

NVRI SEMINAR SERIES 2009

This seminar series is a publication of seminar papers presented by staff and visiting scientists to the National Veterinary Research Institute, Vom during 2009

Compiled and edited by Dr (Mrs.) M. Muhammad and Dr. A. T. Oladokun (Secretary)

NVRI Seminar Committee 2009
Dr. (Mrs) M. Muhammad (Chairman)
Dr. M. O. Odugbo
Dr. (Mrs). L. Ta'ama
Mr. Canice Ugbong
Dr. (Mrs.) Y. Akalusi
Dr. A. H. Bakari
Dr. B. O Akanbi
Mrs C. O. Abolarin
Dr. (Mrs)C. I. Oditia
Mr. E. J. Okpala
Mrs. S. O. Ekundayo
Mr. P. Adeleye
Dr. A. T. Oladokun (Secretary)

National Veterinary Research institute, Vom
PMB 01, Vom
Website www.nvri.gov.ng
Email edvr@nvri.gov.ng or nvri1924@yahoo.com
Website:www.nvri.gov.ng

TABLE OF CONTENTS

Making Scientific Presentations.....	1
The use of Wild <i>Ganoderma Lucidum</i> in the Treatment of Caecal Coccidiosis in Chicken	6
Livestock Technology Development, Packaging and Dissemination.....	16
A Vision for Diagnostic and Experimental Pathology at National Veterinary Research Institute (NVRI), Vom, Plateau State, Nigeria	18
User Satisfaction With Library Resources and Services in Nigerian Agricultural Research Institutes.....	23
Current Management Strategies Against Ticks: A Contribution To Knowledge	25
Preliminary Investigation of Newcastle Disease and Highly Pathogenic Avian Influenza Viruses in Live Bird Markets in Ibadan, Nigeria.....	28
Phenotypic and Genotypic Characterization of <i>Listeria</i> Species Isolated in Parts of Plateau State, Nigeria.....	32
Adopted Village Concept and Livestock Technology Dissemination	39
Serological and Molecular Studies of <i>Mycoplasma mycoides mycoides</i> small colony (MmmSC) in Northern Nigeria	41
Vaccination and Comparison of the Potency of Newcastle Disease Vaccine Strains I₂ and Lasota in Rural Chickens in Plateau State, Nigeria	43
Sero-Epidemiology of Brucellosis in Small Ruminants in Plateau State	46
Evaluation of Efficacy of Aqueous Extract of <i>Senna occidentalis</i> in the Amelioration of Tetracycline-Induced Hepatotoxicity and Nephrotoxicity in Rabbits.....	49
Ameliorative Effects of <i>Phyllanthus niruri</i> Aqueous Extract on Hepatorenal Changes Following Sub-Acute Exposure to Acetaminophen in Rabbits	53
Assessment and Control of Moulds in Houses with Moisture Problem	60
Molecular Epidemiology of Dog Rabies in Nigeria:Phylogeny Based on N and G Gene Sequences.....	62
Distribution of <i>Campylobacter jejuni</i> in Abattoir Specimens in Jos and Environs.....	65
Evaluation of the Contribution of Major T Cell Subpopulations to IFN-γ Production in TB Infection by ELISPOT	68

Conducting Thematic and Diagnostic Surveys in Animal Health Research and Development.....	72
Antimicrobial Screening of Commercial Chicken Eggs In Plateau State	74
Guidelines for Presentation and Manuscript Preparation	76

INTRODUCTION

The 2009 series of seminars kicked off with a presentation on how to make a presentation with the aim of fully addressing the goals of seminars and to build confidence in participants' presentation skills. The seminars were sustained throughout the year with presentations from research staff, visiting scientists and administration. Seminars from administration were aimed mainly to acquaint staff of some new government policies but have not been included in this series. Seminars presented by interns on the toxicology programme jointly funded by the Africa Education Foundation and NVRI are also included in the series.

¹MAKING SCIENTIFIC PRESENTATIONS

M. Muhammad

Bacterial Research Division

National Veterinary Research Institute, Vom

E-mail: lawal54@yahoo.com or maryam.muhammad@nvri.gov.ng

Introduction

Presentations at seminars and other scientific meetings are extremely important to scientific communication and to an individual's reputation. Scientists build on the work of others through communication. Seminars can provide information on research and for both the listener and speaker; seminars are a unique educational opportunity for broadening one's education. When you prepare and present a seminar, your perception of your own study increases, thereby improving the quality of your research. Seminars can also provide a means for uncovering errors, gaining new perspective and strengthening research. Seminars also help to improve communication skills. We attempt to achieve excellence in written presentations for journal publication; we should achieve no less at seminars. Whether an orator or a novice speaker this presentation is intended to improve your oral presentation skills through Planning, Preparation, and Performance.

Preparing a Presentation

Begin with defining your purpose. What do you want to communicate? What will your presentation achieve? Focus on the purpose of presentation at every stage to ensure relevance and efficiency. With the audience foremost on your mind, consider your purpose and subject. Consider the type of message, nature of audience and venue. Presentations should be relevant, simple and to the point. Material should be clear, precise, and easy to follow. Group similar ideas together to establish a theme. Ensure that the audience leaves room better informed. With small groups, a two-way communication is possible. With large groups, ensure that everyone can hear you, link facts, sum up, repeat points, and speak slowly. Presentations require preparation, research and hard work- **take time to prepare**. For every hour of presentation set aside ten hours for preparation.

Knowing Your Audience

Find out details of expected size of audience, standard of knowledge of subject matter, purpose and be adaptable. In a professional presentation, keep in mind that you are talking to those who know you best professionally. If presentation is formal and the audience is small, establish eye contact with all members of the group. If the audience is large, ensure that everyone can hear you clearly especially at the back and repeat main points.

Dealing with Logistics

Visit venue in advance to become familiar with its layout. If organising a group presentation discuss beforehand the order of speaking and group speakers with similar ideas together. Know your venue including the positioning of audio visuals and light switches well in advance. Ensure that you know how the public address system works. If there are immovable features in the room that could obstruct audience's view, plan seating around these

¹ Seminar presented 5th February 2009 at NVRI auditorium

Clarifying Objectives

Before preparing a presentation it is important to think about objectives e.g. to pass on vital information, present progress report of research, present a research proposal or results of research. It is important to know how your research was planned, designed, and carried out including how data were analysed and how your results compare with those of others who have done similar research. Be logically consistent and adopt a well-structured approach to the subject matter.

Use your knowledge effectively and authoritatively. Limit yourself to three or four main points and ensure that you deliver the main concepts of your presentation clearly. A research report must be centred on the research question or hypothesis around which the objectives are woven. A logical pattern and progression in arranging ideas is essential to scientific communication. Almost any scientific report can use the following generic guideline:

1. Introduction
 - a. The scientific topic and background or literature review
 - b. Justification
 - c. Objectives of study
2. Materials and Methods
 - a. Materials and location of experiment
 - b. Procedures and processes
 - c. Data collection and analysis
 - d. Statistical evaluations
3. Results and Discussion
 - a. Synopsis of results
 - b. Presentation of data in tables, figures and supporting text
 - c. Discussion of significance of study, application, and relationship to other studies.
4. Conclusions

Using Your Knowledge

The main objective of making an oral presentation is to provide information and to capture and hold attention. You must show a passion for the subject and deliver the main concepts of the presentation clearly. Always summarise main points in one sentence. Every presentation has three essential objectives: to educate, to entertain and to explain.

Putting Material Together for an Oral Presentation

A successful presentation always begin with careful background research. Research requires initiative and hard work and can be time consuming. Allocate sufficient time for research before presentation. Always keep main objective in mind while writing the presentation. Make the audience feel that you are giving new information. Do not ignore a good source just because the information is not readily available or accessible to you.

Selecting Key Points

Most adults have an attention span of 45 minutes; limit yourself to key points and emphasise main points at the beginning, middle, and end of the presentation. Audiences are usually more open when they have a clear idea of the subject of presentation so it is important to structure material to suit the objective of the presentation.

Structuring Material

The order in which you present the main points of your presentation will affect the message that the audience takes away. Use the most appropriate structure to give the audience the right message. Decide how many points you intend to make and match the presentation structure to the material. Present the points in a sequence that suits the particular subject. In scientific communication, scientists are more likely to organise material in terms of introduction, materials and methods, results and discussion. Let your presentation have a recognisable beginning, middle and end. Always prepare a written outline of your presentation as an outline helps clarify the structure of presentation and to jog your memory. Keep outline simple so they are easy to read at a glance.

Writing the Presentation

Written material can sound very different when it is delivered to an audience in spoken form. Learn to write your prose in a natural oral style and be particular about what you include in your presentation. Do not fill speech with irrelevant points. You do not have to write out your whole speech so as not to overwhelm the audience with too much detail.

Using Audio-Visual Aids (AVs)

Used effectively, AVs can be central to a presentation as they can be used to illustrate difficult concepts and strengthen the scientific message. AVs include handouts, slide projectors, video, audio, flip chart, multimedia, and computer graphics. In scientific communication, the AV is used to clarify concepts beyond what the written or spoken word alone can do. Visual aids also help to compensate for language barriers especially where accents differ. However, although slides contribute to the success of an oral presentation, it is better to have no visual than to have a poor visual aid. Different AVs suit different sizes of audiences. It is always preferable to allow time to prepare AVs. If you cannot prepare your own slides, enlist the help of a knowledgeable person, always rehearse your presentation using your chosen AV. Use bold colours, keep design elements simple, and use a laser pointer to indicate items on a screen. Make a trial run through of slides before presentation to enable you change if your chosen aid does not work properly. Coordinate slides properly with text and speech so that the audience is not distracted, ensure that the visual is relevant to the point being made, and **slow down** when presenting AVs. Generally, slides should be prepared for presentation in a large room; they can then be used for a small room as well. For Microsoft Power Point® presentations, the text on the slide should be between 32 and 40 point size and no text should be smaller than 28. Each slide should make a basic point with one or two sub points.

For a slide presentation on research, your data are the most important support to your objectives and conclusion. Present only data that are essential to the presentation. It must be recognised that the number of data points in tables and figures have to be reduced in order to be able to present representative data. This will enable verbal discussion of the data and make it visible to the whole audience. **Never expect to simply transfer tables and figures from a publication to a slide without some editing.**

The typical contents of a slide presentation should be

Speech Content	Slide Content
Title	Title and authors
Introduction	Full statement Key words or pictures
i. Hypothesis	
ii. Justification	
iii. Literature	Ideas and references
iv. Objectives	Full statements
Methods	
i. Equipment	Photos or illustrations
ii. Sampling and technique	Lists or flow charts
iii. Methods of analysis	Statement or key words
Results and discussion	
i. Objectives accomplished	Statement and pictures
ii. Data	Tables, figures, key words, photographs
Conclusions	
i. Main outcomes	Full statements
ii. Accuracy of hypothesis	Statement or photograph
iii. Application of results	List or picture

From: Davis (2005)

Paramount to making your slides to take central position as an aid is to make sure that they are well organised and carefully coordinated with your speech. A title slide, an objective slide, and a conclusion slide form the foundations around which other slides and the whole presentation should be organised.

Rehearsing the Presentation

Rehearsal is a vital part of preparing for a successful presentation. It is an ideal opportunity to memorise your material and smooth-over any rough edges. Rehearsals should rely less and less on script each time it is done.

Preparing Your Self

It is as important to prepare yourself, as it is to prepare the presentation. The overall impact of the presentation will be determined as much by how you appear as by what you say. A positive image is important for delivering a positive presentation, so behave normally and the audience will warm to you. Do not wear anything that may distract the audience. The tone and volume of your voice have a critical effect on a presentation it is therefore important that you speak and act naturally.

Delivering the Presentation

Only highly experienced speakers will not feel nervous before making a presentation. Nervousness can prevent you from being natural so you need to do everything to control your nerves. The key to a good delivery is to be yourself by acting and sounding natural. Prepare thoroughly as this is the major key to a successful delivery. Use a checklist each time you prepare a presentation. To make a strong effective presentation, relax before hand, and get a good night's sleep. Only smile if it feels natural to do so. The delivery is as important as the message itself. Start strongly by speaking confidently and use eye contact to establish a rapport with your audience. Make initial eye contact with someone approachable but learn to develop your own style and do not read your slides to your audience. Only the title and conclusions should be read verbatim. Open effectively by appearing confident and positive (you must be well prepared). However, save the strongest points for later as most audiences are not alert at the beginning of a presentation. Plan a logical flow to ideas and themes to help the audience follow the presentation easily. Sum up each theme before introducing a new one.

Pacing the Speech

Use your natural voice, pace your speech and use body language to enhance the understanding of what you have to say. Keep to allocated time. A good presentation is characterised by good timing. Both the silent parts of a speech and the spoken parts are important in communicating content. If you are given thirty minutes for a presentation, then prepare for 25 minutes and if given 15 minutes, prepare a 12-minute talk.

Closing Effectively

It is vital to have a strong conclusion to your presentation as it forms the basis of what your audience takes away. Reiterate major points in a summarised form and always close with a good summary. Encapsulate presentation in a few powerful sentences to emphasise key points. Concentrate on presenting accurate, well-researched facts and base conclusion firmly on the facts presented and do not leave AVs on for too long after the presentation.

Handling an Audience

A presentation is made for the benefit of the audience so learn to read the audience's reaction and adjust your presentation to suit the mood of the audience.

Dealing with Questions

In any formal presentation, interaction with the audience is crucial for success. Question and Answer (Q & A) sessions allow the audience to clarify points, add to knowledge of the subject matter, and establish the presenter's reputation as a scientist. It is important to go into your presentation fully prepared to answer any questions. The key to this is careful research and writing. Invite questions during rehearsal. Question-and-answer sessions are as important as the presentation itself. A good presentation can be ruined by a poor question-and-answer session and a mediocre presentation can be redeemed by a confident Q and A session. Answering questions can increase your credibility by demonstrating a wider knowledge of your subject. All questions must be answered with respect and courtesy and all answers addressed to the whole audience not just the questioner. Follow formal procedures when presenting at a formal gathering.

The Role of the Moderator

A moderator serves to chair a presentation and helps both presenter and audience feel comfortable. Moderators should have information to be able to introduce the speaker properly and coordinate efforts related to time, requests for questions, and lights. He/she should obtain the full names, title, qualifications, background, institutional affiliations, and any distinctions of the presenter and the title of the presentation. If there is more than one presentation, the moderator provides the transition from one presenter to the next and ensures that every speaker keeps to the time schedule. A moderator should obtain abstracts of the presentation and prepare a few relevant questions for each speaker in order to get the discussion started if the audience does not. A moderator should be sensitive to any problems the speaker may have, coordinate all efforts with the speaker, buffer him or her from a hostile questioning environment, and discharge all responsibilities in a professional manner.

Bibliography

1. Davis M. (2005). *Scientific Papers and Presentations*. Second Edition. Elsevier Academic Press, Massachusetts USA.
2. Hellar R. and Hindle T. (1998). *Presenting Successfully*. Tombesi-Walton D (Ed). In: *Essential Manager's Manual*. Dorling Kindersley, UK.
3. www.oregonstate.edu/instruction/bb311/discussion4.html

²THE USE OF WILD *GANODERMA LUCIDUM* IN THE TREATMENT OF CAECAL COCCIDIOSIS IN CHICKEN

A.O. Ogbe

Federal College of Animal Health and Production Technology, National Veterinary Research Institute (NVRI), Vom

Email: ogbeadamu@yahoo.com

Introduction

Ganoderma lucidum basidiomycetes (mushrooms) are the fruiting bodies of fungi in the family *Polyporaceae* which grows on wood logs and tree stumps. In tropical Africa including Nigeria wild *Ganoderma* mushrooms grow in abundance during the rainy season every year. In Asia, this mushroom is cultivated artificially for use as source of feed supplements and medicines (Anke, 1989; Anon, 2006). The biologically active compounds in the mushroom are known to have pharmacological effects against microbes, parasites and insects and also enhance wound healing (Teow, 1986; Hobbs, 1995; Anon, 2007). The objectives of this study was to determine if the aqueous extract of wild *Ganoderma* sp. harvested from Vom could reduce *Eimeria tenella* (parasite) oocysts output in infected chickens and improve their live body weight gain.

Methodology

One hundred and twenty day-old Ross broilers were obtained from a hatchery in Jos. The birds were selected for uniformity and fitness and randomly distributed into six treatment groups of 20 chicks each and housed on wire cages, each measuring 80 x 100 cm and labelled A-F. They were fed with broiler starter feed containing 22% crude protein (CP) *ad libitum*. Groups A, B and C were inoculated with *E. tenella* (Houghton strain) at the rate of 36,250 sporulated oocysts/ml/bird using insulin syringe introduced directly into the crop of each bird at 6 weeks of age. By day 6 post-inoculation (PI) birds in group A were treated with 1:5% w/v of aqueous extract of *Ganoderma lucidum*. B with 200 mg/ml of Amprolium® in drinking water. Each treatment was for 7 days consecutively *ad libitum*. Group C and D were not treated and D, E and F were not inoculated. E and F were treated with *Ganoderma* extract and Amprolium respectively. Clinical signs and post-mortem lesions were observed in seven sacrificed birds per group. Faecal samples were also collected from each group daily for parasitological examination to determine *E. tenella* oocysts output per gram faeces using McMaster counting chamber (Long and Powell, 1958). Feed: gain ratio per group was also determined according to the method of Conway *et al.*, (1993).

Results and Discussion

The results showed that all the birds that were inoculated with *E. tenella*, group A, B and C appeared dull and weak with reduced appetite. The morbidity was high (100%) on day 4 post-inoculation (PI). By day 5 PI their faeces became bloody and watery (Table 1). The birds that were not inoculated with *E. tenella* were normal (Groups D, E and F). In addition, there was slight drop in feed intake by birds in groups A, B and C from 1.6 kg each at 7 weeks of age to 1.5 kg at 8 weeks of age, with a further drop in group C, 1.48 kg (Table 1). This was followed by a compensatory increase in feed intake after treatment for 7 days in groups A and B (1.8 kg each) at 9 weeks of age. The feed intake in group C was lower (1.6 kg) and the feed: weight gain ratio was significantly higher, 3.8 ($P < 0.05$). Mean final weight

² Seminar presented on 19th February 2009 at NVRI auditorium

(gain) in C was also significantly lower, 2.5 kg/bird ($P < 0.05$), when compared with other groups (Table 1). In all the groups there was no mortality of birds. However, prior to treatment of the birds the oocysts output were 30,000 oocysts/gram faeces (group A), 28,000 oocysts/gram faeces (group B), and 25,000 oocysts/gram faeces (group C) (Figure 1). The faeces of birds that were not infected (group D, E and F) were free of coccidial oocysts. On day 3 post-treatment (PT) the oocysts detected in birds treated with *Ganoderma* extract (group A) had reduced significantly (2,500 oocysts/gram faeces), compared to the Amprolium treated group, B (5,900 oocysts/gram faeces). Group C showed a significant increase in oocysts count (32,600 oocysts/gram faeces). By day 7 PT, birds in group A and B were negative for oocysts, while group C continued to discharge *E. tenella* oocysts up to day 17 PI. The uninfected controls (group D, E and F) did not shed or pass any oocyst during the entire period of the experiment. Grossly, group A and B had 4 birds out of 7 with haemorrhagic caeca (57.14%). In addition, 2 had rough caecal mucosa and *E. tenella* was observed in 1 (14.29%). In group C, 7 birds (100%) had haemorrhagic caeca and *E. tenella* oocysts. The caecal mucosae were also rough in 6 (85.7%) and ballooned in 3 (42.9%). These lesions were absent in birds that were not infected with *E. tenella* (group D, E and F). There was a successful infection of broilers with *E. tenella* (Houghton strain) and all the infected birds showed clinical signs. There was significant reduction ($P < 0.05$) in faecal oocysts output in treated birds using either *Ganoderma* extract or Amprolium. *Ganoderma* extract like Amprolium has anticoccidial activity against *E. tenella*; so in the absence of further development of the parasites there was improved feed intake and weight gain. The feed: weight gain ratio of the treated birds was also better than those not treated, because of the depressive effect on feed intake by *E. tenella*. Conway *et al.*, (1993) earlier reported high feed: gain ratio of 1.61 in broilers that were infected with *E. tenella* and this was able to produce significant reduction in body weight due to the depressive effect of *E. tenella* on feed intake. However, this may be followed by a period of compensatory feed intake and growth, lasting up to about 14 days in which the birds will partly catch up with their losses due to the infection (Braem and Suls, 1992). Hughes *et al.*, (1958) and Guo *et al.*, (2003) reported that bioactive compounds or polysaccharides in mushroom played vital roles in enhancing health. They block colonization of intestine by pathogens thereby enhancing their removal. The wild *Ganoderma* mushroom used in this study was found in previous studies to contain bioactive compounds (Ogbe, 2008). However, the mechanism of action of these biologically active compounds was not elucidated. It was concluded that future work be done in this area.

Table 1: Clinical observations and performance of broilers infected with *Eimeria tenella* and treated with aqueous extract of wild *Ganoderma lucidum*.

Parameters	Group/Clinical observations and performance of broilers					
	A	B	C	D	E	F
1. Observations						
i. Temperament	Dull	Dull	Dull	Alert	Alert	Alert
ii. Morbidity	7(100)	7(100)	7(100)	0(0)	0(0)	0(0)
iii. Mortality	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
iv. Faeces	bloody	bloody	bloody	normal	normal	normal
2. Performance of broilers						
a. mean feed intake pre-treatment at 7 weeks of age (kg)	1.63	1.62	1.61	1.65	1.68	1.69
b. mean feed intake 3 days after treatment at 8 weeks of age (kg)	1.52	1.57	1.48	1.63	1.76	1.79
c. mean feed intake 7 days after treatment at 9 weeks of age (kg)	1.81	1.80	1.62	1.71	1.74	1.79
d. mean final weight gain (kg)	3.12(2.91)	3.09(2.83)	2.75(2.54)	3.06(2.85)	3.26(3.05)	3.20(2.99)
e. Feed: weight gain ratio	3.41	3.50	3.80*	3.44	3.30	3.40

*indicate the value is significantly different ($P < 0.05$) along the same horizontal column. A = infected/treated with *Ganoderma*, B = infected/treated with Amprolium, C = infected/not treated, D = not infected/not treated, E = not infected/treated with *Ganoderma*, F = not infected/treated with Amprolium.

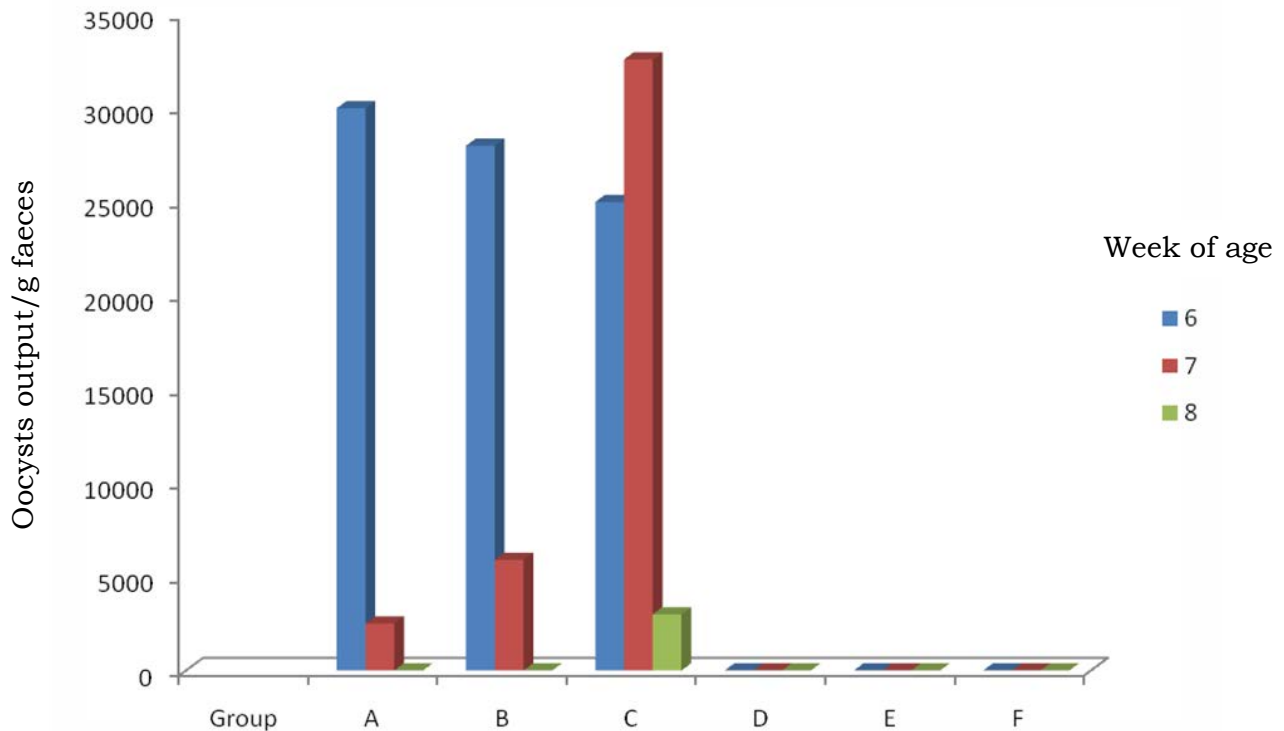


Figure 1: Faecal oocysts output of broilers infected with *Eimeria tenella* and treated with aqueous extract of wild *Ganoderma lucidum*.

References

1. Anke, T. (1989): Basidiomycetes: a source of new bioactive secondary metabolites. *Progress in Industry Microbiology*, 27:51-66.
2. Anonymous (2006): Cultivation, utilization and medicinal effects of *Ganoderma lucidum* in Malaysia. File:///:/reishi97d-9.htm, 30/08/2006; Time: 14:00.
3. Anonymous (2007): Mushrooms may be active against fowl parasite. *Thisday*, Vol. 11, Number 4280, January 9, 2007; Page 36.
4. Braem, G. and Suls, L. (1992): A strategic approach to coccidiosis prevention. *Poultry International*, 12-18.
5. Conway, D.P; Sasai, K; Gaafar, S.M. and Smothers, C.D. (1993): Effects of different levels of oocysts inocula of *Eimeria acervulina*, *E. Ganoderma* and *E. maxima* on plasma constituents, packed cell volume, lesion scores and performance in chickens. *Avian Diseases*, 37: 118-123.
6. Guo, F.C; Savelkoul, H.F.J; Kwakkel, R.P; Williams, B.A. and Verstegen, M.W.A. (2003): Immunoactive, medicinal properties of mushroom and herb polysaccharides and their potential use in chicken diets. *World's Poultry Science Journal*, 59: 427-440.
7. Hobbs, C. (1995): *Medicinal Mushrooms: An exploration of tradition, healing and culture*. Santa Cruz, C.A: Botanica Press, Pp. 96-251.
8. Hughes, D.H; Lynch, D.L. and Somers, G.F. (1958): Chromatographic identification of the amino acids and carbohydrates in cultivated mushroom. *Journal of Agriculture and Food Chemistry*, 6: 850-853.
9. Long, P.L. and Powell, J.G. (1958): Counting oocysts of chicken coccidia. *Laboratory Practice*, 7: 515.
10. Oei, P. (2003): Mushroom cultivation, In: *Appropriate Technology for the Mushroom Grower*; CTA, 3rd Edition, Backhuys Publishers, Leiden. The Netherlands, Pp. 1-7.
11. Ogbe, A.O. (2008): The use of *Ganoderma lucidum* in improvement of antibody response to infectious bursal disease vaccination and treatment of caecal coccidiosis in chickens. PhD Dissertation. Department of Veterinary Surgery and Medicine, Ahmadu Bello University, Zaria, Nigeria, Pp. 73-97.
12. Teow, S.S. (1986): Cultivation of *Ganoderma lucidum* and its medicinal value. Extended Abstract, 9th Malaysian Microbiology Symposium, Pp. 79-82.
13. Wasser, S.P. (2002): Medicinal mushrooms, as source of antitumor and immune- modulating polysaccharides. *Applied Microbiology and Biotechnology*, 6 (3): 258-274.

³SERO-EPIDEMIOLOGY OF AFRICAN SWINE FEVER IN NIGERIA, 2006-2008

Fasina, F. O.; Adedeji, A. J.; Yakubu, M. B.; Mantip, S.; Benschak, A. J.; Okeke, I.; Anagor, P.; Mandeng, D. C.; Akanbi, B. O.; Lazarus, D. D.; Rufai, S. A.; Adamu, S. S. and Makinde, A. A.

African Swine Fever Task Team, National Veterinary Research Institute, Vom

Introduction

African swine fever (ASF) is a highly lethal haemorrhagic disease of domestic pigs that may result in up to 100% mortality (Penrith, Thomson and Bastos, 2005). It manifests in per-acute, acute, sub-acute or chronic forms and was originally described in East Africa by Montgomery in 1921. Initial sporadic infections were reported in Cape Verde (1960), Nigeria (1973), Senegal (1978) and Cameroon (1982), and were eradicated. The disease re-emerged and became endemic in the West African sub-region between 1996 and 2007. It appears that following outbreaks in the Abidjan area of Cote d'Ivoire around April 1996, the disease spread to Benin and then Togo in 1997. Nigeria experienced outbreaks before the end of 1997 and this infection was linked to pig and pig product movement especially in the border areas between Nigeria and Benin Republic in the Ogun-Lagos area. The likely routes of introduction and spread of the 1997-1999 ASF outbreaks in Nigeria is depicted in Figure 1. The disease impacts huge economic costs and estimates put losses due to ASF at \$18 million in Cote d'Ivoire (FAO, 2002), \$3.5 million in Nigeria (El Hicheri, 1998), \$18 million in Benin and \$3.5 million in Cape Verde (FAO, 2002). Even countries that are not affected like the USA spend about \$3.5 million annually.

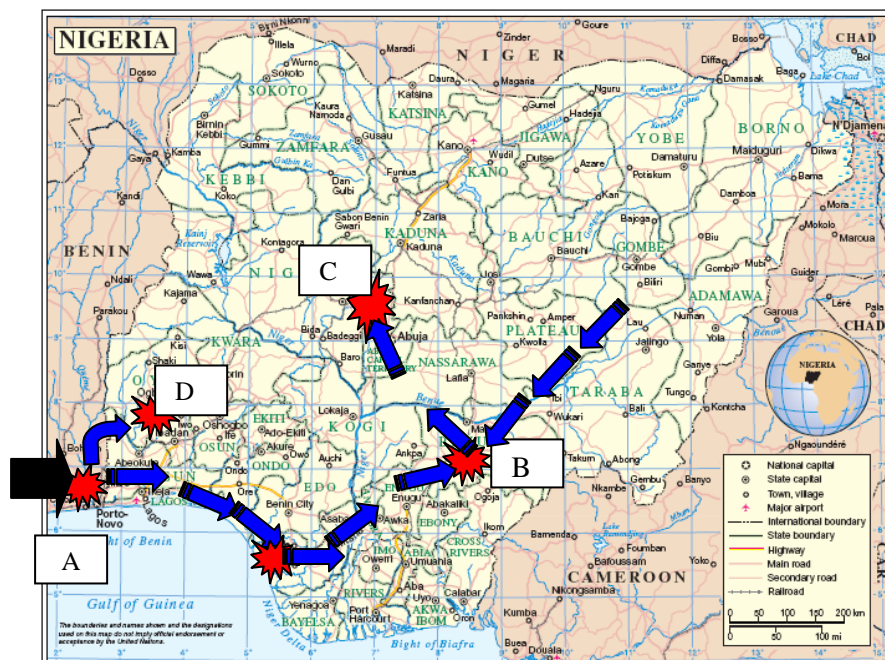


Figure 1: Probable spread of ASF within Nigeria 1997-1998 and 1999-date based on available data

Legend:

Initial Entry point



Pattern of Pig trade movement



Major outbreak locations



³ Seminar presented on 5th March 2009 at NVRI auditorium

Methodology

Key pig producing, consuming and marketing areas within each agro ecological zone of Nigeria were selected. Surveillance visits were conducted and samples collected at farms, abattoirs and markets. One thousand and ninety two (1092) sera samples were collected between October 2006 and December 2008 from 16 States. The samples were transported on wet ice and stored at -20°C until tested. The sera were tested by African Swine Fever indirect enzyme-linked immunosorbent assay (iELISA) using a commercial test kit produced by the Laboratoire National de l'Élevage et de Recherches Veterinaires, Senegal, using the protocol provided. Doubtful results were confirmed using immunoblotting assay. The prevalence of ASF was assessed by evaluating the number of positive sera against the total number of sera tested for the group. All values were subjected to statistical analyses using StatGraphics Plus®. Frequency cross-tabulations of derived values were done to determine the true prevalence of the samples. Confidence intervals, standard deviations and P-values were determined according to standard procedure. Normality of the sample distribution was determined by Standard skewness and Standard kurtosis.

Results

Results are presented in Tables 1 and 2. The national sero-prevalence of ASF between 2006 and 2008 was 21.34% ($X^2 = 40.21$; P-value = 0.0000; DF = 5). Regional prevalences were; North-Central 23%; North-East 35%; North-West 7%; South-East 6%; South-South 15% and South-West 26%. The Farms accounted for 58.52% of all sera collected while the abattoirs/markets accounted for 41.48%. Seroprevalence of antibodies were 19% on farms and 25% in abattoirs and markets ($X^2 6.47$; P-value 0.0110; DF 1) with Yates' correction. The sample distributions were normal according to values recorded for Standard skewness (1.79404) and Standard kurtosis (1.78146). Adamawa, Lagos and Plateau, had the highest seroprevalence of 71%, 55% and 28%, respectively.

Discussion

Our results indicate that ASF is still prevalent in Nigeria. Discussions with stakeholders in the field indicated that pigs and pig products are moved from farms to markets and abattoirs without prior testing. Furthermore, farmers indicated a preference of disposing of sick, off-feed and unthrifty animals first before presenting healthy ones for sale. This increases the chances of spreading infection and is probably responsible for the relatively higher prevalence rate recorded in the abattoir and market. Regional sero-prevalence varied and was highest in the regions with large pig markets and pig-related activities. It is well known that once ASF is established in swine populations, infected animals become the most important sources of spread of the virus. On location basis, Numan, Oke Aro, and Lagos had the highest sero-prevalence to ASF. There is currently no vaccine to control ASF. biosecurity at farm level, quarantine and other hygiene measures are the most effective means of controlling ASF for the near future. Government should pay compensation to farmers who lose their herds to ASF, as this will encourage them to report outbreaks and assist in the control and eradication of the disease.

Table 1: National Seroprevalence of ASF by Agro Ecological Zone.

Zone	Sample Per Zone	Samples Positive	Samples Negative
North-central	526 (48.17)	123 (11.26)	403 (36.90)
North-east	128 (11.72)	45 (4.12)	83 (7.60)
North-west	74 (6.78)	5 (0.46)	69 (6.32)
South-east	68 (6.23)	4 (0.37)	64 (5.86)
South-south	195 (17.86)	30 (2.75)	165 (15.11)
South-west	101 (9.25)	26 (2.38)	75 (6.87)
Total	1092 (100)	233 (21.34)	859 (78.66)

Table 2: National Seroprevalence of ASF by Pig Location

	Total (%)	Positive samples (%)	Negative samples (%)
Abattoirs/Markets	453 (41.48)	114 (25)	338
Farms	639 (58.52)	119 (19)	519
	1092 (100)	233 (21.34)	857 (78.48)

References

1. Awa D.N., Njoya A., Tama A.C.N. & Ekue F.N. (1999). The health status of pigs in North Cameroon. *Rev. Elev. Méd. Vét. Pays trop.*, 52 (2), 93-98.
2. Bastos A.D.S, Penrith M.-L., Cruciere C., Edrich J.L., Hutchings G., Roger F., Couacy-Hyman E. & Thomson G.R. (2003). – Genotyping field strains of African swine fever virus by partial p72 gene characterisation. *Arch. Virol.*, 148 (4), 693-706.
3. El-Hicheri K., Gomez-Tejedor C., Penrith M.-L., Davies G., Douati A., Edoukou G.J. & Wojciechowski W. (1998). – L'épizootie de peste porcine africaine de 1996 en Côte d'Ivoire. *Rev. sci. tech. Off. int. Epiz.*, 17 (3), 660-673.
4. El-Hicheri K., (1998). Emergency Assistance on control and eradication of an outbreak of African swine fever in Western Nigeria. Report of the FAO Consultancy Mission to Nigeria. FAO, Rome, 1998.
5. Food and Agriculture Organization of the United Nations. (2000). Recognizing African swine fever: A field manual. FAO Animal Health Manual No.9, FAO, Rome.
6. Gonzague M., Roger F., Bastos A., Burger C., Randriamparany T., Smondack S. & Cruciere C. (2001). – Isolation of a non-haemadsorbing, non-cytopathic strain of African swine fever virus in Madagascar. *Epidemiol. Infect.*, 126 (3), 453-459.
7. Haresnape J.M., Lungu S.A.M. & Mamu F.D. (1985). – A four-year survey of African swine fever in Malawi. *J. Hyg. (Camb.)*, 95 (2), 309-323.
8. Nsalambi D. (1987). – Particularidades da epizootiologia, do quadro clinico e da patomorfologia da peste suína africana na República Popular de Angola. Doctoral thesis, Academy of Sciences of Ukraine, Kiev.
9. Odemuyiwa S.O., Adebayo I.A., Ammerlaan W., Ajuwape A.T.P., Alaka O.O., Oyedele O.I., Soyelu K.O., Olaleye D.O., Otesile E.B. & Muller C.P. (2000). – An outbreak of African swine fever in Nigeria: virus isolation and molecular characterization of the VP72 gene of a first isolate from West Africa. *Virus Genes*, 20 (2), 139-142.
10. Penrith, M. L., Thomson, G. R. & Bastos, A. D. S. (2005) African swine fever. In: Coetzer JAW, Tustin RC (eds) *Infectious diseases of Livestock*. Oxford University Press, Southern Africa, pp 1087–1119.
11. Saliki, J. T. (1988) ASF: An overview. In: *Viral Diseases of Animals in Africa*. A. O. Williams and W. N. Masiga (eds). OAU/STRC & CTA of ACP/EEC. 1988. Pp 219-234.
12. Sanchez-Botija, C. (1982). – La peste porcine africaine: nouveaux développements. *Rev. sci. tech. Off. int. Epiz.*, 1 (4), 1031-1064.
13. Wilkinson P.J., Wardley R.C. & Williams S.M. (1983). – Studies in pigs infected with African swine fever virus (Malta/78). In *African swine fever* (P.J. Wilkinson, ed.). EUR. 8466 EN. Commission of the European Communities, Luxembourg, 74-84.

4FIELD OUTBREAKS OF PPR IN SHEEP & GOATS AND MOLECULAR CHARACTERIZATION OF SOME STRAINS IN NIGERIA

Kazeem, H. M. [FCVSN]

Viral Research Division,

National Veterinary Research Institute, Vom (Current Address)

E-Mail: haruna_kazeem@yahoo.com

Introduction

Peste des petits ruminants (PPR), also known as goat plague, kata or stomatitis-pneumoenteritis complex is a highly contagious viral disease of goats and sheep. It is caused by a paramyxovirus of the Genus *Morbillivirus* in the Family *Paramyxoviridae*. It was first described in 1942 in Cote d'Ivoire. Reports of PPR have been documented in the Middle East and India (Dhar 2002). It is closely related to rinderpest, canine distemper, human measles, Phocine (seal) distemper viruses, Dolphin *morbillivirus*, Porpoise *morbillivirus* and Cetacean *morbillivirus*. The six structural proteins of the virus include Nucleocapsid protein (N), Phosphoprotein (P), Matrix protein (M), Fusion protein (F), Hemagglutinin-Neuramidase (HN) and Large protein (L) (OIE 2002). Several strains of PPR have been documented across Africa where the outbreaks have been reported (Saliki, 1998; Taylor *et al.* 2002). In Nigeria, strains of PPR virus obtained from sheep and goats include Plumb Island, Eruwa, Nig.75/1, Nig.75/2, Nig.75/3 and Nig.76/1. Since 1976, no new strains have been isolated in Nigeria. Genetic characterization of Indian PPR viruses by sequencing and phylogenetic analysis of nucleoprotein gene segments yield three distinct clusters for the Nigerian, Turkey and Indian isolates. Classification of PPR virus into lineages based on the N gene sequences appeared to yield better picture of molecular epidemiology for PPRV (Kerur *et al.*, 2007). The objective of this study was to collect samples from natural cases of PPR in different ecological zones of Nigeria for characterization and to compare them to the existing strains in Nigeria.

Methodology

Samples including lungs, spleen and mesenteric lymph nodes of 17 suspected cases from natural outbreaks of PPR in goats and sheep were obtained from Akwa Ibom, Bauchi, Edo, Enugu, Kaduna, Kebbi, Osun, Plateau and Sokoto States. Cold chains were maintained for the samples until they were stored at -80°C before testing. Counter current Immunoelectrophoresis (CIE) and Reverse Transcriptase – Polymerase Chain Reactions (RT-PCR) were used to confirm the cases. Primers directed to the Nucleoprotein were used for the RT-PCR and the products were amplified and sent to South Africa for sequencing.

Results and Discussion

All cases except one from Edo State which had undergone putrefaction as a result of break in cold chain were positive by CIE. Similar results were obtained from RT-PCR results. The RT-PCR sequenced results were received in graphic form via Finch TV. The results were edited, converted to nucleotides and aligned using ClustalW; a software for multiple sequence alignment from Georgetown University. Each isolate was BLAST against existing sequences in the GenBank using National Centre for Biotechnology Information (NCBI). The Nsukka (Nig.08/1) and Jos (Nig.08/2) samples showed new strains of PPR following their gene sequence distant from all existing PPR strains in Nigeria. The Nig.08/1 was found to be similar to Tibet/0701 and Turkey/00 strains with 96% identity. There were deletion and substitution in

4 Seminar presented on 2nd April 2009 at NVRI auditorium

the genes of these two strains when compared to the existing Nigerian strains. The protein translation from the Nucleoproteins of these two new strains were compared and found to be 90% related. There was a report of PPR virus studies from India showing a similar lineage with the Nigerian and Turkish strains (Kerur, *et al.*, 2007). It is therefore not surprising to observe a similar lineage with Tibet/0701 and Turkey/00 strains from this study. There is a need to complete this project including isolation of PPR virus so as to have an update in the PPR strains currently ravaging animals across the different geo-ecological zones and to obtain a reliable and current epidemiological data of PPR virus in Nigeria.

Conclusion

Two new strains of PPR Virus have been identified in Nigeria from natural outbreaks: Nig.08/1 PPR strain [from Nsukka] and Nig.08/2 PPR strain [from Jos]

Acknowledgments

The Executive Director NVRI Vom, Directors (Research, Diagnostics & Extension), staff of Molecular Biology Unit, staff of Virology Division, NVRI, my PG Students in NVRI, friends and colleagues at NVRI and the Vice Chancellor, ABU are all gratefully acknowledged.

References

1. Dhar, P; Sreenivasa, B.P. and Barrett, T. (2002). Recent epidemiology of peste des petits ruminants virus (PPRV). *Vet Microbiol.* 88(2):153-9
2. Kerur, N; Jhala, M.K. and Joshi, C.G. (2007). Genetic characterization of Indian peste des petits ruminants virus (PPRV) by sequencing and phylogenetic analysis of fusion protein and nucleoprotein gene segments. *Research in Veterinary Science*, 85 (1). pp. 176-183.
3. OIE (Office International des Epizooties/World Organization for Animal Health; Apr 22, 2002). Peste des petits ruminants. Technical disease card database.
4. Saliki, J.T. (1998). Peste des petits ruminants. In: US Animal Health Association, Committee on Foreign Animal Disease. *Foreign animal diseases: the gray book*. Ed 6. Part IV. Richmond, VA: US Animal Health Assoc.
5. Taylor, W.P; Diallo, A. and Gopalakrishna, S. (2002). Peste des petits ruminants has been widely present in southern India since, if not before, the late 1980s. *Prev. Vet Med.* 52(3-4):305-12

S/NO	Location	Animal species	Sex & Age	Organ	CIE-Test	PCR-TEST
1	Edo 1 (Benin)	Caprine	Female/Adult	Lungs, Spleen & Liver	Positive	
2	Edo 2 (Benin)	Caprine	Female/Adult	Lungs, Spleen (autolized)	Negative	Negative
3	Nsukka 1	Caprine	Male/Adult	Lungs, Spleen & L/N.	Positive	Positive
4	Nsukka 2	Ovine	Female/Adult	Lungs, Spleen, L/N & Liver	Positive	Positive
5	Nsukka 3	Caprine	Female/Young	Lungs, Spleen, & Liver	Positive	Positive
6	Eket 1	Caprine	Male/Adult	Lungs, Spleen.	Positive	Positive
7	Zaria 1	Caprine	Female/Adult	Lungs, Spleen & Liver	Positive	Positive
8	Zaria 2	Caprine	Male/Adult	Lungs, Spleen, Liver & L/N	Positive	Positive
9	Jos 1 (Jos-N)	Caprine	Female/Adult	Lungs, Spleen & Liver	Positive	Positive
10	Jos 2 (Rukuba)	Caprine	Female/Adult	Lungs, Spleen, Liver & N/swabs	Positive	Positive
11	Ilesa 1	Caprine	Female/Young	Lungs, Spleen	Positive	
12	Vom	Ovine	Male/Adult	Lungs, L/N & Trachea (DSI-168)	Positive	
13	Vom	Caprine	Male/Adult	Lungs & Trachea (DSI-171)	Positive	
14	Vom	Ovine	Female/Young	L/N (DSI-173)	Positive	
15	Azare	Caprine	Female/Adult	Lungs, Spleen	Positive	
16	Kebbi 1	Caprine	Male/Adult	Lungs, Spleen & L/N.	Positive	
17	Kebbi 2	Caprine	Male/Adult	Lungs, Spleen	Positive	
18	Sokoto 1	Caprine	Male/Adult	Lungs, Spleen & L/N.	Positive	
19	Sokoto 2	Caprine	Female/Adult	Lungs, Spleen & L/N.	Negative	
20	Misau	Ovine	Female/Adult	Lungs, Spleen & L/N		

PPR Samples and their Sources (2008)

E.I.IKANI

Ikani50@ yahoo.com

Veterinary Extension Division, NVRI, Vom (current address)

Introduction

Livestock extension delivery service in Nigeria has suffered a great deal for so long, majorly due to inappropriate technology development and packaging among other factors. This has resulted into low and very poor animal productivity in the nation. Livestock husbandry practice in Nigeria is largely traditional and extensive with the attendant characteristics of little or no output. The highest percentage of our livestock population is hosted and managed by the nomads. The poor performance of the sector can also be attributed to poor livestock extension delivery services that are based on livestock technologies given out to farmers in piece meals. However, unlike crops, all the production variables in animal agriculture must be given equal and adequate attention or the alternative leads to very serious disaster (IFAD, 1989).

Nigerian Livestock Farmers' Attributes;

Most livestock keepers in Nigeria are illiterates and ignorant of modern production techniques. They are small-scale producers and are not market-oriented. Husbandry practices are extensive/free-range with the scenario of constant movement of both the farm and the farmer. Inability to source, process and handle new/useful livestock information. The livestock keeper is landless and of low social status, however, he is intelligent in his traditional values practices. He is also poor and has no access to modern facilities. These attributes have continued to serve as serious challenges in livestock technology dissemination and adoption coupled with constant conflicts between pastoralists and crop farmers.

Factors to Consider

It has been established that adoption of livestock innovations is very low as compared to the crop sub-sector. A major factor can be traced to inappropriate technology development. For instance, in Nigeria, most livestock Research Institutes develop technologies that are products of intensive animal husbandry practices targeted at end-users who use extensive husbandry practices. These technologies are not always compatible with real field challenges resulting in low up-take and adoption.

Some essential factors to consider when developing livestock technologies are:

1. Which group of farmers?
2. What is the production environment?
3. What are the production constraints?
4. Which are the traditional practices and beliefs?
5. What interventions are needed?
6. What are the expected impacts?
7. What financial implication?

Livestock Technology Packaging

Technology is about a new and better entity, innovation, idea etc. A technology package is therefore, about putting together a complete set of livestock recommended practices targeted at better production results and quality livelihood

⁵ **Seminar presented on 16th April 2009 at NVRI auditorium**

among farmers. The premise on which livestock technologies be packaged should be based on appropriateness coupled with being holistic. Unlike crops, packaging of recommended practices in animal agriculture, must be mutually inclusive i.e. all or nothing. To package an effective livestock recommended production practices all the factors of production must be engaged (Kaimowitz, 1990). Such factors include: breed, feed, shelter, health, husbandry, processing and marketing. Any missing link in the chain leads to serious consequences in livestock production. Therefore technology packaging in animal agriculture must be complete and holistic to attain desired results.

Communication of Livestock Technologies:

Communication is a process by which two or more people exchange/share ideas, facts, feelings and emotions in such a way that each gains a common understanding. This could be verbal or non-verbal. However, feedback is important in response, for communication to be complete. It is important that appropriate channels of communication be used for effectiveness of information transfer. For instance, print media cannot be appropriate for the illiterate nomadic herdsmen, but radio in their own language will be of great help. However, for the literate commercial livestock farmer the print media are effective.

Conclusion

Livestock farmers and product consumers are looking up to researchers and extensionists for direction. They should be major sources of motivation/encouragement. These issues are vital, if the vision 2020 of the present administration is to be realized.

References:

IFAD (1989) IFAD's Experiences in Research and Extension. Rome.
Kaimowitz, D. (ed.) 1990. Making the Link: Agricultural Research and Technology transfer in Developing Countries. Boulder West view Press. pp. 197-226.

**6A VISION FOR DIAGNOSTIC AND EXPERIMENTAL PATHOLOGY AT THE
NATIONAL VETERINARY RESEARCH INSTITUTE (NVRI), VOM, PLATEAU
STATE, NIGERIA**

**B. O. Ikede, BVetMed, DVM, PhD, Dip. Diagn. Path, FCVSN
Retired Professor of Veterinary Pathology & Chairman
Dr. Basil Ikede & Associates Inc
5 Gates Drive, Charlottetown,
Prince Edward Island, Canada, C1E 1R3
Email: ikede@upei.ca**

Pathology and Pathologists

The discipline of pathology has waxed and waned over time in both human and veterinary medicine worldwide. Today, not many graduates from medical and veterinary schools are eager to study pathology. This is largely because they believe that pathology is less lucrative and less glamorous. It is true that the practice of pathology is not as “sexy” as surgery or biotechnology, and yet it is quite demanding for the student. This combination has led to a general shortage of human and veterinary pathologists in many countries including Canada where recently, there was a big scandal about wrong diagnosis of breast cancer in one hospital. In Nigeria, qualified veterinary pathologists are almost extinct. I believe that urgent steps need to be taken to replace the few grey-haired or bald veterinary pathologists remaining.

Introduction

Simply defined, pathology is the study of the science of disease. It is “the medical science, and specialty practice, concerned with all aspects of disease, but with special reference to the essential nature, causes, and development of abnormal conditions, as well as the structural and functional changes that result from the disease process” (Stedman’s Medical Dictionary). Pathology emphasizes the responses of cells, tissues and organs to any form of injury. Consequently, pathologists examine carcasses, organs, tissues, cells or body fluids in order to produce a pathology report. Clinicians, owners of livestock, poultry and pets, collaborating researchers, pharmaceutical companies involved in pre-clinical and clinical trials, as well as the police, need such reports. In places where canine rabies is endemic and dogs are a delicacy, victims of dog bites ask for veterinary pathology reports, even when the dog may not be available for examination.

The study and practice of pathology can be grouped under 5 major sub-disciplines:-

1. Diagnostic pathology
2. Surgical pathology
3. Clinical pathology
4. Experimental pathology
5. Toxicological pathology

Each of these sub-disciplines requires several years of postgraduate training and practice before one can truly be called a pathologist. Medical doctors without specialist training can do some quick postmortem examination and determine the cause of death but they do not sign their reports as pathologists. In the same way, any good veterinarian should be able to open up a cow, dog or chicken in the farm

⁶ Seminar presented on 21st April 2009 at NVRI auditorium

or clinic, but he or she would not sign the report as a pathologist. I would like to talk a little more about the major subdivisions of pathology.

Diagnostic, Surgical Pathology & Clinical Pathology

Diagnostic, surgical and clinical pathology are the “bread and butter” of pathology. When an animal dies on a farm, or a dog is suddenly found dead, the owners and clinicians want to know what really happened. The diagnostic pathologist examines the carcass and provides the clients information that will help them to manage disease outbreaks in the herd or flock or improve management of individual cases. If a zoonotic disease such as rabies is diagnosed, the implications are even wider since a positive diagnosis will require referring affected persons to a medical doctor. Apart from examining traditional domestic animals, diagnostic pathologists also examine wild animals in zoos and parks, fish in aquaria, aquaculture or in the wild, and marine animals. The process is very labour intensive because pathologists have to be on their feet to do necropsy. Diagnostic pathology is also an important avenue for disease surveillance and disease reporting. In addition, it should lead to publications. For example, my chapters in the 8th, 9th and 10th editions of Radostits *et al.* (1994, 2000 & 2007) are based partly on my diagnostic experience with tropical animal diseases in Nigeria and in Malaysia.

Surgical pathology is the examination of tissues (biopsies) taken from the live animal to provide diagnoses that help clinicians treat the animal more specifically. In countries like Canada and the United States where there is greater care for the animals and more disposable income, biopsies on dogs and cats are far more common than necropsies. In addition, because full necropsy may be denied by owners who want befitting burials for their pets, pathologists in such countries are more involved with biopsies than necropsies.

People practicing diagnostic and surgical pathology are often referred to as anatomic or morphologic pathologists or simply histopathologists. They are distinct from clinical pathologists who examine mostly fluids or smears of cells from live animals. Clinical pathology has grown very rapidly in developed countries where the value of individual animals is high. Clinicians routinely send samples of body fluids and tissue aspirates from organs and tumors for clinical pathology examinations and they expect results same day to guide them in their treatment of patients. To achieve this, clinical pathology labs have computerized, sophisticated equipment with automation especially as regards blood chemistry, cell counts, etc. In many veterinary diagnostic laboratories, pathology reports are available much faster than in human hospitals.

Experimental Pathology & Toxicological Pathology

Experimental pathologists study tissue, cellular and molecular mechanisms of human and animal diseases, often by using various agents to induce the changes to be studied in experimental animal models. Experimental pathologists can be in the fields of infectious and nutritional diseases, biomedicine and biomedical engineering. Toxicological pathology is a branch of experimental pathology. It involves using specific toxic agents or chemicals in experimental animals with a view to studying the changes they can induce. Such changes are observed and measured over time. Invariably, the experimental pathologist is a member of a team engaged in elucidating the mechanisms of disease so as to come up with better therapeutic or preventive measures. They work mostly with laboratory animals but also with tissue culture, domestic animals, primates (preclinical trials) and humans (clinical trials). For example, as an experimental pathologist, I worked with colleagues for many years studying the pathogenesis of trypanosome infections in

laboratory rodents and in domestic animals using techniques in histopathology, electron microscopy, hematology, immunopathology as well as clinical and field experience. In Canada, we carried out similar studies on a viral disease of fish, infectious salmon anaemia virus (ISA), using *in situ* hybridization techniques along with histopathology and tissue culture.

Veterinary Pathology in North America today

It is probably true that the veterinary profession generally, and veterinary pathology in particular, are at their highest level of development today in the United States and Canada. This is largely due to the fact that many North Americans regard their pets as part of the family (like the Obama family adopting a dog), and are willing to spend heavily to ensure the health and well-being of their dogs and cats. Furthermore, since livestock are kept as a money-spinning enterprise (a true business venture), owners are willing to spend money on diagnostic activities that will lead to disease prevention and overall net profit as our poultry farmers do in Nigeria. Consequently, there are diagnostic laboratories in veterinary schools, at the level of the state or province, and at national level in both countries. National laboratories usually operate at a higher biosafety level so as to handle highly infectious and contagious diseases. In the past few decades, private diagnostic labs have been established to provide more timely reports for a fee. IDEXX is an example of a multi-national private diagnostic laboratory in the US, Australia, etc. The majority of specimens submitted to such labs are clinical pathology samples, biopsies and postmortem tissues collected and preserved in formalin for shipment by courier. The labs strive to provide accurate and timely diagnostic reports which the clinician demands within a few days of submission. For quality control, the cases are usually reviewed by a team of pathologists before the report is sent out. Two heads are better than one, many heads even better! Peer review is important because a wrong diagnosis is worse than no diagnosis.

Pharmaceutical companies like Pfizer, Roche etc employ veterinary pathologists who are mostly involved with experimental or toxicological pathology. Before any drug can be approved for human or veterinary use, extensive testing and trials must be carried out to check for chemical toxicity, carcinogenesis, drug interactions, etc. Large-scale tests are run in mice, rats, dogs and primates to establish safe dose levels, side effects and consequences of prolonged use. Experimental pathologists examine hundreds and thousands of sections from such animals and determine what changes have taken place when compared with controls. Lesions are quantified through digital imaging and analyzed with appropriate computer soft-wares.

To qualify as a veterinary pathologist in North America or the developed world in general, one must undergo a rigorous and intensive training post-DVM. Most veterinary schools run a residency programme alone or in combination with a Master's or PhD degree lasting from 3 to 5 years and more. At the end of the residency, trainees are eligible to sit for a highly competitive examination in order to be board-certified as Diplomates of the American College of Veterinary Pathologists (ACVP and ASVCP). The passing rate of the exam is often around 50% at the first attempt and one is allowed only three attempts to pass all four parts. Good-paying jobs are readily available after the residency (but not necessarily after a straight PhD) and salaries can be double once the pathologist becomes board-certified.

The Central Diagnostic Laboratory at NVRI

A diagnostic laboratory at NVRI has probably been in existence since the beginning of the Institute. For the first time in many decades, it is receiving the attention it deserves due to the interest and commitment of the Executive Director. I share her vision that the CDL will be the hub of the Institute and will set the pace for research. The CDL is a full Division in a recently renovated edifice located in a well-landscaped area of the Institute. Many new staff members have been recruited and the Division is being equipped to meet the Institute's status as the OIE/FAO Regional Centre for the control of transboundary animal diseases in West and Central Africa. The CDL is no doubt the busiest and best-equipped veterinary diagnostic lab in the country. Its post-mortem room is the hub of the Division. Carcasses are submitted from the Institute's farms, and the entire country for postmortem examination. In 2008, there were over 2,000 carcasses, mostly chickens, dogs or dog heads for rabies. Others were cattle, sheep, goats, pigs, horses, rabbits, wildlife and fish. At necropsy, specimens are collected and submitted to other labs such as bacteriology, clinical pathology, histopathology, parasitology and virology for further examination. Blood and serum samples are also received from live animals for clinical pathology. Surgical pathology on live animals is done only occasionally. However, an overwhelming number of the veterinary research officers involved in providing these diagnostic services are not trained pathologists. As for experimental pathology, a few staff in the Division have carried out or are carrying out research for MSc or PhD degrees in universities, but they can hardly find time for additional collaborative research. There is therefore an urgent need for specific training in pathology.

Reflections on the past three months

This is the second time I am working in the Institute, the first was as a veterinary student from the University of Ibadan on vacation job 44 years ago! When I arrived here nearly three months ago as a visiting professor, the EDVR requested me to do a professional assessment of the Central Diagnostic Laboratory and recommend ways for its improvement. The assignment was completed in about 6 weeks after which I submitted a detailed written report on Friday, March 13 to the EDVR. By Monday afternoon, she had read the report and I was invited to discuss it with her. I am pleased to say that many of my recommendations were accepted and are already being implemented by the EDVR and Dr. Okewole, the Head of Division.

During the period, I have also been involved with training the veterinary research officers in the CDL through power point presentations twice a week and gross rounds once or twice a week. I have also assisted younger colleagues in writing postmortem reports and I am now facilitating some changes in the histology lab, which has been an outstanding bottle-neck for histopathology. These efforts are aimed at capacity development of staff involved in necropsy and pathology in general. It has been an exciting and productive visit so far and I look forward to further collaboration with the Division and with NVRI in the future.

Concluding remarks

I see the CDL as a pentagon of labs surrounding the postmortem room. Each unit has an important and crucial role to play and must be utilized fully to achieve the desired objective. However, in the Nigerian setting for the foreseeable future, the postmortem room will truly be the first among equally important labs in CDL. My vision for the CDL is that it will in the near future, have the required complement of trained pathologists to provide reliable and prompt diagnostic services not only around Vom but also throughout the country. Trained pathologists will have the confidence and expertise to collaborate more actively with at least some of the eight

faculties of veterinary medicine in the country directly and through NVRI's Out-station laboratories. I envisage that CDL services will also reach out more to neighbouring countries in West and Central Africa in due course. I must stress that for this vision to be realized, CDL must engage in active and specific training of veterinary research officers who are qualified anatomic and clinical pathologists able to make timely and reliable diagnosis. Such diagnoses must be backed with histopathology, cytology, etc. The pathologists must also have the expertise to correlate other laboratory tests with their own findings as they apply their knowledge of medicine and pathology to the cases being handled. Trained veterinary specialists in the appropriate disciplines should also supervise every other lab in the CDL. The specialists should also be willing and able to collaborate with other scientists in the Institute and beyond in designing and executing research projects involving infectious and non-infectious diseases, biomedicine and biotechnology. I see a bright future for the CDL and NVRI.

Acknowledgements

I thank Dr. Lombin for inviting me here and giving me the first opportunity to utilize my expertise again in my native land after retirement in Canada. Special thanks go to Drs Shamaki, Makinde and Okewole who facilitated my orientation when I first arrived. It has been a pleasure working with staff of the CDL and interacting with many NVRI staff. I thank my wife Joy who is maintaining our home in Canada while I am here. I thank God for making this visit possible.

Basil O. Ikede, DVM, BVetMed, PhD, Dip Diagn Path, FCVSN.



Prof Basil Ikede has been a professor of Veterinary Pathology for over thirty years. He received his DVM degree from Ahmadu Bello University, Zaria, and his BVet Med from University of Ibadan both in 1967. He obtained a graduate diploma in Diagnostic Pathology for the Ontario Veterinary College, university of Guelph, Canada in 1971 and a PhD from the University of Ibadan in 1972. He was promoted full professor of Veterinary Pathology in 1977, and appointed Head of Department from 1979 to 1987. At Ibadan, Professor Ikede's research and diagnostic activities were focused on major animal diseases that were limiting livestock production in Africa especially Trypanosomosis and PPR. In 1976, he served as a member of the WHO first Scientific Working group on African Trypanosomosis in Geneva, Switzerland. In 1985/86, he was a member of the FAO second External Programme Review of the International Laboratory on Research in Animal Diseases (ILRAD) in Nairobi, Kenya. Dr. Ikede was the founding editor-in-chief of *Tropical Veterinarian* an international journal, first published in 1983 by S. Karger, Switzerland. He has also served as national coordinator of the EEC-funded programme on the control of animal Trypanosomosis in Nigeria. In 1989, Dr. Ikede was appointed Professor of Anatomic Pathology at the Atlantic Veterinary College, University of Prince Edward Island, Charlottetown, Canada. He later served two terms as Chair, Department of Pathology and Microbiology in the same University. He has contributed chapters on tropical animal diseases in the 8th and subsequent editions of the highly acclaimed text book; *Veterinary Medicine: A Textbook of Diseases of Cattle, Sheep, Goats and Horses* edited by Radostits and others. At the time of retirement in 2008, he had 80 publications in peer-reviewed journals and made several presentations at conferences and seminars around the world. He has been major supervisor or on the supervisory Committees of over 50 MSc and PhD students and has contributed to the education of veterinary students for over forty years. Prof. Ikede now resides in Canada with his wife Joy. They have four children and four grand children. Prof. Ikede was at NVRI as visiting Professor and consultant between January and April 2009.

7USER SATISFACTION WITH LIBRARY RESOURCES AND SERVICES IN NIGERIAN AGRICULTURAL RESEARCH INSTITUTES

Lily Oluebube Ezeala

Library, Documentation and Information Division

NVRI, Vom

lilyval02@yahoo.com

Introduction

Agricultural research institute libraries in Nigeria were established to provide literature support for research and agricultural development activities. Unfortunately, the researchers are not using the libraries as expected. There is a need for periodic performance evaluation of these libraries to determine how well they are meeting the objectives for which they were established. The most popular way to achieve this is by performance assessment of the research libraries by user approach method. This research was conducted in order to assess user satisfaction with libraries resources and services in 14 Agricultural Research Institutes. The research objective was to discover how to improve the effectiveness of agricultural research institute libraries with emphasis on harnessing the strengths of the library effectiveness variables of interest; Library collection, electronic resources, library services and library utilization

Methodology

Data were collected using the survey design and structured interview. The questionnaire, which was validated, by library science experts and pre-tests with reliability test of 0.77 were administered on 340 researchers in all 14 agricultural research institutes in Nigeria.

Results and Discussion

The overall response rate was 73.5%. The research data were analysed using frequency counts and percentages, Regression Analysis, Correlation, and Analysis of Variance (ANOVA). Four hypotheses were tested at 0.05 alpha levels. The study revealed that there was no significant relationship between user satisfaction with the research libraries and the number of publications output by the researchers. ($\bar{X} = 3.0$). A weak and insignificant inverse correlation was established between the variables ($r = -.002$, $P > 0.05$). With respect to user-satisfaction with electronic resources and user output, ($\bar{X} = 2.0$) a positive but weak insignificant correlation between these two variables was observed ($r = 0.04$, $P > 0.05$). Correlation analysis revealed a weak correlation between user satisfaction with library collection and publications output ($r = 0.007$ and $P > 0.05$) No significant relationship was established between use of library by the researchers and their publications output ($\bar{X} = 3.15$). Table1 shows that the weighted mean effectiveness for all 14 agricultural research institutes is 2.6. This shows that the libraries are generally ineffective. On individual basis, NCRI (3.4), CRIN (3.1) and NIOMR (3.0) were found to be the most effective. The effectiveness of NIOMR may have been influenced by the Internet services in that library. It was observed that NIOMR was the only agricultural research institute in the study that had Internet services linked to the library. Effectiveness of CRIN library may be because of the size of the library collections. (6,500 journal titles, 3,200 book titles and 1,200 monographs). This library had the second largest size of journal collections in all the fourteen

⁷ Seminar presented on 7th May 2009 at NVRI auditorium

agricultural research institutes. It appeared that libraries under study were generally ineffective.

Conclusion

Based on analysis of research data, one concludes that the agricultural research institute libraries in Nigeria are ineffective in supporting research in their institutes'. This ineffectiveness has resulted from gross under-funding of the libraries by the parent institutions, and this has adversely affected the resources and services. Based on the vital role of the library in the research institute, the low productivity of the agricultural research officers in terms of publications output is attributable to the ill-equipped libraries. Had the libraries been well provided for, the publications output of the research officers could have been much more. One hopes that the management of libraries in Nigeria will not wait for a revolt by the deprived users before appropriate steps are taken to improve library effectiveness.

References

1. Adegbola, C.O.B (1997) Resource sharing and the prospects of networking in agricultural research libraries in Ibadan, Unpublished MLS project, University of Ibadan, Nigeria.
2. Brookes Cork Library, Shelton State Community College 2005 Handbook on Library Effectiveness- Posted on: [WWW.Sheltonstate.edu/library Effectiveness.Intml](http://WWW.Sheltonstate.edu/library_Effectiveness.Intml). Accessed 28/08/2005
3. Childers, S. (2003). Computer Literacy: Necessity or Buzzword? Information Technology and libraries. Retrieved July 15, 2004 from <http://www.lita.org/ala/litapublications/ital/2203childers.htm>
4. Lancaster, F.W. (1993) If you want to evaluate your library... London: The Library Association,
5. Nwalo, K.I.N. (1997). Measures of Library Effectiveness in Nigerian polytechnic libraries with emphasis on User satisfaction. Unpublished doctoral dissertation, University of Ibadan.

Table1: Assessment of Agricultural Research Institute Libraries' Overall Library Effectiveness

INSTITUTION	Overall Assessment						Mean	Total
	Very effective	Effective	Undecided	Ineffective	Very Ineffective			
CRIN	0	8	8	12	0	3.15	27	
IAR	1	12	12	2	0	2.20	15	
IAR&T	0	4	4	1	0	2.63	8	
LCRI	4	10	10	3	1	2.32	19	
NAPRI	1	9	9	1	0	2.09	11	
NCRI	0	3	3	10	0	3.35	18	
NIFFR	1	19	19	0	0	1.95	20	
NIHORT	0	8	8	5	0	2.82	21	
NRCRI	2	18	18	5	0	2.31	26	
NVRI	1	12	12	9	0	2.86	37	
NIOMR	0	9	9	7	1	3.00	21	
NIFOR	0	6	6	5	0	2.92	12	
NSPRI	1	7	7	0	0	2.10	10	
RRIN	1	2	2	0	0	2.00	5	
TOTAL	12(4.8)	127(50.8%)	127(50.8%)	60(24%)	2(.8%)	2.64	250(100%)	

Mean score ≥ 3.0 = Reject. Mean score < 3.0 = Accept

8 CURRENT MANAGEMENT STRATEGIES AGAINST TICKS: A CONTRIBUTION TO KNOWLEDGE

R.I.S. Agbede

Department of Parasitology & Entomology

Faculty of Veterinary Medicine

Ahmadu Bello University, Zaria

E-mail: risagbede@yahoo.co.uk

Introduction

Ticks surpass all other arthropods in the number and variety of pathogens that they transmit to domestic animals and rank second only to mosquitoes as vectors of human pathogens. Damage done by ticks could be direct, primarily caused by the tick bite; abscesses develop, resulting in appreciable loss in the value of hides and skins. In case of dairy animals, such abscesses frequently involve the udder resulting in loss of one or more quarters and thus loss of milk production (McCosker, 1979). The tick bite also provides the portal of entry for bacteria and parasitic flies, predisposing cattle and other animals to streptothricosis (Oduye and Lloyd, 1971). Tick worry [irritation] has a depressing effect on milk and meat production, which is proportional to the number of ticks carried by the animal. In Australia, infestation results in reduction of live weight gain ranging from 0.28kg to 0.8kg per tick, per year (Gee et al., 1971). Blood loss caused by engorging females can result in severe anaemia and even death. (Callow, 1978). Mortalities have been reported because of infestation with *Boophilus decoloratus* and *Amblyomma variegatum* (Agbede, 1984). Injection of tick derived toxins into cattle results in different clinical manifestations such as paralysis caused by *Ixodes holocyclus*, *I. rubicundus* and *Dermacentor andersoni*. Sweating sickness is caused by *Hyalomma truncatum* and tick toxicosis caused by *Rhipicephalus spp.* All effects occur with variable mortality depending on tick burden. The indirect effects are through the diseases they transmit. In Nigeria alone, within the last three decades evidence has emerged for the role of ticks in the transmission of Dugbe, Thogoto, Congo and Bhanja viruses (Obi, 1978; Durojaiye, 1981). Three emerging infectious diseases of man identified in the last two decades in the USA include, Human Monocytic Ehrlichiosis [HME], Human Granulocytic Ehrlichiosis [HGE] and Lyme disease (Walker and Dumler, (1996) and are all tick borne.

Tick Control

The need therefore to control ticks cannot be overemphasized. Current control methods are de-ticking, use of Natural predators of ticks [birds, chickens, ants etc], intensive use of acaricides. Currently the main stay, acaricides are very effective but have problems. Problems of chemical or acaricidal control include toxicity, resistance, and environmental pollution, residues in milk and meat products.

Immunological Control

One approach has been to seek ways to enhance the Natural immunity often acquired by animals in response to tick infestation [Acquired Immunity]. Acquired immunity to ticks reduces tick burden only slightly. Females still engorge on the host.

Vaccination with tick gut antigens

The other approach is via 'concealed antigens' not normally encountered by the host during the feeding of the tick and so the antigens stimulate a different immune effector mechanism (Kemp and Agbede 1986; Willadson and Kemp 1988). This product is the crude midgut vaccine against *Boophilus microplus*. Tick vaccines constitute a cost effective and environmentally friendly alternative to chemical control. Bio-tech Australia bought the patent from C.S.I.R.O and isolated the Basement Membrane (BM 86) protective antigen from the gut of *B. microplus*. The protective antigen produced by recombinant DNA technology has been shown to protect cattle against *B. microplus* infestations. To emphasize the diversity of species, there are five species of *B. sp* worldwide. These include *B. calaratus* (Russia, Antarctica) *B. microplus* (Russia, Australia, Europe, and South Africa and parts of U.S.A.), *B. decoloratus*, *B. geigy* and *B. annulatus* are present in West Africa and so far there is no evidence that they are protected against by BM 86.

Newer Control Techniques

The decoy technology exploits the natural aggregation, attraction, and attachment behaviour of the *Amblyomma* species. Use of Attraction/ Aggregation/ Attachment Pheromone (AAP) of *Amblyomma variegatum* and *A. hebraeum* impregnated in special belt. This technology was developed at the Centre for Tropical Diseases, University of Florida conjoint with Old Dominion University. Trials are on in the USA, Republic of Benin, Zimbabwe, and the West Indies. It is aptly called the tick decoy technology. Food and Agriculture Organization (FAO) is still evaluating the product.

My Vision for NVRI

Vom will be a centre for tick vaccine production. We must build capacity. Two staff of the division of Parasitology are pursuing PhD degrees. They will be the CORE staff of the TICK VACCINE production unit. They need extra training. Dr. Goni Dogo will do further work on Bm86 . The gene encoding Bm86 will be PCR amplified from *B. annulatus* and *B. decoloratus* gut. Funding is required for Dr. Dogo to spend about three months at the State University of Utrecht, The Netherlands. Dr. I.I. Ogo will try to isolate a similar protein from *Amblyomma variegatum*. Funding is required for Dr. Ogo to travel to Spain's National Laboratory for nine months to learn relevant techniques. We have started with histology of the GUT of *A. variegatum*, but there are slight set backs from the histology of *A. variegatum*. Our initial histology studies were not successful due to problems with tick embedding. The Diagnostic Laboratory has no vacuum embedding facility.

Acknowledgements

The Executive Director, Dr. (Mrs) L. Lombin, Dr. David Shamaki & other Directors, Drs Goni Dogo & Okewole plus other Heads of Departments, Dr.(Mrs) Maryam Muhammad , Staff of Division of Parasitology and Officer in charge of the Internet



R. I. S. Agbede DVM, MVSc, PhD, FCVSN

Professor Rowland Ibrahim Shehu Agbede was born in Kaduna on the 3rd of February 1949 and is a staff of the Department of Parasitology & Entomology, Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria, Nigeria. He qualified with a DVM from A.B.U, Zaria in 1975; an MVSc from the University of Liverpool, England in 1977; and a PhD from the University of Queensland, Australia in 1984. He is a Fellow, Parasitology and Public Health Society of Nigeria, [FPPSN] Fellow, College of Veterinary Surgeons, Nigeria [FCVSN]. He lectures both

undergraduate and postgraduate students and has supervised over 12MSc students and 17PhD research theses. He also serves as external examiner for both undergraduate and postgraduate {MSc and PhD} to all Veterinary schools in Nigeria. Professor Agbede has served as Editor in Chief, Nigerian Journal of Parasitology for 8years and President, Nigerian Society for Parasitology. In 1996, he was awarded a Fulbright Senior African Research Scholarship. He is in Who-is-Who in Science and Technology in Nigeria, Newswatch First Edition of Who-is-Who in Nigeria, African academy of sciences, who is who in Science and Technology in Africa. He has published over 60 papers/proceedings. His special areas of interest include ticks and the immunological control of ticks, tsetse and trypanosomiasis control using genetic methods and integrated Pest Management [IPM] packages, formulation and application. He is married with four children. Professor Agbede was a visiting scientist at the Parasitology Division of NVRI in 2009.

⁹PRELIMINARY INVESTIGATION OF NEWCASTLE DISEASE AND HIGHLY PATHOGENIC AVIAN INFLUENZA VIRUSES IN LIVE BIRD MARKETS IN IBADAN, NIGERIA

Shittu I¹, Joannis TM¹, and Olaleye OD²

¹Viral Research Division, National Veterinary Research Institute, Vom

²Department of Virology, University College Hospital, College of Medicine, University of Ibadan

***Corresponding author: shismailus@yahoo.com**

Introduction

The poultry production system in Nigeria is comprised of both commercial and backyard birds estimated at 140 million. Commercial poultry production accounts for a majority (over 70%) of the Nigerian poultry industry. The poultry industry is faced with outbreaks of diseases. Highly pathogenic avian Influenza (HPAI) and Newcastle disease (ND) have been rated the most devastating diseases amongst poultry. These viruses are associated with transmission from wild to domestic birds, and can lead to human infections such as conjunctivitis and influenza-like syndrome (Artois *et al.*, 2002; Capua and Alexander, 2004). Wild birds may function as a reservoir for both viruses, playing a role as potential vectors with few or no clinical signs (Alexander, 2000). Newcastle disease virus (NDV) and avian influenza viruses (AIV) are caused by type A *Orthomyxoviruses* and avian *Paramyxovirus* type 1 (APMV 1) respectively (Swayne and Suarez, 2000). These highly virulent viruses have severe impact on poultry health and limit international trade in poultry and poultry products. Global awareness regarding HPAI (H5N1) has heightened since the 1997 outbreak of the disease in Asia and emphasis on surveillance of possible reservoirs (wild migratory birds, domestic ducks etc) and other risk factors including the Live Bird Markets (LBMs) has been advocated. A live bird market or 'wet market' is a public place where transactions of birds of different breeds/species and occasionally small animals are carried out. Birds or animals entering the market emanate from various sources, including farms that raise birds commercially for sale, backyard and village poultry flocks. ND is endemic in Nigeria and controlling it has been a major problem both in vaccinated and unvaccinated backyard and commercial poultry settings in Nigeria and determining the epidemiology of the disease will enhance the control efforts.

Methodology

Study Area and Sample Collection

The study was conducted in two major live bird markets (LBMs) Molete and Sasa in Ibadan (southwest Nigeria). A questionnaire was administered together with sample collection. Three hundred (300) pooled cloacal swabs (three swab sticks per vial) were collected from apparently healthy live chickens, ducks, pigeons, guinea fowls, and turkeys in both LBMs between March and April 2008. No sick or dead birds were observed in the market during the period of sampling. Swabs from same bird species and from the same market were pooled in viral transport medium (VTM) giving 100 vials. The VTM was composed of glycerol and phosphate-buffered saline (1/1) containing antibiotics concentration of 10,000 IU/ml penicillin, 10 mg/ml streptomycin, 0.25 mg/ml gentamycin and 0.0125 mg/ml amphotericin B adjusted to a final pH of 7.2. The samples were transported to the laboratory on ice and stored at -80°C until analyzed. Specimens were analyzed at the Avian Viral Research Laboratory (AVRL) in Vom. Cloacal swabs were thawed, vortexed and

⁹ Seminar presented on 21st May 2009 at NVRI auditorium

sterile forceps used to remove swab sticks. Suspensions of swab contents were then centrifuged at 3000 rpm for 10 minutes.

Virus Isolation and Identification

Virus isolation was attempted in 9-11 day-old specific antibody negative (SAN) embryonating chicken eggs according to standard protocol (OIE, 2004). Inoculated eggs were incubated at 37 °C for 5 days, examined daily by candling for evidence of embryo death. Embryonic deaths above 24 hours of inoculation were refrigerated. The allantoic fluid (ALF) of the refrigerated dead egg was harvested and tested for haemagglutinating (HA) activity. ALF was checked for bacterial contamination by plating on bacteriological medium (Blood agar). Uncontaminated, HA positive ALFs were screened for avian influenza ribo-nucleoprotein group specific antigen. Standard haemagglutination and haemagglutination-inhibition (HI) tests were used in identifying NDV using ready prepared antisera from our laboratory.

Results

Most of the birds sold in Molete market were commercial and local chickens, ducks, pigeons, guinea fowls, turkeys. Other animals included dogs, rabbits, guinea pigs and cats. Most of the birds sold in this market were sourced from commercial poultry farms within and outside Ibadan including Abeokuta, Ijebu, Ogbomoso, Akure where birds were raised purposely for meat and eggs. The bird population in Sasa market comprised of pigeons, local chickens, ducks, guinea fowls, and turkeys. Animals sold in the market included guinea pigs and rabbits. Most of the birds sold in this market were brought Jigawa, Katsina, Kebbi, and Zamfara States.

The pooled cloacal swab samples comprised of chickens 41 (41%), ducks 15 (15%), guinea fowls 16 (16%), pigeons 27 (27%), and turkeys 1 (1%) as shown in Table 1. The 100 pooled cloacal swabs yielded 21 isolates identified as NDV by the HI test. This result shows an overall prevalence of 21.0% of detectable NDV infections in the total sample from the two markets (Table 1). Out of the 49 pooled cloacal swabs collected, eight (16.3%) and 13 (25.5%) NDV isolates were obtained respectively. NDV was isolated from 10 (24.4%) of 41 pooled cloacal swabs of chicken from the 2 markets. Out of the 15 pooled duck samples, 3 (20.0%) NDV isolates were obtained. Three (18.8%) NDV isolates were obtained from the 16 pooled guinea fowls sampled. The 27 pooled pigeon samples yielded 5 (18.5%) NDV isolates while no isolate was obtained from the turkey samples. No AIV isolate was obtained from all the samples tested by virus isolation during the study period.

Discussion

The result of 21.0% prevalence obtained in this study is higher than the prevalence of 12.7% for NDV reported in a similar study in 10 Vietnamese LBMs in 2001 over a two day sampling period (Nguyen *et al.*, 2005). According to Manchang *et al.* (2004), the incidence of NDV outbreak is higher during the dry season (October-March) in Nigeria. This coincides with the period when this study was conducted and may explain the high isolation rate for NDV found in this study. A comparison of the prevalence of NDV between the two markets showed that Sasa market had a higher rate (25.5%) than Molete (16.3%). The higher rate observed from Sasa could be attributed to the predominance of local chickens, ducks, pigeons and guinea fowls which have been reported to harbour NDV by previous workers (Echeonwu *et al.*, 1993; Nwanta *et al.*, 2008). Sasa market supplies Molete market and other LBMs in Ibadan and environs with local chickens, pigeons, guinea fowls and ducks. The movements of these live infected birds have been suggested as a source of transmission of the virus (Nwanta *et al.*, 2008). NDV was isolated from four of the

five species of birds sampled. The highest isolation of 24.4% was from chickens followed by ducks 20.0%, guinea fowls 18.8% and pigeons 18.5%. Ducks have been implicated in harbouring the virus in its apathogenic forms and subsequently transmitting it to domestic poultry where the virus may undergo mutation to its virulent forms. From the results of this study, it can be deduced that NDV is circulating in healthy chickens, ducks, pigeons and guinea fowls in the two LBMs. Chickens were identified as the primary host harbouring NDV in the markets which is in agreement with the report of Saidu *et al.* (2006). The result of this study also affirms the endemicity of NDV in Nigeria as reported by several authors (Manchang *et al.*, 2004; Oyekunle *et al.*, 2006) and it seems LBMs play a vital role in the maintenance and spread of NDV. Further work is required to determine the pathotypes and phylogenetic studies of the isolates.

Table 1: Distribution of collected samples and NDV isolates by Specie from Molete and Sasa markets

Species	Molete (MO)		Sasa (SA)		Total pooled specimen	Total isolates (%)
	Number of pooled samples	Number of Isolate (s)	Number of pooled samples	Number of Isolate (s)		
Chickens	30	4	11	6	41	10 (24.4)
Ducks	6	1	9	2	15	3 (20.0)
G/Fowl	4	1	12	2	16	3 (18.8)
Pigeons	8	2	19	3	27	5 (18.5)
Turkeys	1	0	0	0	1	0 (0)
Total	49	8	51	13	100	21

Acknowledgments

The Executive Director, NVRI, all staff of Viral Research Department, and the LBM workers

References

- Alexander D. J. 2000. Newcastle disease and other avian paramyxoviruses. *Rev. Sci. Tech.* 19: 443–462.
- Artois, M., Manvell, R., Fromont, E. and Schweyer, J.B. 2002. Sero-survey for Newcastle disease and avian influenza A virus antibodies in great cormorants from France. *J. Wildlife Dis.* 38: 169–171.
- Capua, I. & Alexander, D.J. 2004. Human health implications of avian influenza viruses and paramyxoviruses. *Eur. J. Clin. Microbiol. Infect. Dis.* 23: 1–6.
- Echeonwu, G. O. N., Iroegbu, C. U. and Emeruwa, A. C. (1993) .Recovery of velogenic Newcastle disease virus from dead and healthy free-roaming birds in Nigeria. *Avian Pathology*, 22.2: 383 – 387.
- Gould, A. R., Kattenbelt, J. A., Selleck, P., Hansson, E., La-Porta, A. and Westbury, H. A. 2001. Virulent Newcastle disease in Australia: molecular epidemiological analysis of viruses isolated prior to and during the outbreaks of 1998–2000. *Virus Res.* 77:51–60.
- King, D. J & Seal, B. S. 1997. Biological and molecular characterization of Newcastle Disease Virus isolates from surveillance of live bird markets in the Northern United States. *Avian Diseases* 41: 683 – 689.
- Majiyagbe, K.A. and Lamorde, A.G. 1997. Nationally coordinated research programme on Livestock diseases: Sub-sectoral goals, performance and medium-term research plans. *Trop. Vet.*, 15: 75-83.

8. Manchang, T.K., Abdu, P.A., and Saidu, L. 2004. Epidemiology and Clinicopathologic Manifestations of Newcastle Disease in Nigerian Local Chickens. *Revue Élev. Méd. vét. Pays trop.* 57.1: 35 – 39.
9. Nguyen, D. C.; Uyeki, T. M.; Jadhao, S.; Maines, T.; Shaw, M.; Matsuoka, Y.; Smith, C.; Rowe, T.; Lu, X.; Hall, H.; Xu, X.; Balish, A.; Klimov, A.; Tumpey, T. M.; Swayne, D. E.; Huynh, L. P. T.; Nghiem, H. K.; Nguyen, H. H. T.; Hoang, L. T.; Cox, N. J.; and Katz, J. M. (2005). Isolation and Characterization of Avian Influenza Viruses, Including Highly Pathogenic H5N1, from Poultry in Live Bird Markets in Hanoi, Vietnam, in 2001. *Journal of Virology* 79(7): 4201–4212.
10. Nwanta, J. A., Abdu, P. A., and Ezema, W. S. 2008. Epidemiology, challenges and prospects for control of Newcastle disease in village poultry in Nigeria. *World's Poultry Science Journal* 64: 119 – 127.
11. Office International des Epizooties (OIE). 2004. Newcastle disease. In: *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals. Part 2, Section 2.1, Chapter 2.1.15* (http://www.oie.int/eng/normes/mmanual/A_00038.htm). Accessed 25 June, 2008.
12. Office International des Epizooties (OIE). Avian influenza. Chap 2.7.12. 2004. In: *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals, 5th edition.* (Updated May 2005) 2004. Accessed on 6th June, 2008 from http://www.oie.int/eng/normes/mmanual/A_00037.htm
13. Oyekunle, M.A., Talabi, A.O. and Okeowo, A.O. 2006. Serological Status for Newcastle Disease Virus in Unvaccinated Indigenous Chickens in Yewa Division of Ogun State, Nigeria. *International Journal of Poultry Science*, 5 (12): 1119-1122
14. Sa'idu, L., Abdu, P. A., Tekdek, L. B., Umoh, J .U. (2006). Retrospective Study of Newcastle Disease Cases in Zaria, Nigeria. *Nigerian Veterinary Journal* 27 (3): 53-62.
15. Seal, B. S., Wise, M. G., Pedersen, J. C., Senne, D. A., Alvarez, R., Scott, M. S., King, D. J., Yu, Q. and Kaczynski, D. R. (2005). Genomic sequences of low-virulence avian paramyxovirus-1 (Newcastle disease virus) isolates obtained from live-bird markets in North America not related to commonly utilized commercial vaccine strains. *Vet. Microbiol.* 106: 7–16.
16. Senne, D. A., Pearson, J. E. and Panigrahy, B. (1992). Live poultry markets: a missing link in the epidemiology of avian influenza. In: *Proc. 3rd International Symposium on Avian Influenza.* U.S. Animal Health Association, Richmond, VA. pp. 50–58.
17. Swayne, D. E. & Suarez, D. L. (2000). Highly Pathogenic Avian Influenza. *Rev Sci Tech.* 19: 463–482.

¹⁰PHENOTYPIC AND GENOTYPIC CHARACTERIZATION OF *LISTERIA* SPECIES ISOLATED IN PARTS OF PLATEAU STATE, NIGERIA

Chukwu O. O. Chukwu

Molecular Biology Department, Federal College of Veterinary and Medical Laboratory Technology, National Veterinary Research Institute, Vom, Nigeria

Email: chyooks@yahoo.com

Phone: +234-8037740663

Abstract

Listeriosis affects domestic and wild animals as well as man. The disease has been widely overlooked, but is found with increasing frequency when adequate techniques are employed. Extraordinary laboratory procedures are required to reliably isolate the causative organism, *Listeria species* especially *Listeria monocytogenes*. Studies were carried out on the distribution, phenotypic and genotypic characterization of *L. monocytogenes* and other *Listeria* species in six Local Government Areas of Plateau State, Nigeria. A total of one hundred and fifty (150) experimental samples comprising of cow, goat, poultry, rabbit and sheep wastes (faecal samples), cultivated soil particles, farm wastes, human wastes (faeces), water from each test Local Government Area, given a grand total of 900 experimental samples. The isolation was based on cold enrichment, selective broth and selective *Listeria* agar plates. Phenotypic differentiation was made using specialised media, Gram reaction, beta- haemolysis on sheep blood agar, motility, sugar fermentation and CAMP test (Christie, Atkins, Munch-Peterson) reduction. Molecular characterisation was conducted using deoxyribonucleic acid (DNA) isolation and polymerase chain reaction (PCR). Out of the 900 samples examined, 189 (21%) were found to contain *Listeria* species which was found to be highly significant at 5% and 10% level of probability ($P = 0.01$; $P = 0.05$). The *Listeria* isolates included *L. monocytogenes* (41.3%), *L. ivanovii* (20.1%), *L. grayi* (14.3%), *L. welshimeri* (8.4%), *L. innocua* (6.9%), *L. murrayi* (5.8%) and *L. seeligeri* (3.2%). *Listeria* species occurrences were found to be highest in rabbit wastes (33.3%), followed by sheep wastes (27.8%), farm debris (23.3%), soil samples (22.4%), cow waste (22.2%), poultry droppings (20.0%), leaves (17.8%), and then water (13.3%). Samples of Human wastes were found to contain the least percentage of *Listeria* species (11.1%). The genotypic characterization of some *L. monocytogenes* isolates through polymerase chain reaction (PCR) technique into serotypes, showed that they belonged to monocytogenes serotypes 1/2a, 1/2b, 1/2c, 3a, 4a, 4ab, 4b, and 4c) Also *L. innocua*, *L. welshimeri*, *L. seeligeri*, *L. ivanovii*, *L. grayi* and *L. grayi* sub-species *murrayi* were present. The economic, medical and public health implications of the results obtained from the studies would serve as a valuable resource baseline for listeriosis surveillance in Nigeria.

References

1. Abdulkadir, I. (1989) In: Infectious Diseases of Livestock in Nigeria pp67-69. Ahmadu Bello University Press Ltd, Zaria.
2. Chukwu, O. O. C., Ogbonna, C. I. C., Goebel, W., Bubert, A., Nwobu, O. G. and Chima, C. J. (1997). Genital infection of a dairy animal attendant associated with *L. monocytogenes* in Vom, Nigeria. J. Med. Lab. Sci.6:12 – 14.
3. Chukwu, O.O.C, Ogo I. N., Antiabong, F.J., Muhammad, J.M., Ogbonna C.I.C. and Chukwukere, C.S. (2006a). Epidemiological evidence of listeriosis in Guinea

- Pigs fed with Cabbage (*Brassica oleracea*) in Nigeria. *Animal Production Research Advances* Vol.2 No.4, 248-252.
4. Chukwu, O.O.C., Ogbonna, C.I.C., Chukwu, D.I., Olabode, O.A., Onwuliri, F.C., and Nwankiti, O.O. (2006b). *Listeria monocytogenes* in Nigerian processed meats and Ready-Eat- Dairy Products. *Nig. J. of Microbiology* Volume 20 No.2, 900-904.
 5. Chukwu, O.O.C., Muhammad, M. J., Ngulukun, S. S., Ogbonna, C.I.C. and Olabode, O. A. (2007). Prevalence of *Listeria* species in slaughtered bovine carcasses in Jos abattoir, Nigeria. *Journal of Advanced Medical and Pharmaceutical Sciences* Vol. 1 No.2, 8-9
 6. Enurah, L. U., Ocholi, A. R., Ibu, O. J. and Nawathe, D. R. (1988). An outbreak of Avian listeriosis in Vom – Nigeria. *Trop. Vet.*6:61 – 62.
 7. Fenlon, D. R. (1985). Wild birds and silage as reservoirs of *Listeria* in the agricultural environment. *J. Appl. Bacteriol.* 59:537 – 543.
 8. Fenlon, D. R. (1986). Rapid quantitative assessment of the distribution of *Listeria* silage implicated in a suspected outbreak of listeriosis in calves. *Vet Rec*; 118:240 – 242.
 9. Hohne, K., Loose, B. and Seeliger, H. R. (1975). Isolation of *Listeria monocytogenes* in slaughter animals and bats of Togo (West Africa). *Ann. Microbiol. (Paris)*. 126 A (4):501 – 507.
 10. Gaillard, J. L., Berche, P., Mounier, J., Richard, S., and Sansonetti, P. (1987). In vitro Model and Intracellular Growth of *Listeria monocytogenes* in the Human enterocyt-like cell line Caco-s. *Infect. Immun.* 55: 2822-2829.
 11. Gray, M. L. and Killinger, H. A. (1966). *Listeria monocytogenes* and listeric infections. *Bacteriol. Rev.*30:309 – 382.
 12. Seeliger, H.P.R. (1987). Listeriosis: History and developments. Paper delivered at World Health Organization Consultation on Prevention and Control of Listeriosis, W. Berlin, December 10th 12th , p.3.
 13. Oni, O. O., Adesiyun, A. A., Adekeye, O. J. and Saidu, A. N. S. (1989). Prevalence and some characteristics of *L. monocytogenes* isolated from cattle and milk in Kaduna State, Nigeria. *Isr. J. Vet Med.* 45:12 – 17.
 14. Onyemelukwe, C. G., Lawande, V. R., Egler, J. L. and Mohammed, I. (1983). *L. monocytogenes* in Northern Nigeria. *J. Infection.*6:141 – 145.
 15. Schuchat, A., Lozano, C., Broome, V. C., Swaminathan, B., Kim, C., Winn, K. (1991a). Outbreak of neonatal listeriosis associated with mineral oil. *Paediatr. Infect. Dis. J.* 10:183 – 189.
 16. Schuchat, A., Deaver, A. K., Wenger, D. J. Reingold, A. L., Broome, V. C. and the *Listeria* study group (1992). Role of foods in sporadic listeriosis. I. Case control study of dietary risk factors. *JAMA.*267:2041 – 2045.
 17. Schuchat, A., Swaminathan, B. and Broome, V. C. (1991b). Epidemiology of human listeriosis. *Clin. Microbiol. Rev.*4:169 – 183.
 18. Schlech, W. F., Lavigne, M. P., Bortolulssi, A. R., Allen, C. A., Haldane, V. E., Wort, J. A., Hightower, W. A., Johnson, E. S., King, H. S., Nicholas, S. E., and Broome, C. V. (1983). Epidemic listeriosis: Evidence of transmission by food. *N. Engl. J. Med.*308:203 – 206.
 19. Vazquez-Boland, A. J., Kuhn, M., Berche, P., Chakraborty, T., Dominguez – Bernal, G., Goebel, W., Gonzalez – Zorn, B., Wehland, J. and Kreft, J. (2001). *Listeria* Pathogenesis and Molecular Virulent Determinants of and. *Clinical Microbiology Reviews.*14:584 – 640.
 20. Vogt, R. L., Donnelly, C., Gellin, B., Bibb, W., Swaminathan, B. (1990). Linking environmental and human strains of *L. monocytogenes* with isoenzyme and ribosomal RNA typing. *Eur. J. Epidemiol.*6:229 – 230.

21. Ward, D. W., Keller, R. and Bateson, M. M., (1990). 16S rRNA Sequences reveal numerous uncultured microorganisms in natural community. *Nature*.345:63 – 65.
22. Watkins, J. and Sleath, P. K. (1981). Isolation and enumeration of *L. monocytogenes* from sewage sludge and river water. *J. Appl. Bacteriol.*50:1 – 9.
23. Weaver, E. R. (1989). Morphological, physiological and biochemical characterization of *Listeria* species. In: Isolation and identification of *Listeria monocytogenes* (CDC Lab. Manual) ed: Jones, L. G. P.39 – 45.
24. Weis, J. and Seeliger, H. P. R. (1975). Incidence of *L. monocytogenes* in nature. *Appl Microbiol.*30:29 – 32.
25. Welshimer, H. J. (1968). Isolation of *L. monocytogenes* from vegetation. *J. Bacteriol.*95:300 – 303.
26. World Health Organization (1987) Report on Prevention and Control of listeriosis. World Health Organization, Geneva, Switzerland, WHO/CDS/VPH87. 69,p.2.
27. World Health Organization (1987) Report on prevention and Control of listeriosis. World Health Organization, Geneva, Switzerland, WHO/CDS/VPH87, 69, p.4.
28. World Health Organization (1988). Report of the informal working group on foodborne listeriosis. *Bull. WHO.* 66:421 – 428. Weis, J. and Seeliger, H. P. R. (1975). Incidence of *L. monocytogenes* in nature. *Appl Microbiol.*30:29 – 32.

¹¹EVALUATION OF THE ANTIVIRAL PROPERTIES OF THREE NIGERIAN PLANTS

L.K. Suleiman
Virology Research Division
NVRI, Vom

Ethno-Veterinary Medicine has been described as a holistic, interdisciplinary study of local knowledge and its associated skills pertaining to the healthcare and healthful husbandry of food, work, and other income-producing animals practical development applications within livestock production and livelihood systems, and with the ultimate goal of increasing human well-being via increased benefits from stock raising (McCorkle, 1986). Newcastle disease remains the most economically important disease of poultry in Nigeria and efforts gingered towards its control (including ethnoveterinary practices) are always considered an important issue in Veterinary circuits. This study was conducted to validate the antiviral properties of three Nigerian plant extracts against Newcastle disease virus, using in-vitro and in-ovo virological techniques. The justification for this includes largely the dearth of veterinary services in the rural poultry communities and the difficult logistics of drug and vaccine administration in small flock-holders.

Ethanollic extracts of *Adansonia digitata* and *Garcinia cola*, and aqueous (hot and cold) and were used in this study against a velogenic Newcastle disease virus strain. The extractions were performed according to AOAC Protocol. Extracts were diluted in antibiotic solution and tested at the concentration of 250mg/ml for toxicity to 10-day-old chicken embryos by inoculating 0.2ml of extract suspension per egg. The infective allantoic fluid containing the virus was titrated to determine the lethal dose ($LD_{50} = 108.3/ml$) and two suspensions of 100MLD/250mg/ml and 100MLD/200mg/ml were prepared as test inoculum. A graded dilution of *A. digitata* extract between 200mg/ml and 5mg/ml was also performed to determine the minimum virucidal concentration. 100MLD viral suspension was used in all cases as positive control while extract suspensions at 250mg/ml were used as negative control. Five eggs were inoculated per dilution while five eggs were also kept as uninoculated controls. 0.1ml of virus/extract suspension was inoculated through the allantoic route in all test and control cases apart from in embryo toxicity check in which 0.2ml of extract suspension was used. The virus/extract suspensions were incubated at +4°C before inoculation into the embryonated eggs.

Embryo mortality record post inoculation showed 0% for *A. digitata* and *A. sativum C* inoculated embryos while *G. cola* and *A. sativum M* inoculated embryos had 100% mortality after 96hours. This suggests good antiviral potential for *A. digitata* and *A. sativum C*. Graded doses of *A. digitata* also showed increasing mortality in embryo as proportional to decrease in extract concentration.

These findings suggest that the methanolic extracts of *A. digitata* and aqueous extracts of *A. sativum* may be of importance as an ethno-veterinary alternative in required to completely validate these findings.

¹¹ Seminar presented on 18th June 2009 at NVRI auditorium

¹²THE HAEMATOLOGY OF TRYPANOSOMIASIS

VICTOR O. ANOSA, D.V.M., M.V.M., Ph.D., FRCPATH
Professor of Veterinary Pathology, University of Ibadan
Currently: Visiting Professor of Veterinary Pathology,
College of Veterinary Medicine,
Michael Okpara University of Agriculture,
Umudike, Abia State

Trypanosomes

Trypanosomes are essentially all initially intravascular parasites.
Some species remain intravascular throughout their existence in the host:

Trypanosoma vivax,
T. congolense,
T. simiae.

Others are initially intravascular, but later spread extravascularly and invade tissues:

T. brucei,
T. evansi,
T. equiperdum,
T. gambiense,*
T. rhodesiense.*

*Cause human sleeping sickness

Key Concepts in the Pathogenesis of Trypanosomiasis

Associated with their intravascular existence, their widespread distribution in tissues resulting in tissue necrosis, as well as their perturbation of the immune system, trypanosome infections cause widespread pathologies in the circulating blood and associated haematopoietic organs. In addition to the haematological changes, trypanosome infections also cause widespread pathologies including encephalitis [hence sleeping sickness in man], myocarditis, nephritis, dermatitis, orchitis and ovarian cystic degeneration resulting in infertility, immunosuppression, etc. This seminar will be limited to the haematological changes induced by trypanosome infections.

Some Trypanosomiasis Models I Studied

- [i]. *T. brucei* infection in CFLP mice [trypanosusceptible] 1973/74
- [ii]. *T. vivax* infection in sheep and goats 1973 - 1976
- [iii]. *T. brucei* infection in deer mice [trypanotolerant] 1980/81
- [iv]. *T. vivax* infection in Guernsey-Ayrshire cattle [trypanosusceptible] 1987/88
- [v]. *T. congolense* infection of *Boran* cattle [trypanotolerant] 1991/92
- [vi]. *T. congolense* infection of *N'Dama* [trypanotolerant] and *Boran* cattle 1993/94
- [vii]. *T. congolense* and *Eimeria bovis* infections of German Holstein cattle [trypanosusceptible] 1996/97

Objectives of the Early Studies

- T. brucei* infections of CFLP mice and *T. vivax* infection of sheep and goats:
1. To evaluate the haematological changes caused by trypanosome infections
 2. To understand the parasite dynamics
 3. To study the gross and microscopic pathology
 4. To evaluate the role of the spleen during infection

¹² Seminar presented on 26th June 2009 at NVRI auditorium

5. To evaluate changes in the bone marrow

Results

The main haematological changes were:

Fluctuating parasitaemia

Anaemia

Leucopaenia due to neutropenia, eosinopaenia, lymphocytopenia and monocytosis

Thrombocytopenia

Therefore, there was **pancytopenia**

Bone Marrow showed:

*Expansion of red marrow in the long bones

*Hyperplasia of erythrocyte precursors

*Hypoplasia of granulocyte [leucocyte] precursors

* Drop in myeloid:erythroid [M:E] ratio

Features of Anaemia

Severe, particularly in sheep and goats:

PCV dropped below 10% in some animals

Appears to have a major haemolytic component based on erythrophagocytosis and haemosiderosis in the spleen and liver, and a reduced survival [life span] of RBCs:

*splenomegaly and hepatomegaly were always present

Moderately responsive in mice infected with *T. brucei*

Poorly responsive in sheep and goats despite severe anaemia:

*Reticulocytes averaged 0.1% [0 to 1.6] in sheep and goats with severe anaemia [mean PCV =14.5% (8 – 21)], compared to 29.5+7.4 [20 – 39] in sheep with haemolytic anemia due to *Anaplasma ovis* and *Babesia ovis* [PCV 13.7+ 3.9% (8 – 18)]

Objectives of Subsequent Studies

Since the bone marrow is the factory of most of the blood cells, I decided to study in several models investigate why:

The anaemia in not responsive in ruminants despite erythroid hyperplasia

There is always leucopaenia despite bone marrow hyperplasia

Why the differences in response between trypanotolerant and trypanosusceptible animals occur

Models Studied

T. vivax infection in Guernsey-Ayrshire cattle [trypanosusceptible] 1987/88

T. congolense infection of *Boran* cattle [trypanotolerant] 1991/92

T. congolense infection of *N'Dama* [trypanotolerant] and *Boran* cattle 1993/94

T. congolense and *Eimeria bovis* infections of German Holstein cattle

Results II

T. vivax and *T. congolense* infections of trypanosusceptible cattle induced similar haematological changes as seen in sheep and goats infected with *T. vivax*: pancytopenia, with poor reticulocyte response.

Thorough study of the bone marrow [BM] by light and transmission electron microscopy showed, for the first time, that the changes in blood, including the unresponsiveness, were determined by events in the BM:

- i. BM response was selective, hence while erythroid, monoblastic and megakaryocytic cells were hyperplastic, granulocyte precursors were hypoplastic.
- ii. Macrophages proliferated in the BM and were markedly activated.
- iii. The activated macrophages literally chewed [phagocytosed] large numbers of erythrocytes, neutrophils and eosinophils and their precursors, thereby reducing the capacity of the BM to respond effectively; thus there was dyshaemopoiesis with dyserythropoiesis and dysgranulopoiesis.

Results III

The role of the BM in bovine trypanotolerance was studied in *T. congolense* infections of trypanotolerant *Boran* [Zebu] cattle and trypanotolerant *N'Dama* cattle.

The *N'Dama* cattle had lower overall mean parasitaemia [$\log_{10} 2.23/\text{ml}$ of blood] than *Boran* cattle [$\log_{10} 3.00/\text{ml}$]. *N'Dama* cattle showed lower anaemia and leucopaenia than *Boran* cattle; while the *N'Dama* recovered to two-thirds of pre-infection levels by day 112, the changes in the *Borans* persisted at half the *N'Dama* levels. The BM of the *N'Dama* were hyperplastic, while those of the *Borans* were hypoplastic. Thus the *N'Dama* produced more blood cells than the *Boran*.

However, while macrophages proliferated equally in both breeds, the macrophages of the *N'Dama*, as measured by the surface area, were significantly [$p < 0.001$] more activated than those of the *Borans*. *N'Dama* macrophages also phagocytosed more erythroid and granulocytic cells and thrombocytes than *Boran* macrophages.

It was concluded that the BM plays a vital role in trypanotolerance, the macrophage serving as a key cell in determining the outcome of infection. The BM of the *N'Dama* produced more blood cells and also destroyed more than the *Boran*. But the balance between cell production and destruction was greater in the trypanotolerant cattle. With more active macrophages, and presumably immune system, the *N'Dama* were able to control parasitaemia better.

Conclusion

So much is now known about the haematology of trypanosomiasis than when I joined the quest for its understanding in 1973. However, wide gaps still remain. One area which has intrigued me is why a given macrophage engulfs several cell types particularly erythrocytes, neutrophils, eosinophils and thrombocytes, but seldom lymphocytes, most of which look normal initially? Could it be that the cells are first coated with trypanosome antigen and subsequently acquire anti-trypanosome antibody, rendering them commonly palatable to macrophages?

VICTOR O. ANOSA, D.V.M., M.V.M., Ph.D., FRCPATH

13 ADOPTED VILLAGE CONCEPT AND LIVESTOCK TECHNOLOGY DISSEMINATION

E.I. Ikani

**Veterinary Extension Research Liaison Service (current address), NVRI, Vom
National Agricultural Extension and Research Liaison Services, (NAERLS),
Zaria**

E-mail: ikani50@yahoo.com

Introduction

Livestock production constitutes a significant and important activity in the Nigerian economy. It forms the basis of over 6 million pastoralists' socio-cultural, economic and socio-political organization. However, despite the huge livestock resources in the nation (about; 24.3 million goats, 26.5 million Sheep, 19.8 million Cattle, 4.9 million Pigs, 1 million donkeys, 204,000 horses, 18,000 camels and 126 million Poultry (Ariyo, 2002), the country is yet to become self sufficient in animal protein intake. Growth rate in the sub-sector still lags behind those of crops and agro-forestry. One major reason for this poor performance can be traced to the little or no up-take of improved livestock technologies by end-users. Several attempts have been made by government with the purpose of overcoming this challenge of poor technology up-takes via extension intervention approaches. One of such intervention outreaches is the adopted village concept. The objective of this seminar is to discuss the Adopted village concept and in particular its principles, purpose and implementation protocols, and its implications for livestock technology dissemination

1. The concept of adopted village

- i. Introduced to the National Agricultural Research Institute System in 1996 at the tail end of NARP.
- ii. A model village for improved technology show-casing
- iii. A technology impact-making village
- iv. Fronting also as a farm service centre to make farm inputs readily available
- v. Strategies for shifting research outputs from the shelf to end-users
- vi. Demand-driven advocacy site for accessing useful information and linkages
- vii. On the whole to ensure enhanced production and productivity

2. Principles of adopted village

- i. A concept that involves many stake holders
- ii. All the stake holders do not get on board at the start and at the same time
- iii. Progressive increase in participants at the site should be expected
- iv. Emphasis is placed on best extension approaches
- v. From the on-set livestock intervention projects must be all inclusive

3. Goal /Purpose

- i. Field Validation of technologies
- ii. Increasing up-take of technologies
- iii. Identify/develop extension approaches for optimum technologies
- iv. Persuasive demonstration of research benefits

¹³ Seminar presented on 2nd June 2009 at NVRI auditorium

4. Implementation protocols

- i. Site selection-2 nearby villages or rural communities
- ii. Conduct baseline survey for the purpose of site characterization
- iii. Community entry / advocacy visits
- iv. Form village committees
- v. Training- at the community level using participatory methods
- vi. Identification of challenges and proffering solutions
- vii. Development of action plan
- viii. Technology demonstration
- ix. Documentation
- x. Field days
- xi. Monitoring and evaluation
- xii. Feed-back to funding agencies/policy makers

5. Implications for Livestock Technologies transfer

- i. Choice of site must be of a willing livestock farming nearby village
- ii. Baseline survey checklist should focus on livestock critical factors of production
- iii. Collaborations with other stakeholders very important
- iv. The intervention must be complete and holistic

Conclusion

Extension is the vehicle by which improved livestock technologies are conveyed to farmers for increased production and productivity. This fact cannot be downplayed if self-sufficiency in food production must be attained.

References

1. Ariyo, A.J. (2002) Livestock and fisheries in Africa Atlases Atlas of Nigeria les editions J.A.57 bis rue of Austenil-75016. Paris France: 98-99
2. Gefu J.O. and Otchere, E.O. (1994) Indigenous knowledge and sustainable livestock health management in West Africa: Livestock farming system research Programme, National Animal Production Research Institute, Zaria
3. Preston, T.R. (1986) Matching livestock systems with available resources in tropical countries. Published b y CTA. Ede-Wagenigen. The Netherlands: 10-13

Emmanuel Ike IKANI BSc, MSc, PhD



Dr Ikani holds a BSc. (Animal Science) from University of Ibadan (1983), MSc (Monogastric nutrition) from Ahmadu Bello University Zaria and a PhD (Livestock Extension) also from Ahmadu Bello University, Zaria. He is a senior Livestock Specialist currently being externally assessed as Associate Professor at the National Agricultural Extension Research and Liaison Services, ABU Zaria. His major area of research is impact assessment of livestock technologies. He has 30 articles in peer-reviewed publications and was in NVRI on sabbatical leave between January and December 2009. Born 5th August, 1958 at Affah in Ibaji LGA of Kogi State, Dr Ikani is married and blessed with six children.

**¹⁴SEROLOGICAL AND MOLECULAR STUDIES OF *MYCOPLASMA MYCOIDES*
MYCOIDES SMALL COLONY (MmmSC) IN NORTHERN NIGERIA**

***Nwankpa¹, N.; Lombin¹, L.; Oboegbulem², I.; Chah², K.; Manso-silvan⁴, L.; Yaya³, A. and Thiaucourt⁴, F.**

¹ National Veterinary Research Institute, Vom, Nigeria

² University of Nigeria, Nsukka, Nigeria

³ LANA VET, Garoua, Cameroun

⁴ CIRAD, Montpellier, France

***Corresponding author- nicknwankpa2004@yahoo.com**

Introduction

Contagious Bovine Pleuropneumonia (CBPP), an infectious and highly contagious disease of cattle caused by *Mycoplasma mycoides* subsp. *mycoides* Small Colony (MmmSC) biotype is currently the most economically important disease of cattle in Africa (Osiyemi, 1981; Terlaak, 1992). Because of its transmissibility and economic impacts, CBPP is now recognized as a priority transboundary disease and has thus been categorized as the only bacterial disease in the International Office of Epizootics (OIE) list A diseases (Litamoi *et al.*, 2004). The disease is considered to be the most economically important cattle disease in Africa causing greater losses in cattle than any other disease including Rinderpest (OIE, 2003). Today, Nigeria cannot export its abundant livestock and livestock products on account of CBPP and other trans-boundary diseases. The increasing importance of the disease emphasizes the need for an accurate data on the sero-prevalence of the infection and the different types of MmmSC isolates currently circulating in the country. This study is therefore aimed at establishing the presence of the disease in Northern Nigeria by serological and cultural techniques and characterising the isolates by current molecular tools. This study will also determine links between isolates and the distribution of the disease in the Northern region.

Methodology

A total of 2026 bovine sera samples from 12 states of Northern Nigeria were screened by c-ELISA technique. Tissue samples and nasal swabs were also collected from 5 Northern States and processed for isolation and identification of *Mycoplasma* organisms. The isolates of MmmSC were analyzed by Multilocus Variable-Number Tandem Repeat Analysis (MLVA) and Multilocus Sequence Analysis (MLSA) on two loci: Loc-PG1-0001(non coding region) and Loc-PG1-0103 (coding for a hypothetical lipoprotein) and Multilocus Variable-Number Tandem Repeat Typing (MLVA) on locus TR34 (within gene nat A). All PCR amplifications were performed using GeneAmp PCR Systems (Perkin Elmer) according to specific protocols. PCR products were controlled by electrophoretic separation in 1 % agarose gels. TR 34 PCR products were analyzed on 4 % agarose and polyacrylamide gels. The samples with relevant features were sequenced with the corresponding primers. The sequences were assembled and aligned with the software Vector NTI Suite TM.

Results

Ten out of twelve states in Northern Nigeria tested positive for CBPP by c-ELISA. The prevalence of infection ranged from 2.1 % to 32.5 %, with an overall prevalence of 10.8 %. Thirteen MmmSC isolates were recovered from the tissue samples processed and characterized into seven strains. With TR34, the isolates were

¹⁴ Seminar presented on 16th June 2009 at NVRI auditorium

characterized into 5 alleles. More precisions were brought by the other two loci with 3 and 4 alleles each. Seven profiles were defined for the thirteen isolates giving more precision in the definition of the origin of the strains. It is now possible with the combination of the three molecular techniques, to trace the source of a particular outbreak of CBPP in Northern Nigeria. A new allele, which had not been described previously, was recorded in two of the 13 isolates. This new allele was assigned no. 7. The isolates in this study are the only MmmSC isolates in Nigeria to be identified and characterized to molecular level. The study also indicated that isolates from bovine are the same with those from sheep making sheep a potential source of infection for bovine.

Acknowledgement

We thank N.V.R.I. Vom and Centre International Recherche Agronomic pour Development (CIRAD) Montpellier, France for funding the study and laboratory analysis respectively. I am most grateful to Dr. (Mrs.) L. H. Lombin, Chief Executive of NVRI Vom, my supervisors Drs. Oboegbulem, S. I., Chah, K. F., Manso-Silvan, L. and Thiaucourt, F., *Mycoplasma* Laboratory Staff Muraina, I., Kigbu, S. and Gyang, M. and the Almighty God.

References

1. Litamoi, J. K., Ayelet, G. and Rweyemamu, M. M. (2004). Evaluation of the xerovac process for the preparation of heat tolerant Contagious Bovine Pleuropneumonia (CBPP) vaccine. ELSEVIER. Article in Press (Received 23 April 2004, accepted 10 November 2004)
2. OIE, (2003). Contagious Bovine Pleuropneumonia , Manual of Standards. Chapter 2.1.6.
3. Osiyemi, T.I.O., (1981). The eradication of contagious pleuropneumonia in Nigeria: prospects and problems. Bull. Anim. Health Prod., Afr. 29, 95-97.
4. Terlaak, E.A., (1992). Contagious Bovine Pleuropneumonia . A review. Vet. Quart. 15, 104-110.

Distribution of alleles for combined MLVN TR, MLSA on loc-PG- 0001 and 0103 in Northern Nigeria MmmSC strains.

Strain ID No.	Location	TR34: f repeats	Loc-PG1-0001	PG1-0103: repeats	Loc-PG1-0103 - Alleles	Profiles
06045-C1	Vom	12	4	6	2	A
6050-C5	Bauchi	12	4	6	2	A
6060-C1	Kano	12	4	6	2	A
6052-C1	Kanam	8	4	8	4	B
6057-C2	Fadan Kaje	8	4	8	4	B
6061-C1	Jos	8	4	8	4	B
6058-C1	Vom	14	1	6	2	C
6059-C1	Kafanchan	4	1	7	3	D
6051-C1	Bauchi	9	1	9	5	E
6054C1	Sanga	12	7	9	5	F
6048-C1	Birnin Kebbi	12	7	9	5	F
6056-C1	Vom/Jos	8	4	7	3	G
6053-C1	B/Ladi	8	4	7	3	G
Global results		5 alleles	3 alleles	4 types	4 alleles	7 profiles

¹⁵VACCINATION AND COMPARISON OF THE POTENCY OF NEWCASTLE DISEASE VACCINE STRAINS I₂ AND LASOTA IN RURAL CHICKENS IN PLATEAU STATE, NIGERIA

Musa, Usman

Poultry Division, National Veterinary Research Institute, Vom

Email: usmanmusagulma@yahoo.com

Newcastle Disease (ND) has been recognized for a long time as one of the major production constraints to rural poultry. Management systems, thermolability, maladministration of vaccines and lack of extension services have been suggested as possible causes of outbreaks, spread and maintenance of ND. A study on management systems in rural household chickens in Plateau State was, therefore, conducted in 32 rural settlements of four Local Government Areas (LGAs) involving 1,240 households using a structured questionnaire. Also studied was ND antibodies and field vaccination with ND vaccine strain I₂ (NDVI₂) on 1,208 chickens raised under traditional management system. Thermostability of NDVI₂, vaccination trial and comparative immunogenicity tests using NDVI₂ and Lasota administered via different routes and vaccine carriers were investigated. The cost benefit of ND control using NDVI₂ via intraocular (i/o) route was estimated. The ND antibodies in rural and commercial chickens were evaluated using haemagglutination inhibition (HI) tests and the vaccines were titrated using embryonated chicken eggs. Diseases were the main (83.2%) causes of losses of rural chickens followed by preying (8.85%) and herbs were main remedies used for managing ND. Out of 1,208 sera tested, 627 (51.9%) had detectable antibodies to ND virus, but only 170 (14.1%) of the chickens had a protective HI antibody titre of $\geq 4\log_2$. About 1,041 (86.2%) of the chickens sampled were at risk of suffering from clinical ND. In the field, NDVI₂ administered by i/o route gave higher geometric mean titre (GMT) of $8.3 + 1.6\log_2$ and 92% protection rate than when administered through drinking water (dw) with GMT of $3.4 + 0.6\log_2$, yielding a 46% antibody protection rate. Reconstituted and lyophilized NDVI₂ vaccines maintained a minimum infective dose of $6.8\log_{10}$ and $8.4\log_{10}$ for 3 and 35 days, respectively from an initial titre of $10.2\log_{10}$. Reconstituted NDVI₂ lost $3.4\log_{10}$ titre after exposure at room temperature (RT) for 3 days. Lyophilized NDVI₂ lost only $1.8\log_{10}$ titre after exposure at RT for 35 days. NDVI₂ or ND Lasota vaccines administered through commercial feed, parboiled sorghum, parboiled sorghum coated with gum Arabic and untreated sorghum gave little protection (0 to 22%) to birds when challenged with velogenic viscerotropic ND virus. The financial losses due to ND outbreak in unvaccinated flocks in terms of the value of birds and eggs amounted to ₦1,250,505.9 and losses avoided following vaccination were ₦10,296,264. The benefit cost ratio arising from adopting NDVI₂ via i/o route for the control of ND in rural chickens is 7.2. It was concluded that the prevalence and risk of ND in rural chicken are very high. The NDVI₂ or NDV Lasota vaccines can protect commercial and rural chickens vaccinated through i/o, im or dw routes, while feeds were not suitable as vaccine carriers. In the field, ND vaccination with NDVI₂ via dw route may not be appropriate but the benefits derived in the use of NDVI₂ via i/o route were greater than the cost of the ND outbreak in unvaccinated flocks.

¹⁵ Seminar presented on 30th July 2009 at NVRI auditorium

Table 1: Distribution of Newcastle disease virus antibodies in chickens in households and live bird markets in four Local Government Areas of Plateau State

LGA	Number of sera samples tested	Number of sera with detectable antibodies		Mean \pm SD	Number of chickens protective for ND		Number of rural chickens at risk	
		F	%		F	%	F	%
Shendam	230	114	49.6	1.4 \pm 2.0	32	13.9	198	86.1
Kanam	226	97	42.9	1.1 \pm 1.8	24	10.6	202	89.4
Jos South	283	165	58.0	0.6 \pm 1.6	18	6.4	265	93.6
Barkin Ladi	223	121	54.3	1.3 \pm 1.7	22	10.0	204	91.5
Jos South (market)	198	103	52.0	2.2 \pm 3.3	58	29.3	140	70.7
Shendam (market)	48	27	56.3	2.5 \pm 3.7	16	33.3	32	66.7

Antibody titres (Log₂) of ≥ 4 Log₂ were considered positive to ND based on OIE recommendation of 2000, F = frequency, % = percentage
LGA = Local Government Area

Table 2: Geometric mean antibody titre (GMT) in sera of rural chickens vaccinated in the field with Newcastle disease vaccine strain I₂ administered through different routes.

Local government	Route of vaccination	Number of birds	GMT + SD		
			Pre-vaccination (n)	3 weeks post vaccination (n)	2 weeks post challenge (n)
Jos South	Intramuscular	15	0.1 + 0.3 (15)	7.6 + 2.8 (15)	10.0 + 0.8 (13)
Barkin Ladi	Control	15	0.2 + 0.1 (15)	0.2 + 0.2 (15)	7.0 + 0.2 (2)
Kanam	Intraocular	15	0.1 + 0.2 (15)	8.3 + 1.6 (15)	10.3 + 1.0 (14)
Shendam	Drinking water	15	0.1 + 0.0 (15)	3.4 + 0.6 (15)	7.0 + 0.6 (8)

(n) = number of chickens

Table 3: Geometric mean antibody titre (GMT) and standard deviation (SD) in sera of chickens vaccinated with NDVI₂ vaccine stored under different temperatures and challenged with Newcastle disease virus Kudu 113 strain.

Group	Storage	Route of administration	GMT + SD (Log ₂)			
			Prevaccination (n)	2 weeks post vaccination (n)	3 weeks post vaccination (n)	2 weeks post challenge(n)
A1	+4° C (365 days)	Drinking water	0.1 + 0.3 (10)	3.3 + 1.2 a (10)	5.1 + 2.1c (10)	8.0 + 0.8 e(10)
A2	+4° C (365 days)	Intraocular	0.0 + 0.0 (10)	5.1 + 0.7 a (10)	7.9 + 2.0 c (10)	8.8 + 2.0 e (10)
B1**	+23-29 °C (3 days)	Drinking water	0.1 + 0.3 (10)	5.1 + 0.2 a (9)	6.1 + 1.3c (9)	8.0 + 1.3 e (9)
B2**	+23-29 °C (3 days)	Intraocular	0.1 + 0.3 (10)	4.5 + 1.0 a (9)	5.9 + 0.9c (9)	8.5 + 2.7 e (9)
C1	On bench for 2 weeks	Drinking water	0.0 + 0.0 (10)	4.4 + 1.3 a (10)	6.1 + 2.1c (10)	6.9 + 0.8 e (9)
C2	On bench for 2 weeks	Intraocular	0.1 + 0.3 (10)	4.6 + 1.2 a (9)	6.1 + 2.0c (9)	9.0 + 1.6 e (9)
D1	On bench for 24 h	Drinking water	0.1 + 0.3 (10)	4.0 + 1.8 a (9)	7.0 + 2.4c (9)	9.3 + 1.8 e (9)
D2	On bench for 2 4 h	Intraocular	0.1 + 0.3 (10)	5.2 + 0.9 a (9)	6.2 + 1.3 c (9)	8.0 + 2.5 e (9)
E1	+45 oC for 24 h	Drinking water	0.0 + 0.0 (10)	4.3 + 0.8 a (9)	5.0 + 0.0 c (9)	6.6 + 0.5 e (9)
E2	+45 oC for 24 h	Intraocular	0.0 + 0.0 (10)	4.2 + 1.0a (8)	5.0 + 1.0 c (8)	6.8 + 0.5 e (8)
E3	Bench 24 hs. In feed	Commercial Feed	0.0 + 0.0 (10)	1.1 + 04 b (9)	0.2 + 1.6 d (9)	5.3 + 1.6 e (3)
Control	None	None	0.3 + 0.1 (10)	0.0+ 0.0 (10)	0.0 + 0.0 (10)	6.3 + 1.5 (3)

(n)* = Number of chicks, ** = Reconstituted NDVI₂

a, b, c, d and e = Means with the same letters in the same column are not significantly different at 0.05

¹⁶SERO-EPIDEMIOLOGY OF BRUCELLOSIS IN SMALL RUMINANTS IN PLATEAU STATE

***Bertu¹, W.J; Ajogi², I; Kwaga², J.K.P; Bale³, J.O.O; Ocholi¹, R.A**

***¹Bacterial Research Division, National Veterinary Research Institute, Vom**

²Department of Veterinary Public Health and Preventive Medicine, Ahmadu Bello University, Zaria

³Animal Reproduction Programme, National Animal Production Research Institute, Shika, Ahmadu Bello University, Zaria

***Corresponding Author (08035046018, wilchris2003@yahoo.com)**

Introduction

Brucellosis is one of the most important reproductive diseases of domestic and wild animals worldwide. It is caused by fastidious, intracellular, nonspore forming, non motile, non encapsulated, gram-negative coccobacillus bacteria of the genus *Brucella*. There are six well known species namely *B. abortus*, *B. melitensis*, *B. ovis*, *B. canis*, *B. suis* and *B. neotomae* which infect primarily Cattle, sheep and goats, sheep, dogs, pigs and desert wood rat respectively. However, cross infection do occur among the species. The major routes of infection in animals and man are by ingestion, inhalation and direct contact (Madkour, 1981). Brucellosis has been reported in Nigeria (Eze, 1978) and is a major cause of economic losses in the livestock industry. It is also a zoonosis of great public health significance. Losses in livestock are in terms of abortion, infertility, low conception rate and low survival rate of neonates (Oyedipe *et al.*, 1981).

Small ruminants make up the bulk of livestock population in Nigeria totalling 51million (FAO, 2006) and are major source of meat supply in Plateau state. The state is blessed with a small ruminant population of 964,188 sheep and 1,865,805 goats (Bourn *et al.*, 1992). Sheep and goats are reared together as well as in combination with cattle in communal grazing areas and this provides an opportunity for the spread of *Brucella* infection from cattle to small ruminants (Ocholi *et al.*, 2005).

The role of Brucellosis in limiting livestock production and its economic impact on the livestock industry in Nigeria is widely recognised (Ajogi, 2001). As a zoonosis of great importance, there is also a significant loss in human productivity due to brucellosis (Madkour, 1981). A comprehensive study on seroprevalence of brucellosis in Plateau state has not been carried out. This study is therefore aimed at determining the status of brucellosis in sheep and goats in Plateau state.

Materials and Method

Study area

The study was carried out in Plateau State. The State was divided into three senatorial zones from which three LGAs were selected from each zone. A total of 9 LGAs were used for the study based on the presence of large population of sheep and goats, presence of well established sheep and goat market and the presence of abattoir or slaughter slaps.

Sampling procedure

One thousand, three hundred and forty seven (1347) small ruminants comprising 851 goats and 496 sheep were sampled during the study using a systematic sampling method in which every third animal was sampled in every herd and abattoir in nine LGAs of Plateau state. 5ml of venous blood was collected from the jugular vein into vacutainer tubes or 10ml syringe in the case of field animals while clean sample bottles were used to collect 5ml of blood from slaughtered animals and were labelled and allowed to clot. They were centrifuged at 1000 rpm for 5 minutes. Serum was then decanted into 5ml plastic tubes and stored in the refrigerator at -20°C until required for testing.

Serological tests

Serum samples were tested for *Brucella* antibodies by Rose Bengal plate test (RBPT) and Serum agglutination test (SAT) as described by Alton *et al* (1988).

The results of the two tests were considered in parallel. Data were analyzed using Chi-square as described by Snedecor and Cochran, (1980).

Results

Out of a total of 496 sheep sampled, 72 (14.5%) were positive for brucellosis by both RBPT and SAT and 137 (16.0%) out of 851 goats sampled were positive by both RBPT and SAT.

Discussion and Conclusions

This study reveals an overall brucellosis prevalence of 14.5% in sheep and 16.1% in goats. This appears to be the first comprehensive serological study of small ruminant brucellosis in Plateau State. The Prevalence is high and widespread all over the state. This is not surprising since small ruminants are not being vaccinated against brucellosis coupled with the traditional practice of communal grazing in the state. This suggests a similarly high prevalence in humans since infection in humans is directly related to the infection in animals. This finding is comparable to that by Bale *et al.*, (1982) and Okoh who reported 14.1% and 14.5% prevalence respectively. The finding is however lower compared to 15.9% reported in a recent study by Bale *et al* (2003). Lower prevalences compared to this finding were by Okewole *et al* (1988), Shehu *et al* (1999) showing 12.05% and 6.6% respectively. In goats, the finding agrees with 16.1% reported by Bale *et al.*, (1982) but lower compared to 34.8% and 45.75% reported in recent surveys by Bale, *et al.*, (2003) and Ojo *et al.*, (2007) respectively. In conclusion, the prevalence of brucellosis is high in Plateau state and is widely distributed. The widespread prevalence is of economic and public health significance. There is need to put in place measures for the immediate control and eradication of brucellosis in the state. Culling of affected animals would be the first step towards a successful control.

Acknowledgements

The Executive Director, Dr Mrs L.H Lombin. My HODs, Dr P.A Okewole and Dr M.O.Odugbo. Dr. R. A. Ocholi, for his encouragement and expert advice. My supervisors Profs. I. Ajogi; J.O.O Bale, J.K.P. Kwaga. My colleagues in *Brucella* Lab. Dr. A.M Gusi, Mrs E. Mwankon, Mr M. Hassan, Mrs Rifkatu Dalyop and my family.

References

1. Alton, G.G., Jones, L.M., Angus, R.D. and Verger, J.M. (1988) Techniques for the brucellosis laboratory. Intitut National de la recherché agronomique, (INRA), Paris PP 63-129.
2. Ajogi, I and Akinwumi, J.A (2001). Cash-flow model of the cost of brucellosis in traditionally managed cattle herds in Nigeria. Bull. Anim. Health and Prod, Africa, 49:169-173.
3. Anonymous (2006). FAOSTAT Database. Food and Agriculture Organization, Rome, Italy.
4. Bale. J.O., Nuru .S. and Addo P.B; (1982) Serological study of sheep and goats Brucellosis in Northern Nigeria: Bull. Anim. Health and Prod. Africa, 30; 73 -79
5. Bale, J .O.O, S. Nuru, P.B. Addo and I.A. Adeyinka (2003). Bacteriological investigation of sheep and goats milk for brucellosis in government farms in northern Nigeria. Nigerian Journal of Animal Production. 30(1): 107 - 116.
6. Bourn, D; Wint, W; Blench, R; Wolley, E (1992) Nigerian Livestock Resources Survey.Environmental Research Group, Oxford Ltd. FAO World animal review, 78 (1): 49-58.
7. Eze E.N (1978) Isolation of *Brucellae* from the Nigerian livestock and the typing of such isolates. Bull. Anim. Hlth. Prod. Afri. 26, 29.
8. Ocholi, R.A; Kwaga, J.K.P; Ajogi, I; Bale, J.O.O (2005). Abortion due to *Brucella abortus* in sheep in Nigeria. Rev. Sci. Tech. Off. Int. Epiz: 24 (3) 973-979.
9. Ojo, O.E; Oyekunle, M.A; Omotainse, S.O; Ocholi, R.A; Ogunleye, A.O and Bertu, W..J (2007). Serological evidence of Brucellosis in a goat flock with recurrent abortion in Abeokuta. Nigeria. Tropical Veterinarian. 25: (1) 26-33.
10. Okewole, P.A.; Eze, E.N.; Okoh, A.E.J.; Oyetunde, I.L. and Odeyemi, P.S. (1988). Small ruminant brucellosis in some parts of Northern Nigeria. Bulletin of Animal Health and Production in Africa, 36:251-254.
11. Okoh, A.E.J. (1980). An investigation of abortion in sheep on Kano LIBC near Kano, Nigeria. Bull. Anim. Hlth. Prod. Afri. 28:135-136.
12. Oyedipe, E.O, Bavanendran,V and Eduvie, L.O (1981) .Factors affecting the reproductive performance of Fulani Cattle .NAPRI Seminar.
13. Shehu, L.M; Yusuf, H; Kudi, A.C and Kalla, D.U (1999). Seroprevalence of Brucellosis in ruminants in Bauchi and environs. Nigerian Veterinary Journal, 20 (1) 67-74.
14. Snedecor, G.W and Cochran, W.G.(1980). Statistical Methods. 7th ed. Iowa state university press, Ames

¹⁷EVALUATION OF THE EFFICACY OF AQUEOUS EXTRACT OF *SENNA OCCIDENTALIS* IN THE AMELIORATION OF TETRACYCLINE-INDUCED HEPATOTOXICITY AND NEPHROTOXICITY IN RABBITS

Abongwa M., ¹ Ahmed G. J., ²Arowolo O .A., ³ Dawurung C .J., ⁴ Oladipo O. O., ⁴ Atiku A., ⁴Okewole P. A.,⁴ Shamaki D⁴

¹University of Yaoundé Cameroun

²Forensic Laboratory, Egypt

³University of Ibadan, Ibadan Nigeria

⁴National Veterinary Research Institute Vom, Nigeria

Introduction

Problems of drug use in clinical practice are challenging the frontiers of the new millennium (Lucena *et al.*, 2001). Observations of toxicity in animals and of adverse reactions in humans are compelling reasons for terminating drug development, and for post-marketing withdrawal (Ballet, 1997). One of the most serious adverse effects of drugs in common use is hepatotoxicity (Bakke *et al.*, 1993; Farrel, 1994). Drugs shown to cause hepatotoxicity include carbon tetrachloride (Demirdag *et al.*, 2004), acetaminophen (Jafri *et al.*, 1999), and antimicrobials like the tetracyclines (Brown and Desmond, 2002). The management of liver disease is still a challenge to modern medicine, especially as hepatotoxicity is the most common single adverse effect causing major drug problems, including withdrawals and refusals to approve. In the absence of reliable liver-protective drugs in allopathic medical practice, herbs play a vital role in the management of liver disorders (Prakash *et al.*, 2008). *Senna occidentalis* (*Fabaceae*) is a leading plant used in African traditional medicine in the amelioration of hepatotoxicity.

Methodology

The shrubs of *Senna occidentalis* used for this study were obtained from Tilden- Fulani Local Government Area in Bauchi State, Nigeria. The plant was collected in June 2008 during morning hours. The plant was identified and confirmed by comparing with voucher specimens at the herbarium section of the Federal College of Forestry, Jos, Plateau State, Nigeria. Fresh leaves of the plant were allowed to dry in an oven at a regulated temperature of 40°C. The dried leaves were pulverized with a mortar and pestle, and 700g of the powdered sample was used to obtain an aqueous extract. The extract was then screened for the following phytochemical principles: alkaloids, anthraquinones, cardiac glycoside, flavonoids, saponins, steroids, and tannins. For the acute toxicity studies, six rabbits (New Zealand white/local cross breeds) were randomly selected and divided into 3 groups of 2 animals each. The OECD method was adopted for this study. For the evaluation of the efficacy of extract in ameliorating tetracycline-induced hepatotoxicity and nephrotoxicity, 18 rabbits were divided into 6 groups of 3 animals each. All groups received oral treatments administered by gastric intubation for 14 days. The control group was given feed and water *ad libitum*, and distilled water intubated daily. A negative control group was given feed and water *ad libitum*, and treated with 1000 mg/kg body weight Tetracycline hydrochloride every 48 hours. A third group received 1000mg/ kg body weight of Tetracycline hydrochloride every 48 hours, and 100 mg/kg body weight of aqueous plant extract daily. A fourth

¹⁷ Seminar presented on 19th August 2009 at NVRI auditorium

group received 1000 mg/kg body weight of Tetracycline hydrochloride every 48 hours, and 50 mg/kg body weight of aqueous plant extract daily. Group V received 1000 mg/kg body weight of Tetracycline hydrochloride every 48 hours, and 25 mg/kg body weight of aqueous plant extract daily. Rabbits in group VI received 100 mg/kg body weight of aqueous plant extract only daily.

At the termination of the experiment, blood samples were collected and divided into two portions, one portion for haematology, and the other for biochemical analysis. The animals were humanely sacrificed for gross pathology and tissues were harvested for histopathology.

Results

The aqueous leaf extract of *Senna occidentalis* produced 18.97% w/w yield, with the extract being dark brown in colour, sticky, readily soluble in water, and not soluble in chloroform. The phytochemicals found are listed below.

Phytochemicals	Results
Alkaloids	–
Anthraquinones	–
Saponins	+
Tannins	+
Flavonoids	+
Cardiac glycosides	+
Steroids and Terpenes	+

Key: + = present; – = absent

For acute toxicity studies, none of the rabbits administered the extract died within the study period. The LD₅₀ of the aqueous leaf extract (Oral, rabbits) is >2000 mg/kg. In the Sub acute toxicity study with the administration tetracycline and *S. occidentalis* aqueous extract, (groups II, III, IV and V); varying degrees of hyperpnoea and hypopnoea, bradycardia and tachycardia were observed immediately after dosing. The animals also showed pale ocular mucous membranes, decrease feed intake, decreased weight, and changes in faecal consistency. Haematological analyses revealed significant increases ($p < 0.05$) in Packed Cell Volume (PCV) and decreases in red blood cell count (RBC). Serum biochemistry indicated significant increase ($p < 0.05$) in the serum level of ALT and AST of rabbits in group II (treated with tetracycline only) when compared to the control group. The serum ALT and AST levels decreased in animals in groups III, IV, and IV (taking varying doses of the plant extract) when compared to group II. There was an insignificant decrease ($p > 0.05$) in the serum albumin level in all the groups (II, III, IV, V, VI) when compared to the control group taking distilled water only. There was also a significant increase ($p < 0.05$) in serum creatinine and urea levels in the group taking tetracycline only when compared to animals in the control group taking distilled water only. Animals varying doses of the extract in addition to tetracycline (groups III, IV, V) also had increased serum creatinine and urea levels. Group VI animals (highest dose of extract only) had increased serum creatinine level and decreased urea level when compared to control group I. Pathological changes were observed in liver, lung, spleen, brain, kidney, large and small intestine which were further confirmed at histopathology.

Discussion

A repeated oral dose of Tetracycline HCl was observed to have caused severe weight loss in the test animals. This was expected because an overdose of the antibiotic

would distort the normal microbial flora in the intestinal tract, especially in the caecum of rabbits. However, *S. occidentalis* aqueous leaf extract seemed to have a dose-dependent ameliorative effect on weight loss. Animals treated with the plant had lesser weight losses than animals on tetracycline only, with the animals taking the highest dose of the extract (100 mg/kg) having the least weight loss. A significant increase in the level of serum liver enzymes, alanine aminotransferase, and aspartate aminotransferase is indicative of tetracycline-induced hepatocellular injury. This was confirmed from histopathology examination of the liver. *S. occidentalis* aqueous extract seemed to have an ameliorative effect on induced hepatocellular injury by causing a reduction in the levels of serum enzymes assayed, and with milder hepatocellular lesions. The increase in serum creatinine and urea levels are indicative of tetracycline-induced renal injury (Schultz *et al.*, 1972), as confirmed by glomerular vacuolar degeneration, tubular necrosis, inflammatory cellular response in intertubular connective tissue, and hyaline degeneration of the tubules following histopathological examination of kidney sections. The plant extract seemed to show some ameliorative effect by reducing the level of these excretory substances in circulation. The presence of flavonoids and tannins, which are strong polyphenolic antioxidant compounds and present in the plant extract could be responsible for the ameliorative effect on tetracycline-induced hepatocellular injury and nephrotoxicity. The compounds stimulate bile production in the liver and increased bile release from the gall bladder thus eliminating toxic substances, normalizing blood cholesterol levels, lowering blood lipids, and providing liver protective qualities (Van *et al.*, 1998; Adzet *et al.*, 1987).

Acknowledgement

We acknowledge the African Education Foundation (NEF) and NVRI for funding this project and all our mentors in NVRI.

References

15. Adzet T, *et al.* (1987): Hepatoprotective Activity of Polyphenolic Compounds from *Cynara scolymus* Against CC14 Toxicity in Isolated Rat Hepatocytes, *Journal of Natural Products*, 50: 612.
16. Bakke, O. M., Manocchia M, de Abajo, F., Kaitin K. L. and Lasagna, L. (1995). Drug safety discontinuations in the United Kingdom, the United States, and Spain from 1974 through 1993: A Regulatory Perspective. *Clin Pharmacol Ther* 58: 108 – 117.
17. Brown, S. J., and Desmond, P. V. (2002). Hepatotoxicity in the twenty-first century. *Seminars in Liver Disease* 22 (2).
18. Demirdag. K., Bakcecioglu, I. H., Ozercan, I. H., Ozden, M., Yilmaz, S. and Kalkan, A. (2004). Role of L-carnitine in the prevention of acute liver damage induced by carbon tetrachloride in rats. *Journal of Gastroenterology and Hepatology* 19: 333 – 338.
19. Jafri, M. A., Subhani, M. J., Javed, K., and Singh S. (1999). Hepatoprotective activity of leaves of *Cassia occidentalis* against paracetamol and ethyl alcohol intoxication in rats. *Journal of Ethno pharmacology* 666: 355 – 361.
20. Lucena, M.I., Carnago, R., Andrade, R., Perez-Sanchez, C.J. and Sanchez De La Cuesta, F. (2001). Comparison of Two Clinical Scales for Causality Assessment in Hepatotoxicity. *Hepatology* 33(1): 123 – 130.
21. [Prakash](#), O., [Singh](#), G. N., [Singh](#), R. M., [Mathur](#), S. C., [Bajpai](#) M. and [Yadav](#), S. (2008): Protective Effect of a Herbal Formula Against Carbon tetrachloride Induced Hepatotoxicity. *International Journal of Pharmacology*. Vol 4(4): 282-286.

22. Schultz, J.C., Adamson, J.S., Workman, W.W, and Norman, T.D. (1972). Fatal liver disease after intravenous administration of tetracycline in high doses. *New Engl. J.Med.* 269:999 – 1004.
23. Van, Acker, S. *et al.* (1998): Structural Aspects of Antioxidant Activity of Flavonoids. *Flavonoids in Health and Disease*, Rice-Evans, C. editor, Marcel Dekker, Inc.

¹⁸AMELIORATIVE EFFECTS OF *PHYLLANTHUS NIRURI* AQUEOUS EXTRACT ON HEPATORENAL CHANGES FOLLOWING SUB-ACUTE EXPOSURE TO ACETAMINOPHEN IN RABBITS

Nwatu, I. L., Adanyeguh, M. I., Makoshi, S. M., Oladipo, O. O., * Atiku, A., Dawurung, C. J., Okewole, P. O. and Shamaki, D.

National Veterinary Research Institute, Vom, Plateau State, NIGERIA

***Corresponding author: oladiposola@ymail.com**

Introduction

Acetaminophen is the main cause of poisoning worldwide. It is a safe and effective analgesic and antipyretic at therapeutic levels, which can cause severe toxicity in experimental animals and humans when given as overdose (Ghanem *et al.*, 2009). There is increasing incidence of acetaminophen abuse leading to increased incidence of hepatotoxicity and nephrotoxicity. The antidote for these toxicities is not readily available and affordable. Acetaminophen relieves pain by elevating the pain threshold (Milton, 1976). It reduces fever through its action on the heat-regulating centre of the brain (Botting, 2000). The toxic dose of paracetamol is highly variable. In adults, single doses above 10 grams or 150 mg/kg have a reasonable likelihood of causing toxicity. Toxicity can also occur when multiple smaller doses within 24 hours exceed these levels, or even with chronic ingestion of doses as low as 4 g/day, and death with as little as 6 g/day (Dart *et al.*, 2006). Acetaminophen in overdose results in hepatotoxicity and nephrotoxicity in man and experimental animals (Vermeulen *et al.*, 1992). Damage to the liver results not from paracetamol itself, but from the formation of the reactive and toxic metabolite, N-acetyl-p-benzoquinoneimine (NAPQI) by the cytochrome P-450 system that covalently binds to cellular macromolecules and initiates cell damage (Vermeulen *et al.*, 1992) leading to glutathione depletion and liver susceptibility to oxidative stress (Kostopanagiotou *et al.*, 2009).

Few plants have been evaluated for their protective effect on drug-induced hepatotoxicity and nephrotoxicity. One of such plants is *Phyllanthus niruri*. This plant is herbaceous and a member of *Euphorbiaceae*. It is native in to India and is found in many parts of the world. It is known for a variety of uses like hepatoprotection, lipid lowering, antidiabetic antimalarial (Chandra *et al.*, 2000; Neraliya *et al.*, 2004) and as an analgesic (Santos, 1994). The primary compounds responsible for its liver healing property are *phyllanthin*, *hypophyllanthin* and *triacontanal*. Its use and awareness of its therapeutic potential is low in this part of the world. Very little research has been conducted to evaluate its efficacy in acetaminophen-induced toxicities. The aim of this study was to evaluate the protective effect of aqueous crude extract of *P. niruri* on acetaminophen-induced hepatotoxicity and nephrotoxicity in rabbits.

Methodology

Plant collection and identification

Fresh plants of *Phyllanthus niruri* used in the study were obtained from Zaria, Nigeria. The plant was identified according to the description of Dalziel (1937) and was further authenticated by a voucher specimen at the herbarium of Ahmadu Bello University, Zaria. An aqueous extract of the plant was prepared according to the method described by Sofowora (1993). The standard methods of Trease and Evans (1989) were used in the analysis of the phytochemical components of the plant.

¹⁸ Seminar presented on 19th August 2009 at NVRI auditorium

Chemical acquisition and preparation

Acetaminophen was obtained from a reputable pharmaceutical company in Jos, Nigeria. It was reconstituted in distilled water to make a 5 g% solution. All other chemicals and reagents used for laboratory analysis were of good laboratory grade and were obtained from reliable chemical stores.

Experimental animals

Twenty-four (24) male grower rabbits were obtained from the small animal experimental unit of the National Veterinary Research Institute, Vom, Nigeria. They were housed and acclimatized to the conditions of the experimental house of the institute for seven days before the commencement of the experiment. Six rabbits were randomly selected for the determination of mean lethal dose of *Phyllanthus niruri*, while the remaining eighteen were randomly divided into six groups of 3 rabbits each. The animals were fed on pellets produced by the NVRI-Vom feed-mill. Feed and water were provided ad libitum. The LD50 of *P. niruri* was determined using the OECD (2001) test for acute toxicity.

Experimental design

This study was conducted to evaluate the protective effect of aqueous crude extract of *Phyllanthus niruri* at varying doses on some haematological and biochemical parameters following sub-acute exposure to acetaminophen (APA) in rabbits. Twenty-four male grower rabbits were used for this study. Six rabbits were randomly selected for the determination of mean lethal dose (LD50) of *P. niruri*, while the remaining rabbits were randomly divided into 6 groups of 3 rabbits each. Rabbits in group I served as control and administered orally with distilled water only. Animals in group II were administered with APAP only at a dose of 300 mg/kg. Groups III, IV and V were treated with 25 mg/kg, 50 mg/kg and 100 mg/kg of aqueous extract of *P. niruri* respectively 30 minutes after administration of APAP. Rabbits in group VI were treated with the aqueous extract of *P. niruri* only at 100 mg/kg. The regimens were administered orally once daily and every other day for the plant and APAP, respectively, for a period of 15 days. The effect of treatments on body weight change was determined every other day throughout the study period. The rabbits were observed for signs of toxicity and death. At the end of the experiment, each rabbit was bled via the Venus auricularis. Heparinised blood sample was used for the evaluation of packed cell volume (PCV), haemoglobin concentration (Hb), total erythrocyte count (RBC), total leukocyte counts (WBC), mean corpuscular haemoglobin (MCH), mean corpuscular volume (MCV) and mean corpuscular haemoglobin concentration (MCHC). Serum biochemical parameters assayed for were; alanine amino transferase (ALT), aspartate amino transferase (AST), total protein, albumin, creatinine and urea. At the end of the experiment, all the rabbits were euthanized by injecting air into their heart to form emboli after which gross and histopathological examinations of the liver and kidneys was conducted. Values obtained were expressed as mean \pm SEM. One-way analysis of variance (ANOVA) with Tukey's multiple range comparison post-hoc tests were performed on data using Graph pad prism to compare the level of significance between the test groups and control. Values of $P < 0.05$ were considered significant.

Results

The phytochemical screening of the aqueous extract revealed the presence of saponin, tannin, cardiac glycoside, steroids and flavonoids. Alkaloid, anthraquinone and terpenes were, however, absent. The mean lethal dose of *P. niruri* was found to be > 2000 mg/Kg. Clinical signs of toxicity were inapparent during the course of the experiment. There was also, no significant ($P > 0.05$) difference in body weight gain between the treatment groups. Rabbits administered APAP

showed a significant ($P < 0.05$) increase in serum albumin and total bilirubin concentration compared with the control. Animals treated with the plant extract showed a decrease ($P < 0.05$) in the values of aspartate aminotransferase (AST) (Fig. 2), albumin (Fig. 1), creatinine and total bilirubin (Table 1) compared with the control and APAP groups. Histopathological examination of the liver revealed periportal cellular infiltration, centrilobular and coagulative necrosis, while the kidney showed vascular congestion compared with the control and the plant-treated groups. The values of PCV and Hb were not significantly ($P > 0.05$) different between the groups. There was, however, a slight increase in the value of these parameters in the rabbits treated with the plant extract, compared with the groups administered acetaminophen. There was also no change in RBC in the animals, which received acetaminophen, but a significant ($P < 0.05$) decrease was observed in the groups treated with the plant extract compared with the control and acetaminophen groups (Fig. 3). An increase in the values of MCV, MCH and MCHC was observed in the groups treated with the extract, and this increase was found to be dose-dependent. Rabbits given APAP only showed an increase in the number of circulating leucocytes, compared with the control and treatment with the plant resulted in a marginal decrease in WBC. (Table 2).

Table 1: Effect of *Phyllanthus niruri* on some serum biomarkers following sub-acute exposure of rabbits to acetaminophen

Treatment groups	Total bilirubin (mg/dl)	Creatinine (mg/dl)	Urea (mg/dl)
Control	0.77 ± 0.06	18.67 ± 2.91	85.90 ± 12.09
APAP	1.56 ± 0.06*	16.33 ± 1.76	130.10 ± 12.68
APAP + 25 mg/kgPN	1.77 ± 0.14*	13.33 ± 1.45	95.86 ± 8.33
APAP + 50 mg/kgPN	2.28 ± 0.27*	39.00 ± 16.74***	112.82 ± 12.39
APAP + 100 mg/kgPN	1.66 ± 0.21*	9.33 ± 0.88**	129.32 ± 15.49
100 mg/kgPN	1.31 ± 0.18*	3.33 ± 0.67**	73.76 ± 14.35**

Values are expressed as mean ± SEM, Values of $P < 0.05$ are considered significant. N = 3, APAP = acetaminophen, PN = *Phyllanthus niruri*

Table 2: Effect of *Phyllanthus niruri* on some haematological parameters in rabbits following sub-acute exposure to acetaminophen

Treatment groups	MCV	MCH	MCHC
Control	47.92 ± 14.94	2.67 ± 0.03	375.33 ± 5.24
APAP	40.72 ± 12.97	2.80 ± 0.12	358.00 ± 14.47
APAP + 25 mg/kgPN	46.27 ± 6.57	2.87 ± 0.03*	349.67 ± 6.67*
APAP + 50 mg/kgPN	44.93 ± 2.44	3.10 ± 0.10*	320.33 ± 11.33*
APAP + 100 mg/kgPN	84.87 ± 1.26**	2.50 ± 0.06	401.00 ± 10.58
100 mg/kgPN	74.59 ± 11.78	2.63 ± 0.07	381.00 ± 7.51

Values are expressed as mean ± SEM, Values of $P < 0.05$ are considered significant. N = 3, MCV = Mean corpuscular volume, MCH = Mean corpuscular haemoglobin, MCHC = Mean corpuscular haemoglobin concentration, APAP = Acetaminophen, PN = *Phyllanthus niruri*. *Different ($P < 0.05$) from the control, **Different ($P < 0.05$) from the control and APAP group.

Discussion

The present study showed that sub acute administration of acetaminophen in rabbits had no effect on body weight. This finding is agreement with that of Ghanem *et al.* (2009). The presence of polyphenolic compounds in the plant extract may be responsible for the pharmacological effects of this plant. Polyphenols has been shown to have antioxidative effects as shown by several authors (Wattanapitayakul *et al.*, 2005; Nwanjo, 2006). The study showed that acetaminophen induced hepatorenal damage in rabbits as demonstrated by the increase in albumin and total bilirubin concentrations. This finding is in agreement with previous reports by several authors (Nwanjo, 2006; Mazer and Perrone, 2008; Ghanem *et al.*, 2009), who demonstrated the elevations in liver and kidney markers in rats and mice, and rabbits (Wershana *et al.*, 2001; Zira *et al.*, 2009). Liver damage impairs protein synthesis and increases protein concentration due to leakage from hepatocytes (Rajesh *et al.*, 2009). Likewise, at the marginally low dose (300 mg/kg) used in this study, the slight but insignificant ($P > 0.05$) increase in the values of AST and ALT further demonstrates the potential of acetaminophen to induce hepatorenal damage. Microscopic examination of the liver and kidneys of acetaminophen-exposed rabbits further lends support to the ability of this analgesic to induce hepatorenal alterations. Again, this result agrees with those of Kuvandik *et al.* (2008), Chen *et al.* (2009) and Wilhelm *et al.* (2009).

Treatment with *P. niruri* aqueous extract showed a protective effect on the liver and kidneys, as demonstrated by the decrease in the levels of the serum biomarkers and this effect was found to be dose-dependent. Those treated with the plant extract showed the best response. The fact that rabbits treated with the plant extract only, showed significant ($P < 0.05$) decrease in most of the parameters further confirms the potential of *P. niruri* to maintain the integrity of the liver and kidneys. The findings of this study agree with those of several authors (Lee *et al.*, 2006; Faremi *et al.*, 2008; Manjrekar *et al.*, 2008; Adeneye and Benebo, 2009). The fact that acetaminophen showed a slight but insignificant ($P > 0.05$) decrease in the blood parameters of rabbits in this study, demonstrates that treatment with this analgesic at a relatively low dose may not be sequelae of haematological alterations. Other studies however showed that an over-dose of acetaminophen results in a significant decrease in blood parameters in rabbits (Wershan *et al.*, 2001). Treatment with the *P. niruri* aqueous extract showed significant reductions in blood parameters. The aqueous extract steroidal contains saponins, which have been shown to possess haemolytic effects (Oda *et al.*, 2000). The decrease in total erythrocyte count observed in the groups treated with the plant extract may be due to the haemolytic effect of saponins. This further resulted in a marginal decrease in PCV and a resultant increase in MCHC. The tissue microscopic findings in this study further supports the postulation that *P. niruri* may cause haematological alterations.

Conclusion

Acetaminophen induces hepatorenal alterations in rabbits as demonstrated by the increases in some liver and kidney biomarkers. It also caused congestion, haemorrhage, coagulative, periportal and centrilobular necrosis of the liver and vascular congestion of the kidney tubules. Treatment with *Phyllanthus niruri* aqueous extract ameliorated these effects probably due to the presence of polyphenolic compounds in the plant. Treatment of rabbits with the plant at high doses resulted in haematological alterations. The effect of *P. niruri* was found to be dose-dependent.

Recommendation

Further work is needed to determine the active principles responsible for the action of *P. niruri*. Molecular studies are needed to elucidate the mechanism of action of this plant.

Acknowledgement

The authors wish to acknowledge the Executive Director, NVRI, Dr (Mrs) L. H. Lombin, MFR and the President, Africa Education Initiative (NEF), USA, Dr Chudy Nduaka for funding this project. We also acknowledge the contributions of staff of NVRI for their support throughout the 3-month Toxicology Internship Programme.

References

1. Adeneye, A. A., and Benebo, A. S. (2008). Protective effect of the aqueous leaf and seed extract of *Phyllanthus amarus* on gentamicin and acetaminophen-induced nephrotoxic rats. *Journal of Ethno pharmacology*, 118 (2): 318-323.
2. Botting R.M. (2000). Mechanism of action of acetaminophen: Is there a cyclooxygenase 3? *Clinical Infectious Disease*; 31(Supp 5):S202-S210.
3. Chandra R. (2000). Lipid lowering activity of *P. niruri*, *Journal of Medicinal and Aromatic Plant Sciences*. 22 (1): 29-30.
4. Chandra R. (2000). Lipid lowering activity of *Phyllanthus niruri*. *Journal of Medicinal and Aromatic Plant Sciences*, 22 (1): 29-30.
5. Chen, Y. H., Lin, F. Y., Liu, P. L., Huang, Y. T., Chiu, J. H., Chang, Y. C., Man, K. M., Hong, C. Y., Ho, Y. Y. and Lai, M. T. (2009). Antioxidative and hepato protective effects of *magnol* on acetaminophen-induced liver damage in rats. *Archives of Pharmacol Research*, 32 (2): 221-228.
6. Dalziel, M. (1937). *The useful plants of West Tropical Africa*. Crown Agents, London. 612 pp.
7. Dart R.C., Erdman A.R., Olson K.R. *et al.*, (2006). American Association of Poison Control Centres. "Acetaminophen poisoning: an evidence-based consensus guideline for out-of-hospital management". *Clinical Toxicology*, 44 (1): 1-8.
8. Faremi, T. Y., Suru, S. M., Fafunso, M. A. and Obioha, U. E. (2008). Hepatoprotective potentials of *Phyllanthus amarus* against ethanol-induced oxidative stress in rats. *Food and Chemical Toxicology*, 46 (8): 2658-2664.
9. Ghanem, C. I., Ruiz, M. L., Villanueva, S. S., Luquita, M., Llesuy, S., Catania, V. A., Bengochea, L. A. and Mottino, A. D. (2009). Effect of repeated administration to rats on enterohepatic recirculation of a subsequent toxic dose. *Biochemical Pharmacology*, 77 (10): 1621-1628.
10. Kostopanagiotou, G. G., Grypioti, A. D., Matsota, P., Mykoniatis, M. G., Demopoulos, C. A., Papadopoulou-Daifoti, Z. and Pandazi, A. (2009). Acetaminophen-induced liver injury and oxidative stress: protective effect of propofol. *European Journal of Anaesthesiology*, 26 (7): 548-553.
11. Kuvandik, G., Duru, M., Nacar, A., Yonden, Z., Helvacı, R., Koc, A., Kozlu, T., Kaya, H. and Sogut, S. (2008). Effects of erdosteine on acetaminophen-induced hepatotoxicity in rats. *Toxicologic Pathology*, 36 (5): 714-719.
12. Lee, C. Y., Peng, W. H., Cheng, H. Y., Chen, F. N., Lai, M. T. and Chiu, T. H. (2006). Hepatoprotective effect of *Phyllanthus* in Taiwan on acute liver damage induced by carbon tetrachloride. *The American Journal of Chinese Medicine*, 34 (3): 471-482.
13. Manjrekar, A. P., Jisha, V., Bag, P. P., Adhikary, B., Pai, M. M., Hegde, A. and Nandini, M. (2008). Effect of *Phyllanthus niruri* Linn. Treatment on liver, kidney and testes in CCl₄-induced hepatotoxic rats. *Indian Journal of Experimental Biology*, 46 (7): 514-520.

14. Mazer, M. and Perrone, J. (2008). Acetaminophen-induced nephrotoxicity: Pathophysiology, clinical manifestations and management. *Journal of Medical Toxicology*, 4 (1): 2-6.
15. Meixa W., Haowei C., Yanjin L. *et al.*, (1995). Herbs of the genus *Phyllanthus* in the treatment of Chronic hepatitis B observation with three preparation from different geographic sites, *Journal of Laboratory and Clinical Medicine*, 126 (2): 350.
16. Milton A.S. (1976). Modern views on the pathogenesis of fever and the mode of action on antipyretic drugs. *Journal of Pharmacy and Pharmacology*, 28: 393-399.
17. Neraliya S., Gaur R. (2004). Juvenoid activity in plant extracts against filarial mosquito *Culex quinquefasciatus*. *Journal of Medicinal and Aromatic Plant Sciences*, 26 (1): 34-38.
18. Nwanjo, H. (2007). Studies on the effect of aqueous extract of *Phyllanthus niruri* leaf on plasma glucose level and some hepatospecific markers in diabetic Wistar rats. *The Internet Journal of Laboratory Medicine*. Volume 2, Number 2.
19. Oda, K., Matsuda, H., Murakami, T., Katayama, S., Ohgitani, T. and Yoshikawa, M. (2000). Adjuvant and haemolytic activities of 47 saponins derived from medicinal and food plants. *Biological Chemistry*, 381: 67-74.
20. OECD (2001). OECD Guideline for Testing of Chemicals: Acute Oral Toxicity-Up-and-Down Procedure. No. 425, Adopted 17 December 2001.
21. Prescott, L. F. (1983). Paracetamol over dosage: Pharmacological considerations and clinical management. *Drugs*, 25:290-314.
22. Rajesh, S. V., Raj Kapoor, B., Kumar, R. S. and Raju, K. (2009). Effect of *Clausena dentata* (Wild.) M. Roem. Against paracetamol-induced hepatotoxicity in rats. *Pakistan Journal of Pharmaceutical Sciences*, 22 (1): 90-93.
23. Santos, A. R. (1994). Analgesic effects of callus culture extracts from selected species of *Phyllanthus* in mice. *Journal of Pharmacy and Pharmacology*. 46 (9): 755-759.
24. Sofowora, A. (1993). *Medicinal Plants and Traditional Medicine in Africa*. University of Ife Press, Nigeria. Pp 1-23.
25. Trease, G. E. and Evans, W. C. (1978). *Pharmacology*. 11th Edition, Bailliere Tindall, Ltd, London. pp 60-75.
26. Vermeulen, N. P. E., Bessems, J. G. M. and Van De Straat, R. (1992). Molecular aspect of paracetamol-induced hepatotoxicity and its mechanism based prevention. *Drug Metabolism*, 24: 367-407.
27. Wershana, K. Z., Daabees, A. Y. and Shaban, W. M. (2001). The role of two antidotes in the prevention of acetaminophen-induced toxicity in male rabbits. *Journal of Medical Sciences*, 1 (3): 157-175.
28. Wilhelm, E. A., Jesse, C. R., Leite, M. R. and Nogueira, C. W. (2009). Studies on preventive effects of diphenyl diselenide on acetaminophen-induced hepatotoxicity in rats. *Pathophysiology*, 16 (1): 31-37.
29. Zira, A., Mikros, E., Giannoiti, K., Galanopoulou, P., Papalois, A., Liapi, C. and Theocharis, S. (2009). Acute liver acetaminophen toxicity in rabbits and the use of antidotes: a metabonomic approach in serum. *Journal of Applied Toxicology*, 29 (5): 395-402.

AFRICAN EDUCATION INITIATIVE (NEF)

Formerly known as the Nduaka Education Foundation (NEF), the African Education Initiative is a US-based non-governmental organisation founded in 2001 with the mission to advance science in Africa. This involves collaboration with various African institutions and the provision of educational materials and essential tools necessary to advance science education including promoting academic research in Institutes, supporting scholars pursuing a vast spectrum of professions. Other initiatives include advancing the knowledge of science and engineering not only on the African continent but also in other schools around the world, assisting students who are in need of internship in making the necessary contacts and helping professors who are seeking information about institutions abroad offering research opportunities. NEF also believes in global education and therefore communicates and exchanges information with other international educational foundations. Grants are provided through five programmes including scholarships, technology enhancement, curriculum enhancement, public health education, and student internships. The foundation has awarded grants to universities and students in Nigeria, Ghana, Cameroon, and Liberia. The student internship programme is specifically on toxicology where students are taught how to plan, design, conduct, and interpret toxicological studies. In 2009, six interns undertook a three-month annual toxicology programme in NVRI, Vom. At the end of the programme, interns together with their supervisors presented two seminars based on the research conducted on two medicinal herbs, *Phyllanthus niruri* and *Senna occidentalis*.



2009 NEF interns with Dr. Chudy Nduaka (middle), President and Board Chairman, Africa Education Initiative

¹⁹ASSESSMENT AND CONTROL OF MOULDS IN HOUSES WITH MOISTURE PROBLEM

Chuku A¹, Ekweozor I.K.E², Eze C.L³, Gobo A.E³

¹Dermatophilosis Research Centre, Bacterial Research Division, National Veterinary Research Institute, Vom.

²Medical Microbiology Department, Faculty of Science,

³Institute of Geo-Science and Space Technology, Rivers State University of Science and Technology, Port Harcourt.

ABSTRACT

Moulds are microscopic organisms that belong to the Fungal Kingdom. Moisture problem in buildings results from water incursion either from internal sources (e.g. leaking pipes) or external sources (e.g. rainwater) thereby causing dampness. The assessment and control of moulds in houses with moisture problem was done from April 2007 to March 2008. Sampling was done once a month on the sites and once in two months on the occupants. The chosen area for this study was divided into two categories, the first were of houses identified as having visible moisture problem and the second were houses identified as not having visible moisture problem. The samples collected for this study were, materials with visible growth of mould colonies on them from the first category of houses, air samples from the second category of houses, human samples from sick occupants of all the study sites through bimonthly medical check-up, air temperature, relative humidity, leakages and flooding incidences at each of the study sites. Sabouraud dextrose agar and Potato dextrose agar were used for culture of the samples while isolation and identification of the moulds were accomplished through microscopy, (cultural characteristics) and Microscopy (wet mount using KOH, tease mount and transparent tape preparation). Results obtained indicated that category A sites had less conducive physical parameters with average temperatures of 29°C to 31°C, relative humidity of 54% to 82.6%, leakage occurrence of 208 and flooding incidence of 253. Category B sites had moderate average parameters with average temperatures of 29°C to 29.8°C, relative humidity of 40.4% to 54.7%, two (2) leakage occurrences and no flooding. More ill health complains and positive mould isolations were recorded from category A sites than category B sites. Sixteen (16) moulds were isolated from the study including; *Aspergillus fumigatus*, *A. flavus*, *A. niger*, *Cladosporium* spp, *Penicillium* spp, *Rhizopus* spp, *Mucor* spp, *Trichophyton rubrum*, *T. mentagrophytes*, *T. schoenleinii*, *Microsporum audouinii*, *M. ferrugineum*, *Exophiala* spp, *Scopulariopsis* spp, *Sporotrix schenckii*, and *Fusarium solani*. The most prevalent moulds were *Mucor* spp, *Penicillium* spp and *T. rubrum* (*dermatophyte*). This study also revealed that moulds could grow on any material that is damp and is of plant or animal origin. High humidity above 50% will attract mould growth and as the relative humidity keeps increasing, so will mould growth increase. Moulds survived and multiplied faster during the rainy season compared to the dry season therefore diseases and infections that were associated with moulds had a greater occurrence during the rainy season. Diseases and infections that were associated with moulds had a greater occurrence during the rainy season. Moulds were the single greatest destroyers of household furniture, fittings and spoiler of food items. Houses with insufficient windows, doors, vents, or south facing windows resulted in poor ventilation which presented a favourable condition for the growth of moulds. In order to control moulds in these homes, humidity level below 50% in the home should be maintained, visual inspection of the building

¹⁹ Seminar presented on 27th August 2009 at NVRI auditorium

should be made regularly to identify the areas with mould growth, clothes should be stored dry and clean. All mould affected material should be dealt with as quickly as possible to limit the damage caused and to prevent contamination of other materials. Houses built at waterfronts or marshy areas, should be built on raised stakes or high foundations to prevent flooding or seepage of water into the house. Skin rashes and irritation should be reported for immediate medical attention. Repairs of all leakages from taps, pipes, sinks, roof, or wall should be carried out immediately. Builders should construct proper drainage. People already living in houses with moisture problem should eliminate every source of moisture and employ the mould remediation company to remove the moulds and finally, public education should be carried out on the health hazards associated with houses prone to fungal infestation.

**²⁰MOLECULAR EPIDEMIOLOGY OF DOG RABIES IN NIGERIA:PHYLOGENY
BASED ON N AND G GENE SEQUENCES**

Ogo^{1,*}, M. F., Nel², L. H., Sabeta³, C. T.

¹Department of Viral Research, National Veterinary Research Institute, PMB 01 Vom Plateau State Nigeria

²Microbiology and Plant Pathology, University of Pretoria, 0002 Pretoria, South Africa

³Rabies Unit, Onderstepoort Research Institute, Onderstepoort, South Africa

***Department of Veterinary Tropical Diseases, University of Pretoria, 0110 Onderstepoort South Africa**

* Corresponding author: P.M.B. 01. Department of Virology, National Veterinary Research Institute Vom, Nigeria

Tel.: +234 7031664000, E-mail addresses: fjmaria@yahoo.com

Introduction

Rabies still poses significant public and veterinary health in many parts of Africa and Asia (Cliquet and Picard-Meyer, 2004). An estimated 55,000 human deaths are recorded annually and 99% of the deaths which occur in these two continents are attributed to canine rabies (WHO, 2005). It is a highly neglected disease in Nigeria largely due to competing priorities of other zoonoses, underreporting and poor surveillance of the disease. There are many instances in Africa where clinical presentation may be misdiagnosed with other tropical diseases such as malaria (Mallewa, *et al.*, 2007). The domestic dog is the major maintenance host and vector species responsible for transmission of the disease to humans and other domestic animals in Nigeria (Fagbami *et al.*, 1981). In this country it accounts for over 99% of all human rabies cases with an annual estimate of over 10,000 (Nawathe, 1980), but the true incidence of rabies in humans is likely to be higher. The causative agent of rabies is a highly neurotropic lyssavirus belonging to the Rhabdoviridae family of the order Mononegavirales and seven genotypes are currently recognised within Lyssavirus genus (Tordo *et al.*, 2006). The genome of the virus is made up of a non-segmented, single-stranded negative-sense RNA which is approximately 12 kb in length. There are five viral proteins which the rabies genome codes for (3'-N-P-M-G-L-5') (Fauquet *et al.*, 2005). In this investigation, the sequence data from the partial G and N genes are presented. We used the data to carry out a molecular epidemiological study of rabies viruses from domestic dogs in Nigeria through determination of nucleotide sequences and to compare the relationship of the rabies variant circulating in Nigeria with those of neighbouring countries of Chad, Cameroon, Benin and Niger. This report has attempted to elucidate the molecular epidemiology of dog rabies in Nigeria.

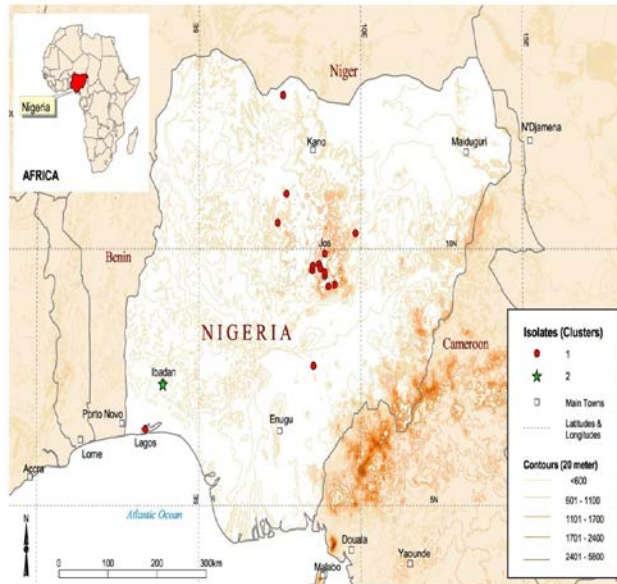
Methodology

A hundred 100 dog brain tissues recovered primarily from the domestic dog in the last 2 decades (1989-2008) were obtained from the National Veterinary Research Institute Vom, Nigeria and transported frozen to OIE Rabies Reference Laboratory in Pretoria (Onderstepoort Veterinary Institute South Africa). Total Viral RNA was extracted from original brain tissues using TRI reagent (Sigma, Aldrich, U.S.A.) as previously described (Chomczynski, 1993). Partial regions of the N and G were reverse transcribed and amplified. The PCR amplicons were purified with spin columns Wizard® purification system, Promega U.S.A Cycle sequencing of purified PCR amplicons were performed with the Big Dye Terminator V3.0 sequencing Kit

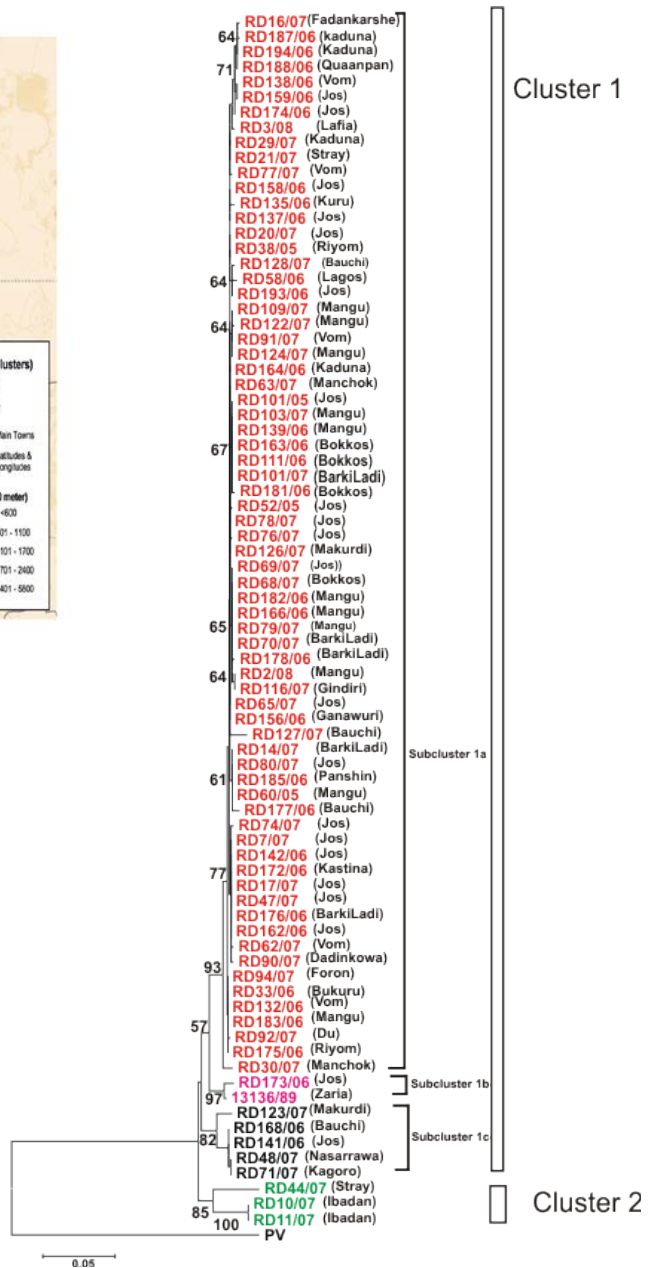
²⁰ Seminar presented on 10th September 2009 at NVRI auditorium

(Applied Biosystems), using the same primers as in the amplification step. The MEGA program was used for the phylogenetic analysis.

Results



A phylogenetic tree showing the relationships of the Nigerian rabies viruses generated by NJ method based on 592 bp sequences of the glycoprotein and the G-L intergenic region and a map of Nigeria depicting the geographical distribution of the isolates



The phylogenetic analysis demonstrated that all the viruses included in this investigation had >99% sequence homology and segregated into two major groups (cluster 1 mainly from northern Nigeria and cluster 2 from southern Nigeria). There were 3 subcluster in group 1 probably representing local outbreaks. The N tree (data not shown) revealed that the dog rabies viruses from Nigeria with those of her neighbours are evolutionary linked and all group into the African 2 rabies virus dog lineage circulating in the West African region.

Discussion and Conclusion

This investigation provides the baseline data on molecular epidemiology of rabies in Nigeria. It has also revealed that one dominant rabies variant is circulating in domestic dogs with a close evolutionary and epidemiologic linkage with those of her

neighbours. The molecular characterization involving PCR, nucleotide sequencing and phylogenetic analysis has demonstrated that the rabies viruses from dogs are closely related with geographical and site inclination. It is then evident that mass vaccination campaigns with parenteral anti-rabies vaccines targeted at dogs including strays remains an effective means of breaking the rabies cycle. There is so the need to have a proper surveillance system for rabies in the country. The concept of one health could be implemented to align and incorporate rabies control programs into existing national public health programs thus availing both human and financial resources available for these national programs for rabies control.

Acknowledgements

This study was funded in part by the International Society of Infectious Diseases (ISID) (Small grant 2007), Poliomyelitis Research Foundation, South Africa (PRF) (grant no. 0859) and Department of Veterinary Tropical Diseases, University of Pretoria, South Africa. The authors are indebted to the Executive Director of NVRI Dr. (Mrs) L.H. Lombin for providing the isolates for this study. We also gratefully acknowledge Dr. D. Shamaki and Dr. T.M. Joannis for helping with the transportation of the samples. M. F. Ogo gratefully acknowledges all the staff of the Rabies OIE Reference Laboratory of the Onderstepoort Veterinary Institute, Pretoria, South Africa where this study was conducted and Dr. N.I. Ogo for all the support given during the course of the study.

References

1. Cliquet, F., and Picard-Meyer, E. 2004. Rabies and rabies-related viruses: a modern perspective on an ancient disease. *Revue Scientifique et Technique-office International des*, 23: 625-642.
2. Chomczynski P. 1993. A reagent for the single-step simultaneous isolation of RNA, DNA and proteins from cell and tissue samples, *Biotechniques*, 15: 532-537.
3. Fagbami, A. H., Anosa, V. O. and Ezebuio, E. O. 1981. Hospital records of human rabies and anti-rabies prophylaxis in Nigeria. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 75: 872-876.
4. Fauquet, C. M., Mayo M.A., Maniloff J., Desselberger, U. and Ball, L. A. 2005. *Virus taxonomy: the classification and nomenclature of viruses. The eighth report of the International Committee on Taxonomy of Viruses*. San Diego: Academic Press, pp 623-631.
5. Mallewa, M., Fooks, A. R., Banda, D., Chikungwa, P., Mankhambo, L., Molyneux, E., Molyneux M. E. and Solomon, T. 2007. Rabies encephalitis in malaria-endemic area, Malawi, Africa. *Emerging Infectious Diseases*, 13: 136-139.
6. Nawathe, D. R. 1980. Rabies control in Nigeria. *Bulletin Office International des Épizooties*, 92: 129-130.
7. Tordo, N., Bahloul, Y., Jacob, C., Jallet, P. and Perrin, H. 2006. Rabies: epidemiological tendencies and control tools. *Developments in Biologicals*, 125: 3-13.
8. World Health Organization. 2005. Expert Consultation on rabies. Technical Report Series 931.

²¹DISTRIBUTION OF *CAMPYLOBACTER JEJUNI* IN ABATTOIR SPECIMENS IN JOS AND ENVIRONS

Spencer, T.H.I.

Bacterial Vaccine Production Division, National Veterinary Research Institute, Vom

E-mail: thispence2005@yahoo.com

Introduction

Awareness of the public health implications of *Campylobacter* infections has evolved over more than a century. McFadyean and Stockman (1) made the first isolation of *Campylobacter* in 1913 from the uterine exudates of aborting sheep.

A few years later, Smith (2) isolated a similar organism from foetuses of aborting cows and named it *Vibrio fetus*. Much later, Florent (3) showed that a form of the infection known as bovine infectious infertility was due to a variety of *V. fetus* transmitted from carrier bulls to cows during coitus. He named the organism, *V. fetus var venerealis* (now *C. fetus* subsp. *venerealis*). Jones *et al*; (4) named as *V. jejuni*, a microaerophilic *Vibrio* isolated from jejunum of calves with diarrhoea. A similar *Vibrio* was isolated from pigs suffering from swine dysentery by Doyle (5) who named it *Vibrio coli*. These became *C. jejuni* and *C. coli* respectively with the formation of the genus, *Campylobacter* by Sebald and Vernon (6). *Campylobacteriosis* is of high economic significance in the livestock industry, affecting the milk and meat production in both large and small ruminants as well as egg production in poultry.

Methodology

One thousand, six hundred (1600) abattoir samples comprising 400 each of gall bladder, intestine, abomasum and foetal heart blood were collected in the wet and dry seasons in 2005. The gall bladder, abomasum, and segments of the intestine were collected intact into sterile receptacles while the foetal heart blood was immediately introduced into Preston *Campylobacter* enrichment broth using a spirit lamp. All samples were immediately transported to the laboratory under cold chain. The gall bladder, abomasal and intestinal contents were emulsified in 3ml of 0.1% sterile peptone water (10). These emulsions were aseptically inoculated onto Preston *Campylobacter* Selective Agar and incubated at 42°C for 48 hours while the enrichment broth containing the foetal heart blood was also incubated at 42°C for 24 hours after which it was sub cultured onto the solid medium and incubated further for 48 hours at 42°C. Pure cultures of the suspected *Campylobacter* colonies were identified based on their non-sporing, non-encapsulating, and non-haemolytic characteristics on Preston *Campylobacter* Selective Agar. Further identification was based on the characteristic darting or corkscrew motility of the organism under phase contrast microscopy, asaccharolytic ability, production of the enzyme, oxidase and its catalase positivity or negativity. All presumptive *Campylobacter* colonies were subjected to nalidixic acid and cephalothin tolerance at concentrations of 30mcg/ml. Ability of the organism to produce hydrogen sulphide on triple sugar iron agar (TSI) and to hydrolyse sodium hippurate to benzoic acid and glycine were tested (11).

Results and Discussion

The zoonotic implication of *Campylobacter jejuni* infection has led to a number of recent reports concerning its carriage by food animals and pets (12, 13). This study

²¹ Seminar presented 8th October 2009 at NVRI auditorium

addresses in part each of these concerns with special reference to animals at slaughter. The study demonstrated that animals could be possible reservoirs of *Campylobacter jejuni* and hence, potential sources of *Campylobacter* enteric infection. The result of the present study showed higher isolation rate of *Campylobacter jejuni* from the gall bladder and intestine with no isolation made from foetal heart blood and abomasum. However, this does not rule out vertical transmission of *C. jejuni* in pregnant heifers. The higher isolation of *C. jejuni* from the gall bladder and intestine agrees with the assertions of (14) and Rosef (1981) (15) that the gall bladder and intestine are major sites of predilection for *C. jejuni*. The wet season distribution of *C. jejuni* was higher (2.38%) than that of the dry season (1.38%). This could be attributed to the verdure nature of pasture with its attendant pollution by these animals at grazing.

Slaughtered animals at the abattoirs may serve as a major source of human transmission of *Campylobacter jejuni*. *Campylobacter* enters the human food chain at slaughter of the animals, thus highlighting the zoonotic and public health implication of this organism. The manner with which butchers handled animal carcasses post slaughter without any protection way encourage the transmission of *Campylobacter*. Many laboratories in most parts of the country do not routinely investigate *Campylobacteriosis* as they do for enteric infections. This might be due to lack of proper awareness of the clinical and economic importance of *C. jejuni* on one hand and the fastidious nature of the organism

Conclusion

As *Campylobacteriosis* is often misdiagnosed in our Nigerian medical and veterinary laboratories, it is advocated that the investigation of *Campylobacteriosis* in human and animals be routinely carried out in both medical and veterinary laboratories in Nigeria as this will go a long way in forestalling the spread of the infection especially from animal to human.

Acknowledgement

The contributions of the following people in various capacities are duly acknowledged: Dr. A. A. Makinde (Director, Diagnostics and Extension, NVRI, Vom), Dr. J. U. Molokwu (Ag. Director, Planning, NVRI, Vom), Professor S. E. Agina (Botany Department, University of Jos), Dr. E. O. Irokanulo (Asst. Director, Bacterial Vaccine Production, NVRI, Vom) and Professor B. A. Ajala (HOD, Botany, University of Jos).

References

1. McFadyean, J. and Stockman, S. (1913). Report of the Departmental Committee appointed by the Board of Agriculture and Fisheries to enquire into epizootic abortion. Appendix to Part II. Abortion in sheep. Pp. 1-64. Wiley, London.
2. Smith, T. and Taylor, M. S. (1919). Some morphological and biological characters of the spirilla (*Vibrio fetus*, N. Sp) associated with disease of the foetal membranes in cattle. Journal of Experimental Medicine, 28: 701-719.
3. Florent, A. (1959). Les deux *Vibrioses* genitales: la *Vibriose* due a et la *Vibriose* de origine intestinale due a *V. fetus* intestinalis. Meded. Vearts School. Gent 3: No 3. Pp. 1-60.
4. The aetiology of infectious diarrhoea (Winter scours) in Cattle. Journal of Experimental Medicine, 53: 845-851.
5. Jones, F. S. and Little, R. B. (1931b). Vibrionic enteritis in calves. Journal of Experimental Medicine, 53: 853-864
6. Sebald, M. and Veron M. (1963). Teneur a bases de' ADN et classification de *Vibrien*. Ann de la Inst. Pasteur, 105: 897-910.

7. King, E. O. (1957). Human infections with *Vibrio fetus* and a closely related *Vibrio*. *Journal of Infectious Diseases*, 101: 119-128.
8. Butzler, J. P. (1973). Related *Vibriosis* in Africa. *Lancet*, 2: 858.
9. Skirrow, M. B. (1977). *Campylobacter* enteritis. A new disease. *British Medical Journal*, 2: 264.
10. *Selective Microbiology for the Clinical Laboratory* (1991). Techniques and Media for the selective isolation of pathogenic organisms. Published by Unipath Ltd, Wade Road, Basingstoke, Hampshire, R. C. 24 OPW. p. 17.
11. Cacho, J. B; Pedro, M; Aguirre, Angel Hernenz and Aurelio, C. Velasco (1989). Evaluation of a disk method for detection of hippurate hydrolysis by *Campylobacter* sp. *J. Clin. Microbiol*, 27: (2), 259-260.
12. Svedhem, A and Kaijser, B. (1981). Isolation of *Campylobacter jejuni* from domestic animals and pets: probable origin of human infection. *J. Infect*, 3: 37-40.
13. Bryner, J. H; P. A O'Berry, P. C. Estes and J. W. Foley (1972). Studies of *Vibriosis* from the gall bladder of market sheep and cattle. *Am. J. Vet. Res*; 33: 1439-1444.

22 EVALUATION OF THE CONTRIBUTION OF MAJOR T CELL SUBPOPULATIONS TO IFN- γ PRODUCTION IN TB INFECTION BY ELISPOT

Moses D Lugos¹, Adetifa Ifedayo², Simon Donkor, Philip C Hill³, Richard A. Adegbola, Martin OC Ota

¹Federal College of Veterinary & Medical Laboratory Technology, National Veterinary Research Institute, Vom-Nigeria

²Bacterial Diseases Programme, Medical Research Council (MRC) Laboratories, Banjul, The Gambia.

³Department of Preventive and Social Medicine, University of Otago School of Medicine, PO Box 913, Dunedin 9054, New Zealand

Correspondence: mlugos2003@yahoo.com Tel: +234 803 486 9343

Introduction

It is estimated that one-third of the world population is latently infected with *Mycobacterium tuberculosis* (Mtb), and 5 to 10% of the infected population will develop the disease during their lifetime (Corbett, 2003; Corbett *et al*, 2003). The outcome of Mtb infection depends on the state of the host immune system: the infection may be eliminated, progress directly to active disease, or become latent in the host with a risk of later reactivation (Lin and Ottenhoff, 2008). A major roadblock in TB control is the lack of understanding of what constitutes a protective immunological response against progression of latent infection to active TB disease (Lalvani and Millington, 2008).

Immunological responses to Mtb infection involve both the innate as well as the adaptive immune system (Ottenhoff *et al*, 2005). A key effector molecule in protective immunity against mycobacteria is IFN- γ produced by several cells (Flynn *et al*, 1993; Newport *et al*, 2003). Studies of T lymphocyte responses show that both CD4⁺ and CD8⁺ T cells are important in controlling TB (Flynn, 2004; Smith and Dockrell, 2000; Smith *et al*, 2000). An analysis of the major T lymphocyte subset responding to the Mtb antigens may inform diagnostic and vaccine development purposes. Therefore, we evaluated the cellular source of the IFN- γ release in an *ex-vivo* ELISPOT assay in TB cases and their contacts.

Methodology

Heparinized blood samples were collected from study participants and depleted of CD4⁺ and CD8⁺ T-lymphocytes using RosetteSep[®] antibody cocktail mix according to the manufacturer's protocol (Stem Cell Technologies, Vancouver).

Test antigens used for this study included two synthetic peptide pools each containing sequential 15-mer peptides overlapping by 10 amino acids spanning the length of ESAT-6 and CFP-10 (ABC, Imperial College, London, UK) respectively. Phytohaemagglutinin (PHA: Sigma- Aldrich, UK) and medium alone were used as positive and negative controls respectively. All antigens and controls were tested in duplicate wells.

Cells were assayed for the frequency of IFN- γ producing cells by ELISPOT assay. The number of IFN- γ spot forming units (SFU) counted in each well were automatically entered into a database minus background (medium wells), and the data expressed as number of spot-forming cells (SFC) / million. All data were entered using double data entry into an ACCESS database and checked for errors. The Kruskal-Wallis test was used to compare responses from the three different cell

²² Seminar presented on 15th October 2009 at NVRI auditorium

populations. Comparison of responses between cases and contacts was by student t-tests. Statistical significance was assumed at a p-value <0.05. All statistical analyses were conducted using Stata software (version 9; Stata Corp, College Station, TX, USA).

Results

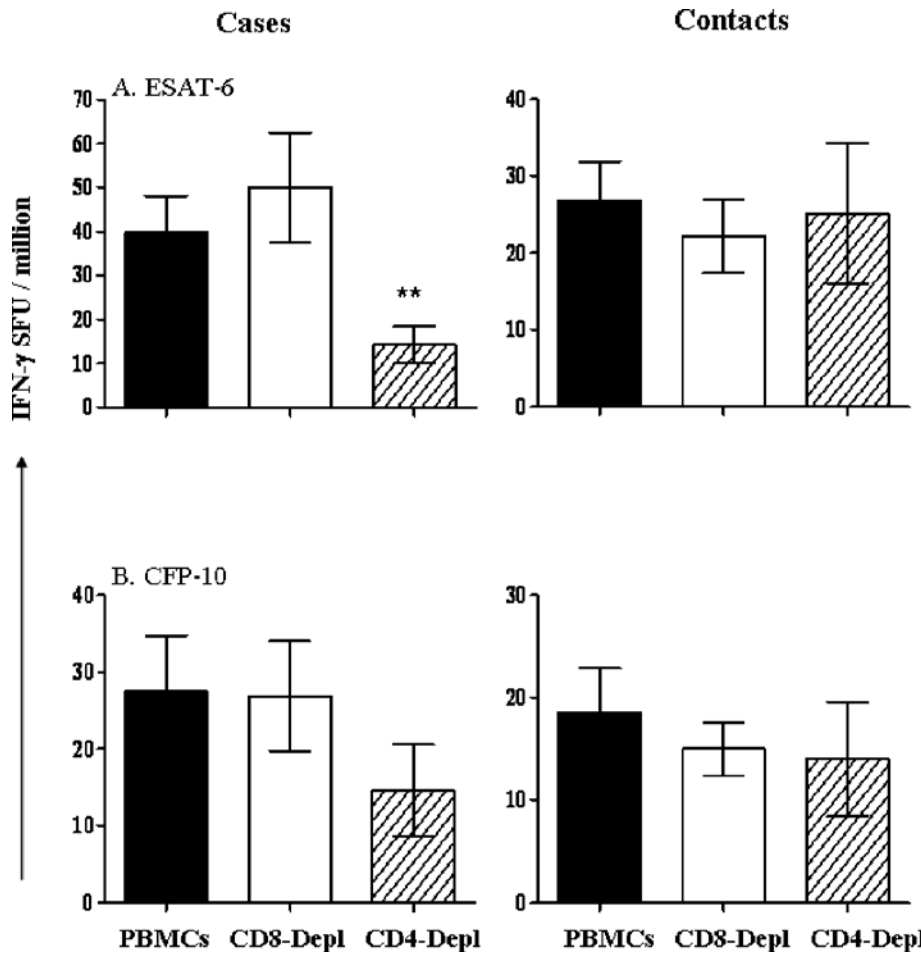


Figure 1: The effect of CD4⁺- or CD8⁺- depletion on the frequency of IFN- γ -secreting cells following stimulation with ESAT-6 (A), and CFP-10 (B) in TB cases and contacts. Averages and SEM of responses from 33 TB cases and 49 household contacts are represented.

**** P-values=0.015 and 0.03 as compared to CD8-depleted and undepleted PBMCs, respectively.**

Discussion

Interferon gamma release assays (IGRAs) that measure IFN- γ production by sensitized T-cells in response to stimulation by relatively Mtb-specific antigens are new methods for detecting Mtb infections (Lalvani, 2007; Lalvani et al., 2001; Menzies et al., 2007). Identification of the major T lymphocyte subset responding to the Mtb antigens during infection or disease may influence the interpretation of interferon-gamma release assays under certain conditions. Our current study assessed the contribution of CD8⁺ and CD4⁺ lymphocytes to the ESAT-6 and CFP-10 antigen-specific IFN- γ response using ELISPOT assay. The ESAT-6 responses tended to be higher than those of CFP-10, and CD4⁺-depletion significantly reduced the response to ESAT-6 in TB cases. Moreover, the response of the CD8⁺-depleted cells to ESAT-6 in cases was higher than that of the contacts. These

suggest that ESAT-6 specific effector cells are predominant in both TB cases and their contacts, and these are mainly due to the participation of CD4+ T lymphocytes.

ESAT-6 and CFP-10 are secreted Mtb protein antigens whose genes are located in the same operon, and share 40% sequence homology (Berthet et al., 1998). The differences in the responses to both proteins could be related to either their expression and/or antigen processing in the host. Although both CD4+ and CD8+ T cells are important to control TB (Flynn, 2004; Smith and Dockrell, 2000; Smith et al., 2000), our data suggest that the responses are mainly due to CD4+ population, particularly in response to ESAT-6. This could be in keeping with the principal role of CD4+ in protection against Mtb in mice (Flynn et al., 1993). Moreover, these responses were obtained at recruitment, which can be considered an early phase of the disease, thus, confirming the suggestion that CD4+ T cells are important during early stages of infection whereas CD8+ T cells become more significant for control of chronic infection (van Pinxteren et al., 2000). Responses from the different cell populations to the Mtb-specific antigens were comparable in both cases and contacts, apart from the CD8+-depleted population response to ESAT-6 that discriminated between cases and contacts, which is likely due to an enriched ESAT-6-specific CD4+ population in the cases. Thus, it is plausible that ESAT-6-specific CD4+ lymphocytes expand extensively during TB disease resulting in the abrogation and enhancement of responses following their depletion and enrichment, respectively. Data have described variable responses to ESAT-6 of TB cases as compared to contacts, and these could be due to variability and difficulty in categorizing the stage of the TB disease in humans (Cardoso et al., 2002; Roberts et al., 2007; Vekemans et al., 2001).

The similarity of the IFN- γ secretion from PBMCs, PBMCs depleted CD4+, or CD8+ T cells in response to CFP-10 is interesting. We used the same number of cells, 2×10^5 , per well for each cell populations studied, which will certainly contain varying proportions of the different lymphocyte subsets. For instance CD4+ cells will predominate in CD8-depleted population, and vice versa. Thus, one obvious implication of the similarity of responses to CFP-10 is that both CD4+ and CD8+ T cells contribute to the response, hence the depletion of one population leads to enhancement of the other, thereby compensating for the loss of response from the depleted cells. Taken together, our data indicate that IFN- γ immune response to CFP-10 is by both CD4+ and CD8+ lymphocytes, while that to ESAT-6 is mainly contributed by CD4+ cells, and these are not compromised by TB disease. This discrepancy in response to ESAT-6 might have consequences on the reliability of IGRAs in conditions associated with low CD4 counts as in HIV/AIDS.

Acknowledgments

We thank the National TB control programme for their ongoing collaboration and our field and laboratory assistants for their hard work. We are grateful to the subjects who took part in this study. The study was motivated by the Training Committee and funded by the Medical Research Council, The Gambia.

References

1. Berthet FX, et al (1998). A Mycobacterium tuberculosis operon encoding ESAT-6 and a novel low-molecular-mass culture filtrate protein (CFP-10). *Microbiology* 144 (Pt 11): 3195-203.
2. Cardoso FL, et al (2002). T-cell responses to the Mycobacterium tuberculosis-specific antigen ESAT-6 in Brazilian tuberculosis patients. *Infect Immun* 70: 6707-14.

3. Corbett EL (2003). HIV and tuberculosis: surveillance revisited. *Int J Tuberc Lung Dis* 7: 709.
4. Corbett EL, et al (2003). Stable incidence rates of tuberculosis (TB) among human immunodeficiency virus (HIV)-negative South African gold miners during a decade of epidemic HIV-associated TB. *J Infect Dis* 188: 1156-63.
5. Flynn JL (2004). Immunology of tuberculosis and implications in vaccine development. *Tuberculosis (Edinb)* 84: 93-101.
6. Flynn JL, et al (1993). An essential role for interferon gamma in resistance to *Mycobacterium tuberculosis* infection. *J Exp Med* 178: 2249-54.
7. Lalvani A (2007). Diagnosing tuberculosis infection in the 21st century: new tools to tackle an old enemy. *Chest* 131: 1898-906.
8. Lalvani A, Millington KA (2008). T Cells and Tuberculosis: Beyond Interferon-gamma. *J Infect Dis* 197: 941-3.
9. Lalvani A, et al (2001). Rapid detection of *Mycobacterium tuberculosis* infection by enumeration of antigen-specific T cells. *Am J Respir Crit Care Med* 163: 824-8.
10. Lin MY, Ottenhoff TH (2008). Not to wake a sleeping giant: new insights into host-pathogen interactions identify new targets for vaccination against latent *Mycobacterium tuberculosis* infection. *Biol Chem*.
11. Menzies D, et al (2007). Meta-analysis: new tests for the diagnosis of latent tuberculosis infection: areas of uncertainty and recommendations for research. *Ann Intern Med* 146: 340-54.
12. Newport MJ, et al (2003). Polymorphism in the interferon-gamma receptor-1 gene and susceptibility to pulmonary tuberculosis in The Gambia. *Scand J Immunol* 58: 383-5.
13. Ottenhoff TH, et al (2005). Control of human host immunity to mycobacteria. *Tuberculosis (Edinb)* 85: 53-64.
14. Roberts T, et al (2007). Immunosuppression during active tuberculosis is characterized by decreased interferon- gamma production and CD25 expression with elevated forkhead box P3, transforming growth factor- beta , and interleukin-4 mRNA levels. *J Infect Dis* 195: 870-8.
15. Smith SM, Dockrell HM (2000). Role of CD8 T cells in mycobacterial infections. *Immunol Cell Biol* 78: 325-33.
16. Smith SM, et al (2000). Human CD8(+) T cells specific for *Mycobacterium tuberculosis* secreted antigens in tuberculosis patients and healthy BCG-vaccinated controls in The Gambia. *Infect Immun* 68: 7144-8.
17. van Pinxteren LA, et al (2000). Control of latent *Mycobacterium tuberculosis* infection is dependent on CD8 T cells. *Eur J Immunol* 30: 3689-98.
18. Vekemans J, et al (2001). Tuberculosis contacts but not patients have higher gamma interferon responses to ESAT-6 than do community controls in The Gambia. *Infect Immun* 69: 6554-7.

23 CONDUCTING THEMATIC AND DIAGNOSTIC SURVEYS IN ANIMAL HEALTH RESEARCH AND DEVELOPMENT

E. I. IKANI

Extension Division, NVRI, Vom (Current address)

National Agricultural Extension and Research Liaison Services, Zaria

E-mail: ikani50@yahoo.com

Introduction

Traditional animals' keepers had hitherto not adopted many of the animal health technologies developed in Research Institutes, to increase their productivity and enhanced livelihood. There is thus the necessity and urgent need to develop new adoption strategies that will be compatible with prevailing husbandry system so that livestock farmers can readily adopt improve animal health management practices. Farming System Research (FSR) approach has been observed over the years to be a methodology of Agricultural Research for the development of improved technologies relevant to farmers' needs and circumstances. Diagnostic/ thematic survey as an important component of FSR process is to describe and understand the farming system of a place and identifies production and animal health constraints along with available opportunities (Auta, 2008). Disease problems can be illnesses that livestock keepers are already very familiar with, but for which they lack information on the pros and cons of different modern or indigenous treatments. For instance, farmers may know that their cattle have worms but they may not know the optimum way to use anthelmintics or the relative merits of their control options. In other situations a disease is recognized by the livestock keepers but the researchers don't know what the disease is. In both of these situations, the starting point is to reach a common understanding of the disease in question (Conroy, 2005). The main objective of this paper is to share thoughts on the question –where does applied animal health research process start from, to bring about relevant and appropriate technologies that will ensure good level of adoption by end-users.



Figure 1 Pastoralists with their animals

effort in adopting animal health technologies that they consider to be unimportant or in appropriate.

Concept of thematic/diagnostic survey

The premise for conducting thematic/diagnostic survey is based on needs assessment. The difference between the two is only in scope. Thematic survey focuses on needs assessment that borders on research theme, while diagnostic survey covers a broader research area. It is important therefore, that any type of animal health research should be based on a sound understanding of what livestock - keepers see as their priority needs, i.e. their main constraints or opportunities. This is an essential foundation or starting point for the whole research process. Farmers are unlikely to invest time and

The principle

Research into animal health care for improve productivity must be considered in relation to various other factors that influence the entire farming system

Decentralized approach which ensures participation of the farmers to avoid top-down approach in diagnosing

Research Team that is multi-disciplinary and multi-purpose

The heterogeneity nature of the pastoral communities demands that the researchers reach out to all the strata of the farming system to be sure that all the identified constraints and opportunities are relevant to all members in the community.

Basic steps in conducting thematic/diagnostic survey

- i. Exploration of the pastoral farming system.
- ii. Diagnosis of real field constraints and opportunities
- iii. Classification, ranking and analysis of identified constraints and opportunities
- iv. Preliminary identification of strategies for planning and implementation of project.

Tools for thematic/diagnostic survey

- i. Semi-structure interviews
- ii. Pastoral farming system diagrams
- iii. Seasonal calendars
- iv. Appraisal of indigenous technical knowledge, attitudes and practices.
- v. Problem analysis
- vi. Work sheet
- vii. Expected out- puts
- viii. At the end of thematic/diagnostic survey the following out-puts are expected;
- ix. Record of identified real field most serious and important animal health care constraints couple with opportunities available.
- x. A clear and general understanding of the production resource base at the disposal of the pastoral communities
- xi. An understanding of the differences among farming families in terms of other factors that have direct or indirect relevance to livestock health care practices
- xii. Participatory selection and validation of potential solutions and strategies for overcoming the constraints faced by the different farming communities.

Conclusion

This discussion focused on the justification for thematic/diagnostic surveys in animal health research, the basic steps for conducting it, the essential tools and expected out-puts of the exercise, all with the purpose of developing appropriate animal health technologies for the desired increased up-take and adoption by livestock farmers.

References

1. Auta, S.J. (2008).Planning a diagnostic survey. A paper presentation at the training of NVRI Extension Staff held August 2008 at Dermatophilosis Conference Hall NVRI, Vom
2. Conroy. C. (2005). Participatory Livestock Research-A Guide. A Publication by ITDG Warwickshire CV23 9QZ, UK

²⁴ANTIMICROBIAL SCREENING OF COMMERCIAL CHICKEN EGGS IN PLATEAU STATE

FAGBAMILA, I.O.

Bacterial Research Division, NVRI, Vom

Email:dridowu4u@yahoo.com

Introduction

Sub therapeutic levels of antibiotics could increase feed efficiency and growth in farm animals and this has led to the widespread incorporation of antibiotics into animal feeds. Antibiotics also reduce the incidence or severity of a number of animal diseases. It has been suggested that antibiotics also prevent irritation of the intestinal lining and improve digestive and metabolic processes in animals. However, addition to positive effects on growth and efficiency, there are associated reductions in excretion of nitrogen, phosphorus and manure by some farm animals (Doyle, 2006). Approximately 80% of all food-producing animals receive medication for part or most of their lives (Lee *et al.*, 2001). The use of antimicrobial agents in food animals has become a very important public health issue because of the effects of some of these drugs on human health (AL-Ghamdi *et al.*, 2000). Human health problems resulting from intake of sub-therapeutic exposure levels of antimicrobial drugs are well documented. Penicillin in chicken has been reported to cause severe anaphylactic reaction in a consumer (Teh and Rigg, 1992). Tetracycline in meat may potentially stain the teeth of young children. Skin allergies in eggs containing sulfonamide residues have also been reported (WHO, 1989).

Methodology

Sampling Commercial Chicken Eggs in Plateau State

Thirty commercial poultry farms were selected from the three senatorial districts of Plateau State using the stratified random sampling method. Information and observations on each farm were collected and recorded using a structured questionnaire. Eggs were sampled according to standard methods (Kabir *et al.*, 2004). Nine hundred eggs were selected using simple random sampling format. The eggs were arranged in a clean container and transported to the laboratory immediately for processing or refrigerated at 4°C until the following day. The disc diffusion method was used to screen eggs for antimicrobial residues. An 18-hour culture of the *Bacillus stearothermophilus* C-953 (DSM, Netherlands) in 10 ml nutrient broth (Oxoid, UK) was used to inoculate Mueller Hinton agar plates. Sterile filter paper discs 0.6cm in diameter dipped in homogenized egg yolk was gently placed on the already inoculated plate and incubated for 24hours (Shahid *et al.*, 2007). Suspected positive samples were then tested using the New Premi®Test Test Kit (DSM, Netherlands).

Results

The screening test gave 32 suspected positive results with the disc diffusion method while the Premi® test method gave 18 confirmed positive results, 10 from Plateau North senatorial district, and 8 from Plateau Central.

²⁴ Seminar presented on 12th November 2009 at NVRI auditorium

Discussion

Plateau south senatorial district is located in the hot and sunny part of Plateau State where there is less commercial activities. Access to veterinary services can be difficult and it is not uncommon to find poultry farmers slaughtering birds when they notice any sign of illness rather than treating them. The zero positive result recorded in the area at the time of this study could be attributed of these reasons. Flock size was a risk factor in the occurrence of drug residues. Farms with larger flock sizes were more likely to take measures towards minimizing financial losses resulting from mortality on the farm such as prophylactic and therapeutic use of antibiotics without adherence to recommendations on antimicrobial drug use. Most commercial poultry farms in Plateau State are established essentially to augment family income and thus observance of withdrawal period was perceived to be a source of financial loss.

Conclusion

This study demonstrated the misuse of veterinary drugs amongst poultry farmers in Plateau State, which resulted in residues in eggs. There is a lack of adequate awareness of the public on the danger of antimicrobial residues in eggs, lack of regular screening for antimicrobial residues and lack of enforcement of laws on the sales, marketing and administration of veterinary drugs.

References

- Al-Ghamdi., M.S., Al-Mustafa, Z. H., El-Morsy, F., Al-Faky, A., Haider, I. and Essa, H. (2000): Residues of tetracycline compounds in poultry products in the eastern province of Saudi Arabia. *Public Health*, 114, 300-304.
- Doyle, M.E. (2006): Food Research Institute University of Wisconsin-Madison Madison, Wisconsin. www.wisc.edu/fri/ 53706 medoyle@wisc.edu. Accessed on 25th October, 2008 at 3:08PM.
- Kabir, J., Umoh, V.J., Audu-okoh, E., Umoh, J.U., Kwaga, J.K.P. (2004): Veterinary drug used in poultry farms and determination of antimicrobial drug residues in commercial eggs and slaughtered chicken in Kaduna State, Nigeria. *Food Control*, 15: 99-105.
- Lee, H.J., Lee, M.H. and Ruy, P.D. (2001): Public health risks: Chemical and antibiotic residues. *Asian-Australian Journal of Animal Science*, 14: 402-413.
- [Shahid](#), M.A., [Siddique](#), M., [Rehman](#), S., [Hameed](#), S. and [Hussain](#), A. (2007): Evaluation of a Microbiological Growth Inhibition Assay as a Screening Test for the Presence of Antibiotic Residues in Poultry Meat. *American Journal of Food Technology*, 2(5): 457-461.
- Teh, W.L., and Rigg, A.S. (1992): Possible penicillin allergy after eating chicken, *Lancet*, ii, 1632.
- World Health Organization, WHO (1989): Evaluation of certain veterinary drug residues in food: *Thirty fourth Report of the Joint FAO/WHO Expert Committee on Food Additives*. WHO Technical Report series, NO. 788, Geneva.

NVRI SEMINAR SERIES

GUIDELINES FOR PRESENTATION AND MANUSCRIPT PREPARATION

The NVRI Seminar Series is an edited publication of seminar presentations made at NVRI during the course of one calendar year. Seminar presentations provide a platform for research staff of the Institute and visiting scientists to present their research findings. The series is published annually.

Types of presentations

Research proposals, progress reports of on-going research, reports of completed studies and thesis are accepted for presentation. Reviews in any area of the Institute's mandate are made by invitation only.

Preparation for of manuscript for presentation

A Microsoft PowerPoint® presentation and an extended abstract are to be submitted to the secretary of the Committee one week before the presentation is due.

Preparation of manuscript

The manuscript should be written as an extended abstract with the following: Title of Paper, Author(s), Address including e-mail, Introduction, Methodology, Results and Discussion, Acknowledgements and References. Only one figure and one table will be accepted. The title of presentation should not be more than 15 words with the names of all authors and their affiliations. Only the email of the corresponding author and telephone numbers are required.

Duration of Presentation

The presentation usually lasts 30 minutes followed by another 30 minutes of discussion.

Check list for slide presentation

1. Visual Aid
Please check that slides are:
 - a. not too many for a thirty-minute presentation
 - b. contents are coordinated with speech
 - c. slides are neat and spaced to fill the screen
 - d. simple and free from excessive data
 - e. free from garish colour and other embellishments that may distract from your message
 - f. have a balance of data, lists, and information with photographs interspersed throughout
2. Speaker
Please ensure that
 - a. You are familiar with speech and slides
 - b. You are dressed properly to make your audience comfortable
 - c. Reduce excessive gestures
 - d. Avoid reading from the slides and notes all the time
 - e. You keep to the time allocated for presentation