doit être mise en place. Celles-ci se limitent à la surveillance épidémiologique intensive suivie de la restriction des mouvements du bétail ainsi que la mise en place du test de dépistage suivi d'abattage des animaux réagissant positivement. Entre les zones libérées et infectées, le cordon sanitaire (comprenant des zones tampon et de surveillance) pour la protection des zones libérées/indemnes de la maladie devrait être établi. Cependant, il faut signaler que dans la zone tampon, la vaccination annuelle est aussi appliquée comme dans les zones endémiques.

Etape 3. Minimisation du risque de réintroduction et d'expansion de la PPCB

Elle doit se faire à travers une évaluation et une gestion adéquates des risques de la PPCB ainsi que des capacités de réaction rapide améliorées sur la base de l'institutionnalisation de la surveillance et des programmes de contrôle de la PPCB, impliquant tous les intervenants et conduisant à l'établissement de différentes zones épidémiologiques. La transparence dans la

communication et l'échange des informations zoo sanitaires relatifs à la PPCB, l'évaluation du système de surveillance et de la disparition de la maladie selon la méthode recommandée par l'OIE devraient permettre la minimisation du risque de réintroduction de la maladie dans le pays.

Cette phase devra ainsi aboutir à l'établissement de barrières sanitaires ou de zones tampons régionales basées sur des facteurs écologiques, sur les systèmes d'élevage et sur les pistes de bétail si on considère la minimisation du risque au niveau régional.

Références bibliographiques

AU/IBAR/PACE (2003). Report of the Workshop on "strategies for control of CBPP", Accra (Ghana) 03-06 February. Final report.

AU/IBAR/PACE (2003). Report of the Technical workshop on CBPP "Recent information made available trough modelling studies on CBPP. Nairobi, 8th May. Final report.

AU/IBARPACE (2001). Report of the Workshop on "Development of policy for control of CBPP in East Africa", Addis Ababa (Ethiopia), 19-21 November. Final report.

Bessin, R. (1998). La péri pneumonie contagieuse bovine en Afrique: Approche d'une stratégie de lutte. Atelier Régional sur la prophylaxie et les stratégies de lutte contre la PPCB en Afrique de l'Ouest, Nouakchott, 10-12 Février 1998.

FAO/IAEA (2003). Report of the final Research Coordination Meeting on the "Monitoring of CBPP in Africa Using ELISA", Bamako (Mali), 17-21 February.

FAO (2000). Report of the second meeting of the FAO/OIE/OAU/IAEA consultative group on pleuropneumonia (CBPP). Rome, Italy, 24-26 October 2000.

FAO (1998). Report of the first meeting of the FAO/OIE/OAU/IBAR consultative group on pleuropneumonia (CBPP). Rome, Italy, 5-7 October 1998.

Hendrikx P. (2000). Etat d'avancement des réseaux d'épidémiosurveillance en Afrique de l'Ouest et du Centre.

Hubschle, O., Lelli, R., Frey, J. and Nicholas, R. (2002). Contagious bovine pleuropneumonia and vaccine strain T1/44. The Veterinary Record (LETTER), May 11th 2002.

Lesnoff, M. (2003). Technical workshop on CBPP "Recent information made available trough modelling studies". Nairobi, 8th May.

Lorenzon, S., Arzul, I., Peyraud, A., Hendrikx, F. and Thiaucourt, F. (2003). Molecular epidemiology of contagious bovine pleuropneumonia by multilocus sequence analysis of *Mycoplasma mycoides* subspecies *mycoides* biotype SC strains. Veterinary Microbiology, **93,** 319-333.

Mariner, J.C. (2003). The dynamics of CBPP endemism and the development of effective control/eradication strategies for pastoral communities: Final modelling report. Project GCP/RAF/365/EC. Food & Agriculture Organization of the UN.

Masiga, W.N., Rossiter, P. and Bessin, R. (1998). Contagious bovine pleuropneumonia. Epidemiology: The present situation in Africa and epidemiological trends. *In:* Report of the FAO/OIE/OAU-IBAR CBPP Consultative Group Meeting, Rome, Italy, 5-7 October 1998. FAO Publication X3960-E, pp 25-31.

Office International des Epizooties, 2003; 2002; 2001; 2000; 1999. International Animal Health Code.

Provost, A., Perreau, P., Breard, A., Le Goff, C., Martel, J.L. and Cottew, G.S. (1987). Contagious bovine pleuropneumonia. Revue Scientifique et Technicque Office International des Epizooties **6,** 625-679.

Windsor, R.S. (2000). The eradication of contagious bovine pleuropneumonia from South-Western Africa: A plan for action. Annals of the New York Academy of Science **916,** 326-32.

The threat of contagious bovine pleuropneumonia and challenges for its control in the SADC region

F. L. Musisi[1], B. Dungu[2], R. Thwala[3], M. E. Mogajane[4], and B. J. Mtei[5]

[1]*FAO, SAFR, Harare, Zimbabwe;*
[2]*Onderstepoort Biological Products, Onderstepoort, Republic of South Africa;*
[3]*Directorate of Veterinary & Livestock Services, Mbabane, Swaziland;*
[4]*National Department of Agriculture, Pretoria, Republic of South Africa;*
[5]*Directorate of Food, Agriculture & Natural Resources, SADC Secretariat, Gaborone, Botswana.*

Introduction

Contagious bovine pleuropneumonia (CBPP) is currently considered one of the main deterrents to the growth of the livestock industry on the African continent. It is the only bacterial disease in the OIE list A diseases. CBPP is caused by *Mycoplasma mycoides* subspecies *mycoides* Small Colony (*Mmm* SC). The endemic form is characterized by hyperthermia, nasal discharge, cough, and rapid and difficult breathing. However, in many African countries where the disease has reached an endemic form, it is difficult to detect affected animals due to reduced clinical manifestations, mostly as a result of antibiotic treatment. In these conditions, a large proportion of animals become chronically infected; the so-called "lungers", and have encapsulated lesions in the lungs.

In the 1960s and 1970s, sustained research on CBPP in Kenya, Chad and other African countries, coupled with a massive international campaign – code-named Joint Project 16 – resulted in the disappearance of clinical disease from most parts of Africa (FAO, 2002). However, because of economic decline and poorly financed veterinary services, the disease made a spectacular comeback in the late 1980s and early 1990s (Kusiluka, 2003; Provost, 1996). Today, more countries are affected by CBPP than there were 20 years ago either as an endemic or re-emerging disease or in epidemic form. Yearly losses directly or indirectly attributable to CBPP are estimated to be around US 2 billion (Masiga *et al.,* 1999).

The Directors of Veterinary Services/Chief Veterinary Officers of the SADC Region have drawn up a 16-year regional strategy for transboundary animal diseases, during their workshop in Pretoria on 22 and 23 July 2003. In doing so, they have categorized TADs for SADC as follows:

- Strategic – FMD, CBPP;
- Tactical – ASF, RVF, NCD, LSD;
- Emerging/Exotic to SADC – rinderpest, PPR, BSE and AI.

Therefore for the SADC region, CBPP is regarded by Member Countries as a disease of strategic importance for which the CVOs will be seeking internal and donor funding for its progressive control leading to a SADC without CBPP over the next 16 years. This paper summarises the outcome of the workshop of the SADC Directors of Veterinary Services/Chief Veterinary Officers.

The threat to the SADC region

Currently CBPP-affected countries within SADC are Angola, Zambia, Tanzania, Democratic Republic of Congo (DRC), and northern Namibia. Countries with affected neighbours are Malawi, Botswana, Mozambique, Zimbabwe and Namibia. Thus these countries are at risk of CBPP incursion or invasion. In Angola, CBPP is endemic and there has been no control strategy for years due to civil strife; thus there is constant incursion into Zambia and Namibia with threats to northern Botswana.

The threat to the cattle industry in SADC is probably best illustrated by SADC's meat export position in comparison to the rest of the African continent. Cattle farming constitute an important component of the agriculture production in the SADC region, occupying a large proportion of the population. Even though its 53 million head of cattle represent only 22% of the cattle population on the continent, the SADC region alone exports more meat than the rest of the continent. In 2001, the SADC exported 84% of meat and meat products from Africa, for a value of US$ 199 million versus US$ 236 million for the entire continent (Figure 1). Most of this export is attributed to Botswana, Namibia, and Zimbabwe, where many rural and urban livelihoods are dependent on this trade. For some of the countries in the region, such as Botswana, meat and meat products export represents the main agricultural product export, accounting for approximately 70% of the total agricultural product exports. The potential for export of meat and meat products from the other countries, namely Tanzania, Zambia and Angola ravaged by CBPP cannot be fully realized.

The lessons from the re-introduction of CBPP into Tanzania and Zambia are that once the disease is allowed to establish itself, it becomes difficult to eradicate, and that where it has been eliminated, it is at a great cost as exemplified by Botswana, which lost more than 300,000 cattle through the stamping out exercise before regaining freedom status. The current CBPP spread will affect trade interests in the region as will be illustrated in countries that experienced in the past or are experiencing the disease now.

The threat is at three levels, household, national and regional and is well illustrated in examples given below from the SADC countries of Botswana, Namibia, Tanzania, and Zambia.

The disruptive effects of CBPP

CBPP is one of the animal disease emergencies whose effects as FAO outlined include, (a) compromising food security through loss of protein/draught power, (b) major production losses, (c) increased production costs due to costs of disease control, (d) disruption of livestock/product trade, (e) inhibition of sustained investment in livestock production, and, (f) pain and suffering to animals (Paskin, 2003; Geering *et al.,* 1999).

According to FAO (1990) animal disease constraints are thought to cause losses of up the 30% of annual livestock output in developing countries and where attempts have been made to measure the actual economic impact of livestock disease outbreaks, the results have been frightening. For instance, in 1995 re-introduction of CBPP in Botswana led to the slaughter of 320 000 cattle at a cost of US$100 million, with further indirect losses estimated at over US$400 million (Geering *et al.,* 1999).

Vulnerability of smallholder households

The CBPP impact at national level indicated above for Botswana seems impressive but it is also necessary to examine the effects on poor resource households with small herds of cattle. This is especially important since according to the Minister of Agriculture, Botswana, nearly 80% of the livestock in Botswana is in the hands of smallholder management (J. K. Swartz, 2003). The effects of CBPP on households in Namibia highlighted below would equally apply to Botswana.

The impact at household level can be very crippling. According to Paskin (1995) this was clearly illustrated in interviews of pastoral households in Namibia who strongly feared CBPP despite the fact that the last epidemic had occurred decades before. The fear of the disease was such that vaccination campaigns instigated by the Veterinary Services enjoyed enthusiastic support, even during a period when CBPP was relatively quiescent. Thus the suffering caused by the well-known CBPP epidemic of 1860 was still well remembered by the Herero people of Namibia and 1860 has been given a special name – Otjipunga – the 'year of the lung' (Schneider, 1994). These families had no source of livelihood other than their stock thus their main source of protein was milk so such an outbreak would have alarming effects at household level.

In a study involving an agro-pastoral society in Namibia, Paskin et al., (1996) reported that the average herd size was 35 but the majority of households surveyed had relatively small herds, ranging from 10–20 animals and CBPP was rampant in the area and most owners reported it as a problem. In such a situation, a mortality rate of only 50% could reduce a herd of 10 animals to only five, with frightening implications in terms of draught power and family milk supply. A high level of dependence on livestock, combined with relatively small herd thus makes smallholder farmers very vulnerable to the effects of epidemic diseases such as CBPP (Paskin, 2003).

Threat in Tanzania

Njau (2003) reported that CBPP and FMD were number one and two priority diseases for Tanzania after rinderpest. CBPP affects Tanzania's participation in international trade of animals and animal products, and is a constraint to improved livestock productivity.

CBPP was re-introduced into the Northeast and Northwestern parts of the country in 1990 and 1991 from Kenya and Uganda, respectively, after being absent for some 25 years and moved with trade stock to Morogoro in central Tanzania. Since its re-introduction, CBPP has been reported to spread to 54 out of 120 administrative districts of Tanzania (Kusiluka and Sudi, 2003). The geographical spread of CBPP is shown in Fig. 2 by the yellow shaded areas. The losses and population at risk for the period 1998 to 2002 are indicated in Table1. The Tanzania–Mozambique international border may not pose serious threat regarding CBPP spread because it has low livestock numbers that are mostly sedentary but the Tanzania–Malawi and Tanzania–Zambia borders (Mbeya, Iringa, Rukwa) are potentially problematic since pastoralists from the north have moved into these areas recently. It is estimated that 350,000 cattle, valued at Tanzanian shillings 40 billion (i.e. about $40 million), have been lost so far. Kitalyi and Njau (2003) estimated that CBPP is responsible for a compounded annual loss of approximately USD 3 million nationally.

Njau (2003) attributed the persistence of CBPP in Tanzania to the following:

- The disease does not lend itself to simple control measures like vaccination because vaccines currently in use do not confer long lasting immunity;
- Re-invasion occurred when Tanzania was in economic turmoil, which impacted the veterinary department for several years thus allowing the disease to flourish to levels that national resources alone cannot contain;
- The lack of comprehensive studies on the socio-economic impact of CBPP thus making it difficult to convince decision-makers to avail money for its control.

As indicated above, CBPP affects the country's international trade in livestock and livestock products. Tanzania aspires and plans to establish a disease free zone (DFZ) in order to increase livestock and livestock product export by 6% per annum starting from 2008. An excellent candidate for DFZ is the area south of the central railway line currently with low livestock numbers but good carrying capacity because of reliable rainfall and low prevalence of transboundary animal diseases, and is far from the traditional entry points for ruminant livestock diseases and historically it is the last to be affected. However, with the threat of CBPP spreading even further south beyond the current distribution shown in Fig 1, it is impossible to establish this DFZ thus further constraining Tanzania's livestock development and its contribution to improvement of the national economy. Thus lost trade opportunities -Tanzania with the largest cattle population in the region cannot benefit from related trade opportunities.

Threat in Zambia

In Zambia, CBPP was re-introduced in 1996 after 23 years in the Western Province of the country from Angola and as late as 2003 outbreaks have been reported (EMPRES-Livestock, 2003). It has since spread to Northwestern Province as shown in Fig.3 and is threatening the Copperbelt Province. It is also feared that the current CBPP situation in Zambia is a great threat to both the cattle population in Zambia and neighboring countries to the south. Like in Tanzania, CBPP is rated the most threatening transboundary animal disease in Zambia. It is blamed mainly on illegal cattle movements. Cases of incursions from Tanzania into Zambia have not been documented but the threat is real as clearly indicated in Fig. 2.

Threat in Angola

CBPP has been considered endemic in Angola since its presumed introduction in the 19th century from South Africa (FAO, 1996). Despite continuous vaccination campaigns, the disease has been established in the Southern provinces, which accounts for 95% of the country's estimated 3.5 million cattle (FAO, 2002). Due to the civil strife that disrupted normal governance including provision of veterinary services and adequate disease surveillance over nearly 3 decades, population movements have contributed to the spread of the disease north- and westward, as well as constituting a continuous threat to neighbouring countries.

The limited vaccination campaigns and other control measures that have been sustained and supported by the Angolan government and the FAO are indicated in Table 2. Sadly, it appears that these measures have not prevented the spread of the disease, which still require a proper assessment.

The neighbouring countries of Namibia and Zambia have suffered from the CBPP incursions resulting from cattle movements with the refugees. However, it is now hoped that with the return of peace, better and realistic assessment of the CBPP situation can be made and national and regional strategies for its control implemented.

Regional level – SADC

In a recent workshop of Chief Veterinary Officers (CVOs) in SADC in July 2003, participants in the workshop identified two areas of concern, namely:

1. Southern Angola/Western Zambia/Northern Namibia;
2. Southern Tanzania/North-eastern Zambia/Northern Malawi.

Thus these are the current epidemiological clusters requiring immediate attention in terms of arresting the spread of the disease in the SADC region.

The challenges to CBPP control in SADC

Over many decades, CBPP control has basically relied upon cattle movement control and vaccination campaigns. Unfortunately, with increasing budgetary constraints and changes in administrative structures, e.g. creation and devolution of power to local government authorities (LGAs) that are demanded by international financial and other donor institutions to be implemented in the affected countries, these two key control measures have become difficult to implement effectively. For instance, the decentralized local administration might not consider CBPP or animal disease control a priority while the neighboring LGA does. Moreover, in some LGAs those in-charge of the decisions on livestock production have either very little or no comprehensive knowledge on animal disease issues. Furthermore, in some countries there is undefined responsibility for disease control between the LGAs and the central veterinary services authority.

The challenges that SADC countries face individually and collectively are implementation of effective cattle movement control, vaccination programs, active and passive surveillance, and plans for emergency preparedness.

Illegal cattle movements

The factors that make effective control of illegal movements of cattle difficult are many. These include socio-cultural ties between cross-border communities, political instability, trade associated with strong and weaker currencies, and lack of credible national animal identification systems. Many cross-border communities have similar ceremonies like dowry, funeral rites, initiation into adulthood, etc. in which donation of cattle is routine and which find restriction of passing on of the gifts offensive. Similarly, political instability linked to civil strife has lead to internal displacements and the imposition of refugee status of people and their livestock, this has been the case in Angola with Namibia and Zambia, and the case in Tanzania in regard to neighbouring countries of Uganda, Rwanda and Burundi. Trade associated with strong and rapidly depreciating currencies of neighboring countries continues to be a major factor; it drives communities from the disadvantaged side to seize the opportunity to acquire the powerful currency and improve the purchasing power while equally the advantaged side views the goods from the other side as cheap. This could be playing out in the Angola-Namibia situation just as it has done in the case of FMD between Botswana and Zimbabwe. Under these situations, the interested parties will do their utmost to avoid using normal cattle routes. Lack of well established national animal identification systems coupled with weak enforcement of legislation further undermine implementation of effective control of illegal movement of animals; the weak legislation was cited by Njau (2003) as contributing to the ineffective CBPP control in Tanzania. Where effective animal movement control exists, there are not always clear or updated

indications on the requirements. The decision on issuance of permits is not always based on verifiable facts. In other instances, there are no clear indications on the required competence of the issuing authority.

Ineffective vaccination programmes

The factors contributing to vaccination programmes being ineffective include failure to adhere to the recommended vaccination regimes, several vaccine administrations in a relatively short period of time, disparities in budgetary support for countries in the region, reluctance of cattle owners to participate fully in the programmes, lack of common strategy and regional control programme, and lack of credible national animal identification systems. Njau (2003) attributed the persistence of CBPP in Tanzania, in part, to the disease not lending itself to simple control measures like vaccination because vaccines currently in use do not confer long lasting immunity. Inability to confer long lasting immunity dictates several administrations of vaccine in a relatively short period imposing undue pressure on logistics required for realisation of a successful vaccination programme. Reluctance of cattle owners to participate fully in vaccination programmes may, at times, result from occurrence of untoward vaccine reactions, cost recovery schemes, and others (Dungu, 2003). Disparities in budgets for disease control in the neighbouring countries coupled with lack of common strategy and regional control programmes further erode the effectiveness of the vaccination programmes; a good example of this is failure of neighbouring countries to harmonise and effect vaccination at the same time along common borders. This equally applies to neighbouring LGAs.

Credible surveillance systems

Many of the countries do not have credible active surveillance systems mainly due to limited availability and/or inappropriate use of available expertise. Njau (2003) reported that, amongst others, inadequate disease information, vaccination coverage and uncontrolled cattle movements pose a serious risk of spreading to neighbouring countries. In many affected countries, indication of the CBPP problem is given by abattoir inspection figures, which are thus derived from passive surveillance. Moreover, while valuable data can be obtained from examining lungs at slaughter, according to Bamhare and Kohrs (1999), in CBPP high-risk regions, animals are subjected to quarantine before slaughter thus eliminating visibly infected animals. Similarly, cattle owners tend to withhold cattle for fear of outright condemnation thus encouraging owners to dispose of such animals at “bush” abattoirs where there is no veterinary supervision. Both of these actions result in a lower estimation of the magnitude of CBPP in given populations. Active surveillance is thus required to make a better assessment of the disease status; this further demands accurate identification of individual herds, sources of infection, traceability of origin and all the contact herds.

Contingency plans

Unfortunately, for various reasons but mainly expertise and budgetary constraints, many countries in the region do not have “Emergency preparedness/Contingency plans” (EPPs) for dealing with CBPP outbreaks. This situation persists despite the recommendation on EPPs at the FAO Expert Consultation on EMPRES - Livestock Diseases Programme (FAO, 1996).

Studies to clarify role of carriers and "lungers"

It is also noted that there is an urgent need in SADC region for proper studies on the role of carriers in the epidemiology of CBPP. In a study in Tanzania small ruminants were implicated in the epidemiology of CBPP but since that report has ever been further investigations to confirm or show otherwise (Dungu, 2003; Kusiluka, 2000). These issues and the role of "lungers" remain a challenge and need to be clarified.

The way forward

In a recent CVOs' workshop, geographical areas of concern were identified into epidemiological clusters that transcend national boundaries. Two phases to tackle the CBPP problem in SADC region were proposed, namely an emergency phase and a recovery phase. Thus the emergency phase would tackle the two geographical areas described earlier, namely Southern Angola/Western Zambia/Northern Namibia and Southern Tanzania/North-eastern Zambia/Northern Malawi.

The emergency strategy devised by the CVO workshop included the following key elements:

- Emergency vaccination of all cattle (i.e. 3 rounds at 0, 3 and 9 months) in the 12 Districts of Southern Tanzania bordering Zambia and Malawi, with possibly buffer vaccination of the contiguous northern areas of Zambia along the Zambia-Tanzania border;
- Emergency vaccination of cattle in Western and North-western Zambia to prevent further spread towards the Copperbelt and central Zambia;
- Heightened surveillance and movement control (Zambia/Malawi/Tanzania/Mozambique);
- Public awareness campaigns (Zambia/Malawi/Tanzania/Mozambique);
- Role of farmers – commitment to controls through heightened awareness (need for awareness tools); animal identification important for tracing and record of vaccination; community-participatory disease searches;
- Role of Veterinary Services – provide access to laboratory diagnostics, responsible for extension, must have a secure and direct chain of command, and a proper contingency plan with elaboration of responsibilities;
- Veterinarians must elaborate strategies that are practical and realistic to convince politicians;
- There is a requirement for economic impact assessments to illustrate importance of CBPP and other TADs.

Other observations of importance:

- Disease reporting to regional unit epidemiology currently poor, needs to be improved;
- SADC lacks a permanent structure for handling emergencies – this must be addressed;
- Industry role – with CBPP, transporters and traders are strongly affected – private sector should be lobbying government, and could assist with public awareness;
- Government must create the climate in which the private sector will feel it safe to invest;
- Vaccination must be free of charge;
- Proper economic assessment important.

The identified milestones of importance were:
1. Baseline study to assess size of population and incidence/distribution of disease;
2. Monthly feedback on vaccination coverage;
3. Assessment of vaccine impact (repeat study);
4. Assessment of status of neighbours at risk.

The workshop also devised a 15-year program for the control of CBPP in the region along the framework indicated in Annex 1 (at the end of this article).

The workshop defined the goal for control of the major transboundary animal diseases (CBPP and FMD) in SADC as “Sustainable food security, poverty reduction and equitable access to markets”. The purpose is “to progressively enhance livestock as a tradable commodity through assured animal health”. The goal and purpose are based on the concept of “progressive control of FMD and CBPP and enhanced preparedness for diseases exotic to the SADC region”. Further, the workshop conceptualised strategic control based on epidemiological clusters that transcend national boundaries and thus requires close liaison and cooperation between the member countries. The epidemiological clusters were defined as follows:

CBPP Cluster 1: Primary endemic disease – Angola, Northern Tanzania.

CBPP Cluster 2: Sporadic/Secondary endemic – Namibia North, Zambia, Tanzania South, DRC East.

CBPP Cluster 3: Presumed free but at immediate risk – Namibia South, Botswana North, Zimbabwe Northwest, Malawi Mozambique North, DRC Rest.

CBPP Cluster 4: Free at low risk - South Africa, Botswana South, Zimbabwe Rest, Lesotho, Swaziland, Mozambique South, Mauritius, Seychelles.

CBPP Cluster 5: Maintaining CBPP freedom – Zimbabwe, Malawi, Mozambique, South Africa, Botswana, Lesotho, Swaziland, Mauritius, Seychelles.

The designation of clusters and the intended actions are summarized in Annex 1.

References

Bamhare, C. and Kohrs, B. (1999). Contagious bovine pleuropneumonia in Namibia. Epidemiology Update, Directorate of Veterinary Services, Internal report.

Dungu, B. (2003). Controlling Contagious bovine pleuropneumonia in Southern Africa. *In:* Report of a Workshop of Chief Veterinary Officers/Directors of Veterinary Services of SADC Member Countries on Transboundary Animal Diseases with special reference to Foot and Mouth Disease and contagious bovine pleuropneumonia in Southern Africa Pretoria, South Africa 21-22 July 2003.

FAO (1990). Cost/benefit analysis for animal health programmes in developing countries. FAO Expert Consultation, Rome, September 1990.

FAO (1996). Evolution de la peripneumonie contagieuse bovine en Angola *in* CBPP prevention and control strategies in Eastern and Southern Africa. Report of the Joint FAO EMPRES and OAU IBAR regional workshop, Arusha, Tanzania, July 1995; page 66-72

FAO (1997). The Emergency control of contagious covine cleuropneumonia (CBPP) in Southern and Eastern Africa. *In:* FAO Animal Production and Health paper 133, 93 – 104.

FAO (2002). Surveillance Et Controle De La Peripneumonie Contagieuse Bovine Et D'autres Maladies Transfrontalieres: ANGOLA*,* Compte rendu final du projet préparé pour le Gouvernement de l'Angola par l'Organisation des Nations Unies pour l'alimentation et l'agriculture, FAO, 2002

FAO (2003). Control of contagious bovine pleuropneumonia in Zambia. (FAO 'EMPRES-Livestock Mail service'. February, 2003.

FAO Electronic conference (2002). Contagious bovine pleuropneumonia – To Eradicate, Control or Live with the Disease June – November, 2001, EMPRES-Livestock, FAO

FAO-EMPRES (2003). Control of contagious bovine pleuropneumonia in Zambia EMPRES publications, 24/02/2003.

Geering, Roeder, Obi, (1999). Manual on the Preparation of National Disease Emergency Plans. FAO, 1999.

Kitalyi J. and, Njau P. (2003). Contagious bovine pleuropneumonia in Tanzania. Implementation of five years control programme: achievements, setbacks and the way forward. *In:* Proceedings of the Tanzania Society of Animal Production.

Kusiluka L. J. M. and Sudi F. F. (2003). Review of successes and failures of contagious bovine pleuropneumonia control strategies in Tanzania. Preventive Veterinary Medicine, **59,** 113-123.

Kusiluka, L. J. M. (2000). Respiratory mycoplasmoses of cattle and goats with special emphasis on molecular epidemiology of Contagious Bovine Pleauropneumonia in Tanzania. Ph. D. Thesis, The Royal Veterinary and Agricultural University of Copenhagen. Pp. 113.

Masiga W. N., Rossitor P., and Bessin R. (1999). Contagious bovine pleuropneumonia. I. Epidemiology: the present situation in Africa and epidemiological trends. *In*: Report of the FAO/OIE/OAU-IBAR CBPP Consultative Group Meeting, Rome, Italy, 5-7 October 1998. FAO publication X3960-E, Rome, pp.25-31.

Njau, P. (2003). Contrasting emergency experiences for rinderpest and CBPP in Tanzania. *In:* Report of a Workshop of Chief Veterinary Officers/Directors of Veterinary Services of SADC Member Countries On Transboundary Animal Diseases with special reference to Foot and Mouth Disease and Contagious bovine pleuropneumonia in Southern Africa Pretoria, South Africa 21-22 July 2003.

Paskin R. (1995). OovaHimba people of Kaokoland: Husbandry Perceptions and Practices. M. Sc. Thesis, University of London, 1995.

Paskin, R. (2003). Economic and social welfare importance of transboundary animal diseases. *In*: Report of a Workshop of Chief Veterinary Officers/Directors of Veterinary Services of SADC Member Countries on Transboundary Animal Diseases with special reference to Foot and Mouth Disease and contagious bovine pleuropneumonia in Southern Africa Pretoria, South Africa 21-22 July 2003.

Paskin, R., Hoffmann, G., Dunkley, K. and Iithete, E. (1996). Socio-economic study: Erongo region. Directorate of Veterinary Services, Namibia.

Provost, A. (1996). Stratégies de prophylaxie et d'eradication de la peripneumonie contagieuse bovine avec ou sans vaccination. *Rev. sci. tech. Off. Int. Epiz.,* **15(4),** 1355-1371

Schneider, H. P. (1994). Animal Health and Veterinary Medicine in Namibia. Agrivet, 1994.

Swartz, J. K., Hon. Minister of Agriculture Botswana (2003). Launching of the SADC Appeal for support to control FMD in the SADC region, 25 September 2003.

Annex 1. A framework designed by the SADC Directors of Veterinary Services for the progressive control of CBPP in southern Africa

(An outline of key elements for a 16-year strategy for the progressive control of transboundary animal diseases in southern Africa)

Stage & Immediate objective	Outputs	Activities	Target year for initiating output / activity															
			1 – 2		3 - 4		5 - 6		7 - 8		9 - 10		11-12		13-14		15-16	
Initial definition of disease status	Defined socio-economic importance of CBPP	Determine size of national herd																
		Determine extent of disease and production / trade effects																
	Defined capacity for regular access to good laboratory diagnostic service	Evaluate national laboratory capacity and access to regional laboratories																
	Defined distribution and epidemiology of CBPP	Collect and analyse epidemiological data with TADinfo																
	Determined primary and secondary endemic areas of CBPP through: disease search, slaughterhouse meat inspections, laboratory culture and serology	Plan and execute active surveillance, departmental staff and farmer training; upgrade local laboratory capabilities																
	Determined presumed CBPP free areas	Undertake critical geographic analysis of data																
	Defined animal movement patterns within country and across borders; defined movement control strategies	Coverage of strategic points																
	Defined targeted vaccination campaigns	Acquire and deliver vaccines																
	Assessed availability of resources for a sustained long period - 10 or more years																	

Stage: Sporadic/Secondary endemic areas	Defined distribution and epidemiology of CBPP	Critically analyze epidemiological data; seek expert opinions																	
Preliminary zoning exercise	Determined primary and secondary endemic areas through: disease search, slaughterhouse meat inspections, laboratory culture and serology	Conduct data analysis; consult with experts "Media consultants"?																	
	Determined presumed CBPP free areas																		
	Instituted communication and community awareness strategies through training of field staff and farmers.	Prepare communication materials; Organize seminars / workshops																	
	Instituted T1/44 3 rounds of vaccinations (0,3,9 months) of all cattle in designated areas	Acquire vaccines; Organize the logistics; Execute the vaccination campaigns																	
	Maintained 3 annual vaccination scheme + test slaughter during the 3rd to 5th year																		
	Maintained strict animal movement control in and out of the designated areas																		
	Sustained surveillance through: village/farm disease search, abattoir slaughter slab meat inspections, microbiological and serological surveys																		
Presumed free but at immediate risk		"Media consultants"?																	
Final disease zoning / movement to freedom from disease and infection	Institution of communication and community awareness strategy through training of field staff and farmers	Prepare training materials Conduct training courses for various target groups																	
	Instituted cross-border control of cattle movement																		

	Sustained surveillance through: programmed clinical, pathological, serological surveys	Deploy field staff; Institute participatory disease diagnosis; Conduct lab diagnosis																
	Reviewed periodically robustness of the CBPP contingency plan	Consult with experts																
	Reviewed effectiveness of national veterinary services	Consult with experts																
	Reviewed and modified accordingly quarantine systems	Consult with experts																
	OIE recognition of freedom from CBPP	Formally apply to the OIE																
Free at low risk	Pay attention to prevention	Consult with stakeholders, experts and political leaders																
	Surveillance and contingency planning																	
	Review effectiveness of national veterinary services																	
	Review quarantine systems																	
	Departmental staff training in CBPP recognition	Prepare training courses and materials; conduct training																
	Disease surveys where/if necessary	Mobilise staff; Conduct surveys																
	OIE recognition of freedom from CBPP	Formally apply to OIE																
Maintaining CBPP freedom	Attention to prevention, surveillance, and contingency planning																	
	Attention to staff training in CBPP recognition																	

Figure 1 (Source: Dungu, 2003)

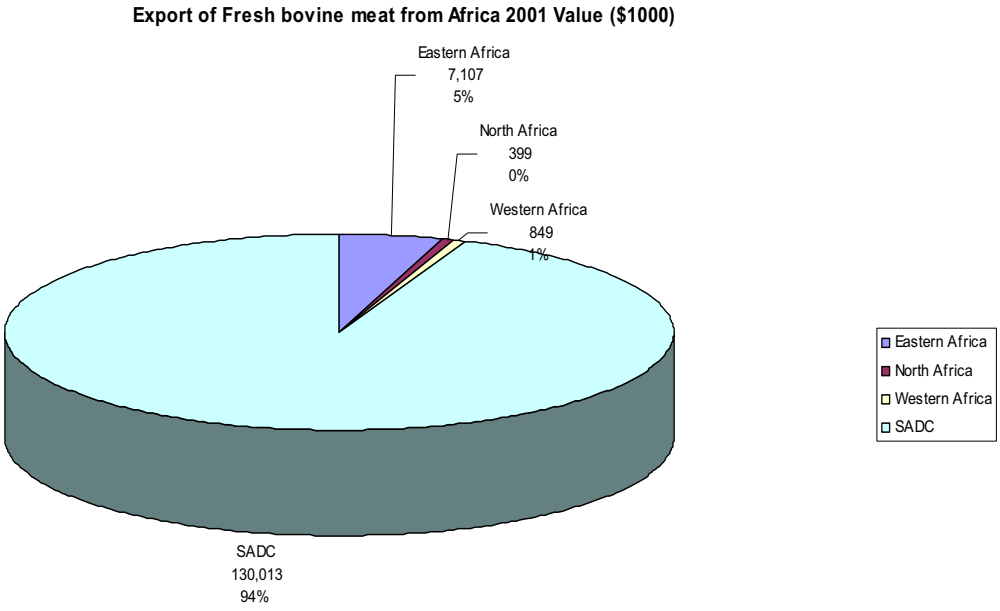


Figure 2. (Source: Njau, 2003)

CBPP infected LGAs 2002

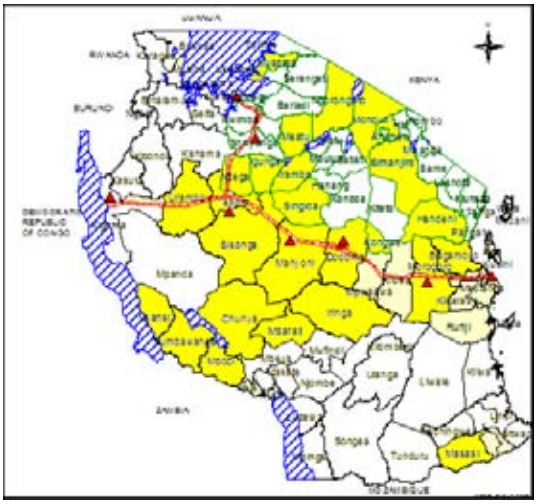


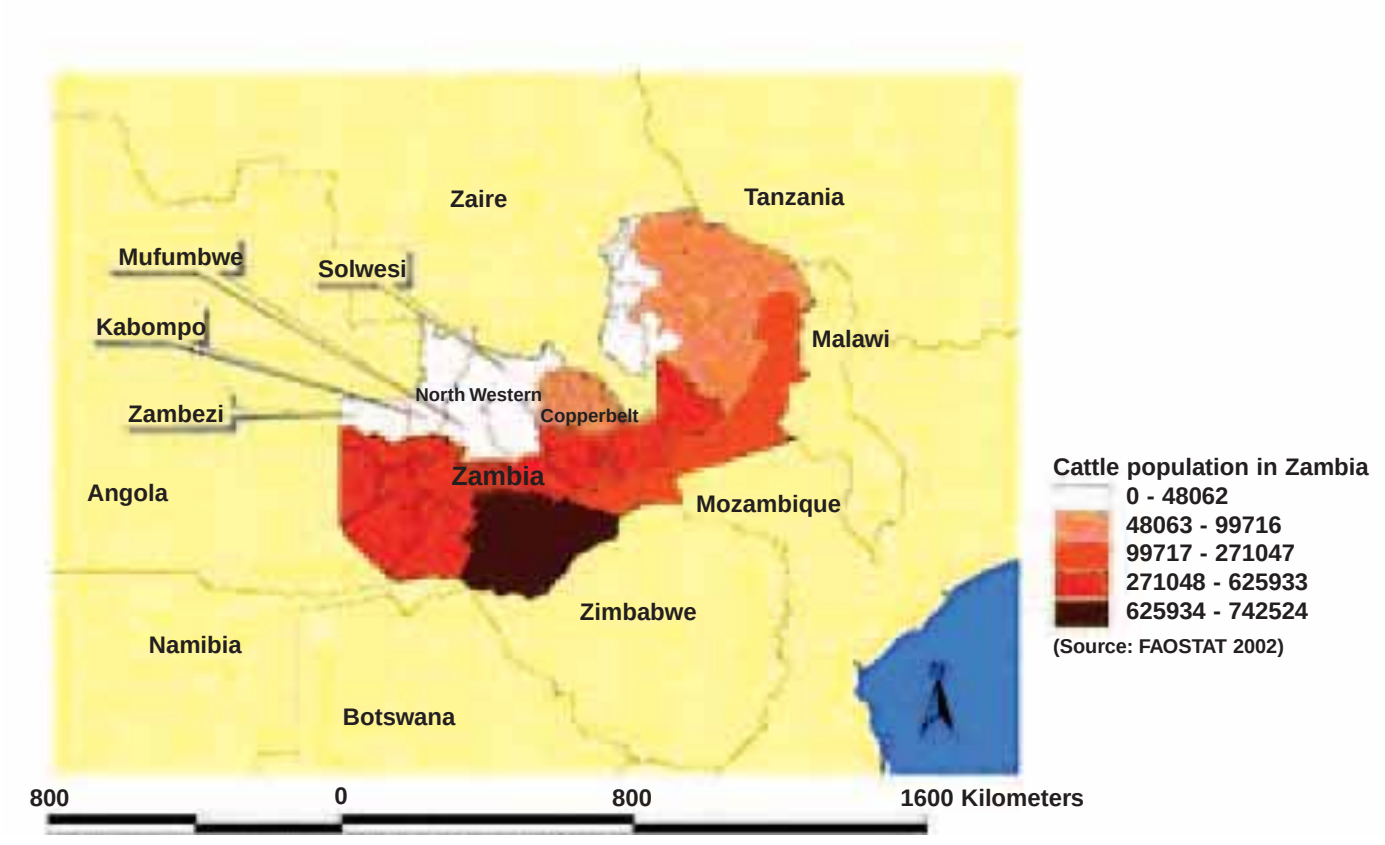
Table 1. CBPP Cases, 1998 – 2002 (Source: Njau, 2003)

Year	Regions affected	Districts affected	Villages affected	Population at Risk	Cases	Deaths
1998	6	11	50	152,854	5,332	3,348
1999	15	29	217	338,220	8,330	3,590
2000	11	18	99	180,157	1,701	1,296
2001	15	28	118	342,072	3,904	3,275
2002	13	26	90	239,035	3,325	1,414
Total					**22,592**	**12,923**

Table 2. Summary of CBPP vaccination campaigns conducted in affected provinces of Angola between 1994 and 2001 (FAO, 2002)

Year	No. vaccinated	%	No. of Outbreaks
1994	555 735	16	12
1995	979 780	28	150
1996	207 355	6	46
1997	481 000	14	62
1998	950 306	27	18
1999	596 209	17	28
2000	527 940	15	18
2001	715 130	20	1

Figure 3. The area affected and the cattle distribution in Zambia (Source:FAOSTAT 2002, in EMPRES, 2002)



The dynamics of CBPP endemism and development of effective control strategies

J. C. Mariner and A. Catley

African Union/Interafrican Bureau for Animal Resources/Community Animal Health and Participatory Epidemiology Project, the PACE Epidemiology Unit and RDP Livestock Services

Introduction

Now that rinderpest is largely controlled in Africa, the attention of animal health authorities has again turned to contagious bovine pleuropneumonia (CBPP). The disease was controlled by vaccination, movement control and slaughter programmes up until the mid-1980s. However, general economic decline led to a reduction in animal health budgets and a resurgence of CBPP in the late 1980s and 1990s. CBPP is now endemic in most pastoral areas of East, Central and West Africa, and is spreading in southern Africa. Many transhumant communities identify CBPP as a major priority for attention. There has been little change in terms of the tools available to control CBPP over the last 30 years, but social, economic and political realities have changed dramatically. Control actions that worked in the past may no longer be feasible.

This report describes the results of a study to model the transmission of endemic contagious bovine pleuropneumonia (CBPP) in transhumant production systems. The objectives of the study were to construct a model of CBPP transmission and then to evaluate the impact of alternative control strategies in an effort to help guide decision-makers in the formulation of realistic recommendations to national governments for the control of CBPP in light of current socio-economic conditions.

The modelling study built upon a previous participatory data collection study (Mariner *et al.,* 2003). Field data and the expert opinion of livestock owners on the behaviour of CBPP in their herds were utilized as a foundation for the modelling. The literature on prevalence, vaccine efficacy, pathology, diagnostic tests and epidemiology has been investigated in detail. The parameter estimates and model structure were developed using data and insights derived participatory epidemiology, serologic studies and an evidence-based literature review. In this way, the analysis was solidly grounded in the reality of the field yet incorporated all available data.

The model has a stochastic, state-transition design and was constructed using @Risk Software. At present, the model includes three inter-linked sub-populations and disease transmission was modelled in both homogeneous and structured populations. Using serological data, the basic reproduction numbers for CBPP in southern Sudan was estimated as 3.2 to 4.6 with a most likely value of 4.1.

Participatory Epidemiology

Participatory epidemiology (PE) is the use of participatory methods to collect epidemiologic data (Mariner, 2000). The participatory approach utilizes and compares all available information through a process called triangulation. Triangulation refers to the confirmation of information using multiple methods and multiple sources. Participatory epidemiology makes full use of sampling, laboratory testing and analytical techniques. Participatory studies provide the proper background for the design of statistical or mathematical

studies and the contextual information that is essential to the correct interpretation of laboratory results.

Pastoralists have a very well developed knowledge of the clinical presentation, epidemiologic patterns and principal pathology of CBPP. In general, livestock owners are aware of the range of clinical presentations of CBPP from mild to severe or acute to chronic. They report that individual cases can last from a few days to up to 12 months. Prolonged clinical cases are often perceived as intermittently episodic. Fully recovered animals are reported to be immune for life. In transhumant communities, a pronounced seasonality in clinical incidence was reported that could be related to grazing movements leading to changes in mixing patterns and contact rates.

Pastoralists clearly recognize that the disease is contagious and that cattle movement, livestock exchanges and contact are major risk factors for the disease. However, the respondents viewed the relative importance of movement in the production systems and the positive socio-economic contributions of cattle exchange to be relatively more important to their pastoral livelihoods than the impact of CBPP. They were risks worth taking relative to the essential benefits entailed.

Livestock owners were also aware of presence of inapparent infection and reported that the source of infection in clinical cases that developed after a period of absence of clinical cases was from within their own herds.

In regard to control interventions, livestock owners and field veterinarians reported treatment with antibiotics had a clear positive effect. For the most part, treatment regimes described could be described as sub-optimal. On the other hand, livestock owners reported that the mass vaccination programs, as currently practiced with available resources, had limited or no impact on the incidence of CBPP.

Summary of the Spatially Heterogeneous Model for CBPP Transmission

The model utilized a stochastic state-transition approach with an open population structure. The final model was spatially heterogeneous in that it incorporated three inter-linked sub-populations. One population was treated as a reference population and the results reported relate to the status of this reference population at the termination of the model. The model incorporated seasonal forcing of contact rates. Vaccination was modelled as pulses delivered in one single time step that could be repeated at the user's discretion. All simulations were allowed to run for six-years.

The model incorporated six principal states, susceptible (S), exposed (E), infectious (I), recovered (R), vaccinated (V) and chronically infected (Q). Vaccination and naturally recovered immunity were modelled as separate states due to the difference in the quality and duration of immunity resulting from vaccination and natural infection. There were two recovery routes for infected animals. The first was full recovery directly to the recovered state (R). The second pathway was recovery through the chronically infected state (Q). This was a process involving sequestration where animals developed encapsulated, infected but non-infectious lesions. The majority of these animals progressed to the recovered state (R), but in a small percentage of cases relapsed, infection was re-activated and the animals re-entered the infectious state (I).

The transmission rate parameters used in the model were derived from serologic studies conducted as part of the research and information available in the literature. The basic reproductive number for CBPP transmission in pastoral communities of southern Sudan was determined from the average age of infection. Model validation consisted of comparison of model predictions of prevalence with prevalence measurements derived from serologic studies.

The effective within population contact rate was based on estimates of the basic reproductive rate. The between population effective contact rate was set as a percentage of the within population contact rate. Unless otherwise specified, between herd contact was set to 10 per cent of the within herd contact.

Principal Results

The model was used to derive estimates of the critical community size for the maintenance of CBPP infection in both heterogeneous populations and single isolated herds. It was found that a heterogeneous population of three inter-liked herds with as few as 50 head per herd were capable of supporting persistent infection over a period six years. On the other hand, in a single isolated herd, more than 300 head was required to support indefinite transmission at equivalent levels of herd prevalence. This points to the efficacy of quarantine and movement control in regard to CBPP control.

A sensitivity analysis was conducted using various levels of inter-herd contact. It was found that when the between herd contact rate was set to 1 per cent of the within herd contact rate, the disease remained endemic in the reference in herds in more than 46 per cent of the iterations of the model. This indicated that only strictly enforced quarantines could be effective. However, the results of the participatory data collection indicated that this level of movement control was not a realistic option under pastoral conditions.

Mass vaccination was modelled for a range of vaccination efficacy (50 to 80 per cent), an 80 per cent level of population coverage and 80 per cent efficiency of vaccination. The average duration of vaccinal immunity was set to 3 years. It was shown that even in 5-year annual or biannual campaigns, vaccination alone was unable to eliminate infection from an important percentage of herds. For example, after 5 years of biannual mass vaccination with a vaccine of 70-80 per cent efficacy the disease persisted in the population in more than 20 per cent of the model iterations.

Elective control methods based on vaccination were modelled by applying vaccination to only one of three sub-populations in the heterogeneous model. The other two sub-populations were left without intervention and served as a potential source for the reintroduction of infection to the vaccinated population. In these simulations, the herd level prevalence of infection among vaccinated herds remained essentially unchanged but the individual animal mortality within the vaccinated herd was substantially reduced. Where 178 deaths could be expected over a six-year period in an unvaccinated herd of 500 head, expected losses were reduced to 124 deaths in a vaccinated herd if a 5-year program of annual vaccination was applied electively. Thus, the livestock owner was able to capture most of the potential benefit in terms of the reduction of losses that could result from mass vaccination in an elective vaccination program. This suggests that elective control is a viable option that has the advantage of being driven by private investment. Such an approach would require the liberalization of CBPP vaccine to empower individual livestock owners and private service providers to embark on elective programs.

The potential impact of treatment was modelled by reducing the average infection period by 25 and 50 per cent and observing the impact of the change on persistence on infection in the reference herd. It was found that a halving of the average infectious period could reduce herd level prevalence at the end of the modelling period from 75.4 to 33.2 per cent. Mortality in a 500 head herd was reduced from 178 to 64 head over a 6-year period. Thus, a reliable treatment regime could have impact as great as or even superior to currently available vaccines.

A combined program of treatment and vaccination was evaluated. Such a program would consist of treatment of all diseased animals and vaccination of the remainder of the herd. A five year program of biannual vaccination combined with treatment of cases was found to be the only option modelled that approached eradication. At the termination of the model, the reference herds remained infected in only 0.4 per cent of the iterations.

The relation between mortality and prevalence of infection was investigated in populations of 10,000 head. When a case fatality rate of 33.2 per cent was incorporated in the model, the predicted prevalence of infection was 6.3 per cent and the total mortality was 1,338 head over the life of the model. On the other hand when a case fatality rate of 4.7 per cent was used, the predicted prevalence of infection was 9.0 per cent and the total mortality loss was 235 head. This simulation illustrates the point that prevalence and impact are not necessarily positively correlated as high mortality rates can decrease the duration of infection leading to a lower prevalence of infection. Prominent epidemiological texts describe this phenomena as a key drawback to the use of prevalence surveys (Martin *et al.,* 1987; Rothman and Greenland, 1998).

Conclusions and Recommendations:

Based upon the results of the field investigations, modelling work and literature review, the following recommendations can be made relative to CBPP control:

- mass vaccination alone with the currently available T1 44 vaccine is unlikely to eradicate CBPP even when applied biannually over a 5-year period;
- a regular programme of mass vaccination can have a major impact on morbidity and mortality losses;
- a proven treatment regime would have considerable impact on both the prevalence of CBPP and the morbidity, mortality and production losses associated with CBPP infection;
- a combined vaccination and treatment programme offers potentially greater impact (up to 44 per cent over annual vaccination campaigns alone) than either approach in isolation;
- individual livestock owners who adopted a regular programme of elective control can capture most of the benefit in terms of reduced disease morbidity, mortality and production losses that would result from a mass vaccination programme even if in-contact herds do not practice vaccination;
- levels of movement control consistent with sustainable pastoral livelihoods are unlikely to have a major impact on CBPP prevalence, and in the current socio-economic climate movement control is unlikely to contribute to CBPP eradication in endemic areas.

The following recommendations can be made regarding CBPP surveillance and economic analysis:

- abattoir surveillance for CBPP lesions is a useful technique for the detection of CBPP in an area but should not be used to estimate prevalence due to problems of sensitivity and bias;

- prevalence surveys are an insufficient approach to the assessment of CBPP impact as prevalence and mortality impact are not necessarily positively correlated;
- broad-based impact assessments conducted at the community level that incorporate participatory approaches and purposive use of laboratory testing are more likely to correctly characterize CBPP morbidity, mortality and production losses than random sero-survey in isolation. Impact assessments can also serve as a reliable basis for the identification of realistic control options and economic analysis that includes livelihood effects.

Given the paucity of socio-economically acceptable tools for CBPP control, the near universal use of antibiotics for treatment of CBPP and the potential impact of well designed treatment regimes, research to validate treatment regimes should be conducted without delay. This neglected area warrants at least as much investment as vaccine development has enjoyed in the past. The objective of the research should be the identification of proven, affordable treatment schedules that can be incorporated into training messages for both veterinary staff and community animal health workers. Provided an effective regime is documented, an approach that combines treatment of clinical cases with vaccination of animals at risk should be developed.

Many national governments lack the means to carry out mass vaccination and international donors are highly unlikely to support an expensive and open-ended control programme. Due to funding constraints, the present policy of government-sponsored mass vaccination actually restrict livestock owner access to vaccine. Strategy guidelines need to be developed focussing on elective vaccination delivered by private and community-based service providers on a strictly commercial basis. Such an approach will require policy reforms that create adequate private sector access to regulated supplies of CBPP vaccine. This will enable livestock owners to develop reliable CBPP herd health programmes in consultation with their veterinarians and community-based service providers.

References

Mariner, J. C. (2000). Manual on Participatory Epidemiology. Food and Agriculture Organization, Rome, pp. 81.

Mariner, J. C., Aluma Araba, and Makungu S. (2003). Consultancy on the Dynamics of CBPP Endemism and the Development of Effective Control/Eradication Strategies for Pastoral Communities: Final Data Collection Report. Nairobi, The Community Animal Health and Participatory Epidemiology Unit of the African Union Interafrican Bureau for Animal Resources.

Martin, S.W., Meek, A.H. and Willeberg, P. (1987). Veterinary Epidemiology: Principles and Methods. Iowa State University Press, Ames, pp. 343.

Rothman, K.J. and Greenland, S. (1998). Modern Epidemiology. Lippincott – Raven, Philadelphia, pp. 738.

Within-herd spread and clinical observations of contagious bovine pleuropneumonia in Ethiopian Highlands (Boji district, West Wellega): methodology and epidemiological results

M. Lesnoff[1], G. Laval[1], S. Abicho[2], A. Workalemahu[3], D. Kifle[3], A. Peyraud[1], P. Bonnet[1], R. Lancelot[1], F. Thiaucourt[1].

[1]*Centre de coopération internationale en recherche agronomique pour le développement (CIRAD), Campus International de Baillarguet, 34398 Montpellier Cedex 5, France;*
[2]National *Animal Health Research Centre, P.O. Box 4, Sebeta, Ethiopia;*
[3]*International Livestock Research Institute (ILRI), P.O. Box 5689, Addis Ababa, Ethiopia*.

Introduction

Contagious bovine pleuropneumonia (CBPP) is a disease caused by the small-colony type of *Mycoplasma mycoides* subspecies *mycoides* (Masiga and Domenech, 1995). Transmission occurs from direct and repeated contacts between sick and healthy animals. It is a major threat for cattle health and production in Africa, where it was reported from 17 countries in 2001 (OIE, 2002). Few field data, however, were reported in the literature on within-herd spread of CBPP during outbreaks. A research programme was set up in a CBPP-infected zone in Ethiopian highlands (Boji district in West Wellega Zone) to estimate the epidemiological parameters of the disease and to assess the effects of different disease management strategies naturally implemented by the local farmers.

Material and methods

Boji district is characterized by a mixed crop-livestock farming system, in which herds are sedentary and small. Cattle are mostly of the Horro breed, an intermediate Sanga-zebu type. Animals are used for agricultural activities, milk, meat production and manure. Cattle exchanges (e.g. for loaning contracts) between farmers are frequent (Laval and Workalemahu, 2002; Lesnoff *et al.,* 2002).

Fifteen newly and naturally CBPP-infected herds (mean herd size = 17 animals) were sampled and followed for 16 months between June 2000 and January 2002. Each animal was ear-tagged. Trained enumerators visited the herds every 2 weeks to record demographic events (entry, birth, mortality, offtake), the symptoms of CBPP clinical cases and the type and cost of veterinary care applied. In case of death, a post-mortem diagnosis was established by the veterinary supervisors according to the clinical signs reported by the farmers and enumerators, and a necropsy (whenever possible) was realized. Blood samples were collected every 3 months from all animals to assess their serological CBPP status. Sera were tested with a competitive enzyme-linked immuno-sorbent assay (cELISA) test.

For each infected herd, a retrospective survey was conducted at the end of the follow-up period to determine the control measures implemented by the farmers to manage CBPP clinical cases. Two measures were identified: treatment of clinical cases with antibiotics and isolation from the rest of the herd. Two CBPP-control strategies were defined according to these practices: herds with complete antibiotic treatment or isolation (coded “C”), herds with no

antibiotic treatment and partial or null isolation (coded “P/N”). Herds for which the strategy was unknown were coded “UNK”.

Logistic-binomial regression models were used to analyse the serological incidence data from the 15 newly CBPP-infected herds. The follow-up was divided into successive 4-month periods starting at the CBPP-onset date. The response was the sero-incidence risk, i.e. the number of positive sero-conversions during each period, over the number of sero-negative cattle at the beginning of the period. Three statistical models were used to take into account for possible within-herd data correlation (Schukken, 2003). The first model was the ordinary logistic regression adjusted with the variance inflation factor (OLR+VIF). The other 2 models were generalized linear mixed-effect models (GLMM) in which herd was the random effect. Parameters of the GLMM were fitted either with the adaptative gaussian quadrature (AGQ) or a Monte-Carlo Markov chain (MCMC) algorithm.

Results

The statistical analysis of the serological incidence provided similar results with the different logistic-regression models (Table 1). The 95% confidence interval of the herd-effect variance in the MCMC model was [0.12; 1.42] and the estimated VIF was 2.2 (H_0: VIF = 1, P< 10^{-3}), which both confirmed the necessity to account for disease clustering. The sero-incidence risk decreased significantly with time (P < 0.01 at the most). For example, sero-incidence risks decreased from 20% in period 1 (t < 4 months after CBPP onset) to 5% in period 4 (MCMC estimates). Risks in period 3 and 4 (i.e. $8 \leq t < 12$ months and $12 \leq t < 16$ after CBPP onset) were > 0. The 16-month cumulative risk was 34% (25; 48) (95% confidence interval in brackets).

No evidence of farmer CBPP-control strategy effect was shown. The difference of the mean random-herd effects between strategies “P/N” and “C” was 0.271 on the logit scale (which represented a variation of 6% in the cumulative sero-incidence risk) and was not significant (randomisation test, B = 2,000 permutations, P = 0.432).

Table 1. CBPP sero-incidence and cumulative sero-incidence risks (%) estimated from 3 logistic-regression models for zebu cattle in 15 CBPP newly infected herds from West Wellega (Ethiopia).

Period (months)	OLR + VIF [a]		GLMM – AGQ [b]		GLMM – MCMC [c]	
	Inc.[d]	Cum. Inc.[d]	Inc.	Cum. Inc.	Inc.	Cum. Inc.
$0 \leq t < 4$	22 (16, 30) [a]	22 (16, 30)	20 (13, 29)	20 (13, 29)	20 (13, 28)	20 (13, 28)
$4 \leq t < 8$	8 (4, 15)	28 (21, 37)	8 (5, 14)	27 (19, 37)	8 (4, 14)	26 (18, 37)
$8 \leq t < 12$	7 (3, 15)	33 (26, 43)	7 (4, 12)	32 (23, 43)	7 (4, 13)	31 (22, 44)
$12 \leq t < 16$	5 (2, 13)	36 (29, 47)	5 (3, 8)	36 (26, 47)	5 (2, 10)	34 (25, 48)

Sample sizes were 278, 212, 197 and 155 cattle during the first, second, third and fourth 4-month periods after CBPP introduction.

[a] Estimated population mean and 95% confidence interval.

Clinical cases were recorded for 39% of the sero-positive cattle. Only 13% of these clinical cases died from CBPP and no antibiotic treatment effect was observed (3/20 untreated animals and 2/19 treated animals, Fisher's exact test P = 1). For animals surviving to the disease, the mean duration of the clinical signs was 4 weeks (median = 3, range = [1, 11]). The difference in the clinical-phase duration between untreated and treated animals was 1.2 weeks and not significant (Kolmogorov-Smirnof test, P = 0.73).

Discussion

The CBPP cumulative incidence and mortality risks observed in Boji district were lower than those reported from experimental challenges or from field outbreak in pastoral herd (incidence risk higher than 80% and mortality risks from 10% to 80%) (Hudson and Turner 19xx; Bygrave *et al.,* 1968; Masiga and Domenech, 1995). The animal confinement conditions, the virulence variability in MmmSC strains or an effect due to the small herd size (cattle management and disease control may be easier than in large herds) might be involved. A number of recovered or vaccinated animals could also have been introduced in Boji district through commercial or loaning flows, thus reducing the proportion of susceptible cattle. Further longitudinal studies are necessary to validate the observed low incidence and mortality in mixed-crop farming systems.

Incidence risks in periods 3 and 4 were not in agreement with the outbreak durations reported in literature (Hudson and Turner 19xx; Bygrave *et al.,* 1968; Provost *et al.,* 1987): in the absence of re-infection, most of the new CBPP sero-conversions should occur within 6-7 months after the initial introduction. The late sero-conversions observed during the present survey might have resulted from secondary CBPP introduction, occurring through unreported cattle importation or unobserved contacts with neighbouring and non-monitored infected herds.

The between-herd variability in CBPP incidence risks observed in the study was difficult to interpret. Antibiotics treatments associated with isolation of sick animals can reduce both incidence and mortalities. However, no effect of the CBPP-control measures, as implemented by the farmers, was observed. This might be related to a lack of power in the statistical analyses due to the small number of infected herds in the sample or to confounding factors (for example, the priority given by the farmers to the treatment of the most severely affected animals). On the other hand, it could reflect a quality problem for the medications used, and more generally, for health-care delivery in the Boji district. Most of the treated cattle during the follow-up survey received a single injection of a 10% oxytetracycline suspension (purchased on the informal market), and administered intra-muscularly at a dose of 10 ml per cattle by the farmers themselves. Such a treatment protocol was probably not appropriate to ensure CBPP recovery.

References

[1] Bygrave A.C. et al., (1968). Bull. epiz. Dis. Afr., 16, 21-46.
[2] OIE, (2002). Handistatus
[3] Hudson J.R., Turner A.W., 196. Aust. Vet. J., 39, 373-385.
[4] Laval G., Workalemahu A., 2002. Eth. J. Anim. Prod., 2, 97-114.
[5] Lesnoff M. et al., 2002. Rev. Elev. Méd. vét. Pays trop., 55,139-147.
[6] Masiga W.N. and Domenech J., 1995. Rev. Sci. tech. Off. int. Epiz., 14, 611-630.
[7] Provost A. et al., 1987. Rev. Sci. tech. Off. int. Epiz., 6 (3), 625-679.
[8] Schukken Y.H. et al., 2003. Prev. Vet. Med., 59, 223-240.

Molecular mechanisms of virulence and antigenicity of *Mycoplasma mycoides* subsp. *mycoides* SC: Conclusions for prevention and control of CBPP

Joachim Frey, Paola Pilo, Edy M. Vilei

Institute of Veterinary Bacteriology, University of Bern, Bern, Switzerland.

Introduction

Mycoplasma mycoides subspecies *mycoides* SC (*Mmm* SC), the etiological agent of contagious bovine pleuropneumonia (CBPP), is a highly virulent *Mycoplasma* species. In spite of the fact that this pathogen has been identified more than 100 years ago, its molecular mechanisms of pathogenicity and its virulence factors are still poorly known. However, in order to design novel rational diagnostic tools and safe and efficient vaccines to control this disease, it is essential to know the mechanisms of pathogenicity of this organism. So far no particular primary virulence factors such as toxins or invasins have been evidenced in *Mmm* SC, nor have primary virulence genes been among those *Mycoplasma* species whose full genomic sequences had been determined. This might be due to the extremely small genome of mycoplasmas leading them to a drastic economization in genetic resources which are reduced to essential functions of life (Razin *et al.,* 1998). Our current studies indicate that mycoplasmas seem to have adopted efficient structural surface antigens and basic metabolic pathway functions as virulence effectors to cause a disease.

Major surface antigens and their potential impact in virulence

Among the prominent surface antigens detected on the cellular membrane of mycoplasmas, and in particular of *Mmm* SC, are a large number of lipoproteins, variable surface antigens, ABC transporter proteins, metabolic pathway enzymes, adhesins and polycarbohydrates such as galactan.

Lipoproteins, in particular of mycoplasmas, are expected to play a role in mechanisms of pathogenicity since they are known to induce pro-inflammatory cytokines and might adopt the function of lipopolysaccharides which are missing in mycoplasmas (Mühlradt and Frisch, 1994; Herbelin *et al.,* 1994; Brenner *et al.,* 1997; Marie *et al.,* 1999; Calcutt *et al.,* 1999). Furthermore, lipoproteins are in general strongly antigenic proteins that might be valuable targets for specific and sensitive serodiagnosis. Their analysis in mycoplasmas, therefore, deserves particular attention. Variable surface antigens (Vsp) have been analysed extensively in a large number of mycoplasmas including *Mmm* SC (Persson *et al.,* 2002), *Mycoplasma bovis*, *Mycoplasma agalactiae*, *Mycoplasma penetrans*, *Mycoplasma gallisepticum* and *Mycoplama pulmonis* (Rosengarten and Wise, 1990; Citti and Rosengarten, 1997; Glew *et al.,* 2000). Vsps seem to play an important role in the mycoplasmas to escape the host's immune system. They have recently also been shown to modulate the susceptibility of mycoplasmas to complement killing hemadsorption and adherence (Simmons and Dybvig, 2003).

ABC carbohydrate transporters are common components in bacterial membranes since they serve the organism for import of various components used as energy source and nutrition and for the export of metabolic substances. Most metabolic pathway enzymes are cytoplasm-located. Surface located metabolic pathway enzymes are commonly involved in metabolic steps

that are closely related to uptake mechanisms or for elimination of substances badly tolerated in the cytoplasm. Blocking of surface located metabolic pathway enzymes by antibodies reduces or inhibits the ability of the organism to grow, a procedure that is currently used in the growth inhibition test.

Adhesins play a crucial role in the primary steps employed by mycoplasmas while interacting with their host eukaryotic cells using specific mammalian membrane receptors, a process that is assumed to be followed by either the invasion of host cells by mycoplasmas or by the production of signals by the mycoplasmas to subvert and damage the host cells.

Polycarbohydrates (glycans) are assumed to give the mycoplasma physico-chemical resistance against the host immune defence, but have also been shown to be involved in pathogenic mechanisms.

Lipoproteins of *M. mycoides* subsp. *mycoides* SC

Currently, a few lipoproteins of *Mmm* SC have been characterized (Table 1). Most of them are strong major antigens and are readily detected in serum of infected cattle on immunoblots. Lipoprotein A of *Mmm* SC is strongly conserved among mycoplasmas of the *M. mycoides* cluster, hence it cannot be used as a specific target for serodetection (Monnerat *et al.,* 1999). Its role in Th1 and Th2 immunity is currently under investigation. Lipoprotein B is found only in *Mmm* SC strains belonging to the African/Australian cluster, but it is not found in strains isolated from the re-emerging European outbreaks in 1980 – 2000. It is, however, also present in other mycoplasmas of the *M. mycoides* cluster (Vilei *et al.,* 2000). The role of two further lipoproteins LppC and LppD is under investigation. Lipoprotein Q (LppQ) seems to be a particular lipoprotein of *Mmm* SC as it is specific to this organism. It has a particularly strong antigenic N-terminal part which is located on the outer surface of the membrane, while its C-terminal part is involved in membrane anchoring (Abdo *et al.,* 2000). The high specificity and strong antigenicity of LppQ have been exploited for the development of a robust indirect ELISA test for serological diagnosis and for epidemiological investigations of CBPP (Bruderer *et al.,* 2002). Structural analysis of LppQ showed strong analogies to proteins with super-antigenic character (Abdo *et al.,* 2000). A recent study has shown that cattle immunized with purified recombinant LppQ, using different adjuvant methods, were significantly more susceptible to challenge with *Mmm* SC than cattle that were not vaccinated with LppQ (Otto Huebschle, personal communication[1]). Hence, LppQ is assumed to play an adverse reaction in vaccination similar to the peptidoglycan-associated lipoprotein PalA of *Actinobacillus pleuropneumoniae*, which was shown to inhibit completely beneficial effects of efficient subunit vaccines when animals were vaccinated simultaneously with PalA (van den Bosch and Frey, 2003). In this respect, it must be noted that the currently used life vaccines express LppQ, a matter to be considered in the development of new vaccine strains.

Variable surface proteins

A variable surface protein, designated Vmm, has recently been discovered in *Mmm* SC (Persson *et al.,* 2002). Vmm is a protein of 16 kDa and is specific to *Mmm* SC. It is expressed by nearly all strains that were analyzed, where it showed a reversible ON-OFF phase variation at a frequency of $9x10^{-4}$ to $5x10^{-5}$ per cell generation. Genes resembling the *vmm* gene were also

[1] Editors Note: Please see Individual Presebtations, Nicholas et al. In this report.

found in other species of mycoplasma, but the Vmm-like proteins in these species could not be detected with a specific monoclonal antibody directed to Vmm of *Mmm* SC. The function of Vmm is currently not known, but it is suggested to provide the pathogen flexibility on its outer surface and to help escape the host's immune system.

Table 1. Currently known antigens of *M. mycoides* subsp. *mycoides* SC.

Antigen	Function	Location	Impact in virulence	Effect in vaccine
LppA	lipoprotein	membrane	? (strong antigen)	under investigation
LppB	lipoprotein	membrane	unknown	probably no
LppC	lipoprotein	membrane	unknown	under investigation
LppQ	lipoprotein	membrane	strong (super) antigen	**adversary**
LppD	lipoprotein	membrane	unclear	?
GtsABC	glycerol uptake + phosphorylation	membrane	yes	possible candidate
GlpO	glycerol-3-phos-phate oxidase	membrane	**yes** (anti-GlpO block cytotoxicity)	**good candidate**
Bgl	6-phospho-beta glucosidase	cytosol	? bacterial survival	?
EIIBCA	sugar transport & phosphorylation	membrane	?	potential candidate
Vss	variable surface protein	membrane	escape of hots's immune defence	probably no

ABC transporter proteins

ABC transporter proteins are integral membrane proteins found in prokaryotes. They play a key role in the import and export of nutrition and energy sources and of metabolites and toxic substances across the membrane. The minimal genomes of *Mycoplasma* species contain an astonishingly large number of ABC transporter genes (Razin *et al.,* 1998). In *Mmm* SC type strain PG1, more than 30 ABC transporter genes have been discovered (Westberg, 2003). At present, only the glycerol uptake system GtsABC has been investigated in detail (Vilei and Frey 2001). In *Mmm* SC, this ABC transporter is involved in active glycerol uptake and glycerol phosphorylation. Glycerol is metabolized in *Mmm* SC after phosphorylation to dihydroxyacetone phosphate (DHAP) by an oxidative process leading to the release of the highly toxic compound H_2O_2. Blocking the glycerol uptake proteins GtsABC by specific antibodies results in a significant reduction of H_2O_2 production. Hence, it is suggested that the glycerol uptake system GtsABC is indirectly involved in virulence of *Mmm* SC. In this respect, it is important to note that, due to a deletion, the new emerging European strains of *Mmm* SC lack the *gtsB* and *gtsC* genes. These strains produce significantly lower amounts of H_2O_2 and also seem to be less virulent than African strains that possess the full *gtsABC* operon and are highly virulent.

Metabolic pathway enzymes

Metabolic pathway enzymes were generally well conserved during the evolution of prokaryotes. Therefore, many metabolic pathway genes of *Mmm* SC can be gained from the genomic sequence (Westberg, 2003). Currently, our laboratory is investigating the L-α-glycerophosphate oxidase GlpO of *Mmm* SC in detail. GlpO is the central enzyme involved in the metabolism, after phosphorylation, of both glycerol imported by the active GtsABC transporter and glycerol imported via the GlpF function to DHAP and H_2O_2. GlpO is membrane-located and allows to synthesize H_2O_2 directly on the surface of the organism and to release it to the environment and to the host cell, respectively, where it causes injury during infection (P. Pilo, E. M. Vilei, J. Frey, unpublished results). Preliminary studies using a cellular model based on calf epithelial cells revealed that the cytoxicity of *Mmm* SC is strongly reduced concomitantly to blocking H_2O_2 production, if the mycoplasmas are treated with monospecific polyclonal anti-GlpO antibodies (P. Pilo, E.M. Vilei, J. Frey, unpublished results). These studies show that GlpO could be an interesting target for the development of vaccines against CBPP.

Adhesins

Adhesins play a highly important role in the early steps of pathogenicity of most microorganisms. Since mycoplasmas do not secrete toxins that could act at long distances, adhesion is particularly important in the virulence of *Mmm* SC. Adhesion plays a central role in the intimate interactions with mammalian cells for long periods, assumed to trigger a cascade of signals which are transduced to the host cell and cause inflammation (Razin *et al.,* 1998). Adhesins also seem to be the basic principles of host specificity of *Mmm* SC for bovines since the other virulence factors, such as toxic metabolic substances and intermediates, are not expected to have any host predilection. Although several adhesins have been identified in various *Mycoplasma* species (Razin *et al.,* 1998; Belloy *et al.,* 2003), adhesins of *Mmm* SC have not yet been detected in spite of their potential primordial role in immune protection.

Polycarbohydrates

Polycarbohydrates, or glycans, have been postulated to play a role in virulence of *Mmm* SC, supposedly by protecting the mycoplasmas from the devasting actions of the cell immune system and by giving them the possibility to closely associate with the host's target cells (Gourlay and Shifrine, 1966). However, neither the chemical composition of the polycarbohydrates nor their direct role in virulence has been described for *Mmm* SC so far. Still certain monoclonal antibodies that are highly specific for the species of *Mmm* SC seem to react specifically with epitopes of polycarbohydrate material.

Conclusions for prevention and control

The knowledge about the molecular mechanisms of pathogenicity of *Mmm* SC has been significantly improved over the last four years, although only few virulence mechanisms are currently known. In particular, there is still no knowledge about the factors involved in the adhesion process, a key step of infection, which would most probably represent an ideal target in immuno-protection, and prevention of CBPP. On the other hand, evidence becomes available on specific targets of *Mmm* SC that are supposed to over-induce the host's immune system and

are consequently considered to cause adversary reactions in vaccines. Such reactions can enhance, rather than reduce, the symptoms caused by an infection by *Mmm* SC.

The corresponding antigens should therefore not be included in vaccines, or their genes must be deleted from vaccine strains. Other antigens, in contrast, are expected to induce antibodies that can block certain metabolic activities, thus avoiding the production of cytotoxic substances. The currently available molecular genetic methods are able both to generate targeted knockout mutants and to complement and genetically modify *Mmm* SC in the view of production of improved vaccine strains (Cordova *et al.,* 2002). Furthermore, efficient expression systems allow the production of substantial amounts of recombinant proteins from *Mmm* SC in heterologous bacterial hosts for use in subunit vaccines or as novel diagnostics (Abdo *et al.,* 2000; Bruderer *et al.,* 2002). It is therefore important to design general strategies or novel concepts of vaccines against CBPP (Table 2), which have to fulfil a broad range of requirements: They must protect efficaciously the animals against infection with *Mmm* SC and prevent from CBPP; they must fulfil veterinary medical and biological safety requirements; they should be economical in production and application; and, finally, they must be socially and politically acceptable.

Table 2. Basic types of vaccines

Type of Vaccine		efficacy	safety	production costs
Knockout mutant of *Mmm*SC	life GMO*	**+**	**++**	**low**
Genetically manipulated MmmSC Knockout and other mutations	life GMO	**++**	**++**	**low**
Subunit vaccine	dead	**+++**	**+++**	**high**
Recombinant carrier strain (e.g. Salmonella vaccinia strain)	life GMO	**+++**	**+++**	**low**
DNA vaccine	dead**	**?**	**?**	**?**

***Only endogenous modifications no foreign DNA**
****often submitted to similar regulations as GMOs**

References

Abdo, E. M., Nicolet, J., and Frey, J. (2000a). Antigenic and genetic characterization of lipoprotein LppQ from *Mycoplasma mycoides* subsp. *mycoides* SC. Clinical and Diagnostic Laboratory Immunology **7,** 588-595.

Belloy, L., Vilei, E. M., Giacometti, M., and Frey, J. (2003). Characterization of LppS, an adhesin of *Mycoplasma conjunctivae*. Microbiology **149,** 185-193.

Brenner, C., Wroblewski, H., Le Henaff, M., Montagnier, L., and Blanchard, A. (1997). Spiralin, a mycoplasmal membrane lipoprotein, induces T-cell-independent B-cell blastogenesis and secretion of proinflammatory cytokines. Infection and Immunity **65,** 4322-4329.

Bruderer, U., Regalla, J., Abdo, E.-M., Huebschle, O. J. B., and Frey, J. (2002). Serodiagnosis and monitoring of contagious bovine pleuropneumonia (CBPP) with an indirect ELISA based on the specific lipoprotein LppQ of *Mycoplasma mycoides* subsp. *mycoides* SC. Veterinary Microbiology **84,** 195-205.

Calcutt, M. J., Kim, M. F., Karpas, A. B., Mühlradt P.F., and Wise, K. S. (1999). Differential posttranslational processing confers intraspecies variation of a major surface lipoprotein and a macrophage-activating lipopeptide of *Mycoplasma fermentans*. Infection and Immunity **67,** 760-771.

Citti, C., and Rosengarten, R. (1997). Mycoplasma genetic variation and its implication for pathogenesis. Wien. Klin. Wochenschr. **109,** 562-568.

Cordova, C. M., Lartigue, C., Sirand-Pugnet, P., Renaudin, J., Cunha, R. A., and Blanchard, A. (2002). Identification of the origin of replication of the *Mycoplasma pulmonis* chromosome and its use in *oriC* replicative plasmids. Journak of Bacteriology **184,** 5426-5435.

Glew, M. D., Papazisi, L., Poumarat, F., Bergonier, D., Rosengarten, R., and Citti, C. (2000). Characterization of a multigene family undergoing high-frequency DNA rearrangements and coding for abundant variable surface proteins in *Mycoplasma agalactiae*. Infection and Immunity **68,** 4539-4548.

Gourlay, R. N., and Shifrine, M. (1966). Antigenic cross-reactions between the galactan from Mycoplasma mycoides and polysaccharides from other sources. Journal of Comparative Pathology **76,** 417-425.

Herbelin, A., Ruuth, E., Delorme, D., Michel-Herbelin, C., and Praz, F. (1994). *Mycoplasma arginini* TUH-14 membrane lipoproteins induce production of interleukin-1, interleukin-6, and tumor necrosis factor alpha by human monocytes. Infection and Immunity **62,** 4690-4694.

Marie, C., Roman-Roman, S., and Rawadi, G. (1999). Involvment of mitogen-activated protein kinase pathways in interleukin-8 production by human monocytes and polymorphonuclear cells stimulated with lipopolysaccharide or *Mycoplasma fermentans* membrane lipoproteins. Infection and Immunity **67,** 688-693.

Monnerat, M. P., Thiaucourt, F., Nicolet, J., and Frey, J. (1999). Comparative analysis of the *lppA* locus in *Mycoplasma capricolum* subsp. *capricolum* and *Mycoplasma capricolum* subsp. *capripneumoniae*. Veterinary Microbiology **69,** 157-172.

Mühlradt P.F. and Frisch M. (1994). Purification and partial biochemical characterization of a *Mycoplasma fermentans*-derived substance that activates macrophages to release nitric oxide, tumor necrosis factor and interleukin-6. Infection and Immunity **62,** 3801-3807.

Persson, A., Jacobsson, K., Frykberg, L., Johansson, K. E., and Poumarat, F. (2002). Variable surface protein Vmm of *Mycoplasma mycoides* subsp. *mycoides* small colony type. Journal of Bacteriology **184,** 3712-3722.

Razin, S., Yogev, D., and Naot, Y. (1998). Molecular biology and pathogenicity of mycoplasmas. Microbiology Molecular Biology Reviews **62,** 1094-1156.

Rosengarten, R., and Wise, K. S. (1990). Phenotypic switching in mycoplasmas: phase variation of diverse surface lipoproteins. Science **247,** 315-318.

Simmons, W. L., and Dybvig, K. (2003). The Vsa proteins modulate susceptibility of *Mycoplasma pulmonis* to complement killing, hemadsorption, and adherence to polystyrene. Infection and Immunity **71,** 5733-5738.

van den Bosch, H., and Frey, J. (2003). Interference of outer membrane protein PalA with protective immunity against *Actinobacillus pleuropneumoniae* infections in vaccinated pigs. Vaccine **21,** 3601-3607.

Vilei, E. M., Abdo, E.-M., Nicolet, J., Botelho, A., Gonçalves, R., and Frey, J. (2000). Genomic and antigenic differences between the European and African/Australian clusters of *Mycoplasma mycoides* subsp. *mycoides* SC. Microbiology **146,** 477-486.

Vilei, E. M., Frey, J., 2001. Genetic and biochemical characterization of glycerol uptake in *Mycoplasma mycoides* subsp. *mycoides* SC: Its impact on H_2O_2 production and virulence. Clinical and Diagnostic Laboratory Immunology **8,** 85-92.

Westberg, J. (2003). Genome sequencing: Analysis of pathogenicity factors in hereditary and bacterial diseases. Thesis/Dissertation, Department of Biotechnology, Albanova University Center, Royal Institute of Technology. Stockholm, Sweden.

An inactivated whole cell vaccine and LppQ subunit vaccine appear to exacerbate the effects of CBPP in adult cattle

R. A. J. Nicholas[1], G. Tjipura-Zaire[2], R. S. Mbulu[2], M. Scacchia[3], F. Mettler[2], J. Frey[4], I. Abusugra[5] and O. J. B. Huebschle[2]

[1]*Veterinary Laboratories Agency (Weybridge)*, *Addlestone, UK*
[2]*Central Veterinary Laboratory, Windhoek, Namibia*
[3]*Istituto Zooprofilattico Sperimentale, Teramo, Italy*
[4]*Institute for Veterinary Bacteriology, University of Bern, Bern, Switzerland*
[5]*Department of Virology, Swedish University of Agricultural Sciences, Uppsala, Sweden*

Introduction

The limitations of the live T1 vaccine have been well documented (Rweymamu *et al.,* 1995). They include the provision of only short term and incomplete immunity and contain residual virulence. A continual appeal from affected countries is the need for better vaccines but these seem a long way off. Inactivated vaccines have been reported for a number of mycoplasma diseases including enzootic pneumonia in pigs, contagious agalactia (Tola *et al.,* 1999) and contagious caprine pleuropneumonia (Rurangirwa *et al.,* 1995). Indeed dead vaccines for CBPP were tried in Africa and shown to be relatively efficacious (Gray *et al.,* 1986; Garba and Terry, 1986). Recent work has shown that a saponin inactivated vaccine against *M. bovis*, the cause of calf pneumonia, provided significant protection against a strong challenge both by aerosol and contact (Nicholas and Ayling, 2003). Lung lesions and mycoplasma dissemination were significantly reduced in vaccinated calves compared to infected controls. We decided to use the same approach to develop an inactivated vaccine for CBPP. In addition some cattle were vaccinated with the purified recombinant LppQ lipoprotein to see whether this could provide protection (Abdo *et al.,* 2000). Cattle infected by intubation were used to challenge all vaccinated cattle by contact.

Materials and methods

Saponin vaccine

The Afadé strain of *Mycoplasma mycoides* subspecies *mycoides* SC (*Mmm* SC) was cultured in bulk, distributed into ampoules of 1 ml and stored at –70°C to provide a standardised source of inocula. The master seed culture was thawed and dispensed into 10 ml quantities of Eaton's broth media. After 48-72 h 10 ml broths were subcultured into 100 ml, and after the same time these broths were inoculated into vessels of greater or equal to 1 l and incubated for a further 2 days. The precise protocol (incubation times, final culture volumes) was established in preliminary experiments. Washed cells were inactivated with 2% saponin (Sigma, UK) at 37°C for 2 h. To check successful inactivation, 0.5ml of saponised cells were inoculated into 5 x 20 ml of Eaton's broth; saponised cells were also directly plated onto Eaton's medium containing agar. After five days, 0.5 ml of incubated broths were subcultured into fresh broths and plates of above media. This procedure was followed every five days for a total of 30 days. Plates were examined daily for 7 days then discarded. No mycoplasma growth was seen at any time.

LppQ

The production of the recombinant N'-terminal half of the lipoprotein Q was described by Abdo *et al.,* (2000).

Vaccination

All animal experiments were carried out in the Mashere district of Northern Namibia where CBPP is endemic. Four 7-8 year old cattle were subcutaneously vaccinated with 1 ml of the saponised vaccine on two separate occasions 6 weeks apart. The same numbers of cattle were inoculated with 1 ml of recombinant LppQ in ISCOM construct at the same time intervals. Approximately 4 months later all animals were placed in contact with intubated cattle (see below).

Challenge

Ten 7–8 year old cattle were intubated tracheo-bronchially, and the procedure which was monitored with the aid of a bronchoscope, was performed by inserting a horse stomach tube into the trachea till the bifurcation and kept in position until completion of the intervention. Each animal was infected with 10 ml of *Mmm* SC culture from the 2nd passage, titre 10^9/ml suspended in 25 ml of 2% agarose. Forty ml of culture medium was used to flush down the suspension to the target site.

To simulate natural disease the saponin and LppQ vaccinated cattle were placed into the same paddock where the intubated cattle were held. In addition 8 unvaccinated control cattle of the same age were also introduced.

For the entire observation period, all cattle were fed with lucerne and had a common drinking trough. They were monitored clinically, serologically and bacteriologically. All testing was done at the Central Veterinary Laboratory, Windhoek. The LppQ ELISA was performed according to Bruderer et al., (2002).

Results

Intubated cattle

On day 10 post-intubation a serological response was detected by CFT in all cattle. Peak titres ranged from 1:1000 to 1:10 000 and the response was maintained at high levels for about 75 days. Although a gradual decrease of CF titres was then recorded all animals still showed a clear serological reactivity (± 1:80) at the time of slaughter. Temperatures exceeding 39.0°C were recorded between day 7 and 15 post-intubation and persisted over a period varying between 2 and 10 days. Animals showed respiratory distress characterised by coughing from day 15 post-intubation onwards. No mortality was observed during the observation period. At slaughter 149 days post-intubation, 5/10 animals showed small but specific CBPP pulmonary changes and *Mmm* SC was isolated from lung tissues (see Table 1).

In contact control cattle

On day 35 post-exposure (p.e.) one cow (9442) died, no lesions due to *Mmm* SC were observed during post-mortem examination and no mycoplasma was isolated from either lung or lymph node specimens collected at post-mortem. Death was due to causes other than CBPP. In the 3 cows that died later, in the course of the experiment, and in the remaining 4 animals that were sacrificed at the end of the experiment, low and sporadic CF antibody response was detected from day 91 onwards but rose massively in the survivors three weeks before the end of the experiment. Temperatures ranging between 39.1°C and 40.3°C were first recorded on day 124. The only exception was cow 9610 that was febrile since day 103 p.e. (not shown) showed serological reactivity on sample taken on day 105 and died on day 119. *Mmm* SC was isolated and antigen detected by PCR and immunocytochemistry (ICC) testing in lung and/or lymph node tissue samples taken from all 7 cattle.

Pathological changes observed either after death or at slaughter were: increased consistency of the lung, enlargement and marmorisation affecting the apical and/or diaphragmatic lobes and in some instances the entire lung, adhesion between visceral and parietal pleura. Sequestra when present were small. In one case both lungs were affected. Peribronchial and mediastinal lymph nodes were hyperplastic, of increased consistency and in some instances haemorrhagic (see Table 1).

Table 1. Pathological findings of vaccinated and contact cattle following exposure to CBPP

Animal number	Pathology	Respiratory distress	CF titre at death
Saponin			
9853	HLN, PF, LS-DL (x2)	Yes	1/10240++++
9734	HLN, LS-DL, HPZ/M	No	1/10240++++
9874	HLN, LS-DL	No	1/2560++++
9405	Died (CBPP) PF, HLN, LS-DL, PP	Yes	1/320++
LppQ			
9623	ES, HLN, HPZ/M-A&CL	No	1/5120++++
9809	HLN, HPZ/M-DL, Adhesions, PF	No	1/2560++++
9231	Died (CBPP)	Yes	1/640+++
9547	Died (CBPP)	Yes	1/2560+++
Contact			
9541	HLNs, Enlarged DL, PP, SS-AL	No	1/2560++
9428	HLNs, LS –AL, HPZ/M-DL, PP	No	1/2560++
9156	HLN, HPZ/M-DL	No	1/1280+
9851	HLN, HPZ/M-entire lung	No	1/5120+++
9719	Died (CBPP)	Yes	1/2560+++
9818	Died (CBPP)	Yes	1/2560+
9610	Died (CBPP)	Yes	1/640++++
9442	Died (not CBPP)	Yes	0

Abreviations:
AL- apical lobe
DL- diaphragmatic lobes
ES- enlarged spleen
HLN- hyperpalstic lymph nodes
HPZ/M-hepatisation/marbling
LS- large sequester
PF- pleural fluid
PP- Pleura parietalis
SS- small sequester

Saponin vaccinated cattle

Antibody titres as detected by CFT developed within two weeks of the first vaccination to significant levels of 1/40-1/320 (Table 2). A second vaccination 6 weeks later boosted 3 of the 4 cows to 1/320. Titres then declined in all cattle to 1/10-1/20 just before exposure to challenge. Titres remained at these levels for the next 4 months, and then there was a sudden massive increase just prior to post mortem which coincided with the death of one of the cows (see Table 1).

Table 2. Serological response of cattle vaccinated with saponin vaccine

Date	CFT titres Animal number			
	#9405	#9734	#9853	#9874
18.01.02*	0	0	0	0
31.01.02	1/40++	1/320+	1/80+++	1/80+
15.02.02	1/20+	1/80+++	1/80+++	1/40+++
05.03.02**	0	1/10++	1/20+	1/10+
19.03.02	1/40+++	1/320+	1/320+	1/320+++
03.04.02	1/160++	1/160+	1/160+++	1/320+
09.04.03	1/10	1/80++	1/160+	1/80+
29.05.02***	1:10+++	1:10+++	1:20+	1:10++++
12.06.02	1:20++	1:40+	1:20++	1:40+
26.06.02	NS	1:10++	1:20++	1:40+
10.07.02	1:10++++	1:20+	1:10++++	1:20+
24.07.02	1:10++++	1:10+++	1:10++++	1:10++++
07.08.02	1:10+	1:10++	1:20+++	0
21.08.02	0	1:10++++	1:20+++	1:10++++
04.09.02	1:40+++	1:20+	1:20+++	1:40+
18.09.02	1:20++++	1:40+	1:20++++	1:10+++
02.10.02	1:320++	1:640++++	1:640+	1:20++++
21.10.02	Dead [CBPP]	1:10240++++	1:10240++++	1:2560++++

* 1st vaccination
** 2nd vaccination
*** animals exposed to infection

LppQ ISCOM vaccinated cattle

LppQ antigen does not elicit CFT antibodies so the homologous ELISA test was performed. Antobody to LppQ was detected within two weeks of the first vaccination and remained high until challenge. Following exposure, titres remained high until 1 month before postmortem when titres rose rapidly. Sporadic low CFT titres were seen over the four months prior to exposure to challenge and remained at this state until one month before post mortem when another massive increase was seen which also saw the death of two cows (Table 3). The two remaining cows had titres of over 1/2560 at post mortem (see Table 1).

Table 3. Serological response of cattle vaccinated with LppQ vaccine

	LppQ CFT/ (ELISA) titres			
	Animal number			
Date	#9231	#9547	#9623	#9809
18.01.02*	0 (57)	0 (3)	0 (2)	0 (90)
31.01.02	0 (284)	0 (117)	1/20+ (234)	0 (295)
15.02.02	0 (255)	1/20+ (103)	1/20++ (205)	0 (312)
05.03.02**	0 (194)	0 (135)	0 (167)	0 (282)
19.03.02	0 (>333))	0 (>333)	1/10+++ (>333))	1/10+++ (>333))
03.04.02	0	0	0	0
09/24.04.02	0 (>333)	1/10++(>333)	0 (>333)	1/10+++ (>333)
29.05.02***	0 (13)	1:10++ (>333)	0 (>333)	1:10+++ (>333)
12.06.02	0 (271)	0 (>333)	1:10+++ (>333)	0 (>333)
26.06.02	0	0	0	0
10.07.02	0 (312)	0 (>333)	0 (>333)	0 (>460)
24.07.02	0	0	0	1:10+++
07.08.02	0 (214)	1:10+ (474)	0 (540)	0 (426)
21.08.02	0	0	0	0
04.09.02	1:10++(>460)	0 (>460)	1:20+ (>460)	1:40+ (349)
18.09.02	1:640+++(>460)	0 (363)	1:10++++ (>460)	1:20++++ (>460)
02.10.02	Dead [CBPP]	1:2560++(>460)	1:80+ (>460)	1:10+++ (>460)
16.10.02		Dead [CBPP]	1:5120+++ (>460)	1:1280++(>460)
22.10.02			1:2560++++(>460)	1:5120+++ (>460)

* 1st vaccination
** 2nd vaccination
*** animals exposed to infection

Discussion

While live vaccines have always been used for CBPP control, there have been a few reports of experiments using inactivated vaccines. Adult cattle that received two large doses of a heat inactivated Blenheim vaccine strain containing complete Freund's adjuvant received better protection than when given a single dose of the same vaccine or T1 broth vaccine (Gray *et al.,* 1986). A formalised preparation of 10^{10} CFU/ml of the Gladysdale strain emulsified with liquid paraffin gave similar protection to the T1 broth vaccine whether the vaccine was used immediately after inactivation or stored for 1 and 2 years or challenged 3 or 6 months after vaccination (Garba and Terry 1986). In spite of these encouraging results no further work on inactivated vaccines has been published.

The present work took two different approaches to vaccination against CBPP: first, the use of a whole cell mycoplasma vaccine inactivated with saponin, an approach which had previously been found to be protective for calf pneumonia, contagious agalactia and CCPP; and second, the use of a recombinant subunit vaccine prepared from the highly immunogenic lipoprotein LppQ. In spite of two vaccinations at 6 weekly intervals there was no evidence in the small number of animals used of any protection afforded by either preparation; indeed, although it was difficult to quantify, there appeared to be an exacerbation of pathology in the vaccinated animals compared to unvaccinated contact animals. Lesions and fibrin were most extensive and pleural fluid more abundant in vaccinated animals. In the LppQ group 2 of 4 cattle died

before the end of the experiment while 1 of 4 died in the saponin group. This compares to 3 of 7 that died in the control group.

An interesting aspect of this experiment was the length of time it took the unvaccinated groups to seroconvert: following challenge apart from the odd sporadic positive CFT titres; it was three months after exposure before cattle seroconverted and then titres rose rapidly. Strong vaccine antibody titres were seen in the saponin group two weeks after the second vaccination and these declined slowly but remained detectable for a further 6 months before rising rapidly prior to post mortem. It would have been interesting, had resourced permitted, to have seen how the T1/44 vaccine performed under these conditions.

References

Abdo, E.-M., Nicolet, J. and Frey, J. (2000). Antigenic and genetic characterization of lipoprotein LppQ from *Mycoplasma mycoides* subsp. *mycoides* SC. Clinical Diagnosis and Laboratory Immunology **7**, 588-595.

Bruderer, U., Regalla, J., Abdo, E. M., Huebschle, O. J. B. and Frey, J. (2002). Serodiagnosis and monitoring of CBPP with an indirect ELISA based on LppQ of *Mycoplasma mycoides* subsp. *mycoides*. Veterinary Microbiology **84,** 195-205.

Garba, S. A. and Terry, R. J. (1986). Immunogenicity of iol based CBPP vaccines in cattle. Vaccine **4,** 266-270.

Gray, M. A., Simam, P. and Smith, G. R. (1986). Observations on experimental inactivated vaccines for CBPP. Journal of Hygeine (Camb) **97,** 305-315.

Nicholas, R. A. J. and Ayling, R. D. (2003). *Mycoplasma bovis*, disease, diagnosis and control. Research in Veterinary Science **74,** 105-112.

Rurangirwa, F. R., McGuire, T. C., Kibor, A. and Chema, S. (1987). An inactivated vaccine for contagious caprine pleuropneumonia. Veterinary Record **121,** 397-402.

Tola, S., Manunta, D., Rocca, S., Rocchiagiani, A. M., Idini, G., Angioi, P. P. and Leori, G. (1999). Experimental vaccination against *Mycoplasma agalactiae* using different inactivated vaccines. Vaccine X, 2764-2768.

CBPP vaccine strain T1 44: possible reversion to virulence

H. Wesonga[1], L. Manso Silvan[2] and F. Thiaucourt[2]

[1]*KARI, Muguga, Kenya,*
[2]*CIRAD-EMVT, Montpellier, France.*

Introduction

The occurrence of post-vaccinal reactions has always been a burden for veterinary services using the T1 44 vaccine strain. These reactions occur with an unpredictable frequency; none sometimes, but up to 11% in some instances (Lindley, 1971). The lesions observed in those cases were strictly similar to what was observed by Louis Willems when he inoculated CBPP pleural fluid subcutaneously. It is characterized by an oedema that appears 10 to 20 days after inoculation. This oedema may vary in size and in some cases it extends to the dewlap and causes the death of the animal. In the past, veterinary services have used antibiotics such as tylosin, in order to treat animals with post-vaccinal reactions.

The origin of these “Willems reactions” was never elucidated. Most authors advocated susceptibility variations in the vaccinated animals. It was unclear if this exacerbated susceptibility was of genetic origin or due to other causes. Another explanation could be a reversion to virulence of the T1 44 strain. An experiment was designed in order to test this hypothesis.

Methods

The protocol included the use of 3 *Mmm* SC strains; the T1 44 vaccine strain as a control for an attenuated strain, a local pathogenic isolate as a control for a virulent strain and T1 B, a strain that had been isolated previously from a “Willems reaction” following vaccination with T1 44 in a previous experiment (Wesonga *et al.,* 2000). Each strain was inoculated subcutaneously to 5 animals and the reaction induced was followed. The size of the local reaction was recorded as well as the body temperature for each animal.

Results

The control groups displayed the expected results. In the T1 44 group, only very small local reactions were measured. None of them exceeded 10 cm in diameter. None of the animals displayed fever. In the pathogenic strain group all animals displayed local lesions that were much more pronounced although there were marked differences between individuals. In two cases the oedema extended to the dewlap and animals were treated with antibiotics to prevent death. The group inoculated with the T1 B strain reacted similarly to that inoculated with the pathogenic strain.

Discussion

This experiment clearly demonstrates that strain T1 B has reverted to virulence. The difference of response is significant in spite of the low number of animals. This result opens new perspectives for research on CBPP. It is an indication that few genes may be involved in virulence. If multiple genes were involved, it would have been very unlikely to observe a full reversion to virulence during a single animal passage. As a consequence, the comparison between T1 44 and T1 B may allow an identification of the most important virulence genes and an understanding of their mechanism. In a future prospect it will also allow the construction of attenuated strains by the specific deletion of these genes (the final proof of their involvement in virulence). From a practical point of view, these results certainly do not call for an abandonment of strain T1 44 that confers good protection rates. It simply calls for caution when using it for the first time in a cattle population. Experience has shown that in subsequent vaccination campaigns the frequency of post-vaccinal reactions was reduced sharply.

References

Lindley, E. P. (1971). Experiences with a lyophilised contagious bovine pleuropneumonia vaccine in the Ivory Coast. Tropical Animal Health and Producton **3,** 32-42.

Wesonga, H. O. and Thiaucourt, F. (2000). Experimental studies on the efficacy of T_1SR and $T_1$44 vaccine strains of MmmSC against a field isolate causing contagious bovine pleuropneumonia in Kenya, Effect of a revaccination. Revue Elevage Medicine Veterinaire Pays Tropicaux **53,** 313-318.

Willems, L. (1852). Mémoire sur la pleuro-pneumonie épizootique du gros bétail. Recueil de médecine vétérinaire pratique **9**, 401-434.

Contagious bovine pleuropneumonia (CBPP) vaccine strains T1 44 and T1 SR: a dose effect trial

Aboubacar Yaya[1] and Francois Thiaucourt[2]

[1]*LANAVET, Garoua,Cameroon*
[2]*CIRAD, EMVT, Montpellier, France*

Introduction

When CBPP was reintroduced in Botswana in 1995 after more than 50 years of absence (Amanfu, 1998), a logical strategy was put in place to contain the spread of the disease by forbidding cattle movement from the infected zone towards free areas and to perform a massive vaccination campaign ahead of the disease front. The vaccine used was the T1 SR strain and it was administered to thousands of cattle in an emergency vaccination campaign. However logical the strategy and correct the implementation plans were, they failed to contain the disease. CBPP soon spread to regions where the vaccination had been performed. Subsequently, an ultimate massive slaughter strategy was put in place and all the cattle population in the infected zone were destroyed. This enabled Botswana to regain its CBPP-free status by the most rapid way although at a high initial cost. One of the tentative first explanations for the failure of the vaccine strategy was to advocate an error that could have been made when handling the seed strain of vaccine, a less potent strain might have replaced the original one. This proved not to be the case, as a subsequent polymerase chain reaction (PCR) technique (Lorenzon, 2002) clearly identified the vaccine used in Botswana as T1. The quality of the vaccine itself was not considered as a possible cause for the failure, given that it had passed all the internal controls by the producer and also from an independent laboratory (PANVAC) in Ethiopia.

Therefore, an international study was put in place to investigate the protection afforded by the two most well known CBPP vaccine strains T1 SR as a control for what happened in Botswana and T1 44, which was considered a more potent strain. When these strains were injected at the minimum dose recommended by the OIE (10^7 live mycoplasmas per dose) the protection afforded three months later was quite disappointing: from 30 to 60%. This would obviously not be sufficient to prevent the contamination of many animals, even if the vaccine coverage was high. A possible way to increase the protection afforded by these types of vaccines may be to increase the number of mycoplasmas per dose. However, there are very few data concerning the relationship between dose of vaccine and protection (Gilbert and Windsor, 1971). This is the reason why a new trial was put in place.

Materials and Methods

Two groups of 45 animals were vaccinated with strain T1 44 and T1 SR respectively. Within each group, 3 subgroups of 15 animals were injected with 10^7, 10^8 and 10^9 live mycoplasmas. Three months later, CBPP was artificially reproduced in a group of 22 animals inoculated by intubation with a culture of a local MmmSC pathogenic strain (Touroua) used in a previous challenge study. These intubated animals were put in contact with the vaccinated animals and a control group of 39 naive animals as soon as clinical signs of CBPP were evident. All the animals were slaughtered two months after the initial signs of disease in the control group. Lesions were monitored and a scoring system, modification from that by Hudson and Turner, was established.

Results and Discussion

CBPP transmission to the control group was successful and the mortality rate was 32%. In addition, most of the surviving animals displayed typical lesions of CBPP with or without seroconversion and presence of MmmSC.

The mortality rate in the vaccinated animals was significantly reduced (6%) and the protection was also evident from the shift in the lesion score distributions. These lesion score distributions seem to indicate a better protection afforded by strain T1 44 (70%) as compared to T1 SR (60%), although differences are not statistically significant. There is also no obvious pattern of correlation between dose and protection. The most likely explanation is that variations in protection are linked to the presence of some highly susceptible animals in one or the other group. The relatively limited size of each group and the degree of variations and individual susceptibility did not allow us to observe any significant trend. However, our results show that if there is a correlation between dose and protection within the range that was tested in this experiment, this correlation is not very marked. Accordingly, it is unlikely that injecting much higher dosages (10^9 mycoplasmas for example) will result in a significant increase in protection level. Increasing the viable content may be very helpful as it may increase the shelf life of the product. It has to be remembered that, although the minimum advocated dose is 10^7 organisms (Lefèvre, 2000), previous authors have advocated the production of vaccine vials containing 10^8 mycoplasmas per dose (Provost, 1987) in order to take into account the very probable losses of titre from the production site to the time and place when the vaccine is actually injected to cattle. Alternative ways to increase the protection rate with the present vaccines may be to perform a booster injection shortly after the first one. Such a trial is going on right now and results should be obtained by next year.

References

Amanfu, W., Masupu, K. V., Adom, E. K., Raborokgwe, M. V. and Bashiruddin, J. B. (1998). An outbreak of contagious bovine pleuropneumonia in Ngamiland district of north-western Botswana. Veterinary Record **143**, 46-48.

Gilbert, F. R. and Windsor, R. S. (1971). The immunizing dose of T1 strain *Mycoplasma mycoides* against contagious bovine pleuropneumonia. Tropical Animal Health and Production **3**, 71-76.

Hudson, J. R. and Turner A. W. (1963). Contagious bovine pleuropneumonia: a comparison of the efficacy of two types of vaccine. Australian Veterinary Journal **39**, 373-385.

Lefèvre, P. C. (2000). Contagious bovine pleuropneumonia in "OIE manual of standards for diagnostic tests and vaccines. 12 rue de Prony 75017 Paris France" pp 957.

Lorenzon, S., David, A., Nadew, M., Wesonga, H. and Thiaucourt, F. (2002). Specific PCR identification of the T1 vaccine strains for contagious bovine pleuropneumonia. Molecular and Cellular Probes **14**, 205-10.

Provost, A., Perreau, P., Breard, A., Le Goff, C., Martel, J. L. and Cottew, G. S. (1987). Contagious bovine pleuropneumonia. Revue Scitifique et Technique Office International des Epizooties **6**, 625-679.

Improved formulations for existing CBPP vaccines: Recommendations for change

John B. March

Moredun Research Institute, Pentlands Science Park, Bush Loan, Penicuik, EH26 0PZ, UK

Introduction and Background to the Problem

Contagious bovine pleuropneumonia (CBPP) is currently the most economically serious disease of cattle in Africa, and in the last decade there has been a substantial re-emergence of the disease, despite vaccination campaigns using freeze dried broth cultures of live attenuated *Mycoplasma mycoides* subspecies *mycoides* small colony biotype (*Mmm* SC) (strain $T_1$44 or T_1SR). The disease was successfully eradicated from the United Kingdom, North America and much of Western Europe by policy of movement restriction and slaughter during the late 19th century. The disease was also eradicated from Australia by the 1960s, following a successful vaccination campaign that began in the mid-1930s (Newton, 1992). Although other control measures undoubtedly played their part in the successful eradication of CBPP from Australia, the CBPP vaccine, using live broth cultures of *Mmm* SC strain V5 has been hailed as 'one of the most important developments in the fight against the disease' (Newton and Norris, 2000). However, such 'non-vaccine based' control measures cannot realistically be applied in Africa due to economic, cultural and social conditions, meaning that effective vaccination is the only realistic policy for CBPP eradication.

Several publications and expert group meetings have reported on the inability of current vaccines to control the disease in Africa (Anon, 1999, 2001; Masiga and Domenech, 1995; Nicholas *et al.,* 2000), and research designed to produce 'next generation' vaccines (e.g. ISCOM, capsular polysaccharide conjugate) has proceeded, although with little apparent success (Absurga *et al.,* 1997; Waite and March, 2002). It is likely that further research findings based upon novel vaccine technologies will also be presented at this meeting. My own research group have also been involved in testing newer technologies including recombinant proteins, DNA vaccines, and even derivatives of wheat carbohydrates have been tested by us (the use of flour as a vaccine for CBPP remains a distant goal!). Unfortunately, for all the scientific interest of these technologies, they are unlikely to produce a usable CBPP vaccine for the foreseeable future. Major issues include:

(i) Cost: The financial burden of developing and testing 'next generation' vaccines is likely to be in the order of several million dollars at the very minimum, even if much of the animal testing can be carried out in Africa, and to a lower standard of safety than otherwise may be demanded for equivalent 'Western' animal vaccines. If equivalent standards of efficacy, safety and licensing were to be demanded then the cost is likely to run into tens if not hundreds of millions of dollars. It is unclear where such funding would come from; the private sector is unlikely to put forward such large sums of money, particularly with little or no possibility of return on investment, and funding by aid agencies and research trusts is few and far between and certainly not of the order of magnitude required.

(ii) Time: Even should sufficient funding be in place, it is likely to take a minimum of 10 years before any new vaccine could be tested, validated and put into production and be ready for widespread use in the field. The intermittent nature of much of today's research and development funding means that the time taken to produce a vaccine may be even longer. ***What vaccines are to be used in the meantime?***

(iii) Efficacy: Although it is assumed that a 'next generation' vaccine against CBPP will prove more efficacious, this is by no means assured. Any such vaccines tested to date have proved to be noticeably unsuccessful (Absurga *et al.,* 1997; Waite and March, 2002), and in some instances have even exacerbated symptoms (Hubschle *et al.,* 2003). Since the basic mechanism(s) of CBPP immunopathology are not yet understood, and since no 'protective' antigens (with the possible exception of the capsular polysaccharide) have been specifically identified, the wait for an effective 'new' CBPP vaccine may be lengthy.

(iv) Production issues: Current CBPP vaccines are very 'low tech' and in fact are exclusively made on location in Africa, with relatively simple equipment, facilities and staff training requirements. It is unlikely that a subunit, recombinant protein or nucleic acid vaccine could be made *in situ* in Africa without a large investment in infrastructure, training and equipment. For example, it cost roughly US $300,000 to produce sufficient GMP-quality DNA for a malaria vaccine trial currently underway in Africa involving only 2000 subjects (Adrian Hill, personal communication). This is quite clearly a totally uneconomic proposition. If 'next generation' vaccines have to be made in the West and imported into Africa this would remove a vital level of autonomy and decision making in the whole process. In addition, where will the funding come from to pay for this infrastructure build or alternatively pay for vaccine importation if it cannot be made locally?

(v) Politics: The control policies involved in a pan-continental disease issues are highly complex, involving both national, international and donor country issues. Agreement and implementation of even the simplest of measures is often extremely difficult to achieve. The complex financial, regulatory, and scientific agendas involved in the development of any new vaccine technology are likely to severely hamper the widespread adoption of the vaccine(s), even assuming that issues (**i-iv**) above can be adequately addressed.

In isolation these difficulties are formidable; when seen as a whole they appear to be almost insurmountable if it is to be hoped that a new and effective CBPP vaccine will be in place during the next decade. Whilst recent initiatives such as the Wellcome Trust's Tropical Animal Health initiative, or charities such as the Gates Foundation may be approachable, the timescales involved before we even begin to understand the basic mechanisms of immuno-pathology, let alone develop 'next generation' vaccines means the we must look to what is available ***now***. It is my contention that it is more important to make the best use of current technologies to try and control CBPP ***today*** rather that concentrate upon the development of technologies that may not prove effective for at least a generation, if at all. This is all the more important since it is clear that current technologies can be effective if properly used. It could be suggested that current research priorities have more to do with the career requirements of Western scientists than the economic and veterinary needs of the developing world.

It is interesting to note that approximately 10 years have passed since the re-emergence of interest in CBPP following outbreaks in Europe and Africa. Despite several million euros spent in research projects, vaccine trials, workshops, expert group meetings and international scientific meetings, little or no meaningful progress has been made with regard to developing new vaccines and other control measures during this period. ***One is tempted to ask; could all this money and resource have been better spent?*** How many vaccination campaigns or serological surveys could this have funded?

Currently used CBPP Vaccines

Almost without exception, all effective CBPP vaccines have been based upon live versions of the disease-causing mycoplasma, either attenuated or not. Current vaccine strains

(T1 44 and T1 SR) for CBPP are made from freeze dried broth cultures of live attenuated (*Mmm* SC) and are generally considered to exhibit poor efficacy and stability (Rweyemamau *et al.,* 1995; Thiaucourt *et al.,* 2000). However, the poor efficacy of vaccines against CBPP appears to be a relatively recent phenomenon, with many reports existing in the literature of successful vaccines based upon live cultures of *Mmm* SC. The first published account of a successful CBPP vaccine appeared as long ago as 1852, although the procedure (involving the implantation under the skin of the forehead of tiny pieces of diseased tissue from a beast that had succumbed to CBPP) had apparently been in use for some time prior to this. By 1926 vaccination and control of this disease was apparently well under control in parts of Africa. To quote from J. Walker, the Chief Veterinary Research Officer in Kenya, "The serum diagnosis of pleuro-pneumonia…. and preparation of pleuro-pneumonia vaccine are now daily technical routine work' (Walker, 1929).

CBPP was successfully eradicated from Australia using the V5 broth vaccine, with no real problems regarding either efficacy or thermostability under field conditions every bit as hostile as those likely to be encountered in Africa (Newton and Norris, 2000). As long as the liquid vaccine was kept wrapped in a damp cloth and protected from direct sunlight this was considered adequate protection (Hudson, 1968). Indeed, even vaccine strain KH_3J (long regarded as being of negligible protective efficacy in Africa) was successfully used in Australia during the early 1960s (Hudson, 1065).

How can these apparent contradictions in reported vaccine efficacy and stability be reconciled? The recent experiences with the T1 44 vaccine in Namibia showed that it was actually highly effective in bringing CBPP under control: reported disease incidence was reduced from 2794 in 1997 to only 87 in 1999 (Bamhare, 2001). Thus the current vaccine was highly effective when administered as part of a well conducted vaccination campaign, in which (i) high levels of coverage were achieved and, (ii) in which the vaccine was used as quickly as possible following reconstitution (before a significant loss in titre occurred). ***If these conditions could be achieved over the entire continent, CBPP would be a disease of the past***. Unfortunately, it is probably wishful thinking to hope that such well-run vaccination campaigns can take place over much of sub-saharan Africa, but based upon recent research findings there are several recommendations that can be made which should have a significant impact upon the efficacy of the current CBPP vaccines and which can be implemented ***immediately***. What are the main issues currently affecting vaccine efficacy?

Recommendations for Change

Effective buffering of growth media: Assuming that the minimum protective dose of MmmSC is 10^7 live mycoplasmas per dose (Gilbert and Windsor, 1971), it obviously becomes vitally important to maintain vaccine titre when in the field. Since it is seems clear that current vaccines can provide effective protection when given correctly, and since the emergence of new 'vaccine resistant' strains has not been reported (March *et al.,* 1999), the major factor behind poor vaccine efficacy is likely to be sub-optimum bacterial titres. It is known that many vaccine production laboratories do not reach the OIE recommendation of delivering a vaccine at 10^8 viable mycoplasmas per animal dose (which allows for losses during lyophilisation, storage and transport (Litamoi and Seck, 1999; Rweyemamu *et al.,* 1995). Why is it difficult to achieve and maintain effective titres? A reduction in vaccine pH during culture growth is the most likely explanation. Apart from excessive heat (greater than 43°C), the pH of the growth medium is the principal factor which affects mycoplasma viability (Garba, 1980; Gourlay and MacLeod, 1966; Miles, 1983; Rodwell and Mitchell, 1979; Windsor, 1978). Current vaccine media (e.g. Gourlay's (Gourlay 1964) and F_{66} (Provost *et al.,* 1970) are poorly buffered, containing a dibasic (Na_2HPO_4) phosphate salt only, and exhibit a sharp drop in pH during MmmSC growth. This is mirrored by a rapid reduction in culture viability once the pH begins to fall. In contrast, the

growth medium used to produce the successful V5 broth vaccine did not contain glucose (a reducing sugar) and as a result, while the final mycoplasmal titre *may* have been slightly reduced compared to media containing glucose, the pH did not fall below neutrality (i.e. pH 7.0) (Turner *et al.,* 1935). The result was that vaccines were highly stable for relatively long periods at high ambient temperatures (at least 1 month at 37°C) (Hudson 1968; Turner *et al.,* 1935). If glucose is to be kept in the growth medium, then a buffer system based upon N-[2-hydroxyethyl] piperazine-N'-[2-ethanesulfonic acid] (HEPES) can be used (Waite and March, 2002). This exhibits a 10-fold increase in final titre and markedly increased culture survival compared to contemporary media due to maintenance of a neutral pH during the growth and stationary phases. Yields in excess of 10^{11} cfu /ml can easily be achieved. Whilst MmmSC cultures in conventional media drop in titre from 10^{10} /ml to completely inactivated within 2 days at 37°C, the simple addition of HEPES buffer means that the titre at 37°C is still above 10^8 /ml after 1 month at 37°C, and is 10^4 /ml after 4 months. An impressive result for such a simple and inexpensive action, with no other changes to current procedures required. ***The benefit to the user – the vaccine is far more stable. The benefit to the producer – the optimum time for vaccine harvesting is increased from a few hours to several days and the yield is increased 10-fold.***

Inclusion of pH indicators: Although high ambient temperatures can evidently lead to MmmSC cell death, cultures will remain stable at 37°C for several weeks as long as a neutral pH is maintained (Turner *et al.,* 1935), and the culture is protected from direct sunlight (Hudson, 1968). It is not until the temperature is above 42°C before rapid cell death occurs. Therefore as long as the culture pH is maintained above roughly pH 6, vaccines should be stable for several days in the field (i.e. without a fall in titre). An extremely simple and inexpensive way to monitor this is to incorporate a simple pH indicator, such as phenol red to the culture medium and /or reconstitution fluid. With a printed pH indicator label attached to the vial, this would provide a rapid and inexpensive visual confirmation of vaccine pH following reconstitution, providing a degree of reassurance and control not currently available to users of CBPP vaccines. A simple visual cut off below which a vaccine should not be used could be incorporated on the label. ***This simple action would provide a direct benefit to the user***.

Similarly, the incorporation of pH indicators ***should also provide a direct benefit for vaccine producers***. Vaccine production should be easier, since a 'real time' check on acidity can be made, without the need for any external sampling and pH monitoring with its associated contamination risks. Again, the cost of incorporating such changes would be minimal – no additional equipment or procedures would be required. To encourage the use of pH indicators, FAO/OIE 'Approved Status' should only be granted to vaccines including such indicators.

Changes in vaccine reconstitution procedure: The current OIE recommended reconstitution procedure for CBPP vaccines ($T_1$44 and T_1SR) is to use a solution of 1 molar $MgSO_4$ (Anon, 2001), which has been reported to increase thermostability (Provost, 1970; Provost *et al.,* 1987). However, this procedure was only ever reported for vaccine strain KH_3J (not in use today) and has apparently never been tested for strains $T_1$44 and T_1SR (current vaccine strains). Unfortunately, these strains result in a much more acidic culture during growth than strain KH_3J (Gourlay and MacLeod, 1966). This is highly significant, since re-suspension of freeze dried CBPP vaccines in 1M $MgSO_4$ makes the vaccine even more acidic due to precipitation of the phosphate buffer component (***present in all current vaccine media***) (March *et al.,* 2002). The Mg^{2+} combines with the phosphate to produce insoluble magnesium orthophosphate, not only removing the buffer component, but releasing free H^+ in the process! It is no wonder the vaccines become so acidic. The acidic pH rapidly leads to mycoplasma death and vaccine inactivation; which will also explain why current vaccines are so unstable when reconstituted in the field following the OIE-recommended procedure. Not only does the $MgSO_4$ solution cause a drop in pH, but the culture pH is lower to begin with due to the presence of the reducing sugar glucose and the use of the highly acidifying strains $T_1$44 and

T_1SR. The cumulative effect of all of these changes is a large reduction in pH and stability for 'modern' vaccines when compared to pre-1970 vaccine formulations: (i) Compared to strain KH_3J, strain $T_1$44 causes an additional drop of 0.6 pH units during growth. (ii) The addition of glucose compared to its absence in identical media results in a further drop of 0.6 pH units. (iii) Reconstitution using 1M $MgSO_4$ causes an additional drop of up to 2.2 pH units. Current reconstituted vaccines can easily be under pH 5! It is hardly surprising that their stability is so poor compared to the Australian vaccine formulations of a generation ago.

In contrast, when a vaccine culture is reconstituted in buffered saline or water it remains stable for many days at 37°C. In 1M $MgSO_4$ the titre drops by 6 $\log_{10}$ over an 8 hour period. We have observed however, that this effect on culture pH can be largely removed if HEPES-buffered vaccine media (as suggested above) are used March *et al.,* 2002).

Use of phosphate-buffered saline (PBS) as a reconstitution solution: An alternative or additional suggestion to the use of HEPES-buffered growth medium is to use PBS as a reconstitution fluid, in place of 1 M $MgSO_4$. While purified water has the advantage that it does not cause a drop in pH following vaccine reconstitution, it cannot provide any additional buffering capacity of its own. PBS would be useful to restore to neutrality the pH of a borderline vaccine batch (for example around about pH 6 at the time of harvesting). The use of PBS at pH 7.5, containing phenol red as a visual indicator to confirm satisfactory pH prior to injection is the best procedure to maintain vaccine pH and titre following reconstitution (March *et al.,* 2002). This is less important if the vaccine medium already contains HEPES, since it will be properly buffered anyway. If it does not, and is therefore inadequately buffered, the use of PBS as a reconstitution fluid should be mandatory I would suggest.

A range of factors can be seen to affect the final pH of reconstituted $T_1$44 vaccines, and it would seem that too many variables are present in current methodology to give consistency, particularly since other factors in the field will also affect final vaccine titre (for example, variable ambient temperature, operator skill, length of time reconstituted vaccine is left prior to use). For optimum vaccine efficacy it is important to minimise these sources of variation. Alterations in methodology in order to maintain a neutral vaccine pH following reconstitution should increase vaccine longevity and thus minimise the effect of these other sources of variation.

Conclusions and Recommendations

It is vitally important to maintain vaccine pH near to neutrality to prevent premature spoiling, and unfortunately, the procedures and reagents currently in use and recommended by the OIE do not achieve this. On the basis of findings discussed in this paper, the following recommendations are made. (**i**) The use of 1 M $MgSO_4$ as a reconstitution fluid in the field should be terminated forthwith. (**ii**) Phosphate buffered saline should be used as an alternative reconstitution fluid. This should provide an extra measure of security against the generation of an acidic pH following reconstitution, even if a vaccine was desiccated under slightly acidic conditions. (**iii**) Ideally, HEPES-buffered growth media should be used to prepare vaccine stocks to ensure the pH never drops below pH 7.0 during the growth cycle. (**iv**) The FAO and OIE to recommend only the use of vaccines and diluents containing pH indicators such as phenol red. With a printed pH indicator label attached to the vial, this would provide rapid and inexpensive visual confirmation of vaccine pH prior to and following reconstitution, providing a degree of reassurance and control not currently available to users of CBPP vaccines.

Hopefully, as the recent experience of Namibia shows, current vaccines can and will be able to make a significant impact upon CBPP disease control. The adoption of these simple and inexpensive measures should have a significant impact upon a disease that is unfortunately

as much of a scourge in Africa today as at any time in the past. With proper implementation, it is plausible to hope that by the time any 'next generation' CBPP vaccines do finally appear, the disease may already be under control (or even eradicated?) in Africa. I would like to propose that rather than necessarily concentrating on promoting further research, the FAO/OIE/OAU-IBAR should perhaps seek large scale funding from agencies such as the Gates Foundation for a concerted vaccination and diagnostic program to eradicate CBPP from the African continent using the tools and technologies already at its disposal. To quote again from the book 'Clearing a Continent', which records the eradication of CBPP from Australia during the years leading up to the 1960s 'On the world scene, eradication under the difficult circumstances in Australia may well encourage countries where the disease persists to embark on a similar course' (Newton and Norris, 2000). Hopefully this will be so.

References

Abusugra, I., Wolf, G., Bölske, G., Thiaucourt, F., and Morein, B. (1997). ISCOM vaccine against contagious bovine pleuropneumonia (CBPP). 1. Biochemical and immunological characterization. Veterinary Immunology and Immunopathology **59,** 31-48.

Anon. (1999). Summary of Presentations and Discussions. Report of the First Meeting of the FAO/OIE/OAU-IBAR Consultative Group on Contagious Bovine Pleuropneumonia (CBPP). Rome, Italy 5-7 October 1998. FAO, Rome. pp 3-16.

Anon. (2001). Report of the second meeting of the FAO/OIE/OAU/IAEA Consultative Group on contagious bovine pleuropneumonia (CBPP), Rome, Italy 24-26 October 2000, FAO, Rome. pp. 11-16.

Bamhare, C. (2001). CBPP surveillance in vaccinated areas: Namibia experience with CBPP vaccines prepared from the T1-44 and T1-SR strains. Report of the second Meeting of the FAO/OIE/OAU/IAEA consultative group on contagious bovine pleuropneumonia (CBPP).Rome, Italy 24-26 Oct 2000, FAO, Rome. pp. 79-87.

Garba, S. A. (1980). Shelf life of wet T1 broth vaccine for contagious bovine pleuropneumonia. Tropical Animal Health and Production **12,** 189-191.

Gilbert, F. R. and Windsor, R. S. (1971). The immunizing dose of T1 strain *Mycoplasma mycoides* against contagious bovine pleuropneumonia. Tropical Animal Health and Production **3,** 71-76.

Gourlay, R. N. (1964). Antigenicity of *Mycoplasma mycoides*. I. Examination of body fluids from cases of contagious bovine pleuropneumonia. Research in Veterinary Science **5,** 473-482.

Gourlay, R. N. and MacLeod, A. K. (1966). Fermentation of glucose by *Mycoplasma mycoides* and its effect on viability. Bull. Epizoot. Dis. Afr **14,** 373-381.

Hubschle, O. J. B., Tjipura-Zaire, G., Abusugra, I., Di Francesca, G., Mettler, F., Pini, A., and Morein, B. (2003). Experimental field trial with an immunostimulating complex (ISCOM) vaccine against contagious bovine pleuropneumonia. Journal of Veterinary Medicine **50,** 298-303.

Hudson, J. R. (1965). Contagious bovine pleuropneumonia: The Immunizing value of the attenuated strain KH_3J. Australian Veterinary Journal **41,** 43-49.

Hudson, J. R. (1968). Contagious bovine pleuropneumonia. The keeping properties of the V5 vaccine used in Australia. Australian Veterinary Journal **44,** 123-129.

Litamoi, J. K. and Seck, B. M. (1999). Vaccines and vaccination. Vaccine quality issues in Africa including safety and efficacy. Report of the first meeting of the FAO/OIE/OAU-IBAR consultative group on contagious bovine pleuropneumonia (CBPP), Rome, Italy 5-7 October 1998. FAO publication X3960-E. pp65-75.

March, J. B., Waite, E. R., and Litamoi, J. (2002). Re-suspension of T144 vaccine cultures of *Mycoplasma mycoides* subsp. *mycoides* SC in 1 molar MgSO4 causes a drop in pH and a rapid reduction in titre. FEMS Immunology and Medical Microbiology **34,** 97-103.

March, J. B., Jones, G. E., Williamson, H. S., and Amanfu, W. (1999). Studies on the immunological diversity of type, vaccine and wild strains of *Mycoplasma mycoides* subsp. *mycoides* SC variant, p. 159-162. *In* L. Stipkovits, R. Rosengarten, and J. Frey (eds.), Mycoplasmas of ruminants: pathogenicity, diagnostics, epidemiology and molecular genetics. Vol III. European Union, Luxembourg.

Masiga, W. N. and Domenech, J. (1995). Overview and epidemiology of contagious bovine pleuropneumonia in Africa. Revue scientifique et technique Office International des Epizooties **14,** 611-630.

Miles, R. J. (1983). Effect of some cultural factors on T1 broth vaccine for contagious bovine pleuropneumonia. Tropical Animal Health and Production **15,** 144-148.

Newton, L. G. (1992). Contagious bovine pleuropneumonia in Australia: some historic highlights from entry to eradication. Australian Veterinary Journal **69,** 306-317.

Newton, L. G. and Norris, R. (2000). An overview of pleuropneumonia in Australia. p 1-14. *In* Clearing a continent. The eradication of bovine pleuropneumonia from Australia. CSIRO Publishing, Collingwood, Australia.

Nicholas, R., Bashiruddin, J., Ayling, R. D., and Miles, R. (2000). Contagious bovine pleuropneumonia: a review of recent developments. Veterinary Bulletin **70,** 827-838.

Provost, A. (1970). Activite thermoprotectrice de la solution molaire de sulfate de magnesium sur l'inactivation thermique de *Mycoplasma mycoides* en phase liquide. Critical Reviews Acad. Sci. Paris **270,** 3156-3157.

Provost, A., Borredon, C., and Queval, R. (1970). Recherches immunologiques sur la peripneumonie VI. Un vaccin vivant mixte antibovipestiques-antiperineumonique inocule en un seul temps conception, production, controles. Revue Elevage Medicine Veterinaire Pays Tropicaux **23,** 143-162.

Provost, A., Perreau, P., Breard, A., Le Goff, C., Martel, J. L., and Cottew, G. S. (1987). Contagious bovine pleuropneumonia. Revue scientifique et technique Office International des Epizooties **6,** 625-679.

Rodwell, A. W. and A. Mitchell . 1979. Nutrition, growth and reproduction, p. 103-139. *In* M. F. Barile and S. Razin (eds.), The Mycoplasmas.I. Cell Biology. Academic Press, New York.

Rweyemamu, M. M., Litamoi, J., Palya, V., and Sylla, D. (1995). Contagious bovine pleuropneumonia vaccines: the need for improvements. Revue scientifique et technique Office International des Epizooties **14,** 593-601.

Thiaucourt, F., Yaya, A., Wesonga, H., Huebschle, O. J., Tulasne, J. J., and Provost, A. (2000). Contagious bovine pleuropneumonia. A reassessment of the efficacy of vaccines used in Africa. Annales New York Academy of Sciences **916,** 71-80.

Turner, A. W., Campbell, A. D., and Dick, A. T. (1935). Recent Work on Pleuro-Pneumonia Contagiosa Boum in North Queensland. Australian Veterinary Journal **11,** 63-71.

Waite, E. R. and March, J. B. (2001). Effect of HEPES buffer systems upon the pH, growth and survival of *Mycoplasma mycoides* subsp. *mycoides* small colony (MmmSC) vaccine cultures. FEMS Microbiology Letters **201,** 291-294.

Waite, E. R. and March, J. B. (2002). Capsular polysaccharide conjugate vaccines against contagious bovine pleuropneumonia: Immune responses and protection in mice. Journal of Comparative Pathology **126,** 171-182.

Walker, J. (1929). Veterinary Record. May 1st pg 403.

Windsor, R. S. (1978). An investigation into the viability of broth cultures of the T1 strain of *Mycoplasma mycoides* sub-species *mycoides*. Research in Veterinary Science **24,** 109-112.

Contagious bovine pleuropneumonia: research trends

F. Thiaucourt

CIRAD-EMVT, Montpellier, France

Introduction

For a cattle owner in Africa, there has been apparently no change in the CBPP situation for many decades. The same vaccines have been used for 40 years, antibiotics are still used in the field. The improvement that was observed after the JP15 vaccination campaigns, associating rinderpest with CBPP vaccination is now gone. As a consequence, CBPP is again a top priority now. However, there are many indications that the situation may change rapidly as research has started to produce some results and more is to come in the near future.

The past decade has seen a number of technological improvements in diagnostic procedures. The direct detection and identification of the causative agent is now obtained by various specific PCR techniques. Furthermore, groups of *Mycoplasma mycoides* subspecies *mycoised* small colony biotype (*Mmm* SC) strains correlated with geographic origins can now be characterized thanks to Southern Blotting and Multilocus sequence typing. Finally, the T_1 vaccine strains can be identified by a specific PCR. Serology techniques have also evolved recently with the development of specific competition assays or ELISA. More is to come in the different steps that can play a role when studying a disease and trying to improve CBPP control. Schematically, four of these steps can be defined: the pathogen itself, the interaction of the pathogen with the host, the transmission of the disease within a herd and, finally, the transmission of the disease on a wider scale.

The pathogen

Dramatic technological improvements have allowed the sequencing of bacterial genomes including those of mycoplasmas that are among the smallest of "free living organisms". The genome sequence of strain PG1 has been obtained by a Swedish team. Unfortunately it has not been put in the public domain, although this project had been started more than 6 years ago. In any case, other strains will be sequenced in the near future, as what is really needed is the sequence of a pathogenic strain. Technological advances are now allowing the study of the transcription of genes or even the totality of the proteins that are synthesized. These tools will allow the comparison of strains of high and low virulence and enable the identification of virulence factors. In addition, novel plasmid constructions allow the transformation of mycoplasmas and the inactivation of some specific genes. This should allow an experimental proof of the involvement of specific genes in virulence mechanisms.

The host pathogen interactions

Unlike other bacteria, *Mmm* SC is devoid of established virulence factors such as toxins for example. The most likely explanation for its virulence is to be found in the interaction between this bacteria and the host immune response. Lesions are the result of an exacerbated local inflammatory response and death occurs when animals are unable to regulate this response rapidly enough. New tools such as microarray analysis will now allow a very fine and comprehensive study of the kinetics of the immune response in infected animals. This will allow us to understand which mycoplasmal antigens are responsible for this response and which

bovine cells are the key effectors of this response. This will help us to design vaccines that are devoid of residual virulence. In parallel, the identification of the type of immune response that leads to protection and not to sensitisation will allow the identification of protective antigens and delivery systems.

The transmission of CBPP in the field

One of the main limitations for CBPP studies is the absence of a laboratory animal model. Accordingly, all experimental trials have to be done in cattle. The situation is even complicated by the fact that there is a very pronounced variation of individual susceptibilities. This is the reason why experimental trials have to include large groups of animals or they are to be duplicated many times before statistically significant results can be obtained. This is the reason why some key parameters for the study of CBPP epidemiology are still uncharacterised. One example is the infectivity of CBPP chronic carriers although this type of cattle may play a very important role in the persistence of the disease or in reintroduction to disease-free zones. Mathematical models may play a very important role. Firstly, they may allow an identification of the key parameters that may influence the long-term persistence of the disease. This, in turn, will pinpoint the biological assays that will have to be performed in order to determine the value of these parameters. Secondly, they may be used to simulate the effect of various combinations of control measures. This should help in defining the most cost-effective strategies.

Whatever new tools are developed in the near future, it should be recognized that the success of CBPP control would depend mainly on the commitment of the various stakeholders in the field. Designing new tools, such as more potent vaccines, should diminish the cost of CBPP control and therefore, make more realistic, the eradication of this disease. This is not a mere dream. CBPP transmission is quite simple compared to other diseases, there is no reservoir in wildlife, transmission is direct and mycoplasmas are rapidly inactivated outside their host. These are very favourable conditions for control.

Use of long acting tetracycline for CBPP: preliminary results

A. Yaya[1], H. Wesonga[2] and F. Thiaucourt[3]

[1]LANAVET, Garoua, Cameroon;
[2]KARI, Muguga, Kenya;
[3]CIRAD-EMVT, Montpellier, France.

Introduction

The use of antibiotics for CBPP control has always been controversial. For some "experts in development", antibiotic therapy could be a valid alternative to vaccination. According to them, the advantage of antibiotics over vaccines is that these drugs are already available in the field and their use may have a direct impact on poverty alleviation. On the contrary, vaccines are often distributed by state veterinary services exclusively and may therefore fail to reach all the cattle owners that need them, as very few veterinary services in Africa are wealthy enough to organize comprehensive vaccination campaigns. On the other hand, there are many arguments that can be put forward that oppose the widespread use of antibiotics, such as the inevitable spread of residues that favour the emergence of resistant bacteria in the environment and the possible long-term carrier state that is allegedly attributed to animals treated with antibiotics.

For these reasons, African veterinary services have some difficulties in defining an official strategy concerning the use of antibiotics. This was exemplified during the recent CBPP AU/IBAR meeting in Accra, Ghana in February 2002. Some Directors of Veterinary Services were of the opinion that the use of antibiotics for the treatment of CBPP must be forbidden (although it is a very common practice in the field); others were eventually considering their use as a possible tool to control the disease. The difficulty is that there are very few data concerning the efficiency of these antibiotic treatments. Many antibiotics have been shown to be active in *in vitro* assays. This is the case of tetracyclines, macrolides, lincosamines, streptogramines and quinolones (Ayling, 2000). In the field, such antibiotics were used successfully to treat post-vaccinal reactions when using strain T1 44 (Lindley, 1971). This is the reason why it was decided to conduct some preliminary trials in order to assess the efficacy of long-acting tetracycline to treat CBPP-infected cattle.

Methods

The first trial was performed at KARI, Muguga. Groups of 5 animals were inoculated subcutaneously, either with a local virulent *Mmm* SC isolate or with a T1 44 vaccine strain that had apparently reverted to virulence. This inoculation was followed by the development of an invading oedema and animals were treated with a single dose of long-acting tetracycline according to the manufacturer's instructions.

The second trial was performed at LANAVET, Garoua. Twenty animals were added to the control group in a T1 vaccine trial. The assay started when the transmission of CBPP to the control group was well established. At this stage, 20 animals were randomly selected for the antibiotic assay and 12 of them displaying CBPP symptoms were subjected to treatment with long-acting tetracycline. Treated animals were housed in a different building in order to prevent reinfection by untreated controls. At the end of the experiment, all animals were slaughtered and their lesion score was evaluated. Comparisons were then established between the treated group and the corresponding controls (animals present at the beginning of the treatment).

Results

In the first trial, out of 10 animals, 2 samples of cutaneous tissue yielded a positive *Mmm* SC culture and, in addition, 2 other animals were positive for isolation of *Mmm* SC from other samples. Therefore, the antibiotic treatment did not prevent 4 animals out of 10 from becoming *Mmm* SC carriers, although the antibiotic treatment definitively stopped the extension of lesions.

In the second trial, in the treated group (12 animals) none died of CBPP, whereas 3 of them did in the untreated group. Furthermore, there was a difference in the distribution of lesion scores between the two groups. However, *Mmm* SC was isolated out of 5 animals (from the 12 treated ones). Preliminary data suggest that *Mmm* SC strains did not develop any resistance to tetracycline. Given that the lung lesions did not consist of well-formed sequestra, the persistence of *Mmm* SC cannot be explained by a problem of antibiotic diffusion to necrotic material within the fibrous capsule.

Discussion

Both trials indicated that tetracyclines had a positive effect on the clinical course of the disease. However, treated animals still suffered from CBPP lesions and some animals that were not treated because they did not display any obvious CBPP signs were nevertheless found to harbour CBPP lesions. These findings clearly show that antibiotic treatments in the field will not allow the disappearance of the infection. Antibiotics will certainly reduce the economic impact of CBPP in affected herds and in addition, they will reduce the infective pressure on susceptible animals. According to these preliminary results, antibiotics may be used in a combined strategy involving vaccinations, bearing in mind the possible drawbacks of the uncontrolled and widespread use of antibiotics.

References

Ayling, R. D., Baker, S. E., Nicholas, R.A., Peek, M. L. and Simon, A. J. (2000). Comparison of in vitro activity of danofloxacin, florfenicol, oxytetracycline, spectinomycin and tilmicosin against MmmSC. Veterinary Record **146**, 243-246.

Lindley, E. P. (1971) La spiramycine et les lésions post-vaccinales au vaccin lyophilisé, "MmmSC souche T1/44" contre la péripneumonie contagieuse des bovidés (PPCB). Cah méd. vét. **40**, 233-236.

Surveillance and testing strategies for the diagnosis of CBPP: Results of the FAO/IAEA Co-ordinated Research Programme on the monitoring of CBPP in Africa

Roland Geiger

Animal Production and Health Section, Joint FAO/IAEA Division of Nuclear Techniques in Food and Agriculture

Introduction and Background

Reliable and efficient diagnostic tests for the serological diagnosis of infectious diseases are the cornerstone of any disease control strategy. The requirements for diagnostic tests depend on the purpose of the diagnostic testing and the epidemiological needs. The monitoring of a region wide vaccination programme has different needs compared to the needs of export certification of individual animals. In the case of contagious bovine pleuropneumonia (CBPP) countries adjacent to infected areas may want to confirm the absence of the disease relying on serology and slaughterhouse inspections. Positive test results will lead to follow up investigations and, depending on the diagnostic tests used, may result in substantial expenses. Highly specific tests with a high positive predictive value are needed to limit the amount of follow up work.

Countries which are operating a disease control programme with no vaccination based on stamping out of positive herds and compensation of the farmers require tests which are highly sensitive and specific at the individual animal level and in the case of positive results several follow up investigations might be undertaken. In countries where the disease is present and control programmes based on movement control and vaccinations are operated the sampling efficiency must be high to enable constant monitoring and redirection of the ongoing programme.

Serological tests

For serological diagnosis the complement fixation test (CFT) is still the most widely used test. Some authors report it as highly sensitive in the acute phase, with lower sensitivity in later stages (OIE, 2002). Other authors report an overall sensitivity of only 63.6% Bellini *et al.,* 1998). Specificity is reported to be high, but some authors report false positive results in up to 3.5% of all sera leading to the misclassification of up to one third of the herds investigated (Stark *et al.,* 1995). An indirect ELISA which showed a high level of sensitivity was found to show many non-specific reactions and consequently a more specific competitive ELISA was developed and introduced into 11 African countries through an FAO/IAEA Co-ordinated Research Project (CRP) on the “Monitoring of contagious bovine pleuropneumonia in Africa using enzyme immunoassays” (Le Goff and Lefevre, 1989; LeGoff and Thiaucourt, 1998). The main objective of this CRP was to compare and validate the main serological tests for the diagnosis of CBPP in particular the CFT and the competitive ELISA. In CBPP free areas the competitive ELISA was reported to have a specificity of close to 100% (Amanfu *et al.,* 1998). An indirect ELISA based on the specific lipoprotein LppQ of *Mycoplasma mycoides* subsp. *mycoides* small colony type (*Mmm* SC) showed a high level of sensitivity and specificity when used in one country but not enough data are available for reliable estimates under different epidemiological conditions (Bruderer *et al.,* 2002).

A rapid latex agglutination test for the detection of circulating capsular polysaccharide (CPS) antigen of *Mmm* SC and for the detection of circulating antibodies to *Mmm* SC which was

reported to have a high specificity was introduced in the last year into the CRP and compared to the results of the CFT and the cELISA (March *et al.,* 2002).

Material and Methods

FAO/IAEA Co-ordinated Research Project

The CRP that was operational from 1997 until 2003 involved 11 African countries with various epidemiological situations. Three Research Agreement Holders (UK, France, Sweden) provided advice and scientific guidance to the CRP. Annual FAO/IAEA Research Contracts awarded to the participating institutions ensured the supply of reagents (primarily for the CFT and the cELISA) and equipment (IAEA 2002). Four meetings were held where the results of the CRP were presented. The results of the final CRP are being published.

Figure 1. Countries participating in the FAO/IAEA Co-ordinated Research Project "The monitoring of contagious bovine pleuropneumonia in Africa using immunoassay"

Research Contract Holders:

1. Nat. Vet. Lab., Botswana
2. Lab. Path. Anim., Côte d'Ivoire
3. NAHRC, Ethiopia
4. CVL, Ghana
5. CVL, Kenya
6. LCV, Mali
7. CVL, Namibia
8. Nat. Vet. Res. Inst., Nigeria
9. ADRI, Tanzania
10. LIRI, Uganda
11. CVRI, Zambia

Research Agreement Holders:

- CIRAD-EMVT, France
- Nat.Vet.Inst., Sweden
- Scient. Agric. Coll., UK

Sera and samples

Namibia and Botswana were the only countries where sera from confirmed CBPP free areas were available. In 9 countries (Ghana, Nigeria, Cote d'Ivoire, Mali, Ethiopia, Kenya, Uganda, Tanzania, Zambia no confirmed negative populations were available. In the endemic areas the Research Contract holders were advised to collect sera preferably from herds where CBPP was diagnosed clinically.

Diagnostic tests

cELISA

The reagents were provided in a kit form containing all the reagents (e.g. polystyrene microplates coated with *Mmm* SC lysate, a monoclonal *Mmm* SC mouse antibody, dilution and washing buffers, a monoclonal anti-mouse IgG peroxidase conjugate and TMB with stopping solution). The initially standard protocol of the competitive ELISA following the FAO/IAEA protocol (FAO/IAEA, 1998) that used freeze-dried antigen was replaced in 2002 through a simplified protocol using precoated plates (FAO/IAEA, 1998; Kit Insert, 2002).

Complement fixation test (CFT)

The CFT was carried out as described before (Campbell and Turner, 1953).

Latex agglutination test

This test was designed as a penside test and that could be carried out under field conditions. The test samples were mixed with latex beads on a glass slide and positive samples showed a visually detectable agglutination. The test was provided in three formulations and was carried out as described in the protocol (March, 2002).

- Blue test: Blue latex beads coated with whole *Mmm* SC. Designed to detect total *Mmm* SC antibodies in serum.
- White test: White latex beads coated with *Mmm* SC capsular polysaccharide (CPS). Designed to detect CPS antibodies in serum.
- Red test: Red latex beads coated with anti-*Mmm* SC CPS IgG antibodies. Designed to detect CPS (principle circulating antigen in serum and other fluids.

Results

cELISA: Sensitivity and specificity

In Namibia and Botswana the specificity of cELISA was between 98% (n=50) and close to 100% (n=1200). The latter value is correct because larger numbers of samples were tested.

Only the relative sensitivity of the cELISA compared to the CFT could be determined since not enough statistically significant numbers of samples from confirmed positive cases were available. Generally the CFT was able to detect more positive sera in newly infected herds whereas the ELISA was able to detect more animals in endemic situations. In an infected herd in Tanzania sequential bleedings of 29 cattle, the cELISA was positive for 25 cattle and the CFT for 21 cattle. In Botswana the testing of stored sera from cattle from the outbreak area with pathognomonic lesions detected in post mortems and which represented most probably true positive cases showed a sensitivity of 90% (n=82) for the cELISA and 93% in the CFT. In Cote d'Ivoire the relative sensitivity of the cELISA to the CFT was very variable and was dependant whether new outbreaks were investigated or whether herds with old infections were investigated (relative sensitivities were: 87% (n=170), 54% (n=176), 0% (n=211, but in the CFT only 4%, e.g. 8 positives) and 88% (n=480)). In Cote d'Ivoire the overall relative sensitivity of the cELISA compared to the CFT was 78% (n=1037).

In Nigeria the relative sensitivity of the cELISA was 100% (n=170). In Namibia the CFT detected 15 out of 44 animals from a herd with a new outbreak where the cELISA could not

detect any animals. In Tanzania in recently vaccinated herds 96% (n=578) of animals reacted positive in the CFT, whereas all the animals were negative in the cELISA resulting in a specificity of the cELISA in vaccinated animals of 100%.

An important aspect in the testing of sera is that the results are repeatable and reproducible and how well a test can be quality controlled. In the ELISA the change to a format with precoated plates reduced the variability in 10 out of 11 countries to acceptable levels. Test results of the quality controls which were included in each kit and consequently on each plate (strong positive serum, weak positive serum, negative serum, conjugate control, monoclonal control) fell within predefined upper and lower control limits indicating that the results of unknown test sera would be equivalent in each of the testing laboratories.

An indirect ELISA based on the specific lipoprotein LppQ of *Mmm* SC (Bruderer *et al.,* 2002) was also included and tested as part of the CRP in one country but the results were not conclusive.

Latex agglutination test

Blue test for the detection of total *Mmm* SC antibodies

Results from five countries (Tanzania, Nigeria, Cote d'Ivoire, Mali, Uganda) showed a high number of positives in infected herds (71% to 100%) that indicated a high sensitivity but probably also a low specificity because of non-specific agglutination. However, no results from infection free areas are yet available which is essential to exclude a possible low specificity of the test.

White test for the detection of anti CPS antibodies

Sensitivity in seven countries ranged from 37% to 100% in new outbreaks. However, all animals (Namibia, n = 50) tested from infection free areas were positive indicating a low specificity, although another report showed a higher specificity (UK, 94%, n = 32) (see Huebschele et al., this report). More testing is presently undertaken in Namibia and Botswana.

Red test for the detection of circulating CPS

No conclusive data on the performance of this test were available. Specificity on sera ranged between 98% in Botswana and 38% in Namibia. Sensitivity using sera was low between 0% (Uganda) and 26% in Namibia and it appears that the test is not suitable for the testing of sera.

Discussion

Studies on the distribution of the disease (surveillance)

For the establishment of successful control programmes it is essential to delineate infected areas from areas that are not infected. If CBPP positive animals are detected by clinical, post mortem or serological investigations in a new area, this area is becoming classified as infected. Abattoir surveillance was recommended as a useful tool for the detection of CBPP (Mariner, 2003). Although the lesions of CBPP in acute cases are pathognomonic the clinical or pathological diagnosis of new outbreaks should be confirmed by laboratory diagnosis through the isolation of the infectious agent.

If serological investigations are undertaken the detection of specific antibodies must be interpreted as a sign of infection until follow up investigations have explained the positive results. Serological positive animals can be the result of vaccinations, cross-reactive sera, or the results of infection with a field strain of *Mmm* SC. The expected serological prevalence in infected herds ranged in Cote d'Ivoire between 0% by the cELISA and 4% by the CFT, in another case 19.6% by the CFT and 20% by the cELISA up to 58% by the CFT and 64% by the cELISA. Modelling suggested that between 3% and 75% of herds if the herd size is between 75 and 500 heads will be infected but only 1.8% to 73% are continuously infected. Cross sectional serological surveys should be designed to detect this basic herd prevalence and within-herd prevalence. Although the established serological tests such as the CFT and the cELISA have low sensitivities, it is possible to compensate for a lower sensitivity by increasing the sample number; e.g. if the sensitivity of a test is only 50% it is possible to compensate for the reduced sensitivity by doubling the sample size. It is more difficult to compensate for a low specificity since the testing of primarily negative samples from supposedly disease free areas will result in high number of (false) positives; e.g. in a scenario with disease at a 5% level where a test with a sensitivity of 100% and a specificity of 100% is applied 59 samples are required to detect at least one positive animal with 95% confidence, if the sensitivity drops to 50% 119 samples are required. If in the same scenario a test with 95% specificity and 100% sensitivity is used 330 samples would be needed and the population would be considered as infected with 95% confidence if 23 or more positives are detected. Because of the reduced specificity of the test there are 5 false positive samples for every 100 true negative samples that have to be clarified through followed up investigations at the field level. This example illustrates that for the surveillance a less than perfect sensitive test is acceptable but a reduced specificity results in rapidly growing sample numbers and associated follow up work.

The specificity of the CFT was not assessed as part of the CRP but the reported results for the specificity of the CFT vary from 96.5% to 99.5%. The results of the FAO/IAEA CRP and other reports suggest a specificity of the cELISA close to 100% if large numbers of samples are tested. To assess the true sensitivity of the cELISA it would be necessary to determine what percentage of animals were detected by the CFT and not detected by the cELISA were vaccinated animals and which were true positive samples, considering that the specificity of the CFT is less than 100%. This was not possible in the present study. On the other hand it is known that the sensitivity of the CFT is less than 100% in particular in chronic infections. In a number of countries the cELISA detected more animals than the CFT. Considering that the specificity of the cELISA is close to 100% it must be assumed that these are animals that were missed by the CFT and that the true sensitivity of the cELISA is considerably higher than the relative sensitivity compared to the CFT. Estimates of the relative sensitivity of the cELISA to the CFT in chronic infection range from 54% (Cote d'Ivoire) to 100% (Nigeria). To calculate the necessary sample sizes for disease surveillance to delineate infected areas from non-infected areas it is therefore suggested to adopt a conservative estimate of 50% sensitivity for the cELISA. This is below the true sensitivity and will lead in the design of the sampling frame to an overestimation of the sample size. At the same time a conservative estimate on the minimum expected prevalence should be adopted which will avoid that control strategies are based on erroneous assumptions that areas are free of disease.

The required sample sizes (number of herds) to detect disease at certain prevalences is shown in Table 1. Table 2 shows the number of animals that have to be sampled to detect disease within a herd with a probability of 95%. Assuming that in an infected herd 20% of animals became infected this would result in a sample size of 15 animals to detect the disease with 95% confidence. If the sensitivity of the test used is only 50% twice the number of samples are required. Modelling suggests that the proportion of herds with active infection in an infected area is above 3%. The serological prevalence will be well above this figure so that the probability that antibody to *Mmm* SC will be detected in at least one herd would be above 95% if 105 herds were sampled.

During the surveillance activities to achieve an international recognition of freedom from rinderpest infection the member countries of PACE are implementing serosurveys which are designed to detect disease at a level of 1% infected herds. These surveys are based on randomized selection of sampling sites and since the stratification for a CBPP survey would be in many countries similar to the stratification of the rinderpest surveys it is possible to make relative precise estimates on the distribution of CBPP.

Table 1. Number of herds which have to be sampled to detect at least one positive herd with a probability of 95% if infection within a herd is also detected with 95% probability.

Herds to be sampled per survey

	Herd prevalence				
Number of strata	1%	2%	3%	4%	5%
1	314	157	105	79	63
2	628	314	210	158	126
3	942	471	315	237	189
4	1256	628	420	316	252

Table 2. Number of samples needed to detect at least one positive in large herds (>10.000). If a diagnostic test with lower sensitivity is used the numbers have to be increased proportionally.

Prevalence	1%	10%	15%	20%	25%	30%	35%
Sample No.	298	30	20	15	12	10	9

Disease detection in buffer zones

Buffer zones or ‘cordon sanitaires’ are established to prevent the spread of infection from endemic areas into disease free areas. This is achieved through a combination of movement control, vaccination, stamping out and intensive surveillance. In this area serological surveillance is difficult since both the CFT and the cELISA will detect vaccinated animals at various levels. Results from Tanzania indicate that the specificity of the cELISA in vaccinated animals might be close to 100% in a situation where the CFT reacts with 96% of the animals. This would result in substantial numbers of false positive results if serological surveillance is used but considering the high specificity of the cELISA combined with abattoir surveillance, clinical surveillance and follow up of positive results through cluster analysis and correct epidemiological analysis serological surveillance can be a useful tool. None of the two tests was shown to be a reliable tool to monitor vaccination coverage.

Disease detection in surveillance zones

Early detection of the outbreak of disease in the surveillance zone is essential to avoid the further spread of the disease. No vaccinations are carried out in the surveillance zones, which is part of the buffer zones. Abattoir, clinical and serological surveillance should be carried out in this zone. The CFT and the cELISA are complementary and a parallel testing strategy

should be adopted where a positive test result in any of the two tests will trigger a follow up action with outbreak investigations.

It is not possible to calculate the sensitivity of such a combined test system consisting of serological tests and clinical observations. Estimates of the combined sensitivity of two independent serological test systems is shown in Table 3). Parallel testing strategies increase the sensitivities considerably but the disadvantage is that the specificity of the test system is reduced proportionally.

Table 3: Combined sensitivities of two independent test systems in parallel testing, e.g. if a serum sample is tested in the CFT (assumed sensitivity 0.6) and in the cELISA (assumed sensitivity 0.7) in parallel and a positive result in either of the tests is used to classify the sample as positive, the combined sensitivity is 88%.

Sensitivity CFT/cELISA	0.3	0.4	0.5	0.6	0.7	0.8	0.9	1
0.3	0.51	0.58	0.65	0.72	0.79	0.86	0.93	1
0.4	0.58	0.64	0.7	0.76	0.82	0.88	0.94	1
0.5	0.65	0.7	0.75	0.8	0.85	0.9	0.95	1
0.6	0.72	0.76	0.8	0.84	0.88	0.92	0.96	1
0.7	0.79	0.82	0.85	0.88	0.91	0.94	0.97	1
0.8	0.86	0.88	0.9	0.92	0.94	0.96	0.98	1
0.9	0.93	0.94	0.95	0.96	0.97	0.98	0.99	1
1	1	1	1	1	1	1	1	1

Prevalence studies in endemic areas

Prevalence studies are carried out in areas where the disease is endemic to assess the impact of the disease or to decide on a more appropriate control strategy (e.g. stamping out versus vaccination). Prevalence studies are best carried out through cross sectional serological studies. Reliable prevalence studies are only possible based on a random selection of the samples and abattoir surveillance is not suitable to estimate the prevalence since the study population will be biased. If the sensitivities and specificities of the tests are known it is possible to correct the prevalence estimates and to use tests with low sensitivities. Both serological tests (CFT and cELISA) might be recommended for mass screening but preference should be given to tests which can be quality controlled such as the cELISA.

Confirmation of outbreak

It is essential to confirm the clinical diagnosis or the post mortem diagnosis through laboratory investigations if outbreaks occur in areas previously not infected. This must include the isolation of *Mmm* SC, the CBPP agent. The serological tests most widely used, the CFT and the cELISA detect antibodies to *Mmm* SC at different stages of infection and no test can detect all infected animals. Serological confirmation should be carried out at the herd basis since both serological tests are not sensitive enough at the animal level to refute the diagnosis based on a single animal. A minimum of 15 blood samples should be collected from clinically affected animals and tested in both tests. The influence of antibiotic treatments on the performance of the serological tests has not been assessed and should be investigated.

Conclusions

None of the validated diagnostic test is sufficient on its own for all the needs of the diagnosis and surveillance of CBPP. However, if the CFT and the cELISA tests are used in an epidemiological and statistical meaningful way and if these tests are combined in correct testing strategies it is possible to detect new disease outbreaks and to make reliable estimates on the distribution of the disease which will enable the implementation of targeted and focused control programmes. The surveys which are needed to assess the distribution of the disease can be undertaken using the existing structures and resources which were established through PACE and through the FAO/IAEA support programmes to carry out rinderpest surveillance. The laboratory capacity to carry out the necessary large scale serological surveys are in place in most African countries, but the diagnostic capacity to isolate *Mmm* SC and to confirm outbreaks of CBPP exists only in few countries.

References

OIE (2000). Chapter 2.1.6., Manual of standards for diagnostic tests and vaccines, 4th edition.

Bellini, S. Giovannini, A., di Francesco, C., Tittarelli, M., Caporale, V., Sensitivity and specificity of serological and bacteriological tests for contagious bovine pleuropneumonia, Rev. sci. tech. Off. Int. Epiz., 1998, 17 (3), 654-659

Stark, K.D.C., Vicari, A., Tontis, A. und Nicolet, J. (1995) Untersuchungen zur Epidemiologie der Lungenseuche in der Schweiz. Schweiz. Arch. Tierheilk. 137, 92-100

Le Goff, C., Lefevre, P.C., 1989. Peripneumonie contagieuse bovine: test immunoenzymatique et cinetique d'apparition des anticorps au cours d'une infection experimentale: relation entre la fixation du complement, l'excretion et la recherché de l'antigene cirulant. Rev. Elev. Med. Vet. Pays Trop. 42, 365-369

Le Goff, C., Thiaucourt, F., (1998) A competitive ELISA for the specific diagnosis of contagious bovine pleuropneumonia (CBPP). Vet. Microbiol. 60, 179-191

Amanfu, W., Sediade, S., Masupu, K.V., Benkirane, A., Geiger, R., Thiaucourt, F., (1998), Field validation of a competitive enzyme-linked immunosorbent assay for the detection of contagious bovine pleuropneumonia in Botswana. Rev. Elev. Med. Pays Trop. 51: 189-193

Bruderer, U., Regalla, J., Abdo, E., Huebschle, O.J.B., Frey, J., (2002) Serodiagnosis and monitoring of contagious bovine pleuropneumonia (CBPP) with an indirect ELISA based on the specific lipoprotein LppQ of Mycoplamsma mycoides subsp. mycoides SC, Vet. Microbiol. 84, 195-205

March, J.B., Kerr, K., Lema, B., (2002), Rapid Detection of Contagious Bovine Pleuropneumonia by a Mycoplamsma mycoides subsp. mycoides SC Capsular Polysaccharide-specific Antigen Detection Latex Agglutination Test, Clin. Diagn. Lab. Immunol.,Vol 10, No. 2

IAEA (2002). Monitoring of contagious bovine pleuropneumonia in Africa using enzyme immunoassays, (in print), Proceedings of the Final Research Coordination Meeting of an FAO/IAEA Co-ordinated Research Project (CRP) on the "Monitoring of contagious bovine pleuropneumonia in Africa using enzyme immunoassays", TECDOC.

FAO/IAEA, (1998). Manual for competitive CBPP ELISA, Bench Protocol Version CBPP 2.1, January 1998, IAEA, Vienna.

Kit Insert (2002). CBPP serum competition ELISA, Vers. P05410/01, Pourquier, Montpellier, France

Campbell, A.B., Turner, A.W. (1953) Studies on Contagious bovine pleuropneumonia of cattle: An improve complement fixation test, Brit. Vet. J. 29, 154-163

March, J., (2002) CBPP Latex agglutination test (LAT) data sheet, Moredun Research Institute, UK.

Mariner, J.C., 2003, The Dynamics of CBPP Endemism and the Development of Effective Control/Eradication Strategies for Pastoral Communities, Final Modelling Report.

Specific diagnostic and epidemiological procedures adopted for the contagious bovine pleuropneumonia eradication programme (1995-2002) in Portugal: A working model for the disease control in African countries

José Regalla

Laboratório Nacional de Investigação Veterinária, Estrada de Benfica 701, 1549-011 Lisboa, Portugal

Introduction

Contagious bovine pleuropneumonia (CBPP) has re-emerged in a few European countries during the last 10 years. This severe contagious disease causes important economic losses and considerable socio-economic problems in many countries worldwide, espacially in Africa. The control of diseases with such impacts is an important objective of National Veterinary Administrations.

Evolution of the disease

CBPP was confirmed in Portugal in 1983 after being absent from the country for about thirty years (1954-1983). It was first recognised in January in a tuberculin positive dairy cow slaughtered at the abattoir of Monção, north of Portugal, near the Spanish border (Regalla, 1984). The disease became endemic in the coastal strip of the agricultural regions of Entre-Douro e Minho (EDM) and Beira Litoral (BL), where the intensive dairy farming is practised and the production of milk is most significant. Besides socio-economic features the most important factors responsible for the spread of the disease and the endemic situation in both infected regions of EDM and BL were the very high densities of bovine population and common milking parlours, as well as the very high numbers of farms very dependent of external replacement. Other important factors were the existence of fairs, traditional cattle-shows and communal pasture lands.

A national serological survey, carried out in 1983 provided an assessment of the incidence and prevalence of the disease, demonstrating a progressive increase from the South to the North with a high number of outbreaks occurring in the central Northern area of the country in the regions of EDM and BL and sporadic outbreaks in Trás-os-Montes (TM) (Regalla, 1984). For disease control and eradication between 1985 and 1989 the country, was divided into three different areas, Endemic, Risk and Disease-free Zones. From 1989 a new reinforced eradication plan was established, co-financially supported by the European Community, and the country was divided into Infected, Buffer and Disease-free Regions (Regalla *et al*., 1996). The following programmes have been developed and adapted according to the epidemiological progression of the disease. A wide programme for 5 years (1985-1989) and further, programmes of 3 and 2 years were established: (1990-1992); (1993-1995) and (1996-1997). The main measures applied between 1985 and 1989 were the following:

i) Endemic Zone, serology on all holdings once a year and ban on bovine movement out of the area;

ii) Risk Zone, serology, once a year, on about 50% of random holdings and control of bovine movement inside the zone;Disease-free Zone, serology, once a year, on 15% of randomly chosen holdings and bovine movement controlled, only allowed from holdings of risk zone where no cases of CBPP or even positive serological results had ever occurred. Further developments lead to changes in serological monitoring during the period between 1989 and 1997. In the Infected Region, serological surveys were increased to

twice a year on all holdings and in the Disease-free Regions, but only on 10% of random selected holdings. All sero-reacting animals were slaughtered, lungs and lymph nodes examined and lesions submitted to laboratory investigations. Two other systems were developed to complement the sero-surveillance: (a) abattoir inspection of routinely slaughtered beef and dairy cattle to detect lung lesions suspicious of CBPP. If infection was confirmed an effective trace-back system was installed to allow the identification of the infected herd; (b) the surveillance of suspicious clinical cases at herd level. All sero-reacting and suspicious clinical cattle were slaughtered, their lungs and lymph nodes examined and the lesions submitted to laboratory investigations. Stamping outs, involving thousands of animals, were made during 1997 in the areas of highest disease prevalence in both regions EDM and BL.

A decreasing trend in CBPP outbreaks was seen in EDM and BL since 1986: 466 in 1986 against 12 in 1998. In EDM the total number of outbreaks in 1998 correspond to a prevalence of 1.1 outbreaks in 10,000 herds. The last outbreak in Portugal was reported in February 1999 where, at the time 91,216 herds were recorded and occurred in the EDM region. Since then no outbreaks have been reported. In EDM, the most affected region, between 1989 and 1999, the decrease in the number of cattle with lesions was significant: 1,141 out of 2,485 had lesions in 1989 versus 19 out of 1,747 in 1998. In 1999, only 1 animal out of 191 slaughtered showed typical lesions.

Adopted tests

Until 1997, within the framework of the eradication programmes, the methodology used for CBPP diagnosis involved the following tests: (i) Complement fixation test; (ii) Bacteriological examination of suspicious material and, (iii) Histological examination of lesions. From 1997, besides the conventional tests, Polymerase Chain Reaction was also been applied (Bashiruddin *et al*., 1994).

The CFT was an essential tool of the eradication campaign with a high specificity of 99.5% but during the last years (Regalla, 1995), due to the low prevalence of the disease, its specificity became critical with false positive results corresponding to about 5,000 cattle slaughtered per year. As a consequence, a strong research effort to improve the test methodology was carried out, culminating with the successful development of a new test: the Immunoblotting test (IBT). The IBT showed a higher specificity than the CFT (Regalla *et al*., 2000) and it has been used, since January 1998, as a confirmatory test in CFT positive animals. The IBT specificity lies in the identification of a IgG core profile of 5 specific immunodominant antigenic bands of *M. mycoides* subspecies *mycoides* SC, with apparent molecular weights of 110, 98, 95, 62/60 and 48 kDa (Regalla *et al*., 2000). This profile is confirmatory of the individual infection. Between 1998 and 2002, a total of 20,588 CFT sero-reacting sera were examined by IBT and a total of 64 IgG immunoblot specific profiles were identified (Table 1). The last specific profile was detected in 2000 with no further confirmation of infection after herd slaughter.

The on-going serial testing (CF test and IBT), within the framework of Portuguese eradication programmes, has allowed the re-arrangement of the regions' division of the territory. Since 1998, all the procedures applied before initiating the eradication programme conformed to a more stringent serological surveillance (CFT and IBT on positive CFT sera) allowing an accurate assessment of the CBPP sanitary situation in the Portuguese territory and provide the assurance of disease eradication.

References

Bashiruddin, J. B., Taylor, T. K. and Gould, A. R. (1994). A PCR-based test for the specific identification of *Mycoplasma mycoides* subspecies *mycoides* SC. Journal Veterinary Diagnostic Investigation **6**, 428-434.

Regalla, J. (1984). Epidemiological aspects of contagious bovine pleuropneumonia in Portugal. *Repositório de Trabalhos do Laboratório Nacional de Investigação Veterinária*, XVI, 13-18

Regalla, J. (1995). La réaction de fixation du complément pour le diagnostic sérologique de la péripneumonie contagieuse bovine: application et interprétation des résultats. *Revue Scientifique et Technique*, 14 (3), 631-644.

Regalla, J., Caporale, V., Giovannini A., Santini, F., Martel, J. L. and Penha Gonçalves, A. (1996). Manifestation and epidemiology of contagious bovine pleuropneumonia in Europe. *Revue Scientifique et Technique*, 15 (4), 1309-1329.

Regalla, J., Gonçalves, R., Niza Ribeiro, J., Duarte, L., Nicholas, R., Bashiruddin, J., De Santis, P., Garrido Abellan, F. and Penha Gonçalves, A. (2000). Development of immunoblotting as a diagnostic tool for contagious bovine pleuropneumonia. *In* COST 826 Agriculture and biotechnology “Mycoplasmas of ruminants: pathogenicity, diagnostics, epidemiology and molecular genetics”, (Ed. D. Bergonier, X. Berthelot, and J. Frey), Rep. No. EUR 19245 EN, European Commission, Luxembourg, 109-112.

Table 1. Results of Complement Fixation test and immunoblotting test serial testing performed between 1998 and 2002, within the frameworks of Portuguese eradication programmes (Direcção Geral de Veterinária – Ministério da Agricultura e Pescas, Portugal)

Sera	Infected Areas						Free Areas					
	1998	1999	2000	2001	2002	Total	1998	1999	2000	2001	2002	Total
Total # of samples Tested in CFT	887,093	863,300	557,229	369,386	431,637	3,108,645	60,546	79,029	64,134	169,726	96,910	470,345
Total # of CFT positive reagents	3,900 (0.4%)	4,573 (0.5%)	3,459 (0.6%)	4,359 (1.2%)	4,454 (1,0%)	20,745 (0.7%)	36 (0.06%)	344 (0.4%)	154 (0.2%)	384 (0.2%)	383 (0.4%)	1,301 (0,3%)
Total # of samples Tested in IBT	3,635	4,311	3,316	4,066	4,119	19,447	26	279	148	357	331	1,141
Total # of IBT positives	41 (1.1%)	11(0.3%)	12 (0.4%)	0	0	64 (0.3%)	0	0	0	0	0	0
	0.005% CFT	0.001% CFT	0.002% CFT	0	0	0.002% CFT	0	0	0	0	0	0

CFT – Complement Fixation Test; IBT – Immunoblotting Test; CFt - Total number of CF tested

Using participatory epidemiology to assess the Impact of livestock diseases

Andy Catley and Berhanu Admassu

Community-based Animal Health and Participatory Epidemiology (CAPE) Unit, Pan African Programme for the Control of Epizootics, African Union's Interafrican Bureau for Animal Resources, PO Box 30786, 00100 Nairobi, Kenya.

Introduction

In an era of declining public sector veterinary services in Africa, priority setting and rational allocation of resources is becoming increasingly important. Regarding livestock disease control, many countries lack the basic epidemiological and economic information that enables disease problems to be prioritised at local or national levels. Furthermore, information deficits are often most evident in those areas characterised by large livestock populations and high levels of poverty.

In recent years the methods of participatory rural appraisal have been adapted by epidemiologists to improve understanding of livestock diseases in resource-poor settings and in areas where conventional methods are difficult to use. The value of this approach is apparent from the emergence of participatory epidemiology (PE) as a distinct branch of veterinary epidemiology, and the application of PE by programmes such as the Pan African Programme for the Control of Epizootics (AU-IBAR) and the Global Rinderpest Eradication Programme (FAO).

This paper provides an overview of PE, outlines how PE has been used in impact assessment to date and proposes how PE can be adapted to understand how and why livestock keepers prioritise diseases.

What is participatory epidemiology?

Participatory epidemiology is the use of participatory methods to improve understanding of animal health issues. Key features are summarised below:

Attitudes and behaviour: Practitioners are required to assess their own professional and cultural biases. Essentially, they needed to be genuinely willing to learn from local people, not lecture to them but actively and patiently listen. This requires respect for local knowledge and culture.

Combined methods and triangulation: Participatory epidemiology uses interviewing, scoring and ranking, and visualisation methods (Table 1). Of these, interviews are the most important group of methods because they are used alone but also complement and form the basis for other methods. The visualisation methods include mapping (natural resource maps, social maps, service maps), seasonal calendars, time-lines, transects, Venn diagrams, flow diagrams. Scoring methods include matrix scoring and proportional piling. These methods are combined with conventional veterinary investigation and epidemiological tools.

The use of key informants: Although pastoral communities are recognised as knowledgeable about animal health matters, certain people are known to possess special livestock knowledge and skills. These local experts are important key informants for participatory epidemiologists.

Action-orientated: Participatory epidemiology aims to generate information that can be verified with communities and leads to agreement on appropriate action. Initially, the aims of a particular study or investigation should be clearly explained to avoid raising expectations. In some situations, further laboratory results will be required and the mechanism for transferring these results back to the community should be defined.

Methodological flexibility, adaptation and development: Participatory epidemiology is a relatively new branch of epidemiology that is still developing. The approach is based on qualitative inquiry and complements the qualitative nature of standard veterinary investigation procedures. According to the needs of a given community or organisation, participatory epidemiology can also combine the benefits of participatory approaches and methods with quantitative inquiry. Methodological adaptation is encouraged.

Table 1. Examples of participatory epidemiology methods

Information required	**PE methods[a]**
Background information:	
System boundary	Natural resource maps, social maps
Social organisation	Social mapping, Venn diagram
Wealth groups	Wealth ranking
Relative livestock ownership	Proportional piling
Preferred types of livestock reared	Livestock species scoring
Food, income and other benefits from livestock	Proportional piling
Marketing systems	Flow diagrams, service maps
Veterinary services	Service map, Venn diagrams, ranking and scoring
Resources available to rear livestock	Natural resource maps, transects.
Disease-specific information:	
Priority livestock diseases, with reasons	Disease scoring
Local characterisation of diseases according to disease signs and causes	Matrix scoring
Estimates of incidence and mortality	Proportional piling; progeny history
Temporal information:	
- history of livestock diseases	Timelines
- seasonal variations in livestock disease, vectors and livestock-wildlife interactions	Seasonal calendars
Spatial information:	
- contact with neighbouring herds, wildlife, disease vectors	Mapping; mobility maps
- areas of disease events	Mapping
- preferred control options, with reasons	Matrix scoring

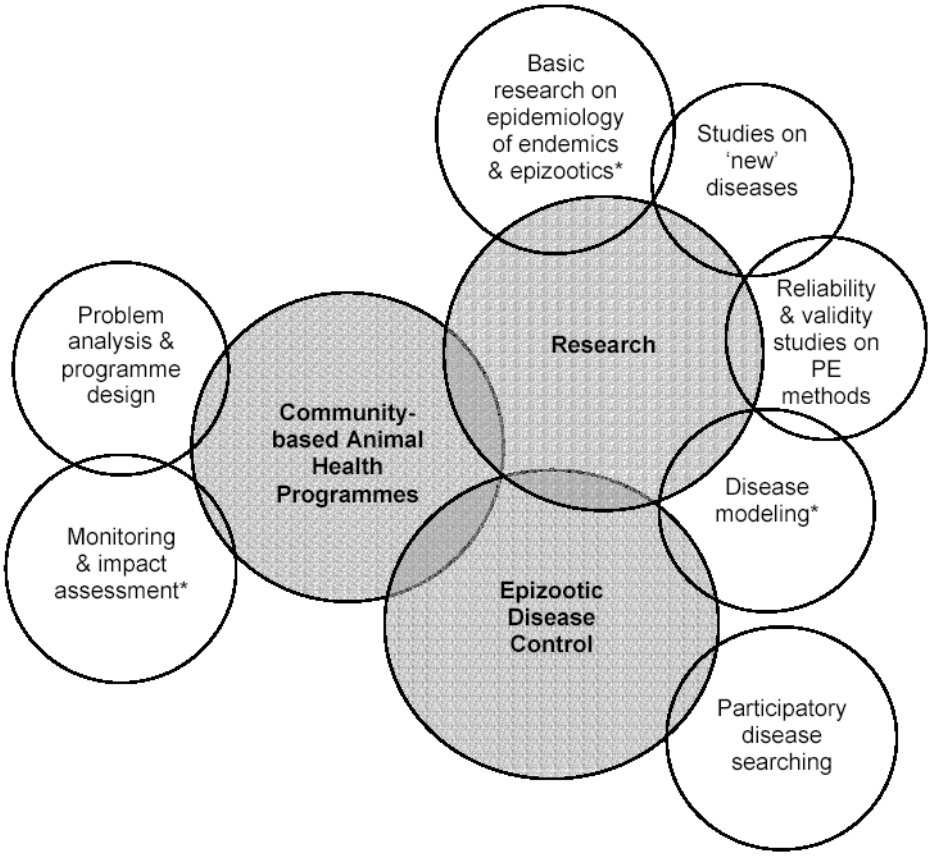
[a]Semi-structured interviews can provide information on all topics

Uses of participatory epidemiology

Uses of PE to date are summarised in Figure 1. Experiences of particular relevance to impact assessment are:

- Basic epidemiological research, including estimates of disease incidence and mortality;
- Methods used in the impact assessment of community-based animal health programmes.

Figure 1. Current uses of participatory epidemiology in pastoral areas of the Horn of Africa



Uses marked with an asterix are particularly relevant to impact assessment of livestock diseases.

Basic epidemiological research: estimates of disease incidence and mortality

Participatory epidemiology studies have included estimation of disease incidence and mortality using methods such as proportional piling. Some of the benefits of the method include:

- Population data in terms of numbers of animals is not required. A population or herd is defined using spatial and temporal criteria. This avoids sensitive questions on herd size and means that the method can be used in areas with limited or no baseline data on population.

- Local definitions of herd structure and age groups are used, together with local disease names. This reduces translation errors and specifically, nondifferential misclassification bias (cf. questionnaires).
- The method is comparative and assesses up to 10 diseases simultaneously. If the researcher has an interest in a particular disease (e.g. CBPP), informants should not be aware of this interest.

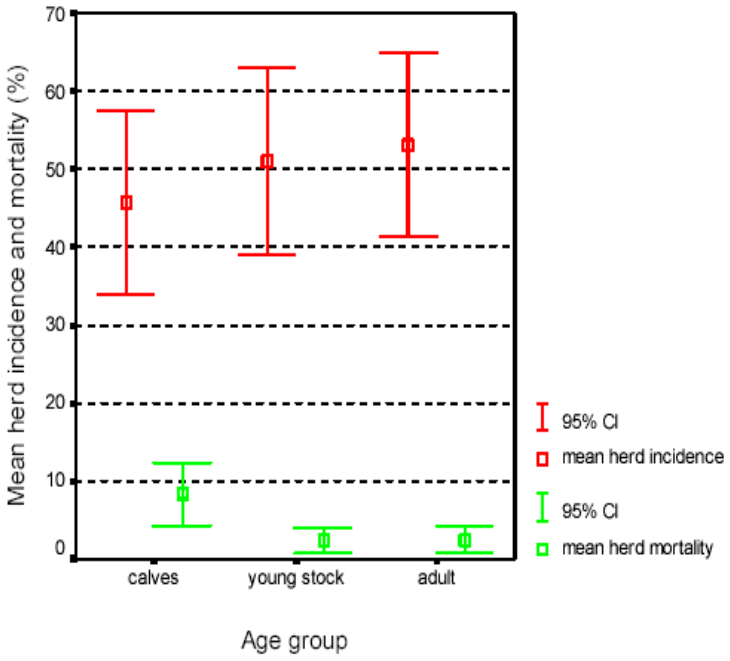
Some of the difficulties or limitations of the method include:

- Very careful explanation of the method and therefore good training of researchers is required.
- Most application so far has been with pastoral or agropastoral informants, with strong diagnostic ability. The method may be less useful with other types of livestock keeper. Crosscheck diagnostic skills with other methods e.g. matrix scoring.
- Recall is an issue. Pastoralists seem able to accurately recall disease events over many years and in specific animals, but what about other livestock keepers? Cross-check with timelines and secondary data on disease outbreaks.

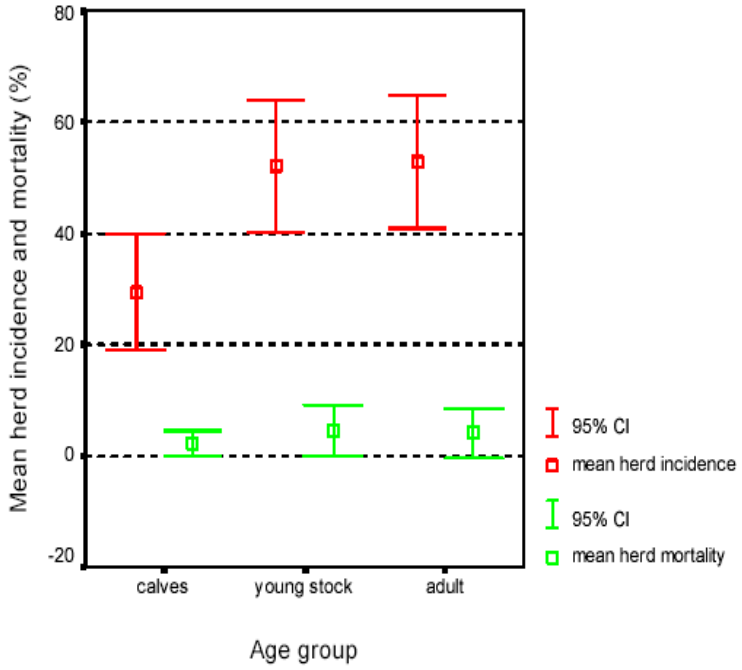
Examples of the type of data that can be produced by proportional piling are shown in Figures 2 and 3.

Figure 2. Mean herd incidence and mortality estimated for three cattle in Maasai herds, Morogoro region, Tanzania, 2000-2001 (n=50 herds) using proportional piling.

a. Olukuluku



b. Endorobo



c. Oltikana

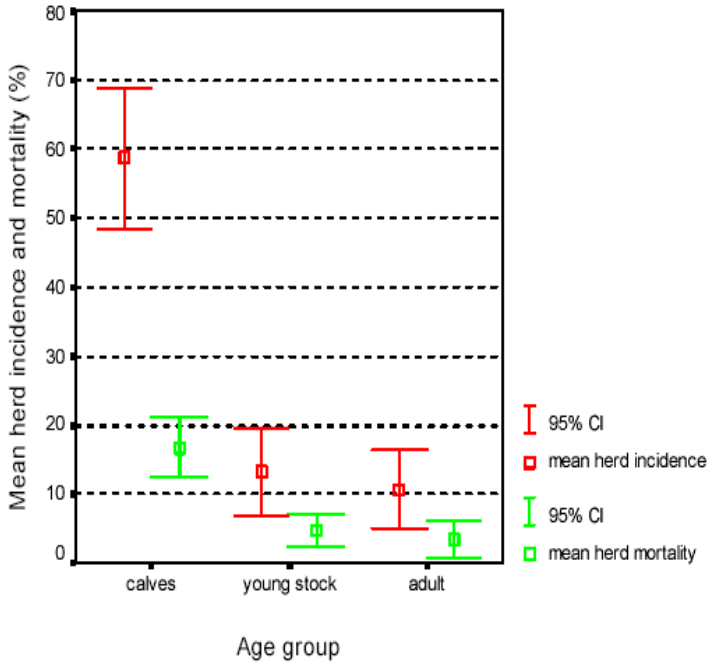
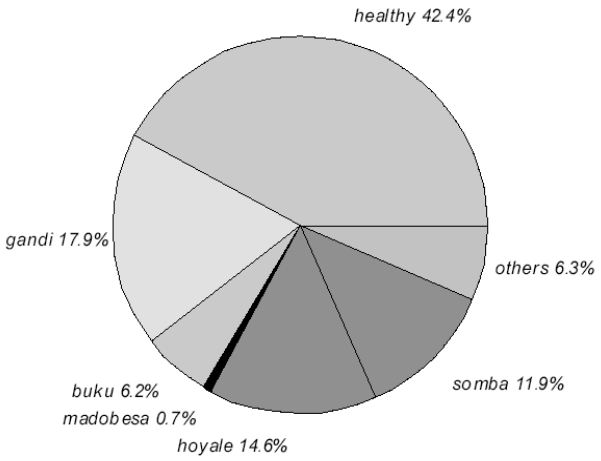


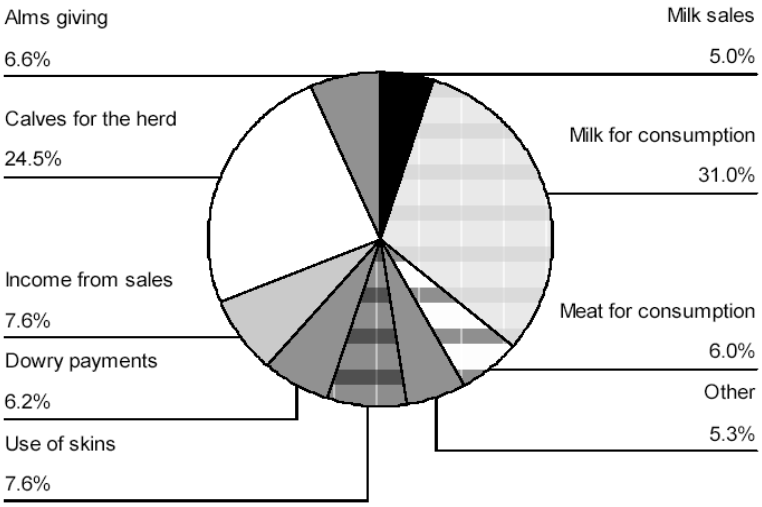
Figure 3. Estimates of cattle disease incidence and healthy cattle in Orma herds, Tana River District, Kenya, 1999-2000 (n=50 herds).



Methods used in the impact assessment of community-based animal health programmes

Impact assessment of community-based animal healthy programmes has included the use of locallydefined indicators of the impact of diseases. One of the principles here is that livestock keepers determine impact using some indicators that veterinarians might overlook. For example, an impact assessment in Ethiopia revealed that Afar herders regarded various 'social payments' such as alms giving and dowry payments as important benefits derived from cattle. In these communities, marriage requires payment of cattle to the bride's father and alms giving includes 'gifts' of livestock to the poor.

Figure 4. Relative importance of benefits derived from cattle in Afar communities, Ethiopia (n=10 informant groups, proportional piling)

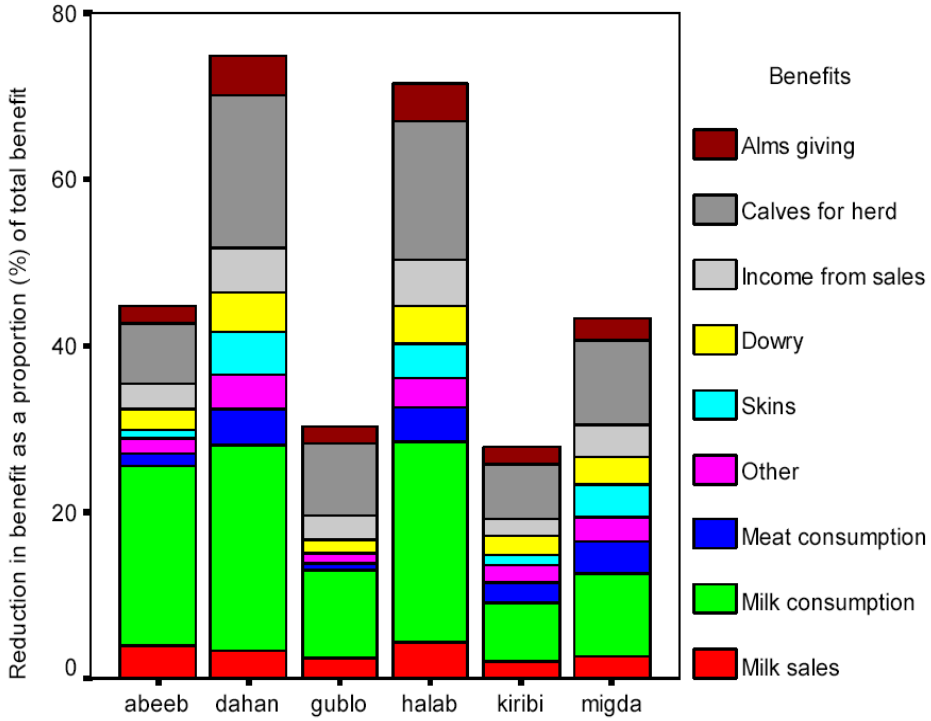


In the Afar example, standardisation of the method and repetition with different informants (or informant groups) allowed a statistical assessment of data reliability.

When livestock keeper perceptions of 'benefit' are known, it is then possible to compare methods such as proportional piling to show the impact of different diseases on

each of these benefits. An example is provided in Figure 5. Note that depending on the specific questions asked, this method can capture perceptions of incidence, mortality and duration of impact as an overall 'reduction in benefit' indicator.

Figure 5. Relative impact of six cattle diseases in Afar communities, Ethiopia.



An outline PE-based methodology for assessing the impact of CBPP

Based on the PE methods outlined above, a draft methodology for the comparative assessment of cattle diseases is presented in Table 2. This involves initial stages of defining a systems boundary and community identification of the 10 'most important' cattle diseases. In the event that CBPP is not mentioned during this initial stage, the research team can choose to add CBPP as an additional disease. However, this risks biasing the research because informants may suspect that the researchers have a particular interest in CBPP.

Table 2. Outline 'minimum' methodology for PE-based impact assessment of cattle diseases.

Information required (per study location)	Participatory appraisal methods:		Conventional methods/sources of secondary data
	Method	Sample size per location	
1. System boundaries: - spatial - temporal	Mapping Timelines	1 key informant group per method	Conventional maps DVO records
2. Livelihood sources by wealth group - sources of food - sources of income - contribution of livestock, by species, to livelihood	Wealth ranking; proportional piling	50 informants/wealth group	Socio-economic reports (if any)
3. Identification of the 10 most important cattle diseases[a]	Simple disease ranking crosschecked with pair-wise ranking	50 informants/ wealth group	DVO records; previous research studies
4. Analysis of impact of the 10 most important cattle diseases - identify local impact indicators[b] - relate impact indicators to diseases[c]	 SSI Matrix scoring	50 informants/ wealth group	Market records for value of livestock and livestock products
5. Incidence and mortality estimates	Proportional piling	50 informants/ wealth group	Previous studies
6. Options for preventing or treating the 10 most important diseases - identify control options used for each disease - rank/analyse preferences - identify & rank main constraints to control for each disease	SSI Ranking/SSI SSI/ranking	50 informants/ wealth group	
7. Market opportunities and constraints	Service maps, SSI, ranking	3 informant groups per wealth group	

Options/notes:
[a] This can be separated out by livestock species, but dramatically increases time inputs.
[b] Requires breakdown of general impact indicators e.g.
General indicator = cash
Specific indicators = uses of cash (food, school fees, clothes, medical etc).
[c] Includes impact in relation to acute or chronic nature of the diseases.

Advanced Veterinary Information Systems (AVIS)

Mark M. Rweyemamu

Technical Director, AVIS College53 Skylines, Limeharbour, LONDON E14 9TS, UK.

The International Animal Health Challenge

During the last decade, animal health has witnessed unprecedented challenges. These stem from a variety of causes, including: rapidly increasing demand for animal protein, the demand-driven "livestock revolution", the ever increasing consumer demand for quality and safe food of animal origin, dynamic changes in livestock farming, animal movement patterns and marketing practices, globalised movement of people and commodities, increasing concern for animal welfare, changes in climate and changes in the world economic order. Concomitant with such events have been changes in the incidence, distribution and dynamics of infectious animal diseases both new and old.

These new animal health challenges demand newer and more dynamic approaches to the acquisition and dissemination of knowledge about the diseases, the standards, the regulation and strategies for their effective prevention and progressive control. This is now the new paradigm in the management of animal health not merely as an on-the-farm technical service, but above all, as an international collective responsibility.

AVIS

AVIS (Advanced Veterinary Information System) was conceived in 1992 as an instrument for accelerating the adoption of the new information technologies into the global arena of animal health in order to facilitate access to authoritative information by animal health specialists throughout the world. It focuses on the development and production of internationally peer-reviewed, multi-media educational programs on infectious animal diseases including zoonoses and on food safety. AVIS has been championed by the consortium/parteneria of TELOS-Aleff Ltd, a specialised multi-media company together with the Institute of Animal Health (IAH), UK, and the two international standards setting and development agencies concerned with animal health, namely the Office International des Épizooties (OIE) and the Food and Agriculture Organization (FAO) of the United Nations. AVIS is a knowledge management tool.

The AVIS College

Ten years after its inception, the AVIS concept has now matured enough to venture into the next stage of a wider involvement of the animal health scientific community in order to bring world expertise within easy reach of all who have a stake in championing the goals of equitable, safe and globalised trade to meet the unprecedented high and increasing demand for animal products and world food security. This is the AVIS College without walls.

The AVIS Offering

The AVIS offering has now stabilized. It is modular in nature, disease and disease management focused. It is now fully internet enabled and offered as full multi-media suite that can be delivered either on CD or via the internet.
There are 3 categories of products:

- The AVIS Overviews - These are freely available on the FAO-EMPRES, Aleff Group and AVIS College websites.
- The AVIS full programs - These are programs that are authored by AVIS editors in close collaboration with the AVIS Consortium Partners. They are peer-reviewed and signed off by the AVIS Consortium Partners. Rinderpest, FMD and CBPP are examples of this category. They are available either on CD or via internet subscription by a cost-recovery system based on equity pricing with discounts for educational establishments and for developing countries.
- Commissioned AVIS Related Programs – Consortium Partners or others may commission special programs on terms agreed between TELOS-Aleff and the client. Examples include the rabies programs for North and Latin America and the GEMP program of EMPRES.

The AVIS Program Structure

The AVIS programs in the pipeline will be based on a 7-module structure, namely The Overview, The Disease, The Cause (including laboratory diagnosis), The Epidemiology (including surveillance and risk analysis), Disease Management, The Human Disease (as applicable) and The Resources (including, Tables, Charts, Images, Videos and interviews).

The CBPP program being demonstrated is the first in the new offering.

Further information may be sought at *http://www.aviscollege.com*

Contagious bovine pleuropneumonia: Possible future strategies for the control of the disease in the PACE region

Gavin Thompson

PACE Programme, AU-IBAR, PO Box 30786, Nairobi, Kenya.

Background

Contagious bovine pleuropneumonia (CBPP) is a disease of cattle caused by *Mycoplasma mycoides* subspecies *mycoides* (small colony) (*Mmm* SC). It has occurred at one time or another in all regions of the world with the exception of South America and Madagascar (Schneider *et al.*, 1994). It remains a significant constraint to cattle production in most of sub-Saharan Africa although a number of southern African countries have been free from the disease for many decades. Annual losses of US$ 2 billion have been ascribed to the disease in Africa although the reliability of this figure is uncertain (Masiga *et al.*, 1999).

The disease is transmitted by direct contact between infected and susceptible individuals. When first introduced into a fully susceptible cattle population CBPP usually results in widespread mortality. For that reason it is included in the Office International des Epizooties (OIE) list A of diseases (OIE, 2003). As an example of the devastation it can cause, within two years of its introduction into South Africa in 1853 it resulted in the deaths of over 100 000 cattle and was a major contributor to the Great Xhosa Cattle-killing Movement of 1856 to 1857 which resulted in the starvation of tens of thousands of Xhosa people and the devastation of that nation (Henning, 1956; Peires, 1989). In endemic situations the disease has a variable course and is often insidious in nature. Clinical forms of the disease include peracute, acute and chronic. There are also cases of inapparent infection when clinical disease does not occur. The latter condition includes carriers (apparently healthy animals that have recovered from the disease) with encapsulated sequestra in the lungs containing live organisms. The extent to which carriers (also referred to as “lungers”) are important in the maintenance and spread of the infection is a matter of contention (Mariner, 2003).

The disease is difficult to reproduce in the laboratory and the study of its epidemiology is problematic in endemic situations because of the insidious nature of the disease. As a result, many aspects of the basic biology, epidemiology, immunology and pathogenesis of CBPP are poorly understood. In particular, determination of fundamental epidemiological parameters such as the basic reproductive number (R_0) that enable inferences to be drawn on factors such as herd immunity levels required to control the disease effectively, have only recently begun to be addressed (Mariner, 2003).

Historically, CBPP was a disease of Europe, the Americas and Asia but was eradicated from the United States, Canada and most of Europe in the 19th Century through clinical diagnosis, movement control and slaughter of suspected cases (Provost *et al.,* 1987). Although CBPP was present in sub-Saharan Africa prior to the colonial era, it was imported by ship into the southern-most part of the continent from Europe in the mid 19th Century, as mentioned above, and subsequently spread as far north as Angola were it has persisted to the present (Windsor, 2000). The complexity of the origins of the infection in Africa is borne out by recent molecular epidemiological studies that have demonstrated three distinct African lineages of CBPP (Lorenzon *et al.,* 2003).

Progress was made in controlling CBPP in Africa during the colonial era and first two decades following independence. Large parts of Southern, Western and Eastern Africa were cleared using slaughter and movement control (Hammond and Branagan, 1965), which later

incorporated testing strategies based on the complement fixation test (CFT) (Campbell and Turner, 1936; Campbell and Turner, 1953; Huddart, 1960). However, a problem has been that the CFT, as well as the more recently developed competitive ELISA, are unable to detect carrier animals or those in the relatively long incubation period efficiently. Furthermore, lack of sensitivity on the part of these tests means that they can only be used to detect infected herds and are not reliable as a means of establishing freedom from infection in individual animals.

As vaccines became available, control programmes increasingly relied upon vaccination combined with movement control. However, vaccine coverage in Central, Eastern and Western Africa has declined since the closure of the Pan-African Rinderpest Campaign (PARC) in 1999 because during PARC vaccines against both rinderpest and CBPP were routinely administered to cattle in Central, Eastern and Western Africa (Kebkiba, 2003). In addition, it appears that the quality of vaccines used in recent times has declined (Waite and March, 2001). There are a number of factors that have contributed to this situation. Lack of independent quality control in some manufacturing facilities in Africa remains a problem and improper handling of the vaccine in the field (poorly maintained cold-chains, for example) have resulted in sub-optimal quantities of vaccine strain mycoplasmas being administered to cattle (Thiaucourt *et al.*, 2003). Occurrence of post-vaccination reactions in as many as 1% of vaccinated animals in some instances, as well as occasional deaths, have contributed to owner reluctance to use existing vaccines in certain areas. Furthermore, available serological tests do not detect vaccinated animals effectively so that sero-monitoring as a means of monitoring vaccination cover and establishing levels of herd immunity resulting from vaccination cannot be conducted effectively (Thiacourt *et al.*, 2003).

In the 1980s and 1990s, economic crises afflicted many African countries and the subsequent structural adjustment programmes resulted in a decline in the funding of public veterinary services. This had an inevitable effect on surveillance and control programmes in Africa, including those for CBPP (Windsor, 2000). Other factors such as increasing public empowerment, recognition of the negative effects of movement control on pastoral livelihoods and a decline in the ability of veterinary services to enforce policies has decreased the effectiveness of measures adopted against CBPP. As a result, the disease is again present throughout most of Eastern, Central and Western Africa (Masiga and Domenech, 1995). The East African focus has advanced south into Tanzania (Bölske *et al.,* 1995) and subsequently spread to most regions of that country. This poses a threat to north-eastern Zambia. The long-standing focus in Angola has again invaded western Zambia and more recently still has spread to north-western Zambia. Furthermore, at least 12 African countries are engaged in or recovering from major internal conflict. These events involve mass movement of people and animals and constitute a significant factor in the spread of CBPP.

As indicated above, African governments are facing acute economic and financial problems that have affected their ability to fund programs of national or regional importance in the animal health field. Livestock and animal health budgets are already small and are being cut further. Most governments rely heavily on complementary donor funding from bi-lateral and multilateral partners to finance animal disease control programs. The sustainability of these programmes is therefore in doubt. Conversely, senior animal health officials of most African countries express the imperative for introducing an integrated regional programme to either eradicate the disease or to greatly lessen its impact on livestock producers (Reports of PACE workshops on CBPP; Addis Ababa, November 2001; Accra, February 2003). In the light of these two incompatible trends a question that needs to be answered is: As national financial resources are limited and declining in absolute and relative terms, how would an integrated regional programme be financed? In this regard it is necessary not only to establish the cost but also the return on such investment. For example, would investing, say, US$150 million in regional CBPP control over a 5 year period

provide a better return than investing that money in human health improvement, education or infra-structure development?

Current policy advocated by PACE and AU-IBAR

In the final report of the Pan-African Rinderpest Campaign (PARC) the recommendation was made that future action against CBPP would require:

- Epidemiological data and information to determine and detect foci of infection;
- Effective control of animal movements from and towards these foci;
- Complete vaccine cover of cattle regularly for at least 5 consecutive years; and
- Repeat vaccination of the same cattle each year (Page 242 of the Draft Final Report: 1986-1999).

More simply, this implies close to 100% vaccination of all cattle twice a year for 5 years in addition to effective movement control.

However, since 2000 there has been limited external financial support for mass vaccination against CBPP in the countries of Central, Eastern and Western Africa. At least partially for this reason the objective of mass vaccine coverage of cattle populations of the region has, with very few reported exceptions, not been achieved. A cost recovery strategy for CBPP vaccination advocated through PACE has been implemented in most countries but this is insufficient to overcome budget deficits for implementation of effective mass vaccination. Nevertheless, as is reflected in the conclusions reached at the PACE workshop on CBPP held in Accra in February 2003, mass vaccination, whether subsidized or supplied free of charge by the veterinary services of countries, continues to be considered by most senior animal health officials in the PACE region as the preferred strategy against the disease. This is despite the fact that both the distribution and prevalence of CBPP have increased in recent years and continue to do so in the tropical regions of Africa (Report of the PACE Workshop on CBPP, Accra, February 2003). The inevitable conclusion is that existing policies are either inappropriate or are not being executed adequately.

There is a growing realisation that eradication of CBPP in the foreseeable future will be more difficult than was the case for rinderpest and probably is not a realistic possibility for the immediate future in the PACE region. If that is so, control of the impact of the disease needs to be the immediate objective (Reports of the PACE workshops on CBPP held in Addis Ababa and Accra in 2001 and 2003 respectively and report on the technical workshop of CBPP experts of 8 May 2003).

Regional options for the future

Judging from the report of the most recent PACE workshop on CBPP (Accra, February 2003) it seems that the weight of opinion among country representatives within the PACE region is that annual mass vaccination should be applied for the next 5 years – although now it is recommended that the cattle be vaccinated only once a year – to achieve control of the disease in infected regions of countries. Once that is achieved it is implied that follow-up action would be aimed at eradication of the disease although precisely what would be required in this respect has so far not been clearly articulated. The recommendations of the Accra meeting refer to a need to re-assess the effects of vaccination after 5 years. It was also recommended that areas free of infection should be protected from incursion of CBPP by vaccination (buffer) zones round the free area(s) and movement control. Ideally, buffer zones should be separated from free zones by surveillance zones in which intensive surveillance should be conducted (Report of the PACE Workshop on CBPP, Accra, February 2003).

It needs to be borne in mind that the recommendation of annual vaccination (in some cases biannual vaccination) for 5 years is based largely on empirical experience. It is only within the relatively recent past that techniques have become available for making extrapolations on the biological consequences of differing herd immunity levels resulting from vaccination. The study by Mariner (2003) is so far the only one that has utilized this approach for CBPP.

Since the Accra workshop held by PACE in February 2003, the report on the consultancy commissioned by PACE (CAPE and PEU, through FAO) and the report on the workshop of CBPP experts held in May 2003 have become available. The consultancy report based on the results of a modelling approach have provided valuable insights into the behaviour of CBPP and what will be required for its control. The study shows, firstly, that eradication of CBPP by mass vaccination alone will probably be unsuccessful. Secondly, it indicates that in order to achieve effective control (as opposed to eradication), high levels of herd immunity (≥80%) need to be attained.

Considering these findings, the workshop of CBPP experts held in May 2003 concluded the following:

- Insufficient funding and physical resources exist within the PACE region to sustain mass vaccination as a mechanism of control; therefore, mass vaccination would only be sustainable if significant outside funding support becomes available;
- Poor quality vaccines that induce ephemeral and/or poor immune responses are a problem for effective CBPP control in the PACE region;
- T1-44 vaccine, because it is inclined to produce post-vaccinal reactions that are unacceptable to livestock owners, constitutes a disincentive to widespread vaccine use for sustained periods of time (at the least it would require funds to compensate owners for losses resulting from post-vaccinal reactions);
- Lack of an *in vitro* test to differentiate vaccinated from unvaccinated cattle complicates control where vaccines are employed;
- Lack of trust and co-operation between some veterinary services and the livestock-owning communities they serve is a significant problem.

This situation leaves AU-IBAR with two alternatives:

1. Devise a regional mass vaccination programme to which donor organizations could be persuaded to contribute that would make up the financial shortfall in funding from country contributions and cost recovery on the one hand and the full cost of mass vaccination on the other; or
2. Develop an alternative strategy.

These two alternatives are discussed in more detail below.

Regional mass vaccination programme

The cost of such a programme, covering a 5-year period (whether the cattle were vaccinated once or twice a year) is likely to be high (see Table 1). The data in Table 1 were derived from information based on the costs of vaccination against CBPP during PARC in 10 countries. It shows the calculated cost of vaccinating 70% of the cattle in the selected countries annually over a 5 year period. This level of vaccine cover was selected because it is: (1) unlikely that all cattle in any country could be consistently vaccinated, and (2) most countries have zones within the country where vaccination is not traditionally practiced because CBPP has little or no impact in those zones (some are claimed to be free of the infection). It is evident that if a high level of vaccination coverage were to be achieved in the >30 countries that constitute Central, Eastern and Western Africa, the overall costs would be

approximately three times greater than those shown in Table 1, i.e. >€ 300 million. It is possible that up to half of this cost could be recovered from the cattle owners but even so a sum of € 150 million is large and probably unrealistic in comparison with previous aid projects directed towards animal health.

Table 1. Total cost (€) of CBPP vaccinations in ten countries (70% mass vaccination scenario)

Country	Year 1	Year 2	Year 3	Year 4	Year 5	Total cost
Ethiopia	6,709,500	5,692,340	5,646,044	5,805,059	5,896,309	29,749,252
Tanzania	4,212,600	3,573,970	3,544,903	3,644,742	3,702,033	18,678,248
Mali	1,909,292	1,619,843	1,606,669	1,651,919	1,677,886	8,465,609
Uganda	2,478,000	2,102,335	2,085,237	2,143,966	2,177,666	10,987,204
Burkina Faso	1,612,800	1,368,300	1,357,171	1,395,395	1,417,329	7,150,995
Senegal	1,040,060	882,387	875,210	899,860	914,005	4,611,522
Cote d'Ivoire	619,920	525,940	521,663	536,355	544,786	2,748,664
Kenya	5,670,000	4,810,428	4,771,305	4,905,684	4,982,796	25,140,213
Ghana	580,580	492,564	488,558	502,318	510,214	2,574,234
Benin	466,550	395,821	392,602	403,659	410,004	2,068,636
Total cost	**25,299,303**	**21,463,930**	**21,289,365**	**21,888,961**	**22,233,033**	**112,174,592**

Assumptions:

1. Cattle population increasing at 1% per year over the base scenario.
2. 70% vaccination coverage.
3. Depreciation of capital equipment from 20% year 1 down to 65% by year 5.

Furthermore, it is unlikely that such a large sum of money would be made available by donors for an animal disease unless there was good reason to believe that it would result in the eradication of the disease and that the extent of the return on the investment could be shown to be at least several-fold higher than the sum invested in the programme. It should also be noted when considering donor support that most European donors link their funding to millennium goals of poverty alleviation and freedom from hunger and the PRSPs whilst USAID is linking its funding to good governance and business development. Merely showing a benefit to cost is unlikely to be sufficient incentive for donor support in the near future.

As already stated, the modelling study of Mariner (2003) indicates that eradication through vaccination alone would be unlikely to succeed. Even effective control over a wide area would require herd immunity levels of around 80% that were hardly ever achieved against rinderpest during PARC. Taking all this into consideration leaves little room for optimism that a mass-vaccination programme against CBPP on its own would be either affordable or effective. Based on experience elsewhere in the world and that from Africa in previous decades, an effective programme would require, in addition to high vaccination coverage, good control over the movement of infected or potentially infected cattle into areas where vaccine is being applied. This would necessitate additional costs, some indirect and essentially hidden resulting from limitations imposed on the choices of livestock keepers, pastoralists especially, in respect of free movement of their animals. Mass vaccination for 5 years therefore has limited prospects for success in the long term.

Alternative strategy

Because CBPP is endemic to many regions of Africa between the Sahara and 15° S, livestock owners have to live on a daily basis with the consequences of the disease, i.e. its erosive effects and periodic epizootics.

It was generally conceded at the two PACE workshops on CBPP held in Addis Ababa and Accra that antibiotic treatment of cases of CBPP is a widespread practice throughout the PACE region. This is despite the fact that treatment of CBPP cases is actively discouraged in many PACE countries and even illegal in some. Antibiotics usually have to be bought by the owners and therefore treatment of cattle for CBPP and other diseases costs owners hard currency which they can ill afford. Conversely, when it comes to administration of vaccine few owners, certainly those in rural areas, have access to vaccine through drug dealers, community-based animal health workers (CAHWs) or private veterinarians. This creates a dichotomy in the approach to CBPP in most countries: the official veterinary service applies vaccine (usually with cost recovery) in areas and at frequencies that they dictate while treatment is discouraged or illegal and at the exclusive cost of the owner. This means that some owners who would like to have their cattle vaccinated have no means of having this done.

The efficacy of treatment for CBPP, as agreed at all the recent workshops held by PACE on CBPP, is largely unknown as are the epidemiological consequences of treatment (possible creation of carriers) despite the fact that livestock owners obviously consider it beneficial. Studies into this issue are ongoing and some are being funded at least partially by PACE. The results are clearly important and anxiously awaited. However, pending the outcome of these studies it is intuitively probable that the best approach to the control of CBPP would be to regularly (say every 6 months) vaccinate cattle in endemically infected areas or those at risk of being infected while treating and, if possible, isolating individual animals when they develop clinical disease. In this way the benefits of both vaccination (creation of high levels of herd immunity) and treatment (enabling animals that would otherwise die or be seriously debilitated to recover) would hopefully act synergistically to reduce losses. The scientific evidence required to recommend the use of a particular antibiotic against a particular microbial agent is based on *in vitro* antibiotic sensitivity tests and determination of minimal inhibitory concentrations reached in various tissues and secretions as well as, where possible, controlled trials. In the case of CBPP, published information on the sensitivity of *M. mycoides* subspecies *mycoides* (small colony) to oxytetracycline (and tylosin) is available and demonstrates sensitivity to these drugs. As there is no scientific evidence to support the hypothesis that the use of these drugs causes a chronic infective CBPP state, there is no scientific reason currently not to use them to treat CBPP.

Mariner (2003) has suggested that elective vaccination of cattle (i.e. the owner decides whether to vaccinate or not and is responsible for the payment for such vaccination) in pastoral areas may be more effective and sustainable than mass vaccination conducted by official veterinary services. He has pointed out that for this to happen would require liberalising the distribution and availability of vaccines against CBPP. Furthermore, Mariner's study has shown that even if elective vaccination was patchily adopted, the benefits to the herd-owners who vaccinate their cattle would not be negated by neighbours who fail to do so.

Bearing the above in mind, a somewhat different approach to CBPP control can be considered. The alternative approach is based on enabling owners, either through private practitioners (or possibly veterinary-supervised paraprofessionals such as CAHWs) operating individually or on behalf of co-operatives, to vaccinate and, where necessary, treat their cattle to control CBPP. This presupposes that official veterinary services would support this process by:

- Enacting enabling legislation to permit non-official vaccination and treatment;

- Supply of vaccine and drugs in locations, under conditions (refrigeration) and quantities (small dose packs) that owners would find helpful.

Ideally, some form of management of the movement of cattle from areas where CBPP is a problem disease to areas that are free or relatively free of the disease, would favour better control than vaccination and treatment alone. Circumstances will dictate where that is possible and where not. However, it needs to be ensured that such movement management measures do not cause greater losses to the livestock industry than the disease itself. Veterinary authorities in Africa have consistently underestimated the knock-on effects of animal health control measures, which is one of the reasons why disease control measures that livestock owners find onerous are often ignored or surreptitiously avoided.

This proposal therefore envisages official veterinary services providing support to livestock owners to control the disease privately rather than physically directing and implementing vaccination and discouraging treatment. This would likely greatly reduce the cost of control to the public sector and enable more effective control for those that want, and are willing, to pay for it.

Clearly, this policy option is contrary to that adopted officially by many countries in the PACE region as well as that recommended internationally. It may also not appeal to many official veterinary services because actions directed towards control/eradication of CBPP are a major focus of their activity. On the other hand, it would overcome the problem of unaffordable – and therefore poorly implemented – vaccination programmes with unrealistic expectations that continually fail to meet their objectives. This unfavourable situation is unlikely to change in the foreseeable future unless, as indicated above, massive additional investment in CBPP control occurs.

CBPP vaccine quality is critical to its efficacy in the field and acceptability to farmers who are expected to pay for the cost of CBPP vaccination. Revival of vaccine quality assurance capacity at regional or pan-African levels is important to ensure good quality products. It is obviously untenable to expect livestock owners to pay for a product that does not reach minimum standards. Ultimately, it is the governments' responsibility to ensure that vaccines and antibiotics available to the public sector are of a suitable standard.

The devolution of CBPP control to the livestock owner would also create difficulties in accepting that a whole region of Africa would tacitly acknowledge that it is unable to eradicate a List A disease that has been eliminated from much of the rest of the world. This is likely to create difficulties in trading live animals but there are other ways of addressing this problem. For example, establishing disease-free or export zones of limited size that could either be kept free of CBPP or where measures could be instituted to reduce the risk of spread of the infection through trade are possible. Such approaches would enable international trade from a country that is otherwise not free of CBPP and would also create nuclei of infection-free cattle that could form the basis of a future eradication strategy.

If this approach were to be endorsed by IBAR and the PACE countries, it may be possible to turn CBPP control into a more palatable option (than mass vaccination) for donor support. Liberalising the use of the vaccine could be viewed as a way of stimulating the private sector delivery of veterinary services. The formation of export zones or systems that create wealth and have a trickle down effect to the poor is similarly likely to be more appealing to some donors than subsidizing mass vaccination campaigns. USAID, for example, is already heavily investing in promotion of livestock trade. The European Commission is increasingly interested in both trade and poverty alleviation.

References

Bolske, G., Msami, H.M., Gunnarsson, A., Kapaga, A.M. & Loomu, P.M., 1995. Contagious bovine pleuropneumonia in northern Tanzania, culture confirmation and serological studies. Tropical Animal Health & Production 27, 193-201.

Bidjeh Kebkiba, 2003. Analyse des strategies de lutte contre la péripneumonie contagieuse bovine (PPCB) dans les pays members du PACE. PACE Epidemiology Unit document, pp 16.

Campbell, A. D. & Turner, A. W., 1936. Bulletin of the Council for Scientific & Industrial Research; Australia, 97, 11.

Campbell, A. D. & Turner, A. W., 1953. Studies of contagious bovine pleuropneumonia of cattle. IV. An improved complement fixation test. Australian Veterinary Journal, 29, 154.

Hammond, J.A. & Branagan, D., 1965. Contagious bovine pleuropneumonia in Tanganyika. Bulletin of Epizootic Disease in Africa, 13, 121-147.

Henning, M.W., 1956. Animal Diseases in South Africa, 3rd edn. Central News Agency Ltd., Pretoria.

Huddart, J.E., 1960. Bovine contagious pleuropneumonia - A new approach to field control in Kenya. Veterinary Record, 72, 1253-1254.

Mariner, J.C., 2003. The dynamics of CBPP endemism and the development of effective control/eradication strategies for pastoral communities: Final modelling report. Project GCP/RAF/365/EC. Food & Agriculture Organization of the UN.

Masiga, W.N., Rossiter, P. & Bessin, R., 1999. Contagious bovine pleuropneumonia. 1. Epidemiology: The present situation in Africa and epidemiological trends. *In:* Report of the FAO/OIE/OAU-IBAR CBPP Consultative Group Meeting, Rome, Italy, 5-7 October 1998. FAO Publication X3960-E, pp 25-31.

Lorenzon, S., Arzul, I., Peyraud, A., Hendrikx, F. & Thiaucourt, F., 2003. Molecular epidemiology of contagious bovine pleuropneumonia by multilocus sequence analysis of *Mycoplasma mycoides* subspecies *mycoides* biotype SC strains. Veterinary Microbiology, 93, 319-333.

Masiga, W.N. & Domenech, J., 1995. Overview and epidemiology of contagious bovine pleuropneumonia in Africa. Scientific & Technical Review, *Office International des Epizooties*, 14, 611-30.

Office International des Epizooties, 2003. International Animal Health Code.

Peires, J.B., 1989. *The Dead will Arise*. Johannesburg: Ravan Press.

Provost, A., Perreau, P., Breard, A., Le Goff, C., Martel, J.L. & Cottew, G.S., 1987. Contagious bovine pleuropneumonia. Scientific & Technical Review, *Office International des Epizooties*, 6, 625-679.

Schneider, H.P., Van der Lugt, J.J. & Hübschle, O.J.B., 1994. Contagious bovine peuropneumonia. *In:* Infectious Diseases of Livestock. JAW Coetzer, GR Thomson & RC Tustin (eds), pp 1485-1494. Oxford University Press, Cape Town.

Thiacourt F., Van der Lugt J.J. & Provost, A., In press. Contagious bovine pleuropneumonia. *In:* Infectious Diseases of Livestock, 2nd edn. JAW Coetzer & RC Tustin, eds. Oxford University Press, Cape Town.

Waite, E.R. & March, J.B., 2001. The effect of HEPES buffer systems upon the pH, growth and survival of *Mycoplasma mycoides* subsp. *mycoides* small colony (MmmSC) vaccine cultures. FEMS Microbiology Letters, 201, 291-294.

Windsor, R.S., 2000. The eradication of contagious bovine pleuropneumonia from south western Africa: A plan for action. Annals of the New York Academy of Science, 916, 326-32.

Immune responses in cattle vaccinated against contagious bovine pleuropneumonia: Preliminary results

F. Mbithi[1, 4], H.Wesonga[2], F. Thiaucourt[3] and E.L.N. Taracha[4]

[1]*Kenya Agricultural Research Institute, Biotechnology laboratory, Nairobi, Kenya;* [2]*Kenya Agricultural Research Institute, Muguga, Kikuyu, Kenya;* [3]*CIRAD-EMVT, Montpelier, France;* [4]*International Livestock Research Institute, Nairobi, Kenya*

Introduction

Contagious bovine pleuropneumonia (CBPP) is one of the most important transboundary animal diseases in Africa. In recent years, CBPP has been found in countries like Botswana from where it was previously eradicated (Amanfu *et al.,* 1998). There is growing evidence to indicate that the incidence of the disease is increasing in endemic areas. These recent increases can be attributed to uncontrolled movement of cattle, poor disease control strategies and application of sub-standard vaccines (FAO, 2000; Masiga, 1996). There is sufficient consensus that efficacious vaccines could contribute substantially to an integrated control program for CBPP (Anonymous, 2000; Tuslane *et al.,* 1996). This would involve improvement of existing and development of a new generation of vaccines. This study was aimed at investigating the immunogenicity and efficacy of two different vaccines against CBPP.

Materials and Methods

Vaccines

The T1 44 vaccine strain and a saponin formulated inactivated virulent strain of *Mycoplasma mycoides* subspecies *mycoides* small colony *(Mmm* SC*),* strain 237*,* were used. Strain 237 was isolated from an outbreak of CBPP in Kenya and confirmed to be *Mmm* SC by the polymerase chain reaction (PCR) assay. Briefly, the preparation of the saponin-adjuvanted vaccine involved centrifugation of a 500 ml culture of strain 237 at 11000 *g*, washing the pellet twice in phosphate buffered saline (PBS) and finally resuspending in 10 ml of PBS. Inactivation of the cultured organisms was achieved at 56°C for 30 minutes. A protein estimation using the Bicinchoninic Acid method was done and the preparation stored at –20°C. On the day of vaccination, 1 ml of saponin obtained from the Kenya Vaccine Production Institute (KEVAPI) at a concentration of 1 mg/ml was added to 1 ml of the inactivated preparation at 2 mg/ml. The vaccine was used at 2 ml per inoculation. The T1 44 vaccine was administered at a dosage of $10^{7.3}$ per ml viable mycoplasmas.

Cattle and vaccination

Bos indicus cattle were obtained from a CBPP free area in Kenya. The cattle were screened for the presence of anti-*Mmm* SC antibodies using the complement fixation test (CFT) and the competitive enzyme linked immunosorbent assay (cELISA) and found to be negative by both tests. The experimental animals were divided into 3 groups of 9 each:

Group 1: Vaccinated twice with the T1 44 at two months interval.
Group 2: Vaccinated once with the T1 44.
Group 3: Vaccinated once with the inactivated saponin-adjuvanted vaccine.

The vaccine was administered subcutaneously on the right shoulder and in the case of group 1 animals, the booster inoculation was performed on the left shoulder.

Preparation of antigens for the proliferative assay

Four antigen preparations were derived from strain 237 including precipitate form, supernatant form, a whole viable mycoplasma and a heat-killed whole mycoplasma. Precipitate and supernatant fractions were obtained after 3 cycles of freeze-thawing mycoplasmal organisms, sonication and centrifugation. The heat-killed antigen preparation was obtained by boiling mycoplasma organisms for 1 hour in a water bath. Protein estimations using the Bicinchoninic Acid method were done to determine concentration of the antigen preparations.

Sampling and T cell assays

Sampling was done twice a week. Blood was collected for the isolation of peripheral blood mononuclear cells (PBMC) and preparation of sera. PBMC at a cell density of 5×10^5 cells/ml were dispensed in aliquots of 100 µl per well of 96-well, round-bottomed microtiter plates. Antigen preparations were added at a concentration of 10 µg/ml and cultures maintained at 37°C in a humidified carbon dioxide incubator for 4 days. Tritiated thymidine was added at 0.5 µCi per well during the last 20 hours. Cells cultured with the mitogen Concanavalin A (Sigma) served as positive controls, while those cultured in medium alone (RPMI 1640 with 20% fetal calf serum) as negative controls. Results were presented as stimulation indices, which were calculated as counts obtained with cells cultured in the presence of antigen/counts obtained with cells cultured in medium alone. For each time point, the mean stimulation index (SI) of the 9 animals in each group was calculated. Flow-cytometric analysis of ex-vivo PBMC and T cell cultures were carried out at selected time-points.

Results

Lymphoproliferative responses

Assays performed before vaccination demonstrated a marginal response in some animals and no response in others. Following vaccination, responses were detected in all vaccinates suggesting that this was a consequence of immunisation. Lymphoproliferative activity was detected to all antigen preparations albeit to varying magnitude. Stimulation indices ranged between 4 and 27. In general, the reactivity detected with the precipitate form of antigen was comparable to responses obtained with viable and heat-killed whole mycoplasma cells. This response was 2-fold more than that obtained with the supernate form of antigen. The proliferative responses were detectable over a period of 19 weeks post vaccination (group 1) and the analysis is on-going up to the time of challenge. Data showing the reactivity of PBMC obtained from the 3 groups of animals to the precipitate form of antigen is depicted in Figures 1, 2 & 3.

Figure 1. Lymphoproliferative responses to the precipitate form antigen in cattle vaccinated twice with T1 44

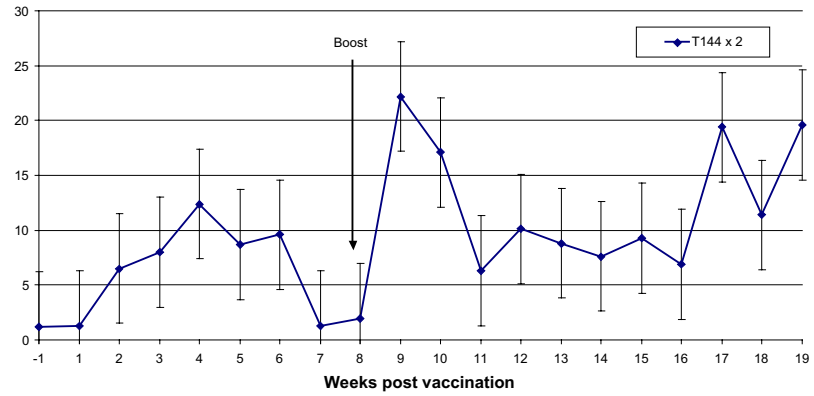


Figure 2. Lymphoproliferative responses to the precipitate form of antigen in cattle vaccinated once with the T1 44 vaccine

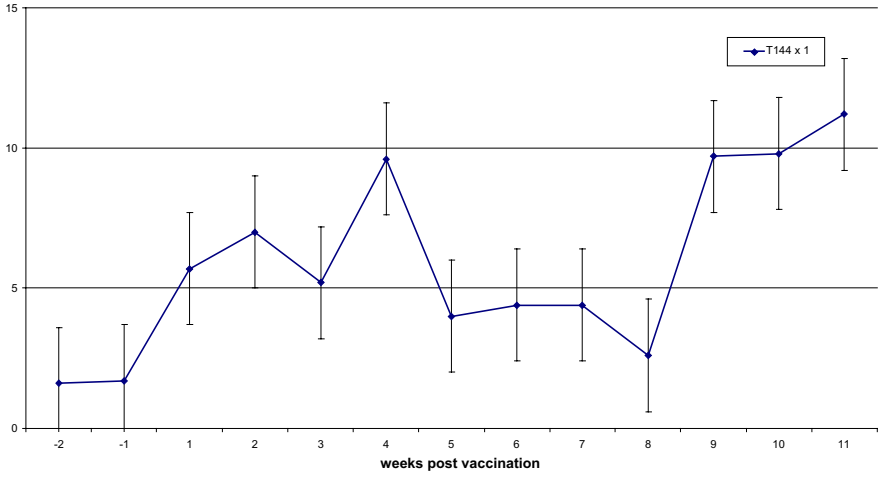
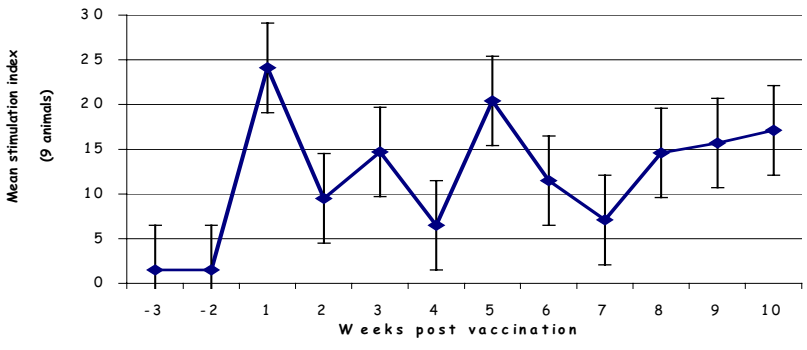


Figure 3: Lymphoproliferative responses to the precipitate form of antigen in cattle vaccinated with saponin-adjuvanted vaccine



As shown in Figures 1 and 2, cattle immunised with $T_1$44 developed a response that peaked at week 4 (mean SI of 10). This response generally reduced in magnitude over the next 4 weeks, even though it was detected at near-peak levels in group 2 animals after this period of time. Significantly, as shown in Figure 1, the responses doubled (mean SI of >20) during the first week following a booster vaccination, and declined gradually to pre-boost levels after 3 weeks. By contrast, responses in cattle that received saponin-formulated vaccine (group3) were detected at high levels (mean SI of 25) by week 1 of vaccination. These responses declined to levels that ranged between mean SI values of 6 and 20 from week 2 to week 11. It is evident that the magnitude of the response observed after boost with T1 44 is comparable to that detected following immunisation with the saponin-adjuvanted vaccine.

Responding cell populations

Flow cytometric analysis of *ex-vivo* PBMC was performed before and after vaccination to define responding cell populations. As shown in Table 1, there was no significant increase of any cell population tested. Preliminary results from analysis of cultured cells, indicate a high proportion of CD4 and $\gamma\delta$ T cells.

Table 1: Phenotypic analysis of PBMC from cattle before and after vaccination

Weeks PV	Media	CD4	CD8	γδTcells	B cells	Monocytes
–3	0.52	29.76	30.62	23.89	37.15	1.43
–1	0.83	29.52	25.80	30.36	26.22	2.11
1	0.26	30.01	23.65	22.10	26.73	0.68
2	0.20	32.55	18.85	15.08	23.98	0.99
3	0.48	35.73	28.68	17.47		0.66

Discussion

The results indicate a lymphoproliferative response as a consequence of vaccination. It is notable that animals vaccinated with the saponin adjuvanted vaccine gave a response comparable to that obtained with the $T_1$44 booster dose, which was about 2-fold higher than that observed after a single $T_1$44 vaccination. There were no marked changes in cell populations in the course of vaccination. Further analyses are being conducted to determine cytokine profiles in *ex-vivo* PBMC. It would be interesting to relate the immune responses observed with the outcome to challenge. This would provide an initial platform from which to develop correlates of immunity.

References

Amanfu, W., Masupu, K. V., Adom, E. K., Raborokgwe, M. V. and Bashiruddin, J. B. (1998) An outbreak of contagious bovine pleuropneumonia in Ngamiland district of north-western Botswana. Veterinary Record **143**, 46-48.

FAO (2000) CBPP status in Africa. In Report of the second meeting of the FAO/OIE/OAU/IAEA consultative group meeting on Contagious Bovine Pleuropneumonia (CBPP) Rome, Italy. 24-26 October.

Masiga, W. N., Domenench, J. and Windsor, R. S. (1996) Manifestation and epidemiology of contagious bovine pleuropneumonia in Africa. Revue Scientifique et technique Office International des Epizooties **15(4)**, 1283-1308.

Tuslane, J. J., Litamoi, J. K., Morein, B., Dedieu, L., Palya V. J., Yami M., Abusugura, I., Sylla, D. and Bensaid, A. (1996) Contagious bovine pleuropneumonia vaccines: the current situation and the need for improvement. Revue Scientifique et technique Office International des Epizooties **15(4),** 1373-1396.

The status of CBPP in west and central Africa and strategies for sustainable control

B. M. Seck[1], M. Kané,[2] W. Amanfu[3]

[1]*Laboratoire Centrale Vétérinaire, Bamako, Mali ;* [2]*Regional Consultant, FAO/TCP/RAF/0172T, Bamako, Mali.*
[3]*Food and Agriculture Organization, Animal Health Service Viale delle Terme di Caracalla, Rome-Italy 00100*

Introduction

CBPP is still a big problem for most cattle producing countries in sub-Saharan Africa, because of its insidious nature and the difficulties associated with controlling the disease. In the late 1960s, expectations for the control of the disease and eventual eradication were high due to the development and the use of freeze-dried CBPP attenuated vaccines (T1 44, KH3J) instead of the previous broth culture vaccines. Later on, the various rinderpest vaccination campaigns gave the opportunity to increase CBPP vaccination rates in many countries using combined (bisec-rinderpest virus vaccine plus CBPP vaccine) vaccines or administering the two vaccines separately. At the end of Pan African Rinderpest Campaign (PARC) programme, outbreaks of CBPP declined dramatically in some countries (i.e. in Senegal, Niger, Chad and Cameroon). Elsewhere, although the disease prevalence declined, more than 20 years ago, it still remains endemic or sporadic in those countries.

In response to this CBPP persistence in Western and Central Africa and due to new developments in disease outbreaks in Eastern and Southern Africa (in mid and the late 1990's), several technical meetings have been held on various issues on CBPP that detailed the major constraints impeding the control of the disease on the continent.

This presentation is intended to provide updated information on CBPP status in West and Central Africa and to propose a strategic plan for its sustainable control. It is based, especially for West Africa, on the activities and the conclusions of the last coordination meeting of the FAO TCP – "Coordinated programme to strengthen capacity for the epidemiosurveillance and control of CBPP", (TCP/RAF/0172) – held in October, 2002 in Ouagadougou, Burkina Faso in conjunction with PACE, and which focused on the development of a proposal for a "Coordinated and Progressive Control of CBPP within the western Africa sub-region". For Central Africa, data were obtained from various PACE programme progress reports and from OIE Zoosanitary status report for year 2002.

Main livestock production systems in West and Central Africa

The main livestock management system, within the cattle producing countries of the two sub-regions, is the extensive pastoral system characterized by livestock transhumance and nomadic livestock system within Sahelian zones. In Mali, the central delta of River Niger is a convergence zone for seasonal grazing for a huge number of transhumant and nomadic cattle herds from Mauritania, Burkina Faso and Niger. In Chad, the Lake Chad zone plays a similar role for transhumant herds from Cameroon, Central Africa Republic Niger and Nigeria. These seasonal traditional movement of transhumant and nomadic cattle herds are accompanied by an important flux of cattle trade movements (more than 500,000 heads yearly) directed southward throughout the year from Sahelian zones of Burkina Faso, Mali, Mauritania, Niger, Cameroon and Chad to coastal countries of Benin, Côte d'Ivoire, Ghana, Liberia, Nigeria, Senegal and Togo.

Within West and Central Africa, cattle movement monitoring by veterinary services is known to be sub-optimal due to lack of resources (human, financial, material). The livestock industry infrastructures (markets, abattoirs, slaughter slabs) are usually monitored by veterinary authorities, but CBPP suspected cases or lesions noticed at their level are not often integrated into the data of the national CBPP surveillance system. In addition, only two countries have made efforts to achieve local cattle identification system (by tattoo in Guinea, by numbered metal ear-tag in Côte d'Ivoire) in order to improve cattle movement monitoring and animal disease trace-back.

Current CBPP status and control in West and Central Africa

With regards to CBPP reported prevalence, West and Central Africa are not under the same burden of disease. While the disease is endemic within most of West African countries, only a few countries in Central Africa currently report it. In terms of cattle population, it must be realized that: (1) There are more cattle producing countries in West than Central Africa; (2) In Central Africa, the Sahelian pastoral zones of Cameroon and Chad have 74% of cattle stocks within the sub-region; (3) West Africa cattle population is 46.5 million cattle heads against 5.9 millions for Central Africa.

CBPP Prevalence and Distribution

In Central Africa, CBPP has never been reported by Congo, Equatorial Guinea and Gabon. In this sub-region, the disease status is far from being clear due to lack or imprecision of available data (Table 1).

CBPP is endemic or sporadic all over West Africa except in the Gambia, Guinea Bissau and Senegal (Table 2). The disease has never been reported by Guinea Bissau, while the last cases in Gambia and in Senegal were reported in 1971 and 1992 respectively. The disease is well established in Burkina Faso, Côte d'Ivoire, Ghana, Mauritania and Mali but is sporadic in Niger and Guinea – Conakry.

Within both West and Central Africa, CBPP outbreak reporting is not usually followed by field investigations to collect more epidemiological, economic or ancillary data. Therefore, in many circumstances, when morbidity and mortality figures are available (Table 3) it is not possible to compute the corresponding morbidity and mortality rates.

Table 1. CBPP Outbreaks in Central Africa (Unit)

Country/ Year	1997	1998	1999	2000	2001	2002
Cameroon	2	nd	nd	nd	nd	2*
Central Africa Republic	nd	nd	nd	nd	nd	+?*
Chad	2	2	4	nd	nd	4*
Congo	0	0	0	0	0	0
Democratic Republic of Congo	nd	nd	nd	nd	nd	nd
Equatorial Guinea	0	0	0	0	0	0
Gabon	0	0	0	0	0	0

nd – no available data. Sources: Country report;
+? – disease suspected
except for * from OIE Countries' zoosanitary status 2002

Table 1. CBPP outbreaks in West Africa (Unit)

Country/ Year	1997	1998	1999	2000	2001	2002
Benin	nd	nd	nd	nd	nd	nd
Burkina Faso	35	42	16	20	10	12
Côte d'Ivoire	10	8	11	7	8	5
Gambia	0	0	0	0	0	0
Ghana	49	51	23	21	4	26
Guinea	22	11	6	0	1	1
Guinea Bissau	0	0	0	0	0	0
Liberia	nd	nd	nd	nd	nd	nd
Mali	15	9	12	12	15	5
Mauritania	10	3	3	1	4	1
Niger	0	7	1	1	0	1
Nigeria	15	16	4	9	31	1
Senegal	0	0	0	0	0	0
Sierra Leone	nd	nd	nd	nd	nd	nd
Togo	nd	nd	nd	nd	nd	15
Total per Year	**156**	**147**	**76**	**71**	**73**	**67**

Source - Country National Veterinary services
nd - no available data

Table 3. Morbidity and morality figures within CBPP outbreaks (in cattle head)

	1998		1999		2000		2001	
	Sick	**Dead**	**Sick**	**Dead**	**Sick**	**Dead**	**Sick**	**Dead**
Burkina Faso	168	63	944	81	389	81	518	46
Côte d'Ivoire	184	84	160	106	152	82	203	130
Ghana	655	45	106	20	50	12	211	15
Guinea	108	57	43	19	0	0	42	30
Mali	244	93	386	140	382	202	241	78
Mauritania	182	133	340	67	3	7	44	7
Niger	75	18	9	2	7	2	0	0
Nigeria	1	76	181	17	1	87	998	219
	793				162			
Total /Year	**3**	**569**	**2 169**	**452**	**2**	**473**	**2**	**525**
	409				**145**		**257**	

CBPP Distribution

West Africa

In the Sahelian countries of this – region, at least three CBPP endemic areas can be distinguished at the bordering provinces between; (1) Burkina Faso, Mali and northern Côte d'Ivoire; (2) Mali and Mauritania; and, (3) Niger and Mali. They include a lot of common dry-season pastures used by transhumant herds from these different countries. Nevertheless, the recent trend of the disease is characterised by spread towards central Côte d'Ivoire, southern Mali and southern Burkina Faso due to the increase of southward flux of

transhumance movement, the gradual settlement of pastoralists in long-lasting pasture zones and the increase of purchase and use of oxen for cotton production.

The current CBPP distribution within West African coastal countries affected by the disease is as follows:

- In Côte d'Ivoire, CBPP occurs every year, mainly in its Central and Northern provinces (sharing borders with Burkina Faso and Mali) inside a "V" shaped area delineated by the provinces of Odienné in the north-west, Bongouanou in the south and Abengourou in the East;
- In Ghana, the disease is endemic and reported in nearly all the ten regions of the country and in many circumstances, the source of the outbreaks has been traced to trade cattle or to transhumant cattle from northern neighbouring countries;
- In Guinea, the eastern part of the country, sharing borders with Mali and Côte d'Ivoire is the traditional CBPP endemic zone while its western part, protected by a sanitary cordon, is free from the disease since the late 1980s';
- In Benin, Nigeria and Togo, the disease prevalences as well as its distributions are unclear or not updated. Although Togo reported to OIE 15 CBPP outbreaks for year 2002, there is no indication about their distribution. Nigeria, with the biggest cattle population of the sub-region (19.8 million out of a total of 46.5 in year 2000), reported only one CBPP outbreak in year 2002 in spite of its transhumant cattle which move from northern Nigeria to Niger, Cameroon and Lake Chad.

In Central Africa, CBPP was not reported from Congo, Gabon and Equatorial Guinea. In year 2002, the two CBPP outbreaks reported from northern Cameroon were both from transhumant cattle herds coming back from Chad. Elsewhere, the absence of data or the imprecision on reported outbreak locations (i.e. the four outbreaks of Chad in 2002) made drawing of geographic distribution of disease difficult. In Democratic Republic of Congo, CBPP started at the border with Uganda in 1981 and within a 10-year period affected 10% of the cattle population of Ituri Province. Since then, the civil unrest in the country made disease surveillance and reporting impossible. The Lake Chad zone, which is a gathering area for a number of transhumant herds from Niger, Nigeria, Cameroon and Central African Republic, used to be considered as the zone where CBPP transmission occurs and from where it is disseminated southwards.

CBPP Surveillance and Reporting

In spite of the setting up of national priority animal disease epidemiosurveillance networks (officially formalised into most of PACE participating countries during the last 2 years), and support from other animal health projects, weaknesses are still noticed in most of the CBPP surveillance systems put in place and at various levels (on field, along cattle roads, in cattle markets, abattoirs and slaughter slabs). In particular, within many countries, the CBPP abattoir/slaughter slab surveillance is not effective or not linked to the CBPP epidemiological surveillance network. Therefore, usually there is no resulting trace-back of field observations of CBPP-like lesions to their herd of origin. The clinical surveillance of herds within affected areas is not regularly undertaken. Cattle movement monitoring is in general sub-optimal as it is not systematic and no permits are issued for inter-provincial movement between areas of different CBPP status (i.e. Mali, Burkina Faso, Côte d'Ivoire). Between ECOWAS countries, transboundary cattle should be (theoretically) provided with International Transhumance Certificate and vaccinated against CBPP. Unfortunately, the border quarantine stations are not often used by transboundary herds and are not provided with veterinary staff.

Two exceptions in the weaknesses of CBPP surveillance and reporting are found in Guinea and Senegal. In Guinea, the CBPP surveillance system receives significant back up

from a livestock breeders group, “Sanitary defence group”, so that stockbreeders are the real first line of early warning and early reaction systems. Senegal has a solid, well structured and functional CBPP surveillance system that is provided with regular and updated information on CBPP. Within both countries, private veterinarians granted sanitary mandates, are formally involved in CBPP field, abattoir surveillance and disease reporting.

Laboratory Confirmation of CBPP Diagnosis

In most of West and Central Africa, only the central veterinary laboratories are in a position to perform CBPP laboratory confirmatory diagnosis. Laboratory testing methods include complement fixation test, enzyme linked immunosorbent assay (ELISA) test, agar gel immunodiffusion (AGID) test and/or bacteriological culture and isolation, identification and characterization. The provincial laboratories are often understaffed, ill equipped and under-supplied with laboratory reagents and media so that their role is limited to serum collection.

Within some countries, suspected field cases or CBPP-like lesions at the abattoirs, are not systematically subjected to confirmatory laboratory diagnosis although everywhere, CBPP is a notifiable disease. The annual figure of field and abattoir samples received for laboratory confirmatory diagnosis ranges between 30 samples (in Mali and Burkina Faso) to one thousand samples (in Côte d’Ivoire and Ghana). In Côte d'Ivoire, Ghana and Guinea, CBPP surveillance and control programmes benefit from excellent laboratory support.

Current CBPP Control Strategies

In Gambia, Guinea Bissau, Congo and Gabon the current strategy to prevent CBPP re-entry is based on increased surveillance of the disease along with increased public awareness. CBPP vaccination has never been done in Guinea Bissau. It was stopped in Gambia and Gabon in 1971 and 1997, respectively. Elsewhere within the two sub-regions, annual vaccination remains the main CBPP control strategy. Although in all countries the veterinary zoosanitary regulations consider CBPP as a notifiable disease and foresee mandatory zoosanitary measures, these are often limited to affected herds for a short period of time and ring vaccination around the disease foci is often carried out. The CBPP control strategy in Ghana is based on annual vaccination in endemic areas complemented by in case of outbreaks, test and slaughter of sick and infected animals and vaccination of animals at risk.

Guinea's PARC programme during the late 1990s focused on CBPP control that led to one of the most comprehensive CBPP control strategies in West Africa due to a combination of factors such as:

- Annual mass vaccination campaign in endemic zones;
- Slaughtering of all clinically affected and exposed cattle within CBPP outbreaks or village based on the extent of the epidemic;
- Country zoning with infected, surveillance and free zone; a sanitary barrier divides the country into two parts with strict ban of cattle movement from the CBPP-infected area into CBPP-free area, except to the abattoir by motorised transportation and under veterinary escort;
- Ear notch branding of all cattle sent to infected area markets;
- Individual identification of cattle by tattoo (started since 1992);
- Active involvement of around 12,000 veterinary auxiliaries and 500 livestock producers "Defence groups" and private veterinarians in cattle movement monitoring and CBPP surveillance and reporting;

- Presence of a central office dedicated to CBPP data collection, analysis and information feed-back to all interested parties;
- Regular technical refresher courses for the veterinary personnel and regular public awareness campaigns.

In 2002, the Federal Government of Nigeria funded a five-year CBPP control programme with a containment phase (foreseeing compulsory annual mass vaccination, transhumance certificate, livestock movement control etc.) to be followed by an eradication phase (with compulsory slaughter of sick or exposed cattle and compensation, active surveillance etc.).

For countries in West Africa, CBPP vaccination campaigns are targeted one-round annual vaccination with T1 44 or with T1 SR vaccine (Table 4). The cattle stock mobility, the cost of vaccine dose and the T1 vaccine side-effects are cited as reasons for the low vaccination rate and the difficulty to do more than one-round vaccination yearly (Table 4).

Table 4. CBPP Vaccination in West Africa

	1999			2000			2001		
	Cattle stocks (Head)	**Vaccinated (Head)**	**Vacci-nation rate (%)**	**Cattle stocks (Head)**	**Vaccinated (Head)**	**Vacci-nation rate (%)**	**Cattle stocks (Head)**	**Vaccinated (Head)**	**Vacci-nation rate (%)**
Benin	1 438 100	Nd	**Nd**	1 500 000	NA	**Nd**	1 500 000	Nd	**Nd**
Burkina Faso	4 704 000	1 309 043	**27.83**	4 798 000	1 242 857	**25.90**	4 798 000	1 004 530	**20.94**
Côte d'Ivoire	1 377 000	1 014 840	**73.70**	1 409 000	874 044	**62.03**	1 409 000	594 400	**42.19**
Gambia	361 400	0	**0.00**	364 100	0	**0.00**	365 000	0	**0.00**
Ghana	1 288 000	835 650	**64.88**	1 302 000	708 190	**54.39**	1 302 000	NA	**NA**
Guinea	2 368 000	835 650	**35.29**	2 679 385	708 197	**26.43**	2 679 385	665 706	**24.85**
Guinea Bissau	499 550	0	**0.00**	512 000	0	**0.00**	515 000	0	**0.00**
Mali	6 427 500	2 849 105	**44.33**	6 620 300	3 321 241	**50.17**	6 818 900	2 971 545	**43.58**
Mauritania	1 433 000	841 976	**58.76**	1 476 000	856 598	**58.04**	1 500 000	700 000	**46.67**
Niger	2 174 000	455 252	**20.94**	2 216 500	571 538	**25.79**	2 260 000	669 333	**29.62**
Nigeria	19 830 000	524 327	**2.64**	19 830 000	644 008	**3.24**	19 830 000	3 200 000	**16.13**
Senegal	2 927 000	1 450 695	**49.56**	3 073 000	1 275 000	**41.49**	3 227 000	1 275 000	**39.51**
Togo	275 200	Nd	**Nd**	277 200	Nd	**Nd**	277 200	Nd	**Nd**
Total/Year	45 102 750	10 116 538	**22.43**	46 057 485	10 201 673	**22.15**	46 481 485	11 080 514	**23.84**

Sources: FAO statistics (2002) for Cattle stocks; National Veterinary Services for immunisation figures

In Central Africa, CBPP vaccination is mandatory in Chad and Cameroon but not in Central Africa Republic. The vaccination has been officially stopped in Gabon in 1997. In Democratic Republic of Congo it was also stopped since 1994 because of lack of resources and civil unrest.

In West Africa and Chad, CBPP vaccination fees are partially or totally supported by livestock owners or traders through a cost recovery scheme. Table 5 indicates the respective cost of one vaccine dose and one cattle vaccination fees (direct ones). The collected revenue is used for cold chain maintenance, vaccination material and purchasing of vaccination certificate cards. The CBPP vaccine (T1 44 or T1 SR) used is supplied by one of the following laboratories: Garoua (Cameroon), Bamako (Mali) and Dakar (Senegal).

Table 5. CBPP vaccine and vaccination cost (Unit)

	Vaccine dose Cost	Vaccination fees (1 unit)	Currency	Amount paid by cattle owner	CBPP vaccine origin
Burkina Faso	45	135 to 175	FCFA	full amount	Cameroon, Mali
Côte d'Ivoire	50	250	FCFA	full amount	Cameroon, Mali
Ghana	0.34	0.5	Cedi	full amount	Cameroon
Guinea	87	300	FG	full amount	Cameroon
Mali	25 to 35	100	FCFA	full amount	Bamako
Mauritania	10.4	30	UM	full amount	Cameroon
Niger	35	100	FCFA	full amount	Cameroon
Senegal	24	110	FCFA	Partial (60FCFA)	Senegal
Chad	?	80	FCFA	full amount	Cameroon

Currency exchange rate: 1Euro=656FCFA 1UM=2.5FCFA; 1FG=0.65FCFA; 1US$=650FCFA= 8,347Cedis

Officially, within the two sub-regions, antibiotics are not allowed to be used for treating CBPP affected animals.

Throughout the two sub-regions, stockbreeders and cattle traders associations are involved in CBPP surveillance and control. Private veterinarians are associated with vaccination campaigns against priority disease through “sanitary mandate”, but except in few countries, they are not yet closely associated with field and abattoir surveillance and disease reporting for CBPP.

Conclusions on CBPP Status in West and Central

Although CBPP status seems relatively clearer in West Africa (except within some coastal countries: Benin, Nigeria and Togo) than in Central Africa, the imprecision of the data on animals at risk, as well as the epidemiologic parameters and economic impact of the disease, demand that studies are carried out to clarify the situation within most of the countries before expecting sustainable CBPP control and minimizing the risk of transboundary spread of the disease.

Strategies for Sustainable CBPP Control

The overall proposed CBPP control strategy, for West and Central Africa, is based on:

- **Preparatory activities** aimed at collecting **epidemiologic and economic** data necessary to justify and properly plan an effective and sustainable CBPP control strategy.
- **Normative activities**, at both national and sub-regional levels, dedicated to reducing as much as possible, the disease burden or risk at national level and to minimize its transboundary spread through a coordinated CBPP risk assessment and management.

Phase1. Defining the Epidemiological Status of CBPP within each Sub-region through:

- enhanced CBPP field, market and abattoir surveillance, reporting and trace-back systems at national and sub-regional levels for improved understanding of CBPP epidemiology and economic impacts;
- improved priority animal disease control infrastructures - including improved veterinary diagnostic laboratories support for CBPP confirmatory diagnosis- to allow development of justified and rationale and economic disease control plans;
- effective involvement of rural communities**,** cattle traders and other stakeholders into all aspects of CBPP surveillance, reporting and control through training, communication and public awareness campaigns;
- improved cattle movements monitoring and trace-back systems at national and sub-regional level – updating transhumance and trade cattle routes; use of official accompanying documents of moving cattle groups and re-enforcement of harmonised International Herd Health Certification usage.

Phase 2. Reduce CBPP Risks at National level by:

- prevention of CBPP re-entry into:
  - **country not currently reporting CBPP** (i.e. Gambia, Guinea Bissau, Senegal, Gabon etc.) by combined intensive active CBPP surveillance, animal movement monitoring and control, establishment of formal buffer and surveillance zones to protect disease high risk areas;
  - **long-lasting CBPP-free zones protected by sanitary** cordon (i.e. western part of Guinea-Conakry) by strengthening or establishing CBPP sanitary cordon, imposing cattle movement control towards CBPP-free zones combined with intensive disease surveillance and re-enforcement of other sanitary prophylactic measures into established surveillance and buffer zones.

  In such zones, an appropriate level of CBPP control activities, by sanitary prophylactic measures mainly, would be undertaken where needed to contain any new CBPP foci as fast as possible and minimise the risk of disease spread.

- reduction of CBPP incidence within endemic countries and limitation of its spread by intensive and regular vaccination of exposed cattle within endemic and high risk areas followed with intensive disease surveillance and cattle movement control.

  Within affected large Sahelian pastoral areas, the testing, vaccinating and control of such diseased herds is currently out of the capacities of most veterinary services. Consequently, control measures would be a compromise between the ideal methods and those that are practically possible. Therefore, in these areas, once the disease incidence has been reduced to a low level, through vaccination, intensive disease surveillance, systematic clinical and pathological surveillance, sero-surveillance and other ancillary actions – other control measures could be justified and implemented.

- development of detailed national and sub-regional control plans.

  While it is not appropriate to develop mid and long-term national CBPP control or eradication plans until the epidemiology of the disease is understood and reliable data available, as well as the priorities for control measures correctly identified, the continuing presence of the disease demands that control measures be initiated. Later on, these control measures would be evaluated and the different CBPP eco-

epidemiological zones re-defined before achieving the mid and long-term control plans for the disease.

Phase 3. Minimise CBPP Transboundary Spread at Sub-regional level through:

- Improved CBPP risk assessment and management – including improved CBPP emergency response capabilities, harmonised disease surveillance, reporting and control strategies;
- Institutionalised CBPP active surveillance and control programmes, involving all stakeholders, in order to establish CBPP free eco-epidemiological zones with recognised international sanitary barriers;
- Established sub-regional mechanisms allowing transparency in CBPP risks assessment and surveillance systems evaluation;
- Harmonised appropriate legislative/regulatory support for CBPP surveillance and control.

This phase should lead to the definition of regional sanitary barriers or buffer zones based on CBPP status, ecological factors, cattle husbandry systems and movement patterns. For instance it could be established, between Senegal and its neighbouring countries, buffer zones of 50 km at least deep at borders and inside Mauritania, Mali and Guinea with their corresponding surveillance zones inside Senegal.

Phase 4. Maintain Optimal Conditions for CBPP-free Status within each Sub-region through:

- improved and continuing sub-regional coordinated efforts and shared resources for CBPP active surveillance and reporting, information dissemination and cattle movement monitoring that could put some countries in the sub-region on the OIE pathway for Declaration of freedom from CBPP.

The above disease control strategy elements would need a high level of political commitment, as CBPP control requires courageous administrative decisions and rigorous application of disease control measures with discipline.

Conclusions

The proposed approach puts emphasis on a good preparatory phase (including improvement of extension services, rural community awareness activities, disease surveillance and control infrastructures, livestock movement monitoring system) followed by normative integrated activities requiring strong and sustained activities at both national and sub-regional level to alleviate the critical factors impeding the two sub-regions in the control of CBPP.

At present, countries in the region have the opportunity through the current animal disease control initiatives**,** to develop and maintain institutional capacity in priority animal disease surveillance and control. Nevertheless, for an appropriate and sustained CBPP control, there is the need for a strong political commitment, in developing strategies for controlling this disease through improved animal health care delivery system, coordinated efforts and resource sharing between countries in the region.

Status of contagious bovine pleuropneumonia (CBPP) in Nigeria with emphasis on control strategies

Foluso E. Fasanmi

Federal Department of Livestock and Pest Control Services, Federal Ministry of Agriculture and Rural Development, Area 11, Garki, Abuja, Nigeria.

Introduction

Projections based on the Livestock population census of 1991, estimated the cattle population in Nigeria at 16 million. Over 90% of these are kept under Nomadic production systems, while about 10% are raised under intensive production systems. The nomadic culture of Nigerian herdsmen coupled with the practice of transhumance has significantly contributed to the spread of diseases in Nigeria particularly contagious bovine pleuropneumonia (CBPP). CBPP is an infectious disease of cattle caused by *Mycoplasma mycoides* subspecies *mycoides* SC**.** It is presently the most important cattle disease in Nigeria. The effects of this disease on livestock production, its productivity and on the rural economy are considerable. Economically, the country suffers direct losses through infection and deaths of cattle, while indirect losses include exclusion from participation in International livestock and livestock product trade.

Over the years, efforts were made by the Nigerian Government to effectively control the disease with varying degrees of success. The disease was virtually eradicated from the country by 1965, following ten years of mass vaccination programme, well organized disease reporting, efficient laboratory diagnosis, effective quarantine and strict control of cattle movement (Anon, 1975a, 1975b; Chima, 1999). Unfortunately, with the outbreak of the Nigeria Civil War and the consequent breakdown of the necessary surveillance measures, the disease regained its prominence. However, the present government in collaboration with the PACE programme in Nigeria is putting measures in place to frontally address the issues.

Epidemiology

The first incidence of the disease in Nigeria was recorded in 1924 when reliable records were first available. This was the year when laboratory facilities for vaccine production and serological diagnosis were carried out in Vom. Between 1924 and 1960 an average of 200 outbreaks occurred each year and mainly in Borno and Kano Provinces of the then Northern Nigeria. During the period 1960 up to 1975, the distribution of CBPP in Nigeria was categorized broadly into three zones with the far North being Enzootic, the Middle/Central belt States Exposed, while the Southern States were regarded as Free of the disease. The North Western State was the most affected as the CBPP status records indicated in Table I. The low vaccination figure for 1973 – 1974 was due to deployment of staff for National Census Operation.

Table I. CBPP Outbreak Record of North Western State (1970 to 1974)

Year	Outbreaks	Mobility	Mortality	Vaccination figure
1970/1971	54	4,222	200	416,855
1971/1972	50	5,444	1,298	886,320
1972/1973	23	10,697	1,939	91,235
1973/1974	12	3,543	346	200,947

During the 1970s up to the 1990s, the socio-economic and cultural settings of the producers/herdsmen changed. Despite the transhumance and trade cattle movement from North to the Southern States of Nigeria, which became more rapid due to adoption of vehicular transportation, there developed large-scale settlements of herdsmen within the South Western States and the South Eastern States. These Pastoral settlements were triggered by these factors; presence of few Grazing Reserves set up then by the Federal Government and the considerable increase in both manpower and infrastructural development in both public and private veterinary sectors. All these changes impacted tremendously on the livestock disease profile in the country, particularly CBPP. The disease therefore had a gradual spread beyond the traditional far North to the Southern States. As at today the whole country is considered as endemic.

Unfortunately the disease reporting system, which was efficient in the 1960s and 1970s, witnessed a setback during the 1990s. The further fragmentation of the country into many more autonomous States coupled with the lack of efficient communication system and the general downturn in the economy overstretched the efficiency of the Federal Department of Livestock and Pest Control Services as well as the State Veterinary Departments. This is evident from the gross under-reporting of outbreaks of CBPP as shown in Table 2.

Table 2. CBPP Outbreak in Nigeria (1995 – 2001)

Year	No. of Outbreaks	No. Involved	Morbidity	Mortality
1995	8	258	147	42
1996	13	516	293	79
1997	15	2,580	894	117
1998	16	1,793	310	76
1999	4	181	68	17
2000	9	1,162	278	87
2001	31	998	987	219
Total	96	7,488	2,977	637

The above table is definitely not a true reflection of outbreak status of the disease. The disease is under-reported to the Federal Department of Livestock and Pest Control Services by the relevant State and Local Government Veterinary agencies as well as from private veterinarians. Judging by the large number of CBPP samples sent and screened by Vom (Personal communication) the disease is definitely on the increase nationwide despite control measures. Gongola and Bauchi States are worst hit by the disease. Frequent cases of “vaccine breaks” were reported in the two areas between 1991 – 1998.

Quoting Dr. Joel Chima (CBPP Laboratory, Vom), “A ‘live with the disease’ attitude has prevailed in the last few years. Farmers hardly report cases but resort to treatment with antibiotics like any other bacteria disease”, (Chima, 1999; 2001). Data on infection within the country is inaccurate and subjective (Chima 2001, Molokwu 2003). Questions as to the disease socio-economic importance, efficiency of currently used vaccines, diagnostic capabilities, and effectiveness of antibiotics treatment remained unresolved. But with the advent of the FAO/IAEA coordinated Research Project on the Monitoring of CBPP in Africa in 1999 and the FAO/OIE/IAEA PACE programme in year 2002, there has been a complete reinforcement of the capacity for CBPP diagnosis in Vom (FAO/IAEA Coordinated Research Project on CELISA diagnosis of CBPP 1997 – 2002). Also with the CBPP Vaccine Revamp Task Force put in place in Vom (2002) and the current PACE programme on surveillance, serological monitoring and Rural participatory appraisal, questions about CBPP epidemiology will be clearly understood and answered soonest.

Strategies for Control of CBPP

Past Efforts

As mentioned earlier, Nigeria has made several efforts in the past to control CBPP. These efforts include the Joint Project (JP 28) of 1960s and the 3-Year National CBPP Programme in the early 90s. Under the JP 28, control of CBPP was attempted through a policy of compulsory mass vaccination in the enzootic areas of the country. This was to be followed by testing, quarantine and slaughter of affected animals. During the 70s, serious problems were encountered with the mass vaccination programme due largely to the refusal of producers to allow vaccination of their animals due to post-vaccination reactions experienced then. Subsequently, emphasis was placed on outbreaks notification, slaughter and compensation for infected animals as well as vaccination of in-contact herds. Unfortunately, the results recorded were not as encouraging as envisaged.

The three-year National CBPP Programme launched by the Federal Government of Nigeria in 1992 was also discontinued due to lack of funds. The inability of past efforts of government to achieve expected goals could be summarized as follows:

- Lack of sustainability especially in relation to continuous and sufficient funding;
- Post-vaccination reactions encountered in some animals with attendant refusal of most pastoralists to present their animals for further vaccination;
- Inability of Government to adequately compensate for condemned animals or parts thereof. The pastoralists considered government as not being sincere and sensitive in her plans to control the disease;
- Inadequate policy framework on livestock disease control in the country.

New Policy Thrust

To adequately address the problem of CBPP, the government in Year 2001 put in place a new control strategy. This strategy comprised two approaches, viz:

(i) **Containment Phase**: In this phase, the strategy is to reduce the incidence of the disease to about 10% through 90% vaccination of the national herd. This involves the following:

  (a) Mass compulsory annual vaccination of cattle for a continuous period of 5 years;
  (b) Control of cattle movement and the introduction of ECOWAS transhumance certificates;
  (c) Strengthening of epidemiological surveillance networks. This is being carried out through Nigerian PACE;
  (d) Improving the epidemiological and economic knowledge of the disease in the country;
  (e) Mass extension programme to enlighten farmers and other stakeholders on the disease.

(ii) **Eradication Phase**: This is aimed at total eradication of the disease from 10% infection rate to zero. This shall basically involve compulsory test, slaughter and compensation in designated areas to be classified as Disease Free Zones (DFZ). The following actions shall be taken in the CBPP disease free zones (but exposed):

  - Establishment of sanitary cordon (buffer zone and sero-surveillance zone) to protect the DFZ;
  - Active surveillance in the DFZ and surveillance zones;

- Mass vaccination of cattle in the buffer zones;
- Implementation of strict procedures for admittance of animal within the DFZ (animals destined for slaughter, breeding stock and transhumants).

Role of NVRI, Vom in these Strategies

NVRI, Vom plays vital role in the strategies for CBPP control through disease diagnosis. CBPP diagnosis involves the use of the cELISA as the standard test along with CFT. For control and prevention, NVRI produces CBPP vaccine, which has undergone recent improvement to enhance its immunogenicity. The just created 5 zonal laboratories in addition to the existing 15 NVRI State Laboratories have improved on the distribution of vaccines nationwide with emphasis in cold chain maintenance and also provide professional technical support to state veterinary services in disease diagnosis.

Conclusion

In any strategy to control disease in livestock, early diagnosis is cardinal. The reporting and documentation of outbreaks is also paramount. The potential for all these requirements is also in place in Nigeria.

The 5 years CBPP mass vaccination, which started 2 years ago, is having a positive effect on getting vaccination up to 70% coverage. This will reduce the incidence of CBPP.

Nigeria had taken a very active part in International disease eradication programmes and continues to show keen interest in liaising with neighbouring countries to control and effect total eradication of diseases that plaque our livestock industry.

Acknowledgements

Many thanks are due to Dr. J. U. Molokwu of NVRI, Vom and for Dr. E. Nwakonobi of my Department for their assistance in collecting the data and to Dr. K. A. Majiyagbe of NVRI, Vom for review of the manuscript. The author is also grateful to Mr. A. O. Edache, Permanent Secretary of the Federal Ministry of Agriculture and Rural Development for permission to publish this paper.

References

Anon (1975a*)* Zoo-Sanitary position of major livestock diseases in Nigeria and regional cooperation in Animal Health. Report, Veterinary Public and Animal Health Division, Federal Livestock Department, Federal Ministry of Agriculture and Rural Development, Nigeria, Bull. Off. Int. Epiz. 83, 901-922.

Anon (1975b) Nigerian Report on Contagious Bovine Pleuropneumonia XLIII Session General Rapport, Bull. Off. Int. Epiz.302.

Chima J.C., Mohammed, A., Lombin, L. H., and Majiyagbe, K. A. (1999). Field Validation of a monoclonal antibody-based competitive ELISA for the detection of antibodies to contagious bovine pleuropneumonia Proceedings, 2nd RCM of IAEA/FAO CRP on Monitoring of CBPP in Africa using ELISA held in Lusaka, Zambia, 27 September – 1 October 1999.

Chima, J.C., Lombin, L.H., Molokwu, J.U., Abiaye, E.A., and Majiyagbe, K.A. (2001). Current situation of contagious bovine pleuropneumonia in Nigeria and the relevance of CELISA in the control of the Disease. A Paper presented at the Research Coordination Meeting of the FAO/IAEA Coordinated Research Programme held in Nairobi, Kenya. June 2001.

Molokwu, J.U. and Nwanepa, N. (2003). Diagnosis and Monitoring of Contagious Bovine Pleuropneumonia: The Nigerian Situation, National Veterinary Research Institute Seminar Series, August 28th 2003. (In press)

Strategie de Controle de la PPCB en Angola

D.L. Simão

Ministério Da Agricultura E Do Desenvolvimento Rural, Instituto De Investigação Veterinária República De Angola.

Introduction

La PPCB est une maladie endémique et de grande importance dans le contexte epidémiologique de l´Angola. Elle sévit sous forme enzootique de la frontière sud avec la Namibie jusqu´ au 14^{e} parallèle où se trouve concentrer 90% du cheptel bovin national estimé de 3.500.000 bovins et où l´élevage est effectué quasi totalement par les éleveurs traditionnels. L´apparition des foyers sporadiques de la PPCB dans les zones traditionnelles de l´élevage caractérise le statut endémique enzootique de cette maladie en Angola.

La transhumance du cheptel durant la fin de la saison pluvieuse vers les grandes fleuves (Kubango et Kunene) pour l´ abreuvement est la cause principale de l´expansion de la maladie à l´intérieur du pays. La traversé permanente du cheptel bovin de part et d´ autre pour le pâturage et l´abreuvement, le commerce illégal, le troc des animaux et sous produits au long de la frontière commune font aussi la cause de l´expansion da la PPCB vers les pays voisins tel que la Namibie et Zambie.

La vaccination massive et systématique des animaux non affecté, suspect et malade deux fois durant l´année est l'une des mesures efficaces qui a été adoptée dans la stratégie de lutte contre cette maladie depuis l´an 2000.

Aperçue Epidemiologique de la PPCB avant 1975

- 1882 Introduction de la PPCB par les émigrants Boers en provenance de la République de l´Afrique du Sud.
- 1887 Diagnostic clinique de la PPCB dans le cheptel bovin situé a Humpata, Huila.
- 1900 Expansion de la PPCB du Sud Este vers le Nord du pays.
- 1921 Création des Services de l'Elevage.
    - Importation des premières doses de vaccin PPCB de l'Institut PASTEUR de Paris, France.
    - Réalisation de la première campagne de vaccination de bovins.
- 1945 Construction du Laboratoire Central de Pathologie à Huambo et c´était à la même année qu´ a été produit les premières doses de vaccin de la PPCB avec la souche local AM_8 que par mesure de prudence (réaction post vaccinal) a été appliquée à la pointe de la queue pour éviter des pertes économiques chez l´animal.

Cause de la Permanence et Prevalence de la PPCB

Il y a de fois on se pause la question pour savoir si la transhumance est une culture des éleveurs Humbi à Kenene, Nhaneka à Huila et Mucubal à Namibe ou bien une nécessité. Il est claire que la manière surtout logistique comme ce mouvement est organisé par les éleveurs peut donner à première vue un mouvement culturel mais au juste c´est une nécessité pour sauvegarder le cheptel durant la fin de la saison pluvieuse vers les zones des grandes fleuves (Kubango et Kunene) à la recherche surtout de l´eau pour l´abreuvement. Ce mouvement constitue le moment fertile pour la transmission et l´expansion de la PPCB

suite à la convivialité entre animaux sains vaccinés ou non, les suspects (affaiblis) et les animaux malades dans les mêmes points d´ abreuvement.

Le commerce des animaux et sous produit, bien comme l´utilisation des animaux pour le transport de biens servent de locomotive des agent infectieux d´un bout à l´autre d´une zone donnée.

Les Services Vétérinaires étant donné comme l´organe de conception, coordination et exécution des mesures de prophylaxies qui visent le contrôle et l´éradication des maladies des animaux sont confrontés à des difficultés énormes du point de vue financière, matérielle et ressources humaines. L´appui apporter par l´Assistance technique international de fois produit des résultats qui sont loin d´être à la solution immédiate des foyers des maladies comme conséquence d´une manque d´accompagnement effectif et permanent de l´état sanitaire des animaux par les services locaux.

Le Gouvernement angolais met en disposition chaque année 1.000.000 de USD pour les deux campagnes de vaccination qui sont réalisées annuellement au niveau national. Mais il est claire et net que beaucoup de moyen manque comme par exemple: une chaîne de froid efficace dans la zone sud du pays, moyens de transport, payement des perdiem aux agents de terrain qui participent directement à des campagnes. Ce budget se limite à l´achat des vaccins (PPCB, Anthrax, Charbon symptomatique, Dermatose nodulaire et la Rage) et les équipement (seringues, aiguilles et glacières). Une fois à l´autre l´achat des bicyclettes et motos. Entre temps d´autres activités complémentaires et essentiels comme le « training », l´entretien des moyens de transport existant et les couloires de vaccination ont aussi besoins des fonts pour être effectués.

Malgré les efforts déployés par le Gouvernement angolais, la guerre civile vécue durant 27 ans a mise en échec à plusieurs reprise la stratégie de lutte et contrôle de la PPCB adoptée en 2000 qui vise fondamentalement accroître chaque année le taux d´ immunité des animaux vaccinés, l´expansion des zones indemnes de la PPCB au niveau national et la protection des intérêts économiques des pays voisins.

Types de Vaccins

Tenant compte de l´écart de temps entre le diagnostic de la PPCB en Angola et la réalisation de la première campagne de vaccination contre cette maladie, des efforts ont été déployés de part et d´autre afin de combattre ce fléau qui au départ empêcher le développement de l´élevage au niveau national. En dehors des efforts scientifiques, des éleveurs ont aussi tenter de trouver des solutions selon leur connaissance en extrayant l´exsudat thoracique des animaux malades afin de l´injecter a d´autres animaux malades ou sains comme mesure de traitement ou de prévention contre la PPCB. Mais cette pratique a énormément contribuée à la contamination et l´expansion de la maladie.

Pour les campagne des vaccinations qui ont démarrées depuis l´année 1921, les vaccins avec souche T1 44 et T1 SR ont été utilisés chaque année sous recommandations de l´ OIE.

L´Institut de Recherche Vétérinaire d´ Angola (IIVA) avait produit à partir de 1945 un vaccin avec souche locale AM8 aussi désignée pour souche Caconda (nom de la municipalité d´où a été récolté le matériel). Ce vaccin a été produit exclusivement pour l´utilisation nationale et n´avait pas été exporté. Des bons résultats ont été produits malgré tout sa production n´a pas continué pour des raisons de la guerre civil qui a détruit complètement les infrastructures du Laboratoire Régional de Vétérinaire de Huambo.

Les vaccins utilisées durant les dernières années contre la PPCB proviennent du BVI, Botswana et du LCP du Mali. Exceptionnellement pour la Dermatose nodulaire des bovins les vaccins sont achetés à Onderstpoort, Afrique du Sud.

Bovins Vaccines et Foyers De PPCB (1970 à 1974)

Année	Foyers	Animaux vaccinés
1970	30	1.725.043
1971	55	1.550.711
1972	39	871.021
1973	54	999.640
1974	34	1.195.878
Total	212	6.342.285

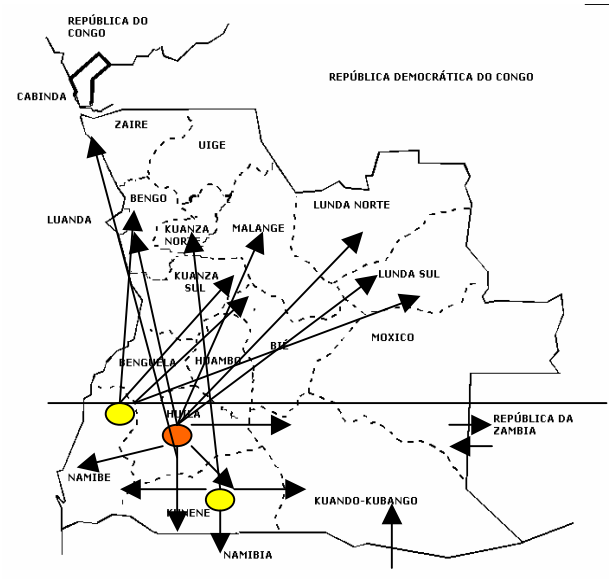
Bovins Vacine de 1990 à 1994

Année	Animaux, Vaccinés
1990	524.924
1991	733.493
1992	818.038
1993	152.653
1994	555.735

Mouvement du Cheptel Bovin de 2002 à 2003

Les effets du processus de la paix définitive en Angola depuis Février 2002 se font remarqués par les mouvements non contrôlé de la population et ses biens vers les zone d´origine. Ce mouvement n´exclu pas les animaux qui font l´objet du capital vif de l´économie des éleveurs en particulier et la population rurale en générale. Ces mouvements ne s´effectuent pas seulement à l´intérieur du pays bien aussi des pays voisins (Namibie, Zambie) vers l´Angola (Figure 1). Ce dernier explique le retour de la population émigrée au pays.

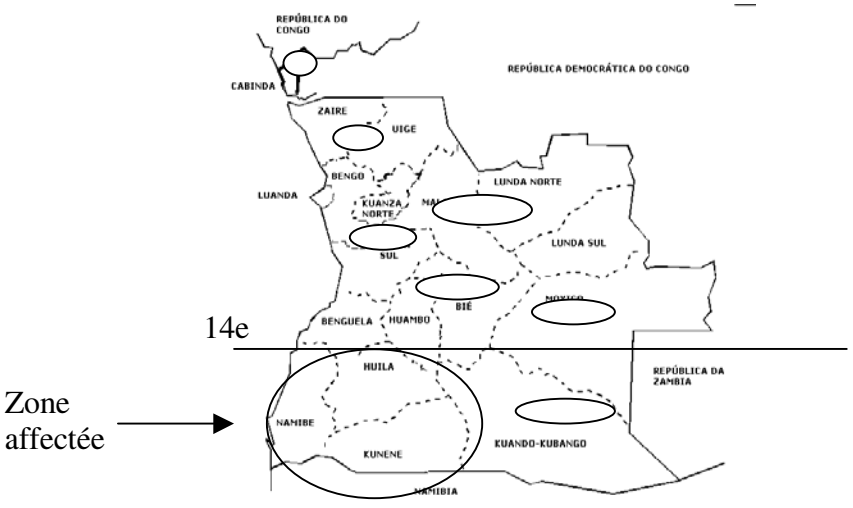
Figure 1



Suite à l´expérience vécue durant le premier processus de paix en 1992, la situation epidemiologique pourra aggravée avec l´expansion des foyers de la PPCB et bien d´autre maladie dans des zones indemne.

Prévision de l' état epidemiologique dans les prochaines années comme conséquence du mouvement actuel du bétail (Figure 2).

Figure 2.



Les Services Vétérinaires s´ engagent à prendre des mesures pour éviter des pertes économique dans le cheptel vue que ces animaux représentent une source de revenue de la population surtout dans la phase actuelle de réinstallation des familles dans les zones d´ origine.

Strategie de Controle de la PPCB

Mesures de Prophylaxie

La vaccination massive et systématique de tous les animaux non affecté, suspect et malade deux fois durant l´année et durant cinq ans reste la meilleur stratégie de lutte mise en œuvre depuis l´ an 2000.

Les services vétérinaires prétendent installer un cordon sanitaire entre le 13^{e} et le 14^{e} parallèles en vue de contrôler avec efficacité le mouvement des animaux du sud du parallèle 14 vers le centre et nord du pays. Mais étant donnée que l´Angola détient un vaste territoire (1.247.000 Km^{2}) ce cordon sanitaire ne sera effectué par une barrière physique mais, plutôt par la police sanitaire, quarantaine et présence des services de diagnostic dans les provinces partiellement affectées (Benguela, Huambo, Bié et Moxico). La présence des services de diagnostic sera marquée sur terrain par des laboratoires mobiles de diagnostic afin de rapidement mettre en place un dispositif fonctionnel pour détecter tous les cas possibles des maladies avant que soit définie une politique sur la réhabilitation et l´extension d´un réseau des laboratoires de vétérinaire au niveau national.

Reglement Sanitaire

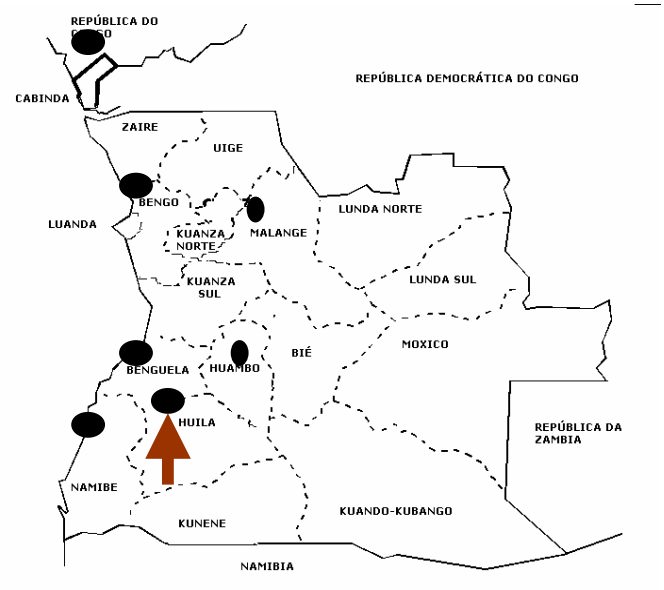
Les SV récemment créés se débattent objectivement sur les aspects de la législation pour mieux structurer l´administration et les services d´ appui technique au niveau national. Pour cela a été soumise le Règlement Général sur la Santé animale a l'approbation du Conseille des ministres depuis début 2003. La législation vétérinaire en Angola date depuis 1932, plusieurs aspects juridique de ce règlement nécessite d´une actualisation. Actuellement du point de vue juridique les services s´appui sur le Code Zoosanitaire qui serve d´ un instrument précieux et efficace pour les cadres vétérinaires et administratives qui exercent leurs activités soit dans l´ appareil d´ Etat ou dans le secteur privé. Comme le règlement générale de santé animale est considéré comme base, son approbation relancera une série de travail sur la réactualisation des règlements sanitaires sur l'exploitation du bétail, les abattoirs, le transport, le commerce, l'exportation et l´importation des animaux et sous produits etc..

Services Specialises

Les SV actuellement créés ont droit à l´autonomie financière, administrative et patrimoniale. Compète au Gouvernement de attribuer un budget qui permettra aux SV de mieux se structurer tout en tenant compte de sa division administrative au niveau national et des programmes en relève pour le contrôle et l´éradication des maladies prioritaires de la région concernée. Cette organisation au départ vient de aboutir à l installation du Système National de Surveillance Epidemiologique (SNSE) malgré tout en phase embryonnaire qui dorénavant traitera les données epidemiologiques sur les foyers et cas de maladies au niveau national.

Les Services de diagnostic sont représentés par l´Institut de recherche vétérinaire (Instituto de Investigação Veterinário de Angola) et sont dotés d´une autonomie financière, administrative et patrimoniale. L´IIV joue un rôle important dans la certitude de la stratégie a appliqué pour le contrôle des maladies prioritaires.

Figure 3. Distribution des Laboratoires Régionaux de Vétérinaires de l´ IIV.



L´enveloppe financier attribué annuellement à l'IIV ne le permet d´ entamer avec une grande ampleur les programmes scientifiques pour l'appui à des solutions des problèmes sanitaires qui affligent l´ Etat angolais. A savoir 80% des infrastructures de l'IIV ont été saccagées durant la guerre civile, raison pour laquelle pour relancer les activités sur le diagnostic des maladies, l'IIV a concentré les ressources humaines e matérielles au Laboratoire Régional de Vétérinaire de Lubango/ Huila, laboratoire situé dans la zone avec 90% du cheptel bovin national, en vue de relancer le diagnostic de la PPCB et d'autres maladies transfrontalière.

Gestion des Financements

Les services spécialisés (SV et IIV) du point de vue financement dépendent totalement du budget de l'Etat, mais aussi bénéficie de l´appui des financements externes surtout quand il s´agit de mettre en place des programmes régionaux. Le Gouvernement angolais mais en disponibilité annuellement 1.000.000 de USD afin de permettre aux services vétérinaires de réaliser les campagnes de vaccination des bovins et d´autres espèces d´ animaux domestiques. Pour améliorer la gestion de cet investissement et donner l´impact que mérite ce financement d´Etat, étant donnée qu´il y a la paix au pays, les SV vont mettre en place un programme national de lutte et contrôle de la PPCB comme priorité en vue de mieux gérer ses ressources.

La communautés internationale joue un rôle important pour la mises en marche de certain programme sanitaire pour la lutte et éradication des maladies prioritaires dans plusieurs régions du monde. L´Angola ne fait pas exception et concrètement en ce qui concerne le diagnostic, l´IIV venait de bénéficier depuis l´an 2000 les financements suivant:

- FAO/ TCP/ ANG/8922 (E) – Surveillance et contrôle de la PPCB et d' autres maladies transfrontalières (1999-2001).
- UE-RPR 146 – Contrôle des maladies des animaux dans la région SADC (1994 2001 suspendu sans justification).
- AIEA TCP/ ANG/5/02 – Modernisation des laboratoires vétérinaires pour le diagnostic des maladies des animaux (2003-2004).

La somme des actions de ces projets a permis à l´IIV d´équiper le Laboratoire Régional de Vétérinaire de Lubango et augmenter la capacité de diagnostic soit de terrain et du laboratoire. Ces actions ont aussi permit d´ améliorer la capacité des SV en matière de communication via Internet et de déplacement sur terrain.

Cette coparticipation de la communauté internationale a poussé les responsables et techniciens des services spécialisés de mieux gérer tous les moyens misent en disponibilité vue l´expérience acquise durant la période de carence.

Conclusion

La PPCB peut être contrôler et éradiquer dans le cas où le Gouvernement angolais continuera a prendre en considération l'importance de cette maladie dans le contexte socio-économique e culturel de l'élevage au niveau national.

L'adoption d' une politique sanitaire bien définie et dotée des ressources financières, matérielles et humaines capable de mettre en œuvre la stratégie en vue est l´ une des décisions souhaité afin de permettre aux SV et IIV d´effectuer le recensement du cheptel bovins existant, de déterminer la prévalence de la PPCB et d´établir les zones indemnes de la PPCB en vue d´un développement économique du secteur de l'élevage permettant dans ce cas la participation des revenues de la production animale dans le budget national.

Les recherches doivent continuer en vue de produire un vaccin capable de conférer une immunité pour plus d'un an vue que le traitement ne stérilise pas l´animal infecté et par contre le transforme d´un porteur sains est capable de transporter et transmettre la maladie à d´ autres animaux.

L'appui international est indispensable pour la défense des intérêts socio - économique et culturel des éleveurs en Afrique.

The situation of CBPP in Namibia in view of a new outbreak in the Caprivi region

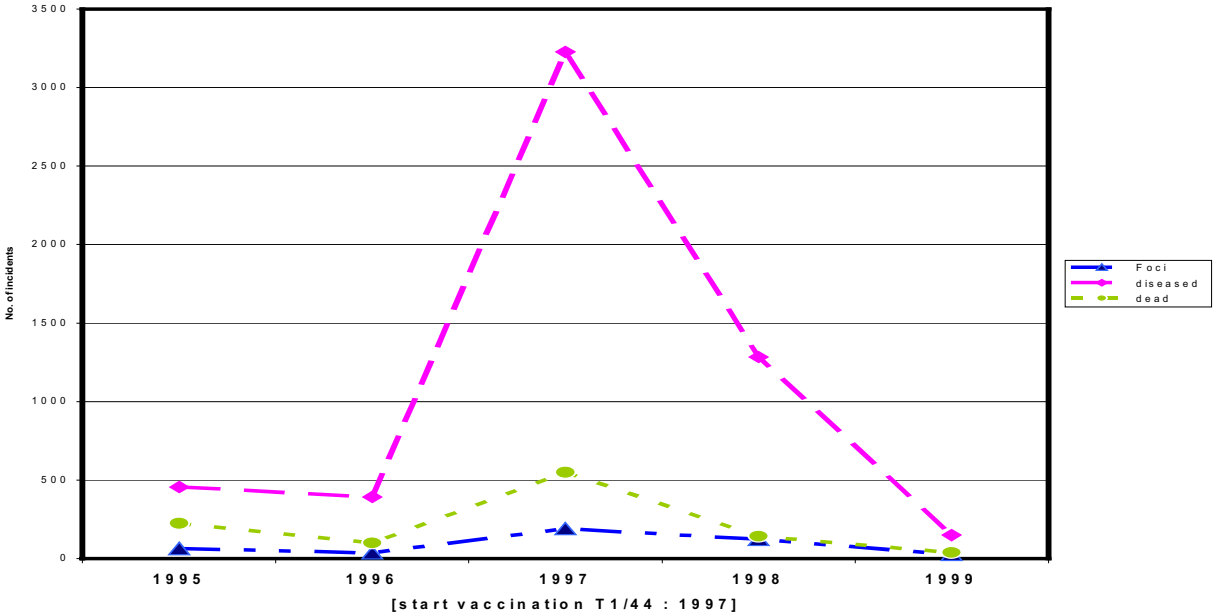
O. J. B. Huebschle[1], C. Bamhare[2], and G. Tjipura-Zaire[1]

[1]*Central Veterinary Laboratory, Ministry of Agriculture, Water and Rural Development, Windhoek, Namibia;* [2]*Epidemiology Division, Ministry of Agriculture, Water and Rural Development, Windhoek, Namibia.*

Introduction

The history of contagious bovine pleuropneumonia (CBPP) in Namibia goes back to pre-colonial times when infected oxen from the Cape Colony carried the disease into the territory at the middle of the 19th century. Though shortly contained after the first outbreak it made its reappearance after only a few years. Early attempts then to contain the disease by vaccination (Wilems) were only partially successful as a result of extensive cattle movement linked with cattle rustling. Further attempts during the early years of the 20th century with a strong emphasis on movement control and vaccination and destruction of infected animals proved successful in eradication of CBPP in the commercial farmlands. However due to the nature of livestock rearing in the communal lands CBPP persisted until today despite many different vaccination campaigns during the last 80 years. According to official documents at least 5 different vaccine formulations have been used for disease control with varying degrees of success. Amongst these vaccines were the Kabete vaccine, the KH_3J strain vaccine, the V_5 strain vaccine, the T1 SR vaccine and presently the T1 44 vaccine. Changes of vaccine formulations were brought about either due to serious side reactions or due to poor immune response as judged by failing protection. The latest vaccine change was done in 1997 after a period of no vaccination, which led to a considerable, renewed upsurge in clinical CBPP cases (Figure1, source: yearly reports).

Figure 1. Clinical/pathological diagnosis of CBPP in Northern Namibia 1995-1999)



As mentioned above, 1997 had a peak number of observed CBPP cases which decreased substantially after initiation of the T1 44 vaccination aimed to reach a near 80-90% vaccine coverage. However, due to civil strive still occurring along the northern border of Namibia, little control of cattle movement (transhumance) in these areas has been possible in the past. The movement of cattle from Angola still persists and veterinary services vaccinate all animals from Angola that are detected. Tables 1 and 2 show the number of confirmed and suspected cases of CBPP that have been diagnosed in the last few years.

Table 1. Confirmed and suspected CBPP cases 2000-2003* in Kavango Region.

	Kavango/ Region 2000		
	Foci	**Diseased animals**	**Dead animals**
Confirmed	4	7	11
Suspicious	10	23	8
	Kavango/ Region 2001		
	Foci	**Diseased animals**	**Dead animals**
Confirmed	4	12	17
Suspicious	11	30	23
	Kavango/ Region 2002		
	Foci	**Diseased animals**	**Dead animals**
Confirmed	0	0	0
Suspicious	2	4	6
	Kavango/ Region 2003 (to 10/2003)		
	Foci	**Diseased animals**	**Dead animals**
Confirmed	1	17	17
Suspicious	1	2	0

Sera submitted to Laboratory for confirmation

Year	**Positive foci/ Foci**	**Sera submitted**	**CFT positive**	**CFT negative**
2000	4/7	62	33	29
2001	1/2	6	1	5
2002	0/3	19	0	19
2003	3/6	37	12	25

* until end October 2003

Despite a recognisable reduction of clinical signs CBPP after the onset of vaccination during 1997, new cases albeit at a distinctly reduced rate have been recorded since the inception of the new vaccine strategy. Over the years the number of cases in traditionally known CBPP infected areas remained at a lower yet recognisable level. In all these cases it could be very often established that CBPP had been brought in via infected animals originating from beyond the northern border of Namibia. However, the areas affected remained restricted to the Kavango region the 4 North-central regions and the Kunene region. At no time since the end of the forties in the previous century had CBPP been recorded in the Caprivi region. This has been achieved by imposing strict movement control from the Kavango region to the further easterly-situated Caprivi region and further on by the fact that certain areas along the relatively narrow Caprivi Strip had been declared a cattle

free zone. Both restrictions allowed curtailing an eventual spread of CBPP from the endemically infected Kavango region into the Caprivi region.

Table 2. Confirmed and suspected CBPP cases 2000-2003* in North Central Namibia

	North Central Regions 2000		
	Foci	**Diseased animals**	**Dead animals**
Confirmed	9	38	5
Suspicious	103	316	68

	North Central Regions 2001		
	Foci	**Diseased animals**	**Dead animals**
Confirmed	5	15	1
Suspicious	93	357	79

	North Central Regions 2002		
	Foci	**Diseased animals**	**Dead animals**
Confirmed	6	73	25
Suspicious	10	46	8

	North Central Regions 2003 (to 10/2003)		
	Foci	**Diseased animals**	**Dead animals**
Confirmed	0	0	0
Suspicious	5	34	2

Sera submitted to Laboratory for confirmation

Year	**PositiveFoci/ Foci**	**Sera submitted**	**CFT positive**	**CFT negative**
2000	16/56	404	40	364
2001	4/13	167	13	154
2002	9/17	171	49	122
2003*	0/3	12	0	12

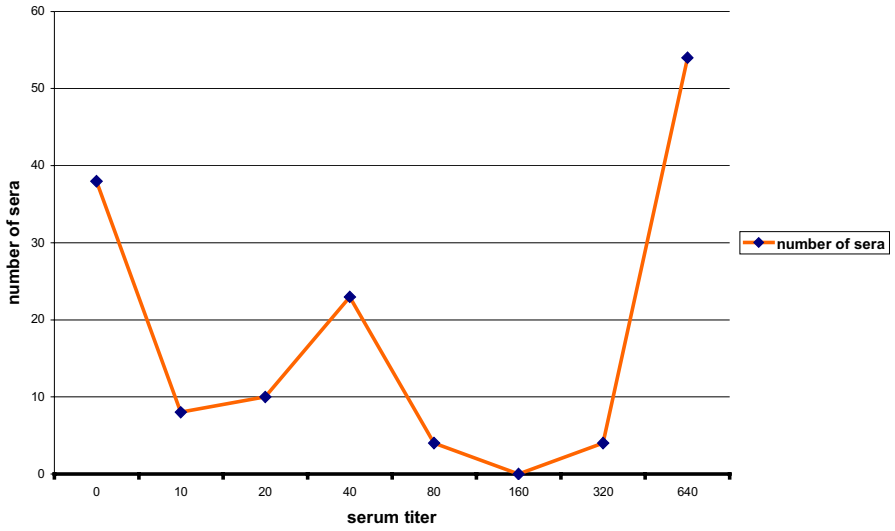
* until end October 2003

Despite a recognisable reduction of clinical signs CBPP after the onset of vaccination during 1997, new cases albeit at a distinctly reduced rate have been recorded since the inception of the new vaccine strategy. Over the years the number of cases in traditionally known CBPP infected areas remained at a lower yet recognisable level. In all these cases it could be very often established that CBPP had been brought in via infected animals originating from beyond the northern border of Namibia. However, the areas affected remained restricted to the Kavango region the 4 North-central regions and the Kunene region. At no time since the end of the forties in the previous century had CBPP been recorded in the Caprivi region. This has been achieved by imposing strict movement control from the Kavango region to the further easterly-situated Caprivi region and further on by the fact that certain areas along the relatively narrow Caprivi Strip had been declared a cattle free zone. Both restrictions allowed curtailing an eventual spread of CBPP from the endemically infected Kavango region into the Caprivi region.

It was only during August 2003 that state veterinarians communicated clinical observations suspicious of CBPP from an area in the Caprivi region, which is relatively

remote from the Kavango border after the region remained free of CBPP since 1938 (Figure 2).

Figure2. Distribution of CFT titers of 141 sera in a new CBPP outbreak of the Caprivi region, Namibia, during 2003.



Post mortem examination and subsequent isolation of MmmSC during September 2003 confirmed the original suspicion. Further enquiries with the owner of the affected animals revealed the specific cattle owner made his living with cattle trading (speculation) and that he might well have brought in animals from Zambia, across the border. As the animal that was inspected at post mortem had advanced lesions it stood to reason that most animals in his herd had contracted CBPP by then. Therefore, all 106 animals in the specific herd were sampled and serum was subjected to a CF test. Seventy six animals in this group (72%) had serum titres ranging from 1:20 to ≥1:640. The animals were then taken to a quarantine facility and slaughtered 3 weeks later. Inspection of carcasses revealed that 80% of them slaughtered had typical pathological lung lesions as seen in MmmSC diseased animals. During a visit to the affected areas another 36 sera from animals known to have been in close contact with the affected herd have been sampled. Of these animals 27 (75%) animals tested again positive in the CFT again with titres ranging from 1:10 to ≥1:640.

Authorities have in the meantime instituted a vaccination programme for the area where CBPP has been detected in the Caprivi, as compulsory culling of animals could not be instituted due to financial constraints and serious objections of the animal owners.

It is noteworthy to have a new, affirmative look at the CFT system that in this very case definitely has passed another validation successfully. All animals in the area were for the first time infected and could distinctively be identified by means of the CF test as CBPP positive animals which was further corroborated by the detection of pathological lesions during post mortem inspection.

[**Editorial Note:** Accompanying notes were not offered for this presentation, and the contents of slides used during the presentation are presented here.]

Experience Guineenne dans le Controle de la Peripneumonie Contagieuse Bovine (PPCB)

D. Bangoura

Direction Nationale Élévage, Republique de Guinée

Introduction

Selon le dernier recensement réalisé en 2000

Bovins: 2.787.058 têtes
Petits Ruminants: 1.719.207 têtes
Porcins: 55.645 têtes
Volaille traditionnelle: 8,6 millions têtes

Système d'élevage

Le mode d'élevage est caractérisé par 3 systèmes extensifs:

- l'élevage semi-familial de petite dimension (entre 1 et 5 têtes);
- l'élevage semi-pastoral de moyenne dimension (taille moyenne de 30 têtes);
- l'élevage pastoral de grande dimension (30 têtes et plus);

Historique de la PPCB en Guinee

- 1936 Premier foyer enregistré dans les mines d'or de Siguiri à l'Est du Pays
- 1945 - 1955 Extension à toute la partie Est du Pays (Haute Guinée et deux préfectures de la Guinée Forestière)
- 1955 Mise en place du cordon sanitaire partageant le pays en deux grandes zones épidémiologiques (zone infectée à l 'Est et zone indemne à l'Ouest)

Situation Epidemiologique de la PPCB en Guinee

Années	Foyers	Sensibles	Cas	Morts	Abattus	Indemnisés
1995	50	22794	335	103	310	0
1996	30	11678	71	11	60	0
1997	36	2644	719	341	1718	0
1998	11	298	61	57	227	0
1999	6	171	24	19	150	34
2000	0	0	0	0	0	0
2001	1	123	42	30	93	0
2002	2	84	3	0	3	0
Total	**136**	**37492**	**1255**	**561**	**2561**	**34**

Strategie Nationale de Controle de la PPCB

Trois périodes caractérisent l'évolution de la lutte contre la PPCB en Guinée:

1. Avant 1987: La lutte était essentiellement basée sur:

   - La vaccination de masse avec le T1 44;
   - La restriction du mouvement du bétail entre la zone infectée à l'Est (Haute Guinée et Région pré-forestière) de celle non in infectée à l'Ouest (Moyenne et Basse Guinée); L'abattage sanitaire dans la zone non infectée à l'Ouest du pays.

2. 1988 à 1994: Cette période a été caractérisée par:
   - La réalisation de campagnes de vaccination mieux planifiées;
   - Le renforcement du contrôle du mouvement du bétail;
   - La réalisation d'enquêtes épidémiologiques pour mieux connaître la répartition de la maladie.
3. De 1995 à nos jours: Cette période se caractérise par:
   - La combinaison de toutes les mesures précédentes de lutte renforcées par la participation active des acteurs du réseau de santé animale;
   - La délimitation de quatre zones épidémiologiques;
   - La surveillance épidémiologique (clinique, sérologique et de lésions);
   - La mise en place d'un cadre législatif et réglementaire;
   - L'identification national des bovins et la communication - Formation.

Contagious bovine pleuropneumonia in Zambia

M.P.C. Mangani

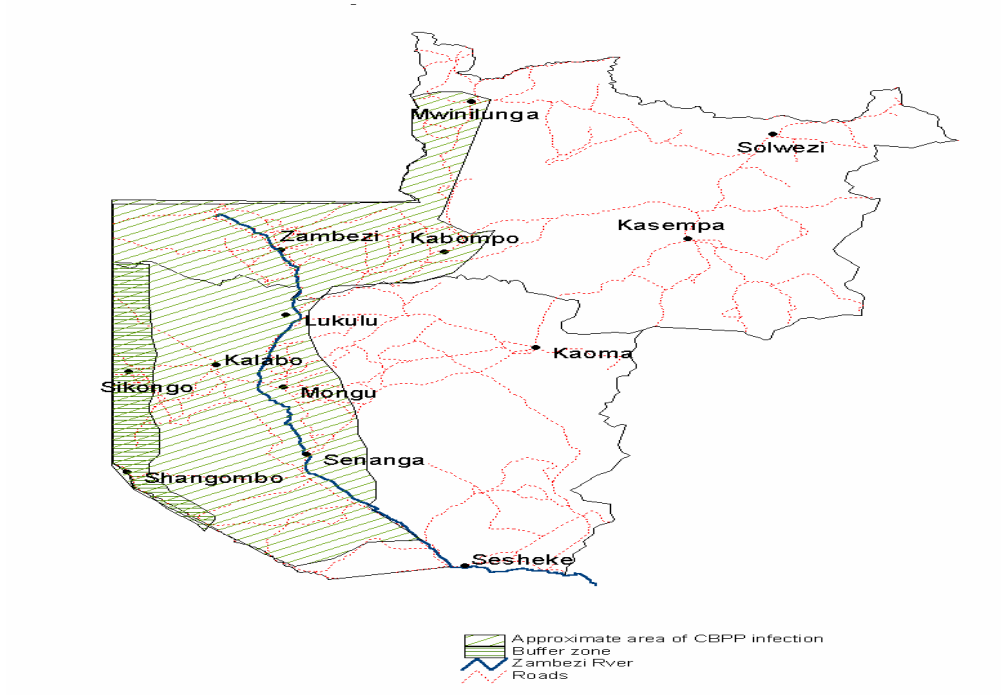
Research and Specialist Services, Box 50060, Lusaka, Zambia

Introduction

Contagious bovine pleuropneumonia (CBPP) was re-introduced into the Western Province of Zambia in 1997 after 23 years of absence. The disease was kept out by a combination of control measures and the creation of a buffer zone. A cordon line was created in the 1970s running for over 300 km parallel to the Angolan border. The buffer zone was maintained with routine vaccinations and other zoo-sanitary measures.

The CBPP situation changed in 1997 and by 2000 the disease had become widespread. In 2002, a further change to the pattern of the disease occurred with the disease spreading from Western Province to Northwestern Province thereby creating a threat to the rest of the Country and the sub-region as a whole.

Districts Affected by CBPP



1997 CBPP Outbreak

This outbreak was first recorded at Sinjemebela in an area not covered by the cordon line and was a consequence of an illegal movement of cattle across the Angolan border. The infected area had a population of 3,000 cattle and this meant that a population of 150,000 cattle was at immediate risk. This was out of a population of more than 500,000 cattle of the Western Province.

To control the disease, control measures were put in place and were based on the creation of control zones as follows:

(1) Focal Area (FA);
(2) Primary Risk Area (PRA);
(3) Secondary Risk Area (SRA).

The test and slaughter with compensation measure was instituted in the FA and PRA and about 1,200 cattle were slaughtered with compensation. The remaining cattle in these zones and those in the Secondary Risk Area were subjected to compulsory vaccination.

Three months after the initial exercise, a follow up test and vaccination exercise was carried out in the PRA and the FA. A total of 3,000 cattle were tested using the CFT and all sero-positive cattle were slaughtered. Over 150,000 cattle were vaccinated during the same exercise. After 6 months another exercise was carried out and only 4 individual cattle were sero-positive. In November of 1998 no sero-positive cattle were recorded and the disease was controlled.

After successfully controlling the outbreak, routine activities that had slackened were strengthened in the following areas:
(1) Buffer Zone;
(2) Surveillance Zone;
(3) Clear Zone.

The following activities were implemented in the different zones:

(1) Buffer zone:

This includes the existing buffer zone in Western and North-Western Provinces along the Angolan border and the area in the Northern Province along the Tanzanian border. In the buffer zone, apart from routine vaccinations, movements of cattle are restricted to only work oxen that are vaccinated and provided with a license for designated routes into the surveillance and clear zones. Cordon guards were deployed at 8 to 10 km intervals but at the time of the outbreak, most of the guards had been laid off.

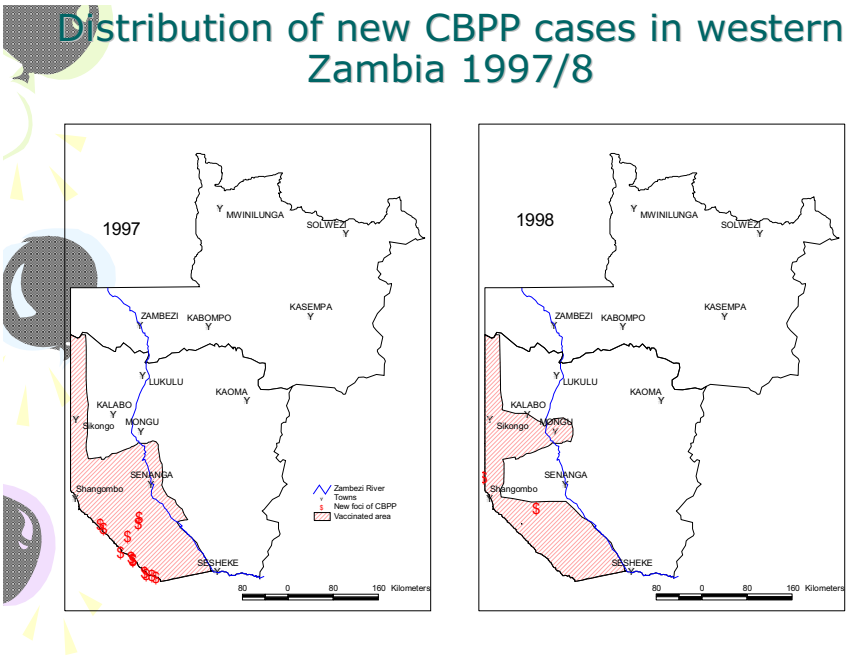
(2) Surveillance zone:

The Western Province apart from the buffer zone was designated the surveillance zone. Herds were inspected at monthly intervals and periodic sero-surveys were conducted based on the epidemiological evidence of the disease. All movements out of this zone were subject to issuance of a movement permit. Cattle were only permitted to move to a designated abattoir within Western Province under veterinary escort and only for slaughter.

(3) Clear zone:

The areas adjacent to Western, Northwestern and Northern Provinces that were free from CBPP were designated as clear zones. In these zones disease search is conducted through abattoir monitoring inspection of herds. Check points were established and maintained at designated points on the routes leading into the clear zones. No live animals are allowed into the clear zones from the surveillance zones.

If disease was detected in the clear zone in a small geographically isolated population and there is certainty that the disease has not spread, the stamping out with compensation will be instituted. Consideration has to be given to the effect of the social disruption that slaughters cause on the communities affected.



Influx of Refugee Cattle 1999 – 2000

In late 1999 due to the escalated civil strife in Angola a fresh re-introduction of the disease was experienced due to influx of refugee cattle into Zambia. The initial 1,767 refugee cattle that arrived in December 1999 were detained and later slaughtered at designated abattoirs and owners were paid compensation. Later on it became difficult to identify refugee cattle as they were disguised as Zambian cattle. This was for fear of having the cattle confiscated. As a consequence in December 2000 a widespread occurrence of CBPP was experienced in the Shangombo, Mambolomoka area. This was to the north of the 1997 focus. Due to the magnitude of the outbreak the Government of Zambia failed to maintain the slaughter and compensation policy. Instead, all herds that had clinical CBPP and those that tested positive on CFT were sent for slaughter under Veterinary escort to the designated abattoir within Western Province.

In effort to control the disease the Zambian Government in 2001 sought assistance from FAO and UNHCR and the support was given to rehabilitate quarantine stations so as to hold the refugee cattle in trust. Two registration centers and two quarantine facilities were established to accommodate refugee cattle at Shangombo and Mabua – Sikoongo.

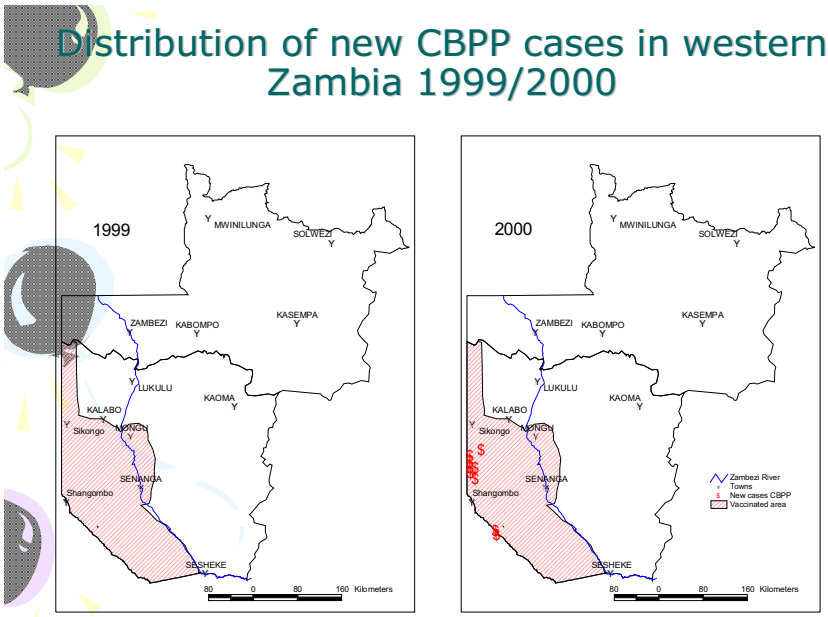
In March 2002, a turn of events was experienced with the following taking place:

(1) Peace was coming to Angola and no refugee cattle were crossing into Zambia
(2) CBPP within Zambia had become widespread and was detected in Zambezi, Northwestern Province.

It was identified that the disease was introduced into the Province through 3 possible routes:

(1) direct introduction through refugee cattle;
(2) llegal cattle movements from Kalabo – Western Province;
(3) trading with Lukulu an infected area of Western province.

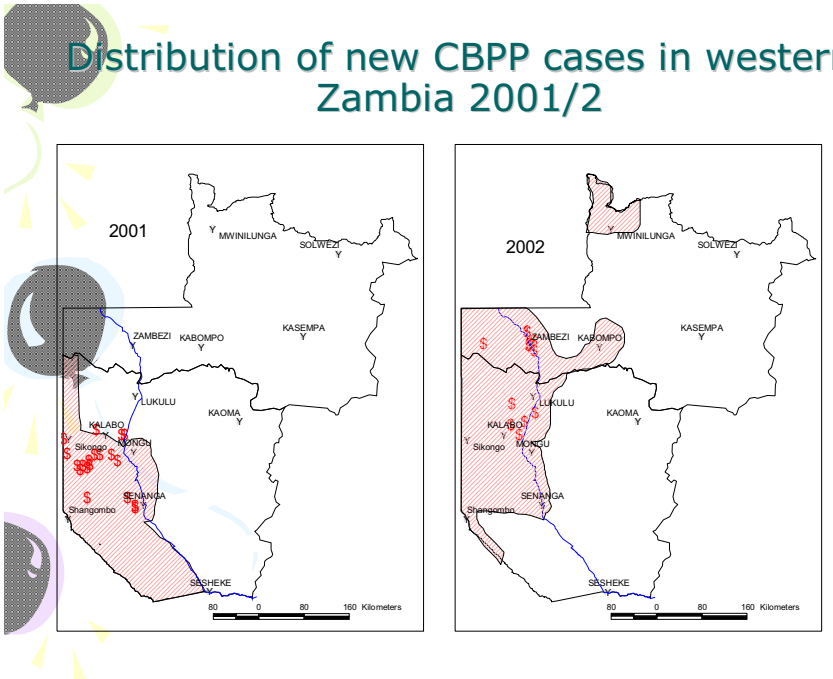
It was also identified that the disease had affected the other districts of Northwestern province namely Chavuma, Kabompo and Mwinilunga.



CBPP in North Western Province 2002

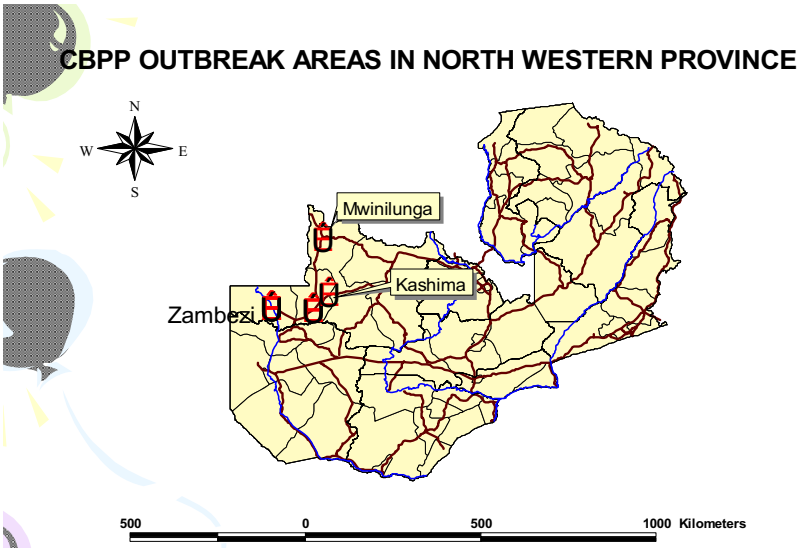
The disease was first detected in Zambezi District in March 2002 at a local slaughter slab. Testing of all herds in Zambezi Central using CFT and cELISA tests showed that the disease was widespread.

The new turn of events necessitated a change in the control strategy as the threat to the whole sub-region became more imminent. At the onset of the disease, slaughter with compensation was attempted but discarded when disease was found to be widespread. The current control measures are therefore based on achieving a high vaccination coverage complemented with disease screening.



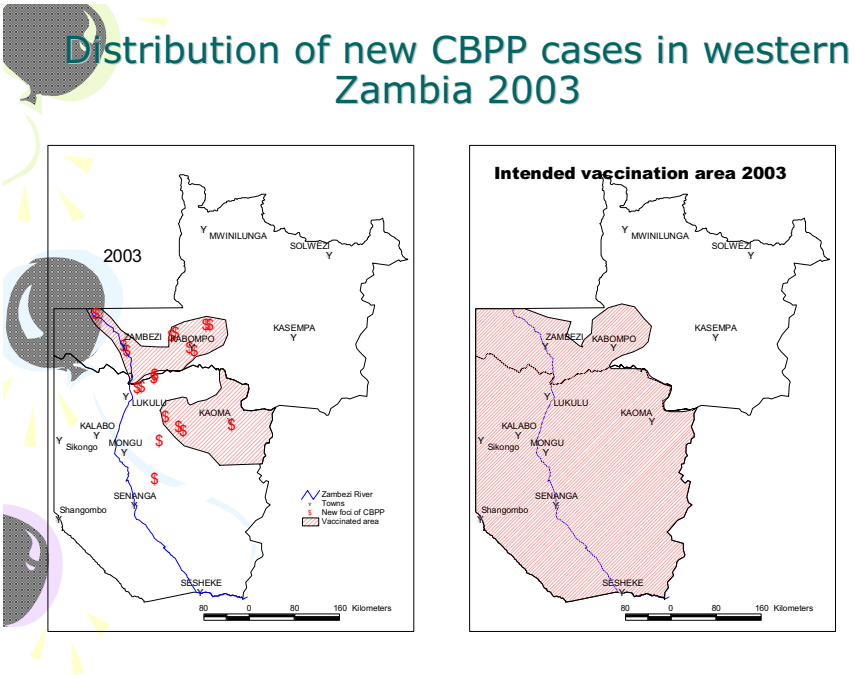
CBPP Outbreak at Mufumbwe – Kashima 2003

In February 2003 CBPP was detected in Mufumbwe District at Kashima area through routine inspections. Two herds were affected due to illegal movement of cattle from an infected area and unvaccinated pocket in Kabompo District. Kashima is on the route to the Copperbelt Province and is the market for beef for Northwestern Province. The affected area has a population of about 200 cattle and an area covering 2500 cattle was vaccinated.



CBPP Outbreak at Kaoma

Kaoma district shares borders with the CBPP infected Districts of Mongu on the West and Senanga on the South. The disease was reported at two Veterinary camps of Winda and Mbayutu in June 2003. Both camps are on the borders of the infected Districts. The disease was being reported for the first time in Kaoma District and the entire District has been vaccinated.



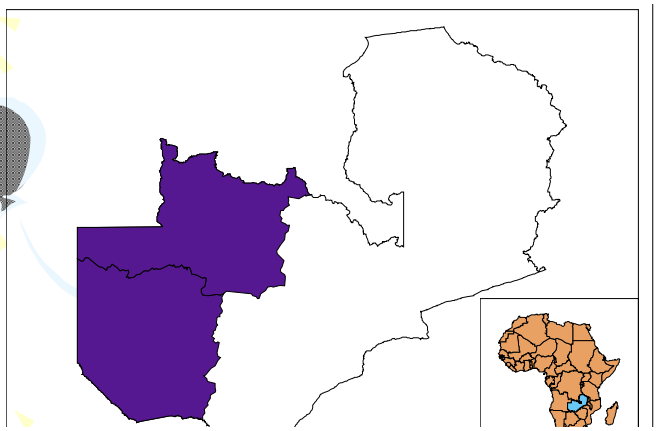
CBPP Intervention Strategy – 2003

The policy of the Government of Zambia is to eradicate CBPP whenever introduced into the country and to contain it wherever it has established itself with a view of eradicating it. Strategies are therefore constantly reviewed according to the disease threat and spread.

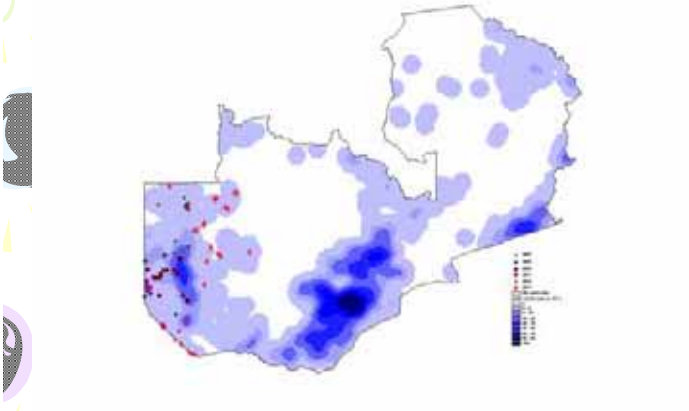
The current CBPP strategy is to contain the disease within the already affected areas with a view to eradicating it and to prevent further spread. The following is the focus:

- Containment of CBPP in newly infected areas through increased surveillance and routine vaccinations to reduce the disease prevalence and to lessen the disease spreading to areas at risk.
- Strengthening the abattoir surveillance through involvement of trained Veterinary staff in meat Inspection to ensure integration of the abattoir surveillance system into the zoo-sanitary information system.
- Implementation of a national identification system and establishment of the herd registry for livestock.
- Sensitizing stakeholders on disease recognition and control in order to improve implementation of zoo-sanitary measures.
- Capacity building of Veterinary staff through continued training in CBPP disease recognition and control.

Provinces of western Zambia affect by CBPP



Distribution of new CBPP outbreaks 1997-2003



[**Editorial Note:** This report was not presented at the Consultative Group Meeting on CBPP in Africa Towards sustainable CBPP control programmes for Africa, Rome, 12 – 14 November 2003, but is included in this document for completeness.]

The use of the field complement fixation test in the diagnosis and control of contagious bovine pleuropneumonia – the Kenyan experience – successes and failures

S.W. Kairu-Wanyoike, A.K. Bengat and W. Lung'aho

Central Veterinary Laboratory, Kabete, Kenya.

Introduction

The zoning of the country with respect to CBPP has constantly been revised since the first case in Kenya was recorded in 1901 following the pattern of disease outbreaks and the situation of the disease in the neighbouring countries (Kariuki, 1971). Up to 1975 3 zones were recognised (though not mapped) as Category A: Infected or severely threatened districts which included the entire north and eastern districts of Turkana, Marsabit, Mandera, Wajir, Isiolo, Garissa and Tana River; Category B: Areas under threat but with no disease consisted of West Pokot, Baringo, Samburu, Kitui, Kilifi and Maasailand; Category C: Clean areas which included the rest of the country mainly the Central and Western highlands and part of the coast (Ministry of Livestock Development and Fisheries, 1978).

Constant threat of CBPP to West Pokot and outbreaks in the early 80s originating from the neighboring Republic of Uganda saw West Pokot added to Category A districts. Illegal movement of cattle from West Pokot to Busia, Bungoma and Siaya districts in the West led to outbreaks in these districts which were then added to category A districts with the neighbouring districts of Trans-Nzoia and Kakamega being added to Category B districts.

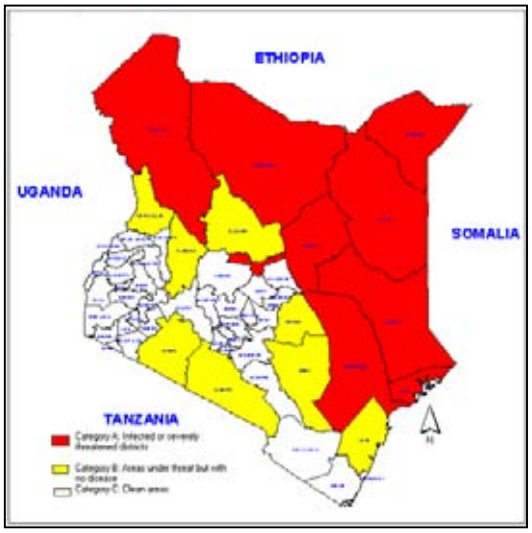
Meanwhile the control policy in Kenya continued along the lines of mass vaccination in enzootic areas, increasing emphasis on field testing with slaughter of reactors within the enzootic areas, rigid control over movement out of the enzootic and suspect areas and rapid stamping out, by testing and possibly vaccination, of any outbreaks that might occur outside the enzootic areas thus the outbreaks in the west were eventually cleaned up (Kane, 1975). Following field-testing with slaughter of reactors and mass vaccination in Maasailand this vast pastoralist area remained free of CBPP from 1968 to 1986. In 1986/87 CBPP re-entered Maasailand following the purchase of ex-Garissa and ex-Isiolo cattle probably from Somalia as this also marked the beginning of the Somalia unrest.

Epidemiological studies by Wanyoike (1999) established that CBPP had partly been re-established in Maasailand and was now threatening Ukambani (Makueni, Machakos, Mwingi and Kitui) Nakuru and Thika districts following transhumant cattle movement due to the decrease in the Maasailand dry season grazing area due to human settlements.

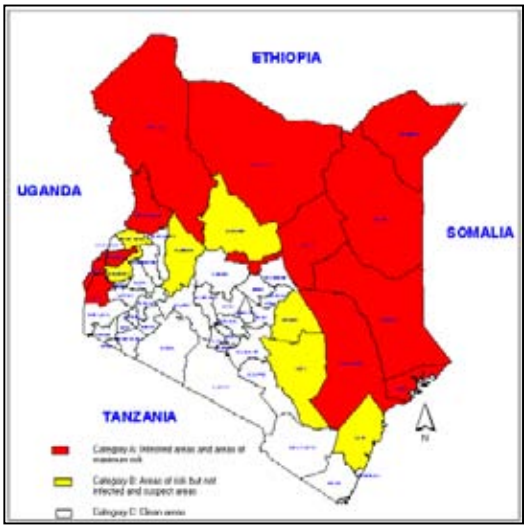
Taking into consideration the recommendations of the CBPP Workshop on Control Strategies in Accra, 2003, abattoir surveillance in 2002 and the recommendations of a Consultancy Report on Livestock Health Quarantine Measure Study in 2000/2001, a new zonation of the country into 4 zones and a redefinition of control strategies was put in place in June 2003. The redefining of the control strategies considers the revitalising of the control measures that saw the eradication of the disease in the greater part of the country in the seventies. These control measures are particularly cattle movement control from infected to non-infected areas with identification of cattle by district brands and field-testing with slaughter of reactors. Of increasing importance is the strengthening of epidemiological

surveillance networks particularly abattoir surveillance along with extension of both field-testing and laboratory confirmation. Vaccination remains an integral part of control of CBPP.

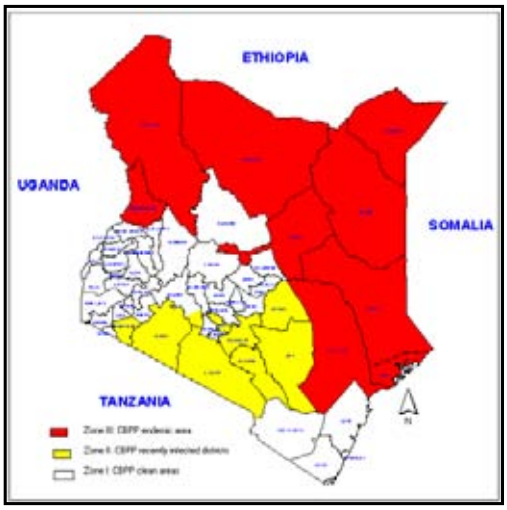
CBPP zones in Kenya 1901 to 2003



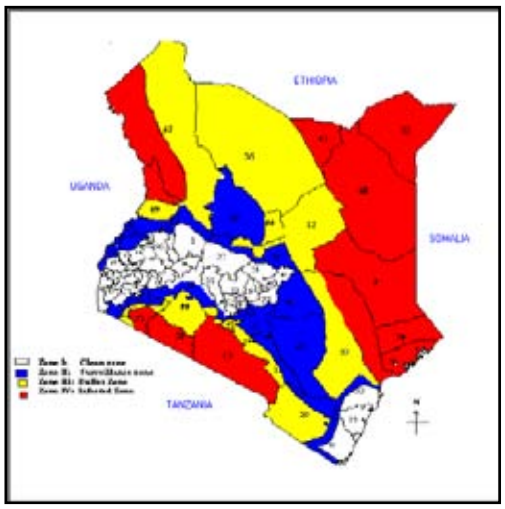
Before1975



1975–1982



1982–2002



June 2003

The history of CBPP field complement fixation test in Kenya

Until 1950 the control of CBPP presented a unique set of problems in Maasailand (and indeed in other pastoralist communities): veterinary cover has never been wholly adequate, information is often difficult to obtain and concealment of infection is common. The Maasai jealously guard their traditional right of unrestricted movement within their territories and indeed transhumance is necessary for survival. The Maasai methods of husbandry include close herding at night in temporary bomas, an ideal situation to perpetuate CBPP. It is against this background that the Kenya Department of Veterinary Services evolved a policy of control based on the removal of infected herds to specially designated quarantine areas (holding grounds) where they remained under maximum possible supervision until free of disease. Difficulties arose in the administration of

quarantine because of shortage of water and grazing and farmers visualization of other epizootics as more important than CBPP leading to widespread attempted evasion of quarantine restrictions.

Though quarantine measures, even when allied to vaccination of infected herds, are not calculated to produce spectacular results in terms of disease eradication, the continued application of such measures during the decade (1950-1959) had by the end of that period resulted in a considerable measure of control, in that CBPP was absent from the eastern half of Maasailand but remained endemic in the western half. To consolidate the progress so far made a large-scale vaccination campaign using egg adapted vaccine was attempted in 1957. The campaign was to be abandoned because of severe inoculation reactions. In 1959 the movement of a single infected herd through the east led to the re-infection of the eastern half and about six other districts (Huddart, 1963).

It was in the face of this considerable distribution of the disease that it was decided that possibilities of control based upon increased use of the complement fixation test be investigated. It is towards this end that a modification of the conventional complement fixation test, suitable for large scale field-testing was devised.

Brief outline of the testing operation

Field-testing was designed to be carried out by a mobile unit, transportable, together with its staff of eight assistants in a 3-ton lorry. The important items of equipment include a large tent, portable tables and chairs, an electric generator, centrifuge, refrigerator, water baths, drums of water and petrol and glassware in specially designed containers. Tents for the staff and personal baggage were also carried.

Testing took place at centres equipped with suitable cattle crush facilities (usually at cattle watering points) and the unit travelled to the testing site and set camp on the day before testing was set to begin. It is usually convenient to spend several days at any one site during which time cattle are brought to the crush from within a radius of ten or more kilometres. The testing tent was erected at least a hundred yards from the crush on the windward side of it to avoid dust, one of the greatest enemies of the test in the field.

Today a mobile laboratory (specially designed landrover) transports equipment while another landrover transports the staff hence replacing the 3 ton lorry. Camp is sometimes set up at permanent public facilities such as schools, chiefs' camps, hospitals and field veterinary offices obviating the need for tents, portable chairs and tables. The sampling team who makes the local runs and delivers the samples to the testing team then uses the second landrover.

When testing is in progress cattle are driven into the crush and there numbered and bled from the tail so as to produce samples of whole blood diluted in saline. Batches of 76 samples at a time are carried to the tent and inactivated to destroy complement, then centrifuged to produce clear diluted plasma that is used in the test. Testing is carried out on plastic agglutination trays incubated by 'floating' on the surface of a water bath maintained at 37°C. Preliminary screening test is followed by a more detailed repeat test the whole testing operation taking 3-4 hours to complete. Test results become available at intervals during the day and reactors are identified either the same day or early the next morning. Reactors are the branded 'PP' for immediate slaughter under the supervision of a veterinary officer upon which samples are taken.

The testing unit is supplied with preserved test reagents sufficient for two weeks stay in the field though it is usually convenient to renew reagents each week. The samples must

be held at 4°C initially otherwise for a days work the reagents are packed in vacuum flasks with a few cubes of ice and adequately padded to resist extreme degrees of vibration in the course of travel over poor road surfaces. The testing capacity of a unit depends greatly on the quality of the crush and local assistance in driving animals and a fair day's testing under average conditions consists of about six hundred individual tests.

The field CFT has its major advantages over the laboratory CFT in that:

1. It makes use of a method of dispensing suitable for use by relatively untrained laboratory assistants,
2. It makes use of a method of bleeding cattle (through the base of the tail) rapidly and in large numbers so as to produce samples capable of being used immediately in the test,
3. Due to 1 and 2 above, there is high sample throughput.

The testing of animals that was previously carried out at the Central Veterinary Laboratory in Kabete on conventional serum samples is now done by a testing unit in the field resulting in a great saving in time and labour. Field-testing obviates the time consuming jugular bleeding and transport of samples to the laboratory and repeated sampling due to anti-complementary samples.

Application of the field CFT in field control

1. Survey and Quarantine testing

As a survey test, the CFT is used to detect infection in districts and identify infected herds. The usual problem in Kenya has been infection either detected or suspected within an area, commonly involving perhaps ten to twenty thousand head of cattle. Testing for a week or two by a field unit enables the incidence of disease to be mapped out with complete certainty and field officers are able to apply appropriate quarantine measures. This type of operation has been carried out on many occasions and contributes substantially to local control of the disease (Table 1).

As a quarantine test the field CFT is applied at the end of the outbreak to remove chronically infected animals.

Sometimes it was possible to begin the survey soon after the outbreak was reported while in some instances two to three months elapsed before the survey begun due to lack of funds. In some areas the survey had already begun before an outbreak was reported as it was highly at risk from a neighbouring infected area.

Quarantine was lifted after no more reactors were reported (Kiambu, Embu) or when there were no more reports of disease (Kilifi, Thika). Survey was on both the focus and in-contact herds /farms and was often followed with vaccination to stop the spread of disease hence the need for 3 to 6 or more months interval between tests.

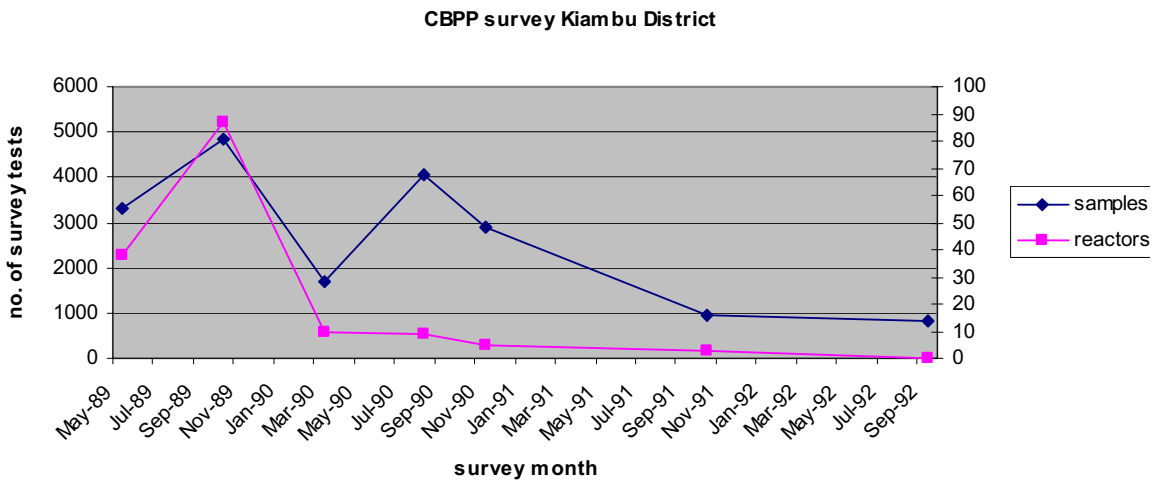
Table 1. The application of survey and quarantine testing in Kenya in recent years.

District	Outbreak location	Period of survey	No. of survey tests	No. of reactors	Last outbreak	Quarantine imposed	Quarantine lifted
Kiambu	Karai/ Ndeiya	May 89–Sept 92	18,598	152	1992	Aug 88	Oct 97
Embu	Karaba (Nov 89)	Nov 89–Feb 92	11,814	14	1993	Nov 89	Apr 93
Kitui	Athi/Ikutha/ Kanziku (Jan 89)	Jan 89–July 90	37,446	111	1996	1986	In force
Kirinyaga	Marurumo (Mwea) (Nov 89)	Nov 89–Nov 90	3,304	8	1989	-	-
Machakos	NYS Yatta /Embakasi/ Lukenya /Boming Range (Jan 89)	Nov 89–Aug 95	23,185	73		Dec 88	In force
Kilifi	Galana/ Mariakani APRS (Feb 90)	1990–1993	70,576	831	1993	Apr 90	Aug 93
Narok	Naroosura (Mar 90)	Apr 90–Oct 91	19,555	429	2003	Apr 90	In force
Lamu	Delta Agencies/ Nairobi Ranch (Nov 90)	Apr 90 – Nov 90	4,973	9	1994	July 88	Oct 88
Nakuru	Marula estate (Jan 1991)	Jan 91–Sept 93	22,847	141		Jan 91	
Nakuru	Gilgil (Jan 91)	Jan 91–Sept 91	11,096	22	2000	Jan 91	In force
Tana-River	Wenje/Galole/ Garsen	Aug 91–July 97	8,713	76	2000	July 00	July 02

Table 1.
(Cont'd)

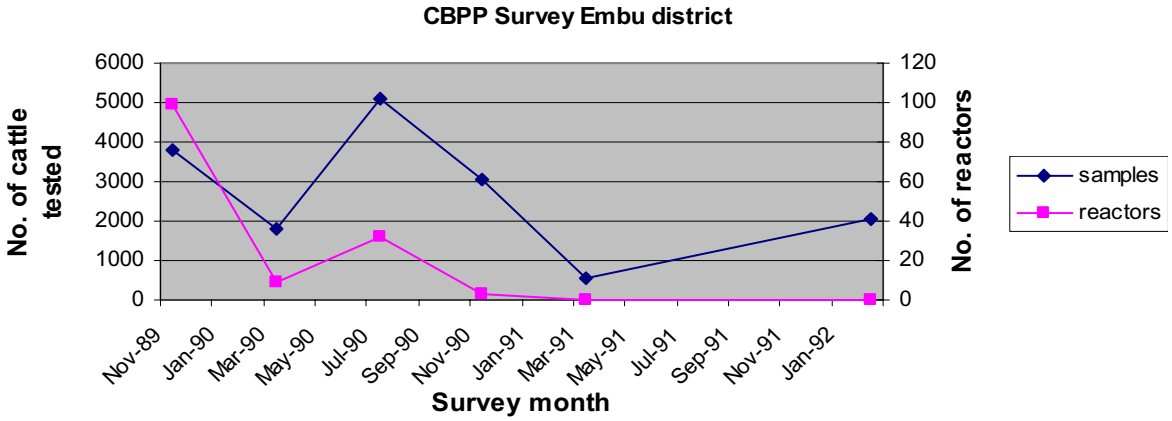
District	Outbreak location	Period of survey	No. of survey tests	No. of reactors	Last outbreak	Quarantine imposed	Quarantine lifted
Taita-Taveta	Ananka/ Bachuma/ Maungu/ Taita ranch (Jul 91)	Jan 91	1,939	0	2003	May 91	In force
Nakuru	Kedong ranch (Mar 92)	Jan 92 – Jun 95	8,999	78	1995	Jan 92	1996
Laikipia	Ereri Ranch (July 92)	July 92–Aug 94	14,370	323	1992	July 92	Sept 95
Mombasa	Mombasa dairies (Mar 92)	Mar 92 – Mar 93	1,005	14	1992	Mar 92	1995
Laikipia	Laikipia airbase (July 93)	July 93 – Sept 95	9,580	264	1993	July 93	Sept 95
Laikipia	Ngobit (Aug 94)	Aug 94–Feb. 95	3,111	20	1994	June 94	Sept 95
Thika	Kiganjo – Witeithie (Mar 97)	Jun 97–Mar 98	3,099	25	1998	July 97	In force
Kajiado	Ngatataek/ Lenkism (Mar 98)	May 98	2,104	0	2001	Apr 98	In force
Narok	Maji-moto/ Olkinyei (Feb 02)	Apr 02	3,280	5	2003	Feb 02	In force
Trans-Nzoia	Macheo/ Gitwamba/ Kedowa (Apr 02)	Apr 02 – May 03	401	40	2003	May 02	In force

Graph 1: CBPP survey Kiambu district (Ndeiya/Karai, Kenya, 1989-1992



The progressive removal of reactors coupled with livestock movement control and ring vaccination of about 3,523 cattle in 1991 and 1992 led to the disappearance of disease from this location such that the disease has not been reported again since 1992.

Graph 2: CBPP survey Embu district (Karaba/Evurori), Kenya, 1989-1992

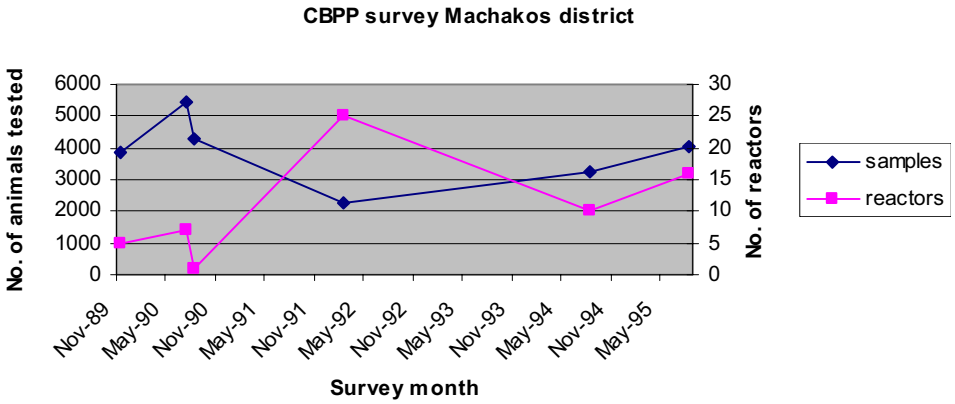


In Embu district the disease was “stamped out” through test and slaughter of the reactors and disease has not been reported since 1993.

Marula estate – Nakuru district

In Marula estate CBPP broke out in January 1991. Attempts to control the disease through test and slaughter were frustrating as each subsequent test yielded more reactors than the previous one. This prompted the manager to attempt treatment but it not being encouraged at the time, a different approach of test and slaughter and vaccination of the remaining animals every 3 months saw the eventual eradication of the disease from the ranch that the disease has not been reported since September 1993.

Graph 3: CBPP screening Machakos district (Yatta, Lukenya/Boming Range), Kenya, 1989-1995



The progressive removal of reactors and ring vaccination of about 31,303 cattle in 1992 and 1995 has not sufficiently controlled CBPP in Machakos district such that the disease persists to date and the affected area is under quarantine since December 1988.

2. Screening testing

In Kenya there exists the frequent necessity for testing large numbers of cattle derived from potentially infected areas and intended for distribution in clean areas. This happens in the case of cattle moved for fattening in ranches and for breeding. Though intended for immediate slaughter, cattle moved from infected areas for slaughter particularly to Nairobi, Kiambu and Mombasa have undergone such screening. Originally cattle were moved only after successfully undergoing consecutive negative CFT tests 2 months apart; 3 for cattle intended for fattening and breeding and 1 for cattle intended for immediate slaughter. Currently only 2 tests 21 days apart are required for cattle moved for fattening and breeding while slaughter cattle are not screened but rather are mouthed and clinically examined.

Field teams

Team	Area of operation	Status	Purpose
Kabete	Booster to all other teams and Central region	Operational	Stamping out of disease in central clean area and backup for other teams
Nakuru	Maasailand in the south and West Pokot district in the west	Non-operational	Removal of reactors for control and screening of cattle for movement from infected to non-infected area
Isiolo	Northern and eastern region (endemic area)	Operational	Removal of reactors for control and screening of cattle for movement from infected to non-infected area
Tana River (Garsen)	Eastern and coast region	Operational	Removal of reactors for control and screening of cattle for movement from infected to non-infected area (mainly coast region)
Malindi	Coast region	Non operational	Screening of cattle for movement to coast region and stamping out in coast region
Garissa	Eastern region	Operational since October 2002	Screening of cattle for movement mainly to coast region and Ukambani
West pokot	Western region	To be established in October 2003	Screening of cattle for movement mainly from Turkana and West Pokot to the clean districts in the west and stamping out in the western clean districts

Table 2: The application of screening testing in Kenya in recent years.

Year	Cattle screened	Number positive	% positive
1989	30856	6	0.02
1990	37976	20	0.05
1991	17099	11	0.06
1992	8737	15	0.17
1993	4951	49	0.02
1994	1308	1	0.08
1995	3683	0	0.00
1996	7655	1	0.01
1997	2012	0	0.00
1998	3165	0	0.00
1999	5877	7	0.12
2000	1591	0	0.00
2001	532	0	0.00
2002	17365	11	0.06
2003 (up to July)	19369	0	0.00

Screening to some extent serves as a survey test. Indications over the years are that the prevalence of the disease as detected by the field CFT in infected areas is very low reaching a maximum of only 0.17% (Table 2). The number of cattle screened fell due to the fact that screening of slaughter cattle was put on hold and also because of increased insecurity. However, in the last 2 years it has picked up with improved security and increased movement of cattle into ranches and the establishment of a new screening unit in Garissa.

3. Eradication testing

On a larger scale, survey testing can be applied to establish disease free areas which by further testing can be extended and so form the basis of a large scale eradication scheme.

Eradication testing has been attempted in Kenya by attaching the field teams to vaccination teams during district wide vaccination programs either for CBPP or for FMD. Testing precedes vaccination. Positives are removed and herds in which there are positives are ear marked for subsequent testing. Problems arise due to the need to concentrate testing resources over a lengthy period and lack of full co-operation of livestock owners in removal of reactors.

Problems facing the mobile field teams

Several problems face the field teams as follows; leading to declining operations.

1. Inadequate personnel and operating funds

Following structural adjustments within the civil service, there has been a decline in the number of field personnel leading to the shrinkage of field teams and team members being charged with additional responsibilities. This has greatly affected screening testing of cattle intended for slaughter (up to 150,000 annually) as the numbers are too high for the teams such that this kind of testing has been put on hold. Field testing also requires a near permanent presence in the field, as cattle are moving all the time, which puts a strain on the national budget.

2. Breakdown of equipment and vehicles

Of major use in field testing are two 4 wheel drive vehicles, a large centrifuge (to handle up to 144 tubes), 1 or 2 large waterbaths, a portable refrigerator. When these equipment and vehicles breakdown, sometimes replacement is not as fast as should be.

3. Insecurity

Field testing requires the rounding up of cattle and remainder in a holding ground for several days. In areas where rustling is a frequent phenomenon, field operations are threatened and so are the lives of personnel involved.

4. Lack of co-operation from stock owners

Resistance is encountered in presentation of cattle twice for testing such that some cattle are moved only after the first test. Some will resist the slaughter of positives but this is not very common, as there is a salvage value to the animals.

5. Fragmentation in the marketing process

Originally, the Livestock Marketing Division of the Ministry would purchase cattle from the livestock owners and would take responsibility for testing and eventual delivery of the cattle to the slaughter points. Following trade liberalisation, individuals assumed responsibility for testing and marketing of their own cattle making the screening exercise more difficult as screening was now of many small groups instead of a few large groups.

6. Disappearing holding grounds

Recently individuals have taken possession of public utility land and holding grounds have not been spared. In the few cases where they have been spared, fencing and watering and grazing facilities have not been properly maintained and crushes at watering points may also have suffered the same fate.

Conclusion and Discussion

Field testing has proved to be useful in the 'stamping out' of the disease from specific enclosed herds such as in ranches and in areas with small plots of private owned grazing land. In certain instances the disease was removed by the test and slaughter policy alone as in Embu district while in Kiambu district vaccination had to be brought in. Where movement control is difficult and herds are not enclosed as in Machakos district, test and slaughter even when coupled with vaccination may not adequately control the disease.

Testing old-standing quarantine herds, with a view to identifying and removing chronic reactors and so enabling the rest of the herd to be discharged from quarantine has achieved little success. Usually the first results in the detection of chronic cases and are removed. A second test six weeks later reveals the presence of a few low titre reactors and even if these are also removed the same thing happens in the third test. This can be ascribed to the presence of persistent low-grade circulating infection, but whether or not this is the case, the practical result is that old standing quarantines cannot be cleared by testing as quickly as would be hoped. Vaccination then becomes of use in preventing the further spread of the infection.

The sensitivity of the field CFT reaches only 40% whilst the specificity reaches 97.8% (Scudamore, 1975). The incubation period of CBPP is 20-40 days (Provost *et al.,* 1987). As the field CFT will not necessarily detect cattle incubating the disease or those with hyperacute or early infections, cattle in Kenya are screened twice at a 21 day interval taking into consideration the incubation period and the economic and practical aspects of how long the cattle can be held for screening.

The screening of trade cattle in Kenya is a workable system in that using the system up to 40,000 cattle per annum have been moved to clean areas without outbreaks in the past (Kane, 1975). Due to lack of economic resources, breakdowns in part of this system have been

encountered and all that is required is a revamping of the system to accommodate the 150,000 cattle moved annually.

Large scale survey testing using the laboratory CFT in 2003 involving 6000 samples proved to be rather disappointing since long storage of samples led to massive anti-complementary reactions and false positives probably due to non-specific reactions from probable contamination. With the higher sample throughput of the field CFT and no need for storage of samples, it is possible that a reasonable level of success could be achieved in large scale survey testing for zonal eradication of CBPP and as an epidemio-surveillance tool.

Acknowledgements

Appreciation goes partly to the GTZ, DAAD and the Universities of Berlin and Addis Ababa and the Kenya Veterinary department of the Ministry of livestock Development and Fisheries through which it was possible to collect and process the data used to synthesize this paper.

References

Consultancy report (2001). The Livestock Health Quarantine Measures Study in Kenya by African Technology Development Link.

Huddart, J. E. (1963). A field modification of the complement fixation test for contagious bovine pleuropneumonia. Rome: FAO, Animal Health Branch Monograph No. 6. Food and Agriculture organisation of the United Nations.

Kane, K. W. S. (1975). The control of contagious bovine pleuropneumonia in Kenya. *Bull. Off. Int. Epiz,* 84, 421-428.

Kariuki, D. P. (1971). History of CBPP in Kenya. *Bull. Epiz. Dis. Afr*. 19, 111-116.

Ministry of Livestock Development and Fisheries (MOLD&F) (1978). Veterinary Departmental Reports. Ministry of Livestock Development and Fisheries, Nairobi.

Provost, A., Perreau, P., Breard, LeGoff, C., Martel, J. L., Cottew, G. S. (1987). Contagious bovine pleuropneumonia. *Rev. Sci. Tech. Off. Int. Epiz*. 6, 565-679.

Scudamore, J. M. (1975). Evaluation of the field complement fixation test in the diagnosis and control of contagious bovine pleuropneumonia. *Trop. Anim. Hlth. Prod*. 7, 73-79.

Wanyoike, S. W. (1999). Assessment and Mapping of CBPP in Kenya: past and present. Master of Science Thesis: Freie Universität Berlin and Addis Ababa University.

REUNION CONSULTATIVE FAO-OIE-AU/IBAR-IAEA SUR LA STRATEGIE DE LUTTE CONTRE LA PERIPNEUMONIE CONTAGIEUSE BOVINE (PPCB) EN AFRIQUE
12 AU 14 NOVEMBRE 2003, ROME-ITALIE

EXPERIENCE GUINEENNE DANS LE CONTROLE DE LA PERIPNEUMONIE CONTAGIEUSE BOVINE (PPCB)

Présentation :

Dr Daouda BANGOURA
DSV-Guinée

I – INTRODUCTION

Selon le dernier recensement réalisé en 2000 :

Bovins : **2.836.600 têtes**
Petits Ruminants: **1.830.181 têtes**
Porcins **55.645 têtes**

Selon les estimations de la DNE en 2000 :

Volaille traditionnelle : **11,9 millions têtes**

Système d'élevage

Le mode d'élevage des bovins est caractérisé par 3 systèmes extensifs:
- l'élevage familial de petite dimension (entre 1 et 5 têtes) ;
- l'élevage semi-pastoral de moyenne dimension (taille moyenne de 30 têtes) ;
- l'élevage pastoral de grande dimension (plus de 30 têtes) ;

II- HISTORIQUE DE LA PPCB EN GUINEE

** 1936 Premier foyer enregistré dans les mines d 'or de Siguiri à l 'Est du Pays*
** 1945 - 1955 Extension à toute la partie Est du Pays (Haute Guinée et deux préfectures de la Guinée Forestière)*
** 1955 Mise en place du cordon sanitaire partageant le pays en deux grandes zones épidémiologiques (zone infectée à l 'Est et zone indemne à l'Ouest)*

III SITUATION EPIDEMIOLOGIQUE DE LA PPCB EN GUINEE

Années	Foyers	Sensibles	Cas	Morts	Abattus	Indemnisés
1995	50	22794	335	103	310	0
1996	30	11678	71	11	60	0
1997	36	2644	719	341	1718	0
1998	11	298	61	57	227	0
1999	6	171	24	19	150	34
2000	0	0	0	0	0	0
2001	1	123	42	30	93	0
2002	2	84	3	0	3	0
TOTAL	136	37492	1255	561	2561	34

IV - STRATEGIE NATIONALE DE CONTROLE DE LA PPCB

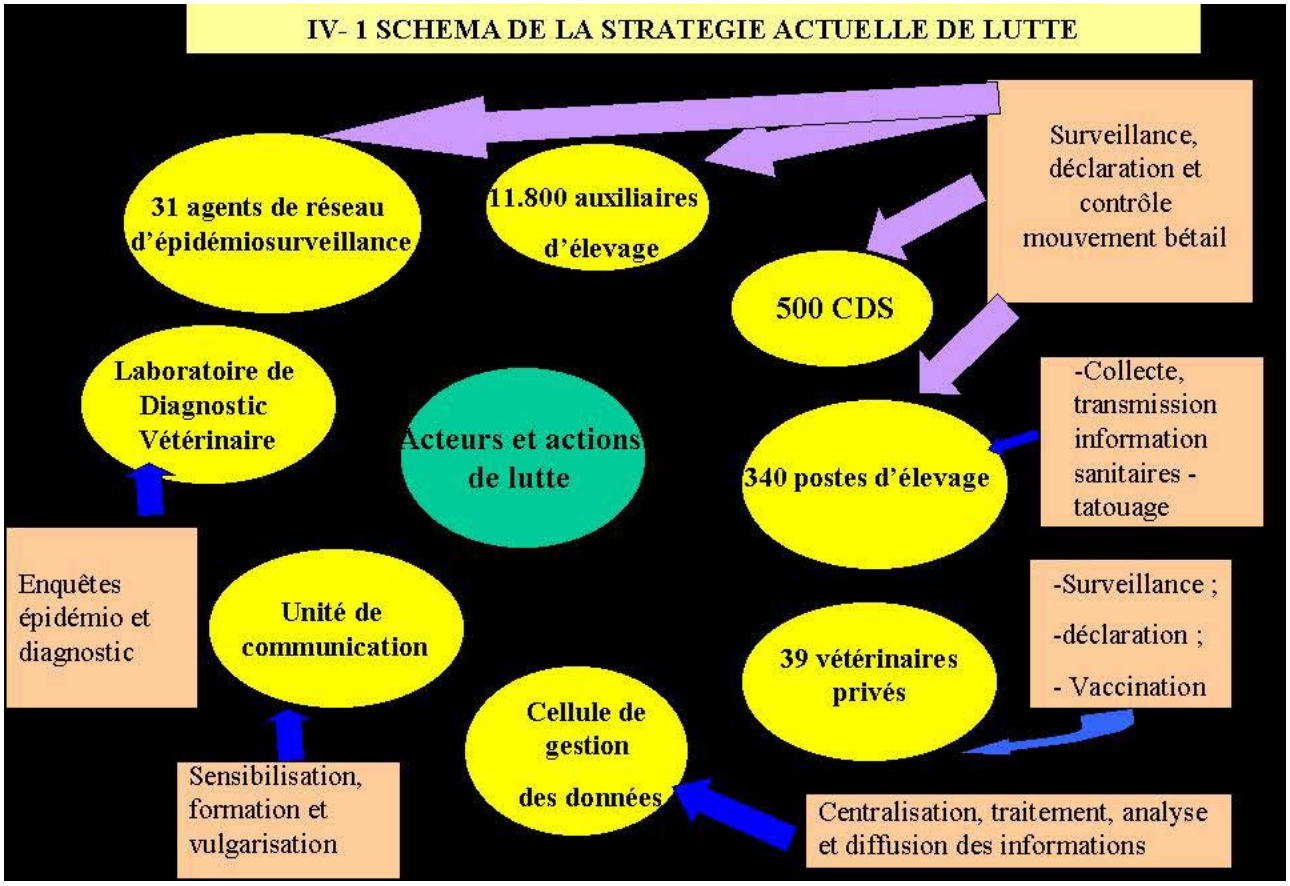
Trois périodes caractérisent l'évolution de la lutte contre la PPCB en Guinée :

 Avant 1987 : La lutte était essentiellement basée sur :

- la vaccination de masse avec le T1-44 ,

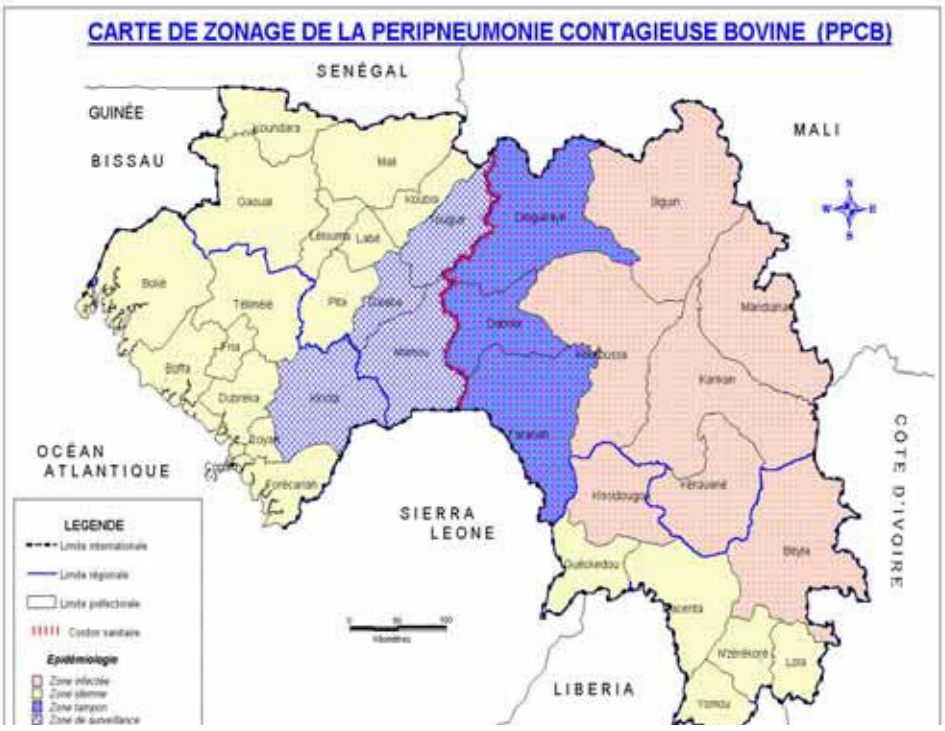
- la restriction du mouvement du bétail entre la zone infectée à l'Est(Haute Guinée et Région pré-forestière) de celle non in infectée à l'Ouest (Moyenne et Basse Guinée).**- l'abattage sanitaire** dans la zone non infectée à l'Ouest du pays

 1988 à 1994 : Cette période a été caractérisée par:
- la réalisation de campagnes de vaccination mieux planifiées ;
- le renforcement du contrôle du mouvement du bétail
- la réalisation d'enquêtes épidémiologiques pour mieux connaître la répartition de la maladie.
 de 1995 à nos jours : Cette période se caractérise par:
•la combinaison de toutes les mesures précédentes de lutte renforcées par la participation active des acteurs du réseau de santé animale;
•la délimitation de quatre zones épidémiologiques;
•la surveillance épidémiologique(clinique, sérologique et de lésions);
•la mise en place d'un cadre législatif et réglementaire;
•l'identification national des bovins et la communication - Formation.



IV - 2 - zonage épidémiologique du pays par rapport à la PPCB :

Sur le plan opérationnel, 4 zones distinctes ont été délimitées suivant leur situation épidémiologique



IV – 3 ACTIONS MISES EN ŒUVRE DANS LES DIFFERENTES ZONES :
□ Zone endémique : Elles portent sur :
□ la vaccination massive annuelle et obligatoire des troupeaux , exécutée par les Vétérinaires privés bénéficiaires de mandats sanitaires ;
□ le contrôle rigoureux du mouvement du bétail assuré par les Chefs de poste d 'élevage,les CDS, les éleveurs et les auxiliaires ;
□ l'interdiction des marchés à bétail de la zone aux animaux de la Basse et de la Moyenne Guinée ;

***** ***En cas d 'éclatement de foyer de la maladie , les mesures suivantes seront appliquées :***
□ le recensement et le cantonnement, de tous les animaux du foyer sous l'autorité de la SPRA ;
□ l'abattage sur place ou à l'abattoir le plus proche, des animaux malades sous contrôle vétérinaire ;
□ la vaccination en anneau et dans le foyer ;
□<u>Zone tampon :</u> Les activités portent sur:
□la surveillance active : clinique, sérologique et de lésions à l 'abattoir, avec les agents du réseau d'épidémio-surveillance, les vétérinaires inspecteurs des abattoirs et aires d 'abattage;

□le contrôle des mouvements des animaux qui mobilisent les agents des secteurs publics et privés, les CDS, les auxiliaires ;
□l'interdiction de déplacement des animaux vers les régions indemnes du pays, sauf ceux convoyés par véhicule adapté, vers les abattoirs sous contrôle vétérinaire ;

***** ***En cas d 'éclatement de foyer de la maladie , les mesures suivantes seront appliquées :***

□le recensement et le cantonnement, de tous les animaux du foyer sous l'autorité de la SPRA ;
□ l'abattage sur place ou à l'abattoir le plus proche, des animaux malades sous contrôle vétérinaire ;
□ la vaccination deux fois par an à six mois d 'intervalle en anneau et dans le foyer si la situation le commande;
<u>zone de surveillance</u> :

Des mesures drastiques y sont appliquées. Elles concernent :
□ la surveillance active et le contrôle des mouvements des animaux ;
En cas d'apparition de foyer ou de confirmation de la maladie par les analyses de laboratoire
□ le recensement et le cantonnement, de tous les animaux du foyer sous l'autorité de la SPRA ;
□ l'abattage systématique des troupeaux et l'indemnisation des éleveurs pour les veaux abattus;
□ la fermeture des marchés et l'interdiction de toutes manifestations impliquant le bétail (foires, fêtes de labour etc.) ;
□ études épidémiologiques approfondies sur les circonstances d'apparition de la maladie ;
□ la vaccination systématique en anneau du cheptel suivant le schéma proposé en 1970 par le groupe d'experts OUA/FAO/OIE.

<u>zone indemne :</u>

Mêmes mesures que dans la zone de surveillance, moins la vaccination

V- SUPPORTS DE LA STRATEGIE DE CONTRÔLE DE LA PPCB

La Communication -Formation:

La Radio-Télévision Nationale
Les Radios Rurales et Communautaires
Les Troupes Artistiques et Théâtrales
La vidéo mobile, les Affiches, les Réunions d'information et de sensibilisation
Les ateliers, les manuels etc……..

La législation :
l'application des mesures de police sanitaire autorisée par la loi portant Code de l'Élevage et des Produits Animaux;

Identification des animaux :
Le tatouage à l 'oreille d 'un numéro combinant des chiffres et des lettres selon un code préétabli.
Le marquage à l 'oreille avec la pince à trèfles dans les marchés à bétail de la zone endémique et dans les foyers actifs.

VI - APPUIS FINANCIERS

Différents projets ont été financés par le Gouvernement guinéen et ses Partenaires au développement, à savoir:
Projet FAO/GUI/78/012 de 1974 - 1981 a permis de réduire le nombre de foyer de 77 à 3;
Projet PRSE de 1988 - 1992, en a fait une de ses priorités.
Projet PARC - GUINEE de 1996 - 1999, qui s'est fixé comme objectif principal en Guinée la lutte contre la PPCB;
Projet PACE - GUINEE, démarré en 2000 en a fait aussi une de ses préoccupations
TCP - FAO/RAF/0172 (T) de 2001 - 2003 a renforcé la lutte contre la PPCB.

VII - ACQUIS DE LA LUTTE CONTRE LA PPCB EN GUINEE

VII - 1 L 'existence d 'un cadre législatif et réglementaire
la loi N°L/95/046/CTRN/ Portant Code de l'élevage et des produits animaux;
le décret N°D/97/217/PRG/SG inscrivant la PPCB sur la liste des maladies réputées contagieuses à déclaration obligatoire;
l'arrêté N° A/2003/5962/MAE/CAB Portant mesures spéciales de police sanitaire contre la PPCB.

VII - 2 L'adhésion de la majorité des éleveurs au programme de lutte contre la PPCB VII - 3 Le transfert de la vaccination aux vétérinaires privés par le biais du mandat sanitaireVII - 4 L'adhésion des éleveurs au programme de vaccination et la prise en charge des coûts en dépit des réactions poste-vaccinales

VII - 5 La participation des organisations d'éleveurs à l'application des mesures de police sanitaire (y compris l'abattage) et du contrôle du mouvement du bétail VII - 6 L'acceptation de l 'abattage sanitaire par les éleveurs
31O (1995); 60 (1996); 1718 (1997); 227 (1998); 150 (1999); 93 (2001) et 03 (2002)
VII - 7 La mise sous le contrôle des vétérinaires et des organisations d 'éleveurs des marchés à bétail
VII - 8 Le suivi permanent des mouvements commerciaux des bovinsVII - 9 L'adhésion de la majorité des éleveurs à la pratique du tatouage et son utilisation comme moyen de contrôle du

mouvement du bétail et de recherche en amont des foyers (beige en zone indemne et rouge en zone endémique)
VII - 10 déclaration de la maladie et la réduction significative du nombre de foyer

VII - 11 l'existence d'un système national de surveillance épidémiologique
En matière d'analyses sérologiques, pour la période de 1997 à 2002:
17 360 sérums appartenant à 728 troupeaux ont été analysés
27 Troupeaux ont été reconnus positifs soit 3,70%.

Dans les abattoirs :
sur 26 052 poumons examinés, 482 étaient porteurs de lésions soit 1,85%.

VIII - CONTRAINTES:

l'absence d'un programme sous - régional et régional pour lutter contre la maladie ;
l'inaccessibilité de certaines zones d'élevage en saison pluvieuse ;
les difficultés de rassembler les animaux pendant les opérations de vaccination et d'assainissement dues au système d'élevage;
l'insuffisance de la structuration du milieu éleveur rendant difficile l'organisation des opérations de lutte en certains endroits ;
et l'inexistence d'un mécanisme formel d'indemnisation des éleveurs pour reconstituer les troupeaux assainis;

IX- PERSPECTIVES :

organiser l'étude socio-économique de la maladie en vue de renforcer le programme de lutte et d'œuvrer pour une démarche régionale;
poursuivre et intensifier l'adhésion de tous les acteurs, en particulier les éleveurs au programme de lutte contre la PPCB dans le but ultime de l'appropriation par ces derniers des actions de lutte et de surveillance de la maladie;
poursuivre la vaccination massive obligatoire du cheptel, couplée avec le contrôle du mouvement du bétail, l'assainissement des troupeaux et la surveillance de la maladie, en attendant de nouvelles orientations scientifiques dans la lutte contre la maladie;
définir un mécanisme formel de financement des opérations d'assainissement des troupeaux associant les éleveurs et leurs organisations;
et renforcer le cadre juridique et réglementaire relatif à la lutte contre la maladie.

X - CONCLUSION

L'ensemble de ces résultats montre l'importance des efforts fournis par l'Etat et les partenaires au développement pour non seulement l'amélioration de la santé animale mais aussi le contrôle et l 'éradication de la PPCB en Guinée.Toutefois, les acquis sont encore fragiles quand on sait que la lutte contre les maladies animales transmissibles et la PPCB en particulier est une opération de longue haleine.

The main objective of the Consultative Group on Contagious Bovine Pleuropneumonia (CBPP) is to foster collaboration among the various stakeholders on CBPP disease management and provide the technical platform for discussions and policy direction for adaptive research relating to various aspects of CBPP disease. The Third Consultative Group meeting held in Rome, Italy from 12 to 14 November 2003, was attended by a wide array of CBPP experts from field veterinary services, diagnostic laboratories, policy-makers, international partner institutions, research and international reference laboratories and FAO Animal Production and Health staff. This report provides an account of the technical presentations made at the meeting, summaries of discussions held, and recommendations from participants. Progress made with diagnostic tests that could facilitate CBPP disease surveillance, research advances that may be useful in clarifying the epidemiology of the disease, aspects of CBPP vaccine development, as well as discussions on control options for CBPP, are reported in the proceedings.

ISBN 92-5-105166-6 ISSN 1810-0732

9 789251 051665

TS/M/Y5610E/1/07.04/1200