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VETERINARY PATHOLOGY REPORT

Australian Society for Veterinary Pathology
Brought to you by:
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Veterinary Clinical Centre
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DEADLINE FOR NEXT VET. PATH REPORT IS: October 31, 1996

SECRETARIAT

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ASVP EXECUTIVE 1995-1997

President	Ron Slocombe	University of Melbourne, Veterinary Clinical Centre Werribee Vic 3030	03 9741 3500
Secretary	Karl Harrigan	University of Melbourne Veterinary Clinical Centre Werribee Vic 3030	03 9741 3500
Treasurer	Mark Williamson	Australian Animal Health Ryrie Street, Geelong Vic 3220	052 27 5000
Committee Members			
	Ian Jerrett	PO Box 1271 Bairnsdale Vic 3875	
	Alison Havadjia	Centaur International PO Box 1284 Bairnsdale Vic 3875	051 52 0800

APPOINTMENTS

Chairperson (Registry of Domestic Animal Pathology)	Tony Ross
Newsletter Editor	Karl Harrigan
Coordinator (Training Committee)	Vacant

CONVENOR - SLIDE OF THE MONTH

Rod Reece	National Registry of Domestic Animal Pathology, EMAI, Private Mail Bag 8, CAMDEN NSW 2570	046 293327

STATE REPRESENTATIVES

Queensland	Bruce Hill, Rockhampton Vet Lab, QDPI Box 6014, Rockhampton MC Q 4702	079 360211
Victoria	Malcolm Lancaster, Victorian Institute of Animal Science, 475 Mickleham Road, Attwood 3049	03 92174200
South Australia	Ruth Reuter, VPS, PO Box 96, Plympton SA 5038	08 3623544
New South Wales	Paul Gill, RVL Wollongbar 2480	066 261261
Western Australia	David Forshaw, Regional Office, WA Dept Ag. Albany 6330	09 8420500
Northern Territory	Anton Janmaat, PO Box 79, Berrimah 0820	089 992240
Tasmania	Barry Munday, Uni Tasmania, PO Box 1214, Launceston 7250	003 243812

Notes from the President;
July, 1996:

With regard to the scientific and social aspects of the combined NZSVCP/ASVP meeting in Christchurch this last June, the meeting was clearly a success and the New Zealanders are congratulated for such a well organised and interesting program. From the perspective of attendance of the ASVP membership, the meeting was not a success and the scheduled AGM failed to reach a quorum, being defined in the statutes as 20% or more of the membership. Less than half of the required number was present.

Under Section 12.4 of these regulations, the Chairman adjourned the meeting on the understanding that written notices to the membership would advise of a new date for a rescheduled AGM. Several alternate dates are currently under consideration, and the committee members and state representatives will be consulted in order to decide the best option.

One matter of emerging concern to us is the relative isolation of Executive from issues affecting State and private diagnostic laboratories. The universities and CSIRO are not affected by changing government policies in the same way as are regional laboratories, and it is clear that the Executive needs continued input from the State Representatives and the membership in order to be prepared and responsive to change. We therefore encourage a greater input from the membership than the executive has previously experienced.

Also noted is a mild decline in membership numbers from 1994 to current, based on fully paid memberships, although the receipt of several new applications for membership is encouraging.

Ron Slocombe
President ASVP

1995/96 ANNUAL GENERAL MEETING

Apologies: Karl Harrigan, Mark Williamson, Robin Giesecke, John Gibson, Bronwyn Smits, Jim Taylor

Quorum: 20% of membership required. 124 paid members were listed as the 1994/95 membership. Therefore, 25 members were required for a quorum and only 7 were present at the opening of the AGM. The President adjourned the meeting on the understanding that members would be advised in writing of a future date for the rescheduled AGM.

President's Report: 1995/96

At the 1995 AGM at Attwood, Victoria, the new executive was installed for the next term. We thank the outgoing executive for their guidance in the transitional period and John Gibson in particular for alerting us to emerging issues. Changes described by John Gibson that affected the membership in the early 1990's continue to evolve, and in many respects political expediency, economic rationalism and imposed strictures without consultation and due process appear to continue, at least in that proportion of the membership that is involved in government diagnostic services.

Since the last AGM, the National Animal Health Committee charter has been established and the representatives on this committee now nominated. The ASVP had no influence in this process; it is not represented on the NAHC and is therefore forced to work indirectly to make matters of importance known to the NAHC. It is my personal view that the lack of affiliation with the AVA has been detrimental in allowing either direct or indirect influence or input of the ASVP executive to the NAHC, particularly since the AVA has direct representation on the NAHC.

Motions arising from the last AGM have been acted on by the executive.

1. That the ASVP) combine with the NZSVCP for a joint conference in 1996...

The meeting took place as scheduled and a small but enthusiastic group of ASVP members attended.

2. Matters raised by a letter of D. Obendorf be brought before the Tasmanian members of the ASVP, The DPIF and the Tasmanian Graziers and Farmers Association.

Matters raised by Dr Obendorf have been investigated, with correspondence between the Executive, Dr. Obendorf, Tasmanian members and the Minister, DPIF taking place. Resolution of these matters is still to take place.

3. The ASVP bring current deficiencies in disease surveillance within Australia to the attention of the NAHC.

4. The ASVP formulate a discussion paper for the purposes of alerting producer organizations of deficiencies in Australian veterinary laboratories...

Letters have been sent to the NAHC foreshadowing a submission from a subcommittee headed by John Mackie regarding the deficiencies in veterinary surveillance and diagnostic services in Australia.

In matters related to above concerns of the membership in this area, the ASVP was represented at formal hearings of the Steering Committee of NSW regarding State Diagnostic Laboratory closures and more recently, Dr Tony Ross and Dr David Obendorf made written and verbal submissions to the Australian Quarantine Review Committee hearings.

Following a letter from Dr John Glastonbury, Wagga Wagga, we contacted the Australian Veterinary Journal to voice our concerns regarding proposed changes to journal style that might negatively affect the membership, particularly proposed changes in policy for the publication of short case reports regarding cases of spontaneous disease. The AVJ acknowledged this concern, indicated that these concerns had already been taken to the AVA, and that policy on journal style had already been finalised and that the matter was now closed. If the membership finds these new policies for publication in the AVJ onerous, then please inform the Executive and we will take these concerns to the new editor.

Subsequent to the recommendations of the Training Subcommittee chaired by Robin Giesecke, we are looking to develop a limited number of training models on a trial basis. The implementation of these recommendations under the stewardship of a new coordinator is currently being pursued. The Executive thanks Robin for her leadership and effort, which should provide tangible assistance for trainees in the near future.

The ASVP is currently represented on a NATA steering committee in the process of developing guidelines for an accreditation scheme for Veterinary Diagnostic Laboratories. These guidelines are likely to be completed by late August, 1996.

Reports from the executive and subcommittees were tabled for consideration at the AGM, and are published in the ASVP newsletter at the request of the membership.

My thanks in particular to Dr Karl Harrigan as secretary, who has undertaken the largest share of the work of the Executive, to Mark Williamson for his work as Treasurer, to the state representatives and the officers on the subcommittees.

Ron Slocombe, President, ASVP.

AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY INC

ANNUAL GENERAL MEETING

20th June 1996 Christchurch, New Zealand

SECRETARY'S REPORT

Transfer of the Society's accumulated paperwork etc. to the new Executive at Werribee was accomplished in the months following the 1995 Annual General Meeting. I thank Jim Taylor for his assistance in facilitating the transfer and guidance in finding my way through the material.

Communications regarding the combined N.Z.S.V.C.P./A.S.V.P.meeting have been satisfactory. Thanks to Alastair Johnstone, Karen Bailey and Brian Cox for their assistance.

A variety of issues have developed over the short period the new Executive has been in office. A number of members have brought issues to the attention of the executive and enabled the Society to provide a response to them. I have attempted to utilize the Society's State Representatives to obtain/disseminate information on certain issues. I thank them for their help and co-operation.

The Secretariat arrangements continue to work well and I would like to acknowledge Mrs Pat Bosence for her untiring and expeditious work on our behalf. Members are reminded that all subscription and membership matters including change of address are handled through the Secretariat.

A.S.V.P. Secretariat (Mrs Pat Bosence)
P.O. Box 114
Walkerville, South Australia, 5081
PH: (08) 3446337
Fax: (08) 3449227

The current membership list indicates a large number who have not yet paid the 1996 subscription. The Secretariat will be gladdened by your positive and expeditious response to this timely reminder on your financial commitment to the Society.

Veterinary Pathology Report - Editor's Report

Number 43 of the V.P.R. was set for publication in March 1996. Unfortunately it was not available until April. Hopefully we will be able to achieve a more timely result in future. We should be able to achieve a publication every 3-4 months.

Again the Society's State Representatives are to be thanked and congratulated for their efforts in obtaining the information which makes up the Report.

I recently received a letter from one of our overseas members concerning the Veterinary Pathology Report and requesting permission to provide case reports for it. The letter also indicated the considerable approval our publication had received from the member's colleagues. They seemed to express a wish that their veterinary pathology group(s) had a similar newsletter.

Members, of course, will not rest on their laurels. They will be busy providing information for the next issue, hopefully to complement some international reports.

K Harrigan
Hon. Secretary

TREASURER'S REPORT FOR ASVP FOR 1995

This report is based on statements issued up to close of business on 1/7/96.

Statement of Income and Expenditure of ASVP for 1995

INCOME

Opening Balance	9,464.62
Subscriptions	6,205.14
Conference Receipts	6,634.30
Credit Interest	306.85
	22,340.91

EXPENDITURE

Conference costs	5,353.80
VPR/Secretarial services	2,436.96
National Registry of Domestic Animal pathology	5,000.00
Training Survey	90.55
Refunds	265.00
Petty Cash	50.00
Bank fees and taxes	45.74
	13,242.05
Closing Balance	9,098.86
	22,340.91

Mark Williamson
Honorary Treasurer

ASVP 1996 ANNUAL REPORT
NATIONAL REGISTRY OF DOMESTIC ANIMAL PATHOLOGY

MISSION STATEMENT: to provide cost-effective continuing professional education and development relevant to veterinary pathologists working in the animal health industry of Australia, and to assist in maintaining and improving professional standards of veterinary pathology by providing a national registry and a quality assurance service.

1. Rod Reece continued his term as Registrar (part-time) during 1995/96. Funding from Federal and State sponsors is secure until February 1999.

2. Cases selected for entry into the Registry were derived from cases referred for second opinion, cases solicited by the Registrar, and cases sent to the registry as good examples of particular conditions. These have been derived from Australia and Overseas, and therefore cover both endemic and exotic diseases.

3. As at late May 1996, the registry contained 965 cattle cases, 527 sheep, 277 pigs, 155 goats, 311 horses, 26 deer, 495 dogs, 184 cats, 657 poultry and 136 other species (total 3733). These are fully indexed and readily retrievable using cards or a computerised system based on PARADOX.

4. A small number of transparencies were donated in 1995/96 by Dr Dave Forshaw (Albany WA) and Dr Roger Kelly (Univ Qld) - for these we are thankful.

5. A 12" Laser Disc player was purchased with funds donated from ASVP. This allows access to the 25,000 transparencies on a laser disc obtained from University of Athens, Georgia, USA. There is both a free form search mode allowing sequential presentation of cases on topics such as horse liver or sheep skin, and also an interactive training set on many pathological conditions such as common post mortem findings in pigs or renal lesions. The player was transported to every state last year (except SA) and used in training courses. Arrangements can be made to borrow this. It requires a standard computer with keyboard and screen for text, a mouse is useful but not essential, and a high resolution screen for displaying the figures from the laser disc.

6. Funds were obtained from ASVP to purchase a camera and transparency copying equipment. Some of this has been obtained.

7. Training courses were held in late 1995-early 1996 with the theme of kidney and liver pathology. The format was a lecture type overview followed by examination of selected specimens from the Registry to demonstrate response of the organs to insult and common diagnostic features. This was followed by case presentations by attendees on the same theme.

SA: staff reductions, absences due to leave and workload precluded SA from participating this year.

Victoria: 12 veterinary pathologists (2 Agriculture, 4 University, 2 CSIRO, 3 private, 1 Centaur) attended a one day session held at the Veterinary Clinical Centre, Werribee. Besides presentations by the registrar and case reviews by attendees, Dr John Dowling from Monash Medical Centre gave a lecture on "Diagnostic approach for human renal biopsies".

TAS: 4 Government veterinary pathologists attended for 3 days instruction at Mount Pleasant Laboratory.

NT: 4 Government veterinary pathologists, including a trainee for 4 days at Berrimah Veterinary Laboratory.

QLD: veterinary pathologists from DPI (4) and University of Queensland (2) for 2 days at the Department of Veterinary Pathology, St Lucia; the Registrar then attended the regional veterinary pathology meeting at Toowoomba where the theme was neuropathology.

WA: veterinary pathologists from Murdoch University (2) and Agriculture (4) for 3 days initially at Murdoch University then at South Perth Laboratory.

NSW: veterinary pathologists from Agriculture (7), Veterinary Pathology Services (1) and a post-graduate student from Sydney University (1) for 2 days at Elizabeth Macarthur Agricultural Institute.

Forty-one veterinary pathologists received continuing professional education and development (CPED) over 1-4 days via these courses. As before, the policy was to charge $100 per day for non-sponsors or significant services in return but this seemed to exclude many of the private practitioners and university departments.

8. A large dossier (5cm thick) on Bovine Spongiform Encephalopathy and related diseases, and possible relationship of BSE to human health, including the newly recognised variants of CJD, was prepared by the registrar in May 1996 for distribution. Copies can be obtained by contacting the Registrar at $15 to cover cost of paper, post and packaging.

9. Preparation for 1996/97 courses has been superseded by impending involvement of the registrar in a national training workshop for the diagnosis of bovine spongiform encephalopathy and related diseases. This is being organised by CSIRO Australian Animal Health Laboratory on behalf of SCAHLS and Dr. G. Wells from UK will be the main presenter. It is anticipated that training courses will be held in the different states in 1997 utulising material presented at the national workshop on BSE as a platform for discussion of neuropathology.

10. The Slide-of-the-Month continued to operate in 1995/96. Some contributors experienced difficulties in supplying sections for distribution due to staff, time and funding difficulties. This is a reflection of constraints imposed upon many of our colleagues at this time. The SOTM is an important contributor to CPED, and many colleagues have commented on its benefit. Some contributors have requested comment on sections but have received little feedback: please reply as it is an important contribution to wider CPED of colleagues.

In summary: the NRDAP provides a unique and valued source of continuing professional education and development, and quality assurance in the area of veterinary pathology. The registry continues to be supported by members of the ASVP and through them by the respective state and federal bodies.

Rod Reece, Registrar
National Registry of Domestic Animal Pathology
EMAI, PMB 8 Camden, NSW 2570
Phone 046 293327; Fax 046 293400

Training Committee Report

MODULAR TRAINING AND CONTINUING PROFESSIONAL DEVELOPMENT

REPORT TO THE ANNUAL GENERAL MEETING, 1996

At the 1995 AGM the Training Committee was given the responsibility of finding a suitable candidate for the pivotal role of national Coordinator. Finding a suitable person has been difficult, as all have been affected by job losses and funding cuts. The committee considered several options, including the possibility of offering a funded position to a retrenched person with suitable qualifications to work with the Registrar of NRDAP. It became apparent that University Veterinary Pathology departments are better resourced to supply credible training programs, in the short term at least, and that the next Coordinator would be better placed in closer communication with these departments and the executive. None of the present committee was able to take on the position due to their added responsibilities but Dr Judith Wilkie-Nimmo has expressed willingness and interest in the role, and enthusiasm for the concept. It is therefore recommended that Judith Wilkie-Nimmo is appointed as the next national training coordinator.

The role of the national coordinator would be to develop a framework for the modules, select and assist the module writers from listed specialists and university staff, identify resource material and liaise with resource providers and executive. An additional role could be the development of the program at the annual conference to include subject and speakers which would enhance the module material. To undertake these tasks, it is recommended that the Coordinator be given up to $3000 per annum from ASVP funds to cover expenses.

There is probably no more important time than this for training programs to be undertaken. As Professor Slocome recently reported (VPR no.43, P. 2) legislation may soon make quality assurance programs necessary, at least for those laboratories in Victoria providing diagnoses for livestock. In the longer term a need for quality assurance or for laboratory accreditation will underpin the need for credible training programs. The development of accessible training programs is therefore vital to the careers of veterinary pathologists everywhere and ASVP is in a position to lead, through initiation of such programs.

Mindful of this, discussions have been held with Professor Wayne Robinson, head of Veterinary Pathology at the University of Queensland, on the financial feasibility of structuring programs within the current restrictive environment. He believes that in the short term, module development could be funded from student fees along the following lines:

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Students would pay $ 2000 for a two stage program, each program consisting of 12 modules.
Module coordinators would be paid $200-$400 from these fees to develop each module.
A minimum of four paying students would make this possible.
Students would proceed through the modules at their own pace, using them for revision, upgrading, or for College Membership preparation. The modules would be based on systems and include material from all species.
In the longer term, modules could be developed as marketable commodities, and placed on the Internet or CD-ROM.

While fully supportive of development of training programs, Rob Rahaley and I will not be continuing on the Committee. The opportunity could be taken to increase the representation of universities on the new committee if so desired. It is also **recommended that Professor Robinson be invited on to the committee or to act as a consultant** with Roger Kelly representing the University of Queensland.

I would like to take this opportunity of thanking the past committee, in particular John Mackie, John Glastonbury and Roger Kelly, for their unflagging support for the development of training for veterinary pathologists and for their support for my efforts to progress it during my term as Coordinator.

Robin Giesecke
(Acting) Training Coordinator.

The following letter is published for the information of all A.S.V.P. members with the approval of Dr Ross.

Secretariat
Australian Quarantine Review Committee
PO Box 4596
Kingston
ACT 2604

22 May, 1996

Submission from Dr Tony Ross
Chairperson, Management Committee
National Registry of Domestic Animal Pathology

The National Registry for Domestic Animal Pathology was established in 1986. It provides continuing education and quality assurance programs to veterinary pathologists throughout Australia. A recent fact sheet on the Registry is attached.

I have worked in Australia, New Zealand and the UK in the private, government and university sectors of veterinary medicine and veterinary pathology for the last 24 years. I am a registered specialist in NSW in the field of veterinary pathobiology. The following is a personal submission and does not necessarily represent the views of the Australian Society of Veterinary Pathology.

1. Privileged Animal Health Status

1.1 There is no doubt that Australia has privileged animal health status due to our remote location and previous policies of risk avoidance.

1.2 Improvements in transportation have increased the risks of accidental and illegal importation of new animal and plant diseases. Australia's animal health status affects all Australians either directly or indirectly.

2. Risk Avoidance VS Risk Assessment and Management

2.1 I understand that our GATT obligations require us to discard risk avoidance and embrace risk assessment and management in determining importation policies.

2.2 This being so it is essential that a critical mass of experienced and qualified experts in animal and plant disease risk assessment be assembled and maintained to support appropriate policy development.

2.3 By definition, risk assessment and management increases the risk of importing disease above that associated with risk avoidance.

3. Managing Risk

3.1 An important component of the success of Australia's quarantine policies is their effectiveness at point of entry. I can provide first hand examples where this has been clearly inadequate including:

- tropical fish
- sheep
- contaminated footwear

3.2 Once a disease has been introduced it may be possible to control and even eradicate it.

3.3 Successful eradication usually requires early detection.

3.4 Early detection requires community awareness and a critical mass of teams of strategically placed field and laboratory veterinary diagnosticians.

4. Critical Mass of Field and Laboratory Diagnosticians

4.1 During my 24 years working in this area I observed an increase in critical mass during the 1970s, most notably in Victoria.

4.2 More recently in the 1990s I have observed severe reductions in trained and experienced field and laboratory diagnosticians particularly in the areas of farm animals, fish, poultry and wildlife. The remaining diagnosticians have an average age of 45-50 years. Young diagnosticians are conspicuous by their absence.

4.3 Competent field and laboratory veterinarians are both needed for a successful diagnostic team. The skill, experience and motivation required are rarely found in private sector veterinary practitioners and pathologists.

4.4 Therefore the loss of diagnostic services in most states can be correlated with the reductions in public sector veterinary services.

4.5 It is my professional opinion that there are several large areas of Australia where adequate risk management cannot be practised due to the provision of severely inadequate diagnostic services.

5. The Essentials of Veterinary Field Diagnostic Service for Risk Management in Australia

5.1 Appropriately trained and resourced field veterinarians are required with skills in epidemiology and pathology as well as clinical medicine.

5.2 They must be funded in a way which encourages rather than discourages investigation of new or unusual events. The financial realities of private veterinary practice do not usually permit such investigations.

5.3 These field veterinarians should be located throughout Australia in regional locations most relevant to the animal industries being monitored.

5.4 They should be supported by an awareness and continuing education network and linked with diagnostic laboratories to provide the information and skills they need.

5.5 They should supply health monitoring data to a centralised facility or facilities. The data are used as the basis for reporting the current animal health status within Australia and to our customers overseas. New Zealand is an excellent structural model for disease surveillance and reporting. Although it falls short in the number of field and laboratory diagnosticians who remain involved in the diagnosis of diseases in commercial livestock.

6. Trends in Veterinary Laboratory Services in Australia

6.1 Adequately staffed and trained regional veterinary laboratories are the field veterinarian's link to the accurate diagnosis of a wide variety of diseases in livestock.

6.2 When regional laboratories are not present or are inadequately resourced, the ability to diagnose many diseases is severely curtailed.

6.3 Through the 1970s and into the 1980s Australia in most states had an effective network of regional veterinary laboratories which was recognised as world class.

6.4 Overseas visitors to these laboratories readily acknowledged their important role in underpinning the superior health status of Australia's herds and flocks.

6.5 During the last decade state governments reduced, and in some states dismantled their regional laboratory networks for largely financial reasons.

6.6 The remaining laboratories vary widely in their mandate and scope of services. There is a trend to embrace non-farm animal diagnostic work - again largely for financial reasons.

6.7 Commercial veterinary laboratories have emerged in several states in the last decade.

6.8 In general terms, public sector funded laboratories in developed countries tend to be full service laboratories offering a wide range of services to perform general and regulatory diagnostic testing. Support for regulatory and non-regulatory testing is generally considered to be for the collective public good.

6.9 Commercial veterinary laboratories are less likely to offer a full service. Some services such as post-mortem examination of large animals, serological testing for a range of diseases and investigation of many virus diseases are not usually cost effective and are therefore not offered.

6.10 Surveillance information retrievable from commercial laboratories is usually poor or non-existent.

6.11 Some areas of Australia claim access to full service laboratories when in reality only a partial service is available.

7. Recommendations

7.1 Australia's animal quarantine policy framework be revised to require a minimum functional network of field and laboratory diagnostic monitoring and reporting services.

7.2 That quarantine risk assessment processes be required to take account of the lack of effective diagnostic, monitoring and reporting services when assessing risk.

7.3 An audit of the current field and laboratory diagnostic, monitoring and reporting services be undertaken to identify areas of adequate and inadequate services. This audit should be conducted by independent experts in the field.

7.4 Prepare a position paper, action plan and a stakeholder and public consultation process aimed at ways to maintain and/or reintroduce adequate services to all major livestock regions in Australia.

I would be grateful for the opportunity to present this submission at a public hearing.

Yours faithfully

A. D. Ross
Chairperson, Management Committee
BVSc, Msc, PhD

Queensland - Bruce Hill

Pigeon Herpesvirus infection and Trichomoniasis - John Gibson, Toowoomba Veterinary Laboratory

A mortality rate of 54% was recorded over 2 months in a mixed age group of young pigeons on a commercial squab enterprise at Dalby. Necropsy of one bird revealed severe necrosis, dilation and thickening of the wall of the oesophagus. The mucosal surface was covered with loose yellow necrotic debris. Direct smears of the lesion revealed large numbers of protozoa consistent with trichomonads. Histologically sections of oesophagus had focal areas of ballooning degeneration extending from the basal cells to the stratum corneum. Many ballooned nuclei had pale eosinophilic intranuclear inclusions. Superimposed on the above lesion was a superficial necrosis with fibrin pseudomembranes adherent to the surface. Transmission EM of the lesion demonstrated enveloped viral particles typical of herpesvirus within nuclear inclusions. Another bird submitted from the same property had severe hepatitis with numerous viral inclusions.

Mycoplasmosis in a young goat kid - John Gibson, Toowoomba Veterinary Laboratory.

Mycoplasma capricolum was isolated from the joints of a young male kid from a property at Oakey. The kid was one of three affected on a property running 1000 goats. The kids had sudden onset of fever (41C), joint stiffness and swelling. One kid necropsied had severe polyarthritis with excess cloudy synovial fluid in most joints. In some joints the fluid contained fibrin. Histologically the lesion was a severe acute fibrinopurulent tenosynovitis.

FNQ Contribution - P.W. Ladds, Department of Biomedical and Tropical Veterinary Science, James Cook University, Townsville.

Interesting recent cases have included:

Canine	Presumed aflatoxicosis in several animals; Babesia canis infection in pups from two litters; "classic" renal lesions of acute leptospirosis with what seemed to be terminal DIC; histiocytic ulcerative colitis; "black grained mycertoma" in sublingual region; preputial abscess in a 4 month-old dog caused by a filamentous (*Actinomyces/Nocardia*) organism; fatal parvoviral enteritis; mycotic {probably *Aspergillus* sp.) rhinitis in a 5 year old dog, and chronic copper toxicosis in a Bedlington Terrier.
Avian	Rickets in a cockatoo; mycotic pneumonia and/or air sacculitis in a sun comure and in ostrich chicks; mycotic coelomitis in a Muscovy duck and severe necrotising enteritis (possibly salmonellosis) in a chicken.
Equine	Pigmented fungal granuloma of skin and chronic renal failure in an old animal with advanced amyloid type change in both kidneys.
Feline	Cryptococcal rhinitis.
Wildlife	Coccidiosis in a Grey kangaroo and cryptococcal pneumonia in a koala found dead on Magnetic Island.

On the research front, **Windia Adnyana** has resumed his PhD studies on diseases and parasites of wild-caught turtles in Indonesia and Gilbert Buenviaje (who completed an MSc degree at JCU a few years ago) has returned to commence an in-depth study of skin diseases in crocodiles.

ASVP members will also be interested to learn that **Dr. Robert Foster**, currently a staff member at Ontario Veterinary College, will be returning to JCU for three months from mid July as a Visiting Lecturer. I am sure he is looking forward to renewing acquaintances with many colleagues.

Victoria - Malcolm Lancaster

Bacillus piliformis in a pup - Fenella Muntz, Central Veterinary Diagnostic Laboratory.

An owner lost one 6 week old pup in each of 3 litters born at around the same time to one German Shepherd bitch and 2 wire-haired Dachshund bitches. Bitches were housed in different areas. All pups presented with bloody diarrhoea and died within 1-2 days. The bitches and merest of the litters remained in good health.

We received multiple post mortem tissue from one pup. Histopathology revealed a multifocal necrotizing hepatitis with bundles of filamentous *Bacillus piliformis* organisms seen within intact hepatocytes at the edge of the necrotic zones (confirmed by Warthin Starry staining).

There was also a moderate neutrophilic enteritis with multifocal erosions and associated (non-Bacillus) bacterial colonies. Low numbers of *Bacillus piliformis* organisms were identified by special stains within the intestinal mucosa. Culture of the intestinal contents grew a heavy growth of mixed organisms. No specific pathogens were isolated. Occasional coccidial organisms were seen within and beneath the small intestinal epithelium (probably *Isospora canis*).

Bacillus piliformis (Tyzzer's disease) occurs most often in lab animals and in foals. It is reported uncommonly in dogs and cats. Infection is usually thought to be via ingestion of bacterial spores (in rodent faeces). It is possible that dogs and cats harbour the organism which, under stressful conditions, may produce disease in the gastrointestinal tract. Intestinal infection disseminates via the portal circulation to the liver and less often to the heart.

Immunodeficiency is though to contribute to the disease as most cases have been seen at around weaning age. Often animals have concurrent immunosuppressive diseases such as canine distemper, feline leukaemia or feline panleukopaenia. In this case the owners had a lot of dogs so that overcrowding may have been a contributing factor. They also had a mouse problem so that may have been the source of the bacteria.

The organisms are very difficult to isolate so diagnosis is usually made on histopathology. Organisms are gram negative. They stain best with silver stains. Giemsa, methylene blue and PAS can enhance visualisation. The organism has recently been shown to belong to the Genus Clostridium by RNA sequence analysis (*Clostridium piliforme*).

References:

Duncan, Carman, Olsen, Wilson. Assignment of the agent of Tyzzer's disease to *Clostridium piliforme* comb. nov. on the basis of 16 Sr RNA sequence analysis. Int. J. System Bacteriol 43:314-318, 1993.

Greene - Infectious diseases of the dog and cat, 1990 Volume 2, p373.

Jubb, Kennedy and Palmer, Pathology of Domestic Animals.

Granulomatous Lymphadentitis and Enteritis in a Thoroughbred Horse - Kathleen Johnston, Central Veterinary Diagnostic Laboratory

A three year old female thoroughbred horse was presented for weight loss, diarrhoea, increased gut sounds and decreased appetite. A complete blood examination at that time demonstrated lymphocyte activation and no other abnormalities. Faecal examination was negative for endoparasites, RBCs, luecocytes and *Salmonella* but positive for *Campylobacter* spp..

In spite of treatment with antibiotics, anthelmintics, corticosteroids and dietary adjustment the condition of the horse continued to deteriorate over the ensuing 7 months and the horse was euthanased. A postmortem examination revealed enlarged, oedematous mesenteric lymph nodes.

Sections of spleen, mesenteric lymph nodes, colon and small intestine were examined histologically. There was evidence of erythrophagocytosis in the spleen and all sections of intestine. The lamina propria of small intestine and colon was infiltrated with large numbers of lymphocytes and fewer macrophages, effacing crypts and filling small intestinal villi, resulting in villous atrophy and fusion. Within the colon there was prolapse of the crypts into Peyer's patches. Throughout the lymph nodes there were zones of necrosis and fibrosis, surrounded by large numbers of histiocytic macrophages. Acid fast stains revealed large numbers of mycobacterial organisms within the macrophages of the lymph nodes.

Granulomatous lymphadenitis of mesenteric lymph nodes associated with coagulation necrosis has frequently been reported as a cause of chronic weight loss, generally in young horses. Often this is accompanied by hypoproteinaemia. Though the horse possesses a strong innate resistance to tuberculosis, infection involves ingestion of the organism and localization in the gut and mesenteric lymph nodes. This may be followed by hematogenous spread to cause generalized infection and hepatic and pulmonary involvement. The course of disease is chronic with progressive debilitation.

Malabsorption and protein loss from the intestinal mucosa is subsequent to chronic inflammation in the lamina propria with secretion of cytokines from activated T-cells. This results in stimulation of crypt epithelial lining cells with increase in the kinetics of epithelial cell turnover. Immature crypt epithelial cells migrate onto the villi and are incapable of normal absorption. They quickly move up the villi and are shed prematurely resulting in villous atrophy and fusion.

These changes have been associated with various mycobacterial species including *M. avium, M. avium-intracellulare*, and *M. paratuberculosis*. Fresh tissue was not submitted in the case, thus there was no attempt to identify the species of mycobacteria involved.

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Cryptosporidiosis in an Ostrich chick - Malcolm Lancaster, VIAS Attwood.

An ostrich breeder reported 100% mortality in 10 birds hatched over the previous 5 weeks. Birds became progressively depressed over 2-3 days before dying. Two birds were submitted for necropsy, both 2 weeks old.

Histological changes were observed in the intestines of both birds. There was accumulation of cellular debris and proteinaceous droplets in spaces beneath and between epithelial cells of the villi. Bird one had lost most of its epithelium due to autolysis. In bird two the epithelium was better preserved and more heterophils were apparent in and beneath the epithelium. In some regions the villi appeared smaller than normal. Many structures morphologically consistent with cryptosporidia were apparent in the brush border of some areas with and without nearby inflammatory changes.

These chicks can be put into the "ostrich fading syndrome" basket, aetiology unknown. The role of cryptosporidia in this syndrome and in this case is not clear. It may simply be an opportunistic pathogen in some birds immunocompromised for other reasons. However, given the degree of autolysis and associated intestinal epithelial loss evident in many ostriches at necropsy, the prevalence of this organism in diseased birds may be underestimated.

Marek's Disease - John Humphrey, VIAS Attwood

Numerous cases of Marek's disease have been seen in commercial poultry over the past 3-4 months, with high mortalities and high production losses reported. In a number of cases affected birds have been submitted with a history of vaccination. Clinically, neuromuscular signs of paralysis, paresis, incoordination, twisted and contorted necks, an inability to stand without the aid of outstretched wings and backwards stretching of one leg are seen. In many cases typical signs are absent and birds present with weakness, depression and reluctance to stand or move. On occasions sub-epidermal tumours are present. Signs generally commence in birds prior to or at point-of-lay.

Gross pathological examination may show no lesions or may show soft, pale, single or multicentric tumours involving a range of organs and tissues. Ovary appears to be a common site of tumour formation, often associated with obstructed oviducts and yolk in the abdominal cavity. On occasions, massive replacement of skeletal muscle by tumour tissue has been seen.

Histologically cells forming tumours are characterised by a high degree of pleomorphism with lymphoblastic, lymphocytic and plasmacytic cells types evident. In many cases large "Marek's disease" cells are present. Lymphoid infiltration of central and peripheral neurological tissue is common but many cases do not have nervous involvement. Locally expansive infiltration of the lamina propria of the intestine with compression of adjacent crypts and loss of villous structure occurs commonly.

Attention is drawn to the frequent occurrence of Marek's disease and to possible changes in the pathogenesis and epidemiology of the disease, especially in the face of reports of a history of vaccination.

Polyarthritis in Lambs - Mike Forsyth, Centaur International, Bendigo.

Two cases were submitted in late May 1996 of 2 Iambs which were 7 to 10 days old on the one hand and one lamb 3 weeks old on the other, where there were lambs in the flock with stiffness and deaths. In the first case, the owner reported losing 30-40 merino lambs out of 1300 ewes at about 7 to 10 days. The affected lambs became stiff, lethargic, recumbent and died after 2-3 days. In the second case, the first cross Merino lambs were stiff and were abandoned by their mothers and died. The weather during this period had been wet early in May but was dry in late May.

At post mortem examination the three lambs had purulent polyarthritis. Bacterial cultures revealed CO2 dependent, clear, glistening colonies of short and long gram negative organisms, catalase and oxidase negative, not growing on McConkey's agar and biochemically inert. These were classified as *Actinobacillus seminis*.

Their antibiotic sensitivity patterns were similar from both properties - sensitive to neomycin and tetracycline and resistant to trivetrin, gentamycin, amoxicillin streptomycin and penicillin.

One owner reported having *Actinobacillus seminis* (*as Histophilus ovis*) diagnosed 7-10 years previously.

Fatal Haematozoan infection in an emu - Ian Jerrett, Centaur International, Bairnsdale.

An autopsy was performed on an 18 month old farmed emu, one of three adult birds to have been found dead within a 2 week period. All deaths occurred in birds introduced to the property 3 weeks previously. On gross examination the liver was enlarged and congested with a pronounced lobular pattern on the cut surface. The spleen was also enlarged.

Microscopic examination of the spleen and liver revealed large numbers of megaloschizonts, some of which had ruptured and released small round merozoites. In the liver the megaloschizonts were most numerous in congested central veins and periacinar sinusoids. No host cell nuclei were identifiable. There was widespread parenchymal macrophage infiltration in both the liver and the spleen. Schizonts were also present in renal veins and in renal tubular epithelium. A few equivocal Leucocytozoan-like gamonts were seen in renal capillaries.

The identity of the Haematozoan is not known. Similar megaloschizonts and lesions have been seen in emus in other states (R. Reece, W. Hartley pers. comm..).

Ovine Johne's Disease in East Gippsland - Ian Jerrett, Centaur International, Bairnsdale.

Johne's disease has been diagnosed in Merino sheep on 8 properties in East Gippsland. Seven of the 8 flocks were related. Sporadic cases of progressive fatal ill-thrift and mild diarrhoea in adults had been reported on each property. Most cases have been in 4-5 year old ewes but one clinically affected animal was 12 months old.

A striking gross finding has been the prominence of serosal and mesenteric lymphatics over the lower small intestine, caecum and proximal colon. The ileo-caecal and mesenteric lymph nodes have generally been slightly enlarged, some have been fibrosed. The ileal mucosa has usually been thickened and either finely nodular or formed into transverse ridges. Caecal and colonic thickening has been variable.

Microscopic granulomatous intestinal and lymph node inflammation has generally been severe and a marked serosal lymphangitis has been a consistent lesion. Acid-fast organisms have been numerous in most cases - in one case few organisms were seen. We have so far isolated *M. paratuberculosis* by radiometric culture from 5 cases. The AGID test was positive in 4 of 7 cases from which blood was obtained.

Infectious Canine Hepatitis Revisited - Alison Havadjia - Centaur International, Bairnsdale.

A five year-old male Kelpie cross was presented to a submitting veterinary clinic with sudden onset of vomiting, anorexia, depression and lethargy. On physical examination there was mild dehydration, pale mucous membranes, temperature of 41°C, slightly increased heart and respiratory rates and an enlarged liver on abdominal palpation with no apparent associated pain.

In clinic, haematology and biochemistry showed mild anaemia, marked leukocytosis, hyperbilirubinaemia and elevated ALP and ALT (the latter off the scale!). The urine was dark orange with 2+ protein and 2+ bilirubin.

Blood was submitted to the laboratory for *Leptospira interrogans* serology. Titres to *L canicola* and *L. icterohaemorrhagiae* were both negative (<1:50).

The dog remained pyrexic and became obviously jaundiced. After three days of supportive and antimicrobial therapy (penicillin and streptomycin), he died. An autopsy performed by the practitioner revealed generalised icterus, hepatomegaly and splenomegaly.

Fresh and formalin fixed samples of liver and spleen were submitted to the laboratory. There was no growth on routine microbial culture of either tissue.

Histopathology revealed a congested spleen with mild haemosiderosis, depleted white pulp and areas of necrosis. The liver was also congested and showed widespread severe degeneration and necrosis of hepatocytes, leaving only a narrow zone of normal appearing hepatocytes in periportal regions. There were heavy periportal to diffuse accumulations of mixed inflammatory cells, predominantly neutrophils. Bile ducts were full of necrotic cells and debris and there was much bile plugging of canaliculi. Moderate numbers of intranuclear eosinophilic inclusions were seen, mostly in necrotic hepatocytes.

A presumptive diagnosis of infectious canine hepatitis was made and on questioning, the dog was reported to have been vaccinated for distemper, hepatitis and parvovirus as a pup and for the first two years of its life but not in the past three years.

The practitioner wanted the diagnosis confirmed beyond doubt if possible. Virus isolation of canine adenovirus is not a popular test anywhere but after numerous phone calls Cyanamid Websters agreed to do serology on the initial serum sample. The SN titre was 2896, which was extremely high, normal vaccination titres being approximately 200 to 250. Confirmation of the disease was thus obtained and no doubt there was a significant boost in the veterinarian's income from routine vaccinations!

Elevated GGT in Horses - Janeen Samuel - Centaur International, Hamilton and John Callinan - Cox Street Veterinary Clinic, Hamilton.

Over a period of 20 months, a standardbred trainer, a veterinary practitioner and a pathologist were all frustrated by their joint inability to find a cause for consistently elevated levels of gamma glutamyltransferase (GGT) in horses under the trainer's care. The levels were detected when biochemical profiles were run on horses showing poor performance. Over the 20 months, 15 horses were found to have levels of GGT at least one-and-a-half times the upper limit of the normal range with some values as high as 5 times the upper limit. There were no haematological abnormalities. Total bilirubin was elevated in some of the horses although none had clinically detectable jaundice. Alkaline phosphatase was significantly raised in about half of the horses in which it was measured but in some of these, non-hepatic sources may have been the cause. Glutamate dehydrogenase was within normal limits in the majority of affected horses and only two horses had levels more than twice the upper normal limit. In four horses that were tested on more than one occasion, GGT levels remained high for periods of up to 12 months.

Both males and females were affected. Most were unrelated and had not been bred on the property. In spite of poor performance none was showing clinical illness and therefore liver biopsy was considered not justifiable. Initially the elevated GGT was attributed to overzealous iron supplementation as many of the affected horses also had higher than normal levels of serum iron: up to 48 umol/1 (normal 12-25). However the problem continued for over a year after mineral supplementation was discontinued. In one horse the serum iron fell gradually over 10 months to normal levels while the GGT remained essentially unchanged. Moreover several horses with high serum iron had normal GGT. The trainer denied using any other drugs or chemicals. There was no evidence of liver fluke infestation.

At length a thorough "farm walk" was undertaken by the practitioner. This revealed no toxic plants in the pasture and no apparent sources of toxins in the stables and feed storage areas. However it happened that a load of feed oats was being delivered and questioning of the grower revealed that he routinely treated the oats with aluminium phosphide (Phostoxin) - "for weevils" although it is in fact a rodenticide. Liver damage is among the effects recorded in acute poisoning by this compound or the related zinc phosphide but we have been unable to find any references to subclinical effects. At the rate at which it was being added to the grain the dose would have been well below that needed to produce acute poisoning. Its use has now been discontinued and we are waiting to see what effect this will have. We would be interested to hear from anyone who has had experience of subclinical poisoning by aluminium or zinc phosphide or who has an alternative explanation for the elevated GGT in these horses.

South Australia - Ruth Reuter

A novel glomerulopathy in transgenic mice - John Finnie, Vetlab, Adelaide

A strain of transgenic mice (Eu-pim-1) was being used in a carcinogenesis study because of its predisposition to malignant lymphoma. A significant proportion of these mice, however, died or was killed due to renal failure.

Microscopic examination of affected kidneys revealed severe glomerular disease involving most if not all glomeruli in an animal. In H & E sections the most striking change was the presence of copious amounts of deposited homogeneous, eosinophilic intracapillary material resulting in obliteration of many capillary loops. The deposited material was PAS-positive, stained green with Masson's trichrome and variably argyrophilic; this material also appeared somewhat laminated with silver stains. Furthermore, the majority of glomeruli showed a proliferative glomerulonephritis, predominantly mesangial in nature with a concomitant increase in mesangial matrix. Some glomeruli also showed mild segmental double contouring as is seen in mesangiocapillary (membranoproliferative) glomerulonephritis and a few manifested minor extracapillary proliferations in the form of small epithelial crescents. Tubulointerstitial reaction was variable with some tubular casts present and, in many, lymphomatous interstitial infiltration.

This glomerulopathy requires further characterisation and does not conform precisely to any one category of human disease. However, it almost certainly represents a reaction to the lymphoproliferative disease process and probably best resembles a cryoprecipitable IgM deposition or Type 1 cryoglobulinaemia.

Giant cell tumour of bone in a cat - Ruth Reuter, VPS, Adelaide

Giant cell tumour of bone is a relatively uncommon and frequently controversial diagnosis in animals. Giant cells of varying morphology can be found in a variety of neoplastic and other disease conditions. The condition most often high on the list of differential diagnoses is the giant cell variant of the osteosarcoma.

Formalised tissue was submitted to the laboratory from a desexed male Siamese cat 11.2 years old, belonging to a local veterinarian. A mass had been noted on the forehead the previous week. X-rays revealed that there was invasion through the cranial bones and into the underlying frontal sinus. The sample submitted was composed of sections of connective tissue, fragments of bone and attached muscle.

On histopathology there were very large numbers of multinucleated giant cells in a stroma of fibrous connective tissue accompanied by some smaller supporting cells. The sheets of giant cells occupied all fields in all sections of tissue and invaded the adjacent muscle. There was little or no production of osteoid in the sample. A diagnosis of giant cell tumour of bone was made.

Giant cell tumours have been reported primarily in dogs and cats, usually involving the long bones. However, cranial lesions have been described. Most of these have been benign although aggressive variants occur with metastasis to the lungs. In the case described here the veterinarian operated on the lesion. The neoplasm was soft and crumbling, invading the bones of the cranium and extending in a projection into the sinus. He removed as much as possible. The cat was doing well 6 weeks later.

Bees attack dogs - Martin Copland, VPS, Adelaide

Inexplicably two Cocker Spaniels were set upon by a swarm of bees. One animal, a two year old named Toby received many stings. Afterwards he showed hypersalivation and diarrhoea and was treated with cortisone, antihistamines and antibiotics. The next day he appeared depressed, slightly dehydrated and displayed regular but apparently involuntary contractions of his abdominal muscles and diaphragm. His urine was dark red.

Biochemical and haematologic tests were conducted on days 2, 4 and 8 after the attack. The initial test, Day 2, showed increased total proteins (120 g/L) which was probably attributable to haemolysis, increased muscle enzymes (CK 4224 U/L, AST 533 U/L) and moderate to severe anaemia (PCV 0.20 L/L). In subsequent tests muscle enzymes waned and the PCV feel to 0.13 L/L. Icterus developed and peaked on Day 4 (total bilirubin 257 U/L). Reticulocytosis was evident and marked spherocytosis was noted on all three occasions.

Toby and his partner, Zac, made a full but slow recovery over several weeks.

This episode is similar to an attack on five men in Brazil by "Africanized" honey bees (*Apis mellifera scutellata*) after which three men died within 3 days. In that case the clinical features were reported as intravascular haemolysis, respiratory distress, hepatic dysfunction, rhabdomyolysis (myoglobinaemia and myoglobinuria), shock, coma and acute renal failure.

In Toby's case the prime lesion appeared to be haemolytic anaemia rather than extensive rhabdomyolysis.

Tasmania - Barry Munday

Barry Munday is reviving work on ulcerative mycosis of platypus in Tasmania and would be grateful for reports on the occurrence of similar disease elsewhere. At present the problem seems to be restricted to streams draining into the Tamar River in northern Tasmania. Niall Stewart, one of Barry's honours students, is working on fungal isolates from affected platypus and has found one recent isolate to be *Mucor circinelloides* (confirmed by David Muir at the Mycology Reference Centre, Royal North Shore Hospital). The finding is probably not surprising as the invading fungi (*M. amphibiorum* and *M. circinelloides* are almost certainly opportunists.

Another of Barry's students is working towards her PhD on the immunology of southern bluefin tuna and would be grateful for sera from this and/or other tuna species. Game-fishermen (fisherpersons) among the readership may be able to assist.

Another fish histopathology workshop is being planned for February 1997. This will be run in conjunction with a fish immunology workshop conducted by Dr. Judith Zelikoff of the Institute of Environmental Medicine, New York University Medical Centre.

Mt. Pleasant laboratory have recently diagnosed Johne's disease in sheep and a viral infection in clams.

New South Wales - Paul Gill

NSW Agriculture Lab. Closures

NSW Agriculture's voluntary redundancy program has closed with about 500 of the Department's previous 2300 full-time-equivalent staff having left. RVL Woolongbar lost 7 technical and office support staff. RVL Wagga Wagga lost 5 support staff and veterinary pathologists John Glastonbury transferred to Menangle and John Searson to regional management. Other RVL's lost fewer staff. Pathologists Jim Rothwell left RVL Menangle to join Blanco and Steve Love left RVL Armidale. The Department refused redundancy to vets at the RVL at Orange and Wollongbar which the Government has resolved not to close.

Following the March 2 Federal Labor loss, the NSW Labor Government abandoned the next 3 year's budget cuts ($21 million) to Agriculture. With voluntary redundancies the Department will meet this year's $11.2 million cut with money to spare. It has now embarked on a staff recruitment program, advertising positions for veterinarians (including pathologists) recently and for technical staff in the near future. Enquires to Dr. Richard Sheldrake, phone (069) 913 317.

The Legislative Council's review of the closure of RVL's Armidale and Wagga Wagga has been extended to 31 July because parliament had been prorogued for 3 months earlier in the year. We understand that the government then has 6 months to respond to the review by which time the closures should have been affected.

Regional Veterinary Laboratory, Wollongbar.

Achondroplasia in Australian Dexter Cattle

We have examined 2 cases of Achondroplasia in neonatal Dexter calves. These Dexter "bulldog" calves had crown/rump lengths of 24 and 25 cms and a range of skeletal defects including short heads, retruded, flattened faces and marked micromelia. The tongues were normal in size and protruded. Large abdominal hernias had occurred through large defects in abdominal skin. The abdominal viscera had herniated between the margins of the skin defect and were covered by an extensive, thin stretched muscle aponeurosis l-3mm thick.

Microscopic examinations found there was an abrupt loss of adnexal structures and rete peg formations in the superficial dermis at the margin of the skin defect. The tissue overlying the defect consisted of thin squamous epithelium overlying a thin dermal aponeurosis. Multiple abnormalities were present in the tiny "long" bones of the limbs including shallow irregular physes and combined zones of hypertrophy and calcification 1-6 cells deep. There was a sharp irregular line of transition to coarse bony trabeculae within the metaphyses.

Further work is needed to detect the molecular and biochemical error which causes the condition and to improve the detection of heterozygous animals. Please report any suspect cases to Melanie Latter or Peter Harper at Grafton Agricultural Research Station, Phone (066) 420 467.

Paspalum distichum - associated pneumonia

Bovine atypical interstitial pneumonia (BAIP) was confirmed in a dyspnoeic cow from a herd pastured on *Paspalum distichum* "water couch". Microscopic examinations showed hyperplastic alveolar epithelium; there were many sloughed alveolar epithelial cells, some multinucleate cells and hyaline membranes and a few alveolar macrophages and granulocytes in the alveoli, alveolar ducts and respiratory bronchioles. This disease has been associated with grazing *Paspalum distichum* "water couch". In a feeding trial at Grafton in 1981, interstitial pneumonia was found at slaughter in clinically normal penned cattle feed *P. distichum.*

"Fog Fever" is a form of BAIP which occurs in cattle overseas and the amino acid tryptophan (TRP) in pasture is supposed to be the toxic principle. A single oral dose of 0.35g TRP/kg body weight causes fatal BAIP. There is little reliable data on TRP in local pastures. European data indicates less than 3g/kg dry matter (DM) is normal.

P. distichum was collected from the property 2 weeks after the cow's death and from 3 other properties with no evidence of BAIP. TRP was quantitated by HPLC.

Property	g TRP/kg dry *P. distichum*
1 recent BAIP	3.6-4.3
2 near 1 (no recent BAIP)	4.4-5.1
3 near 1 (no recent BAIP)	3.1-4.1
4 remote (no recent BAIP)	5.4-6.7

These concentrations of TRP are such that a 450 kg cow consuming 15kg dry matter daily, if it were entirely *P. distichum*, would take in the equivalence of the single toxic dose of TRP within 2-3 days grazing. How the rate of intake affects toxicity is not known. The *P. distichum* contains 3 to 7g TRP/kg DM and that the plant's pneumotoxicity is not associated with variation in that content.

Screw worm fly investigation

We were sent a 3rd instar human botfly larva (*Dermatobia hominis*) which had been removed by a local medical practitioner from a boil-like lesion on the leg of a woman who had returned a week previously from a 6 week research trip in the rainforest of Costa Rica. The boil-like lesion was first noted 2 weeks after arrival in Costa Rica. *Dermatobia hominis* is a serious pest of cattle in South America and also attacks sheep, dogs, cats, rabbits and humans. It has been previously found in small boils in travelers returning to Australia from South America. Establishment of this species in Australia could have severe implications for human and animal health.

The mature larva is about 25mm long and has a few rows of sharp spines on most body segments. Botfly larvae are understandably unidentifiable using Spradbery's "A Manual for the Diagnosis of Screw-Worm Fly", the only text on maggots available in most regional veterinary laboratories. The larva in this case was identified by Dr. Gary Levot, currently at BCRI, Rydalmere.

Spina bifida and Arnold-Chiari malformation

A stillborn Brahman-x calf had spina bifida and Arnold-Chiari malformation. Immunoglobulin was not detected in its pleural fluid (<80 µg IgG/ml fluid) so congenital infection by a teratogenic agent was not implicated.

There was 3 x 6cm area of epitheliogenesis imperfecta over the sacrum, deep to which the cranial sacral vertebrae lacked dorsal arches. Caudal to the sacrum the vertebral column sharply recurved so that the tail emerged caudal to the imperfect skin and projected cranially. The spinal cord terminated in the lumbar area and the leptomeninges continued as a tubular narrow meningocoele through the incomplete sacral vertebrae into the dermis. The hind legs were rigidly straight.

The cranial cavity was somewhat dorso-ventrally flattened. The occipital lobes of the cerebrum had parallel, saggital gyri and were coned into the caudal fossa of the cavity. The cerebellum was elongated and displaced caudally, its vermis extending into the spinal canal at C1. The dorsal cerebellar folia were also sagittal and parallel and slightly swollen. The caudal medulla was not kinked. There were no other schises, hydrocephalus or cardiac defects.

The folia of the cerebellar vermis lacked the atrophy, neuronal degeneration and axonopathy detected microscopically by Madarame and others (Cerebellar hypoplasia associated with Arnold-Chiari malformation in a Japanese Shorthorn calf. *J. comp Path 1991*,**104**: 1-5).

There are several variations of the Arnold-Chiari malformation. They often accompany spina bifida. The aetiology is undetermined.

UNIVERSITY OF SYDNEY

Cervical lymphadenitis in guinea pigs (Zoonosis risk?) - Malcolm France.

Severe suppurative cervical lymphadenitis with occasional sinus tract formation was observed in several guinea pigs in a research colony. Most affected animals were culled although when allowed to survive, the nodes would eventually reduce to nearly their normal size and become firm. Of 3 animals submitted for necropsy one also had numerous pulmonary abscesses in addition to lymphadenitis. Anaerobic cultures of 2 nodes and a lung abscess each yielded small colonies of pleomorphic Gram negative rods of similar appearance to organisms seen in smears of pus and in silver stained tissue sections. Aerobic culture results were equivocal.

Cervical lymphadenitis is said to be a common condition in guinea pigs and *Streptobacillus moniliformis* (a pleomorphic Gram negative rod) is regarded as an important cause. Rats are considered the usual reservoir for this organism and disease can occur in humans through direct contact ('rat bite fever') or through contamination of food or water with rat emanations ('Haverhill fever'). While the organism recovered in the present case resembled *S. moniliformis* morphologically, its strong preference for anaerobic conditions differs from most descriptions which are based on rat isolates and which state that growth occurs readily aerobically. An earlier report of *S. moniliformis* recovered from a guinea pig, however, noted that better growth was obtained anaerobically suggesting strains may differ between hosts. Superficially similar lesions have also been seen by the author in a guinea pig with lymphosarcoma.

ELIZABETH MACARTHUR INSTITUTE

Pilchard Deaths - Richard Whittington

An epizootic in the Australasian pilchard *Sardinops sagax neopilchardus* between March and September 1995 was widely reported in the media. It covered more than 5000 km of the Australian coastline and 500 km of the New Zealand coastline. Affected fish died within a few minutes of clinical signs of respiratory distress; hypoxaemia and hypercapnea were demonstrated by blood gas analysis in NZ. Significant lesions were confined to the gills. In affected/dead fish they comprised subacute inflammation with bizarre epithelial hypertrophy and hyperplasia. Interpretation of these lesions was difficult and only became clear after sequential sampling of fish from Iluka NSW, before, during and after the mortality event. Lesions were initially focal with exudation of fluid, mixed inflammatory cells and chloride cells into the subepithelial space. Inflammation spread to become locally extensive and generalised, then subsided and was replaced by epithelial hypertrophy and hyperplasia over about 4 days. In dead or clinically affected fish locally extensive to generalised inflammation and focal to locally extensive epithelial lesions were always observed; severe epithelial hyperplasia was not an essential precursor to death. This suggests that early lesions compromised respiratory, ion regulation and excretory functions of the gills but that compensation occurred. Underwater observations in NZ confirmed reports from observers in boats in Australia that an external stressor such as chasing the school was associated with decompensation and death. The pathology in affected fish across the distribution of the disease was similar, suggesting a common aetiology. The lesions were unlike those associated with known gill pathogens or toxins in other species of fish. Amoebae were seen in large numbers in the gills of some affected fish and are being studied at Mount Pleasant Labs, TAS. However, a herpesvirus was the only factor consistently associated with the lesions. This was found by examination of ultrathin gill sections and by negative contrast EM in labs in WA, AAHL and NZ. No viruses were isolated in fish cell lines in WA, NSW or NZ but an unexplained CPE was consistently found

at AAHL. Strong circumstantial evidence implicates the herpesvirus but transmission trials have not been possible to date. The origin of the herpesvirus is unknown but the behaviour of the disease was consistent with a new pathogen in a naive population. Some observers believe the herpesvirus was introduced into Australian waters with shipments of pilchards from South America. These are fed to sea ranched tuna in South Australia, near the origin of the outbreak.

REGIONAL VETERINARY LABORATORY, WAGGA WAGGA

Major mortalities in Turkeys due to fowl cholera (*Pasteurella multocida*) infection with exclusion of Avian influenza - Barbara Moloney and Ian Links.

History: A semi-intensive turkey farm with 2500 mixed age birds had a sudden onset of major mortality in 6-12 week old birds. When losses commenced on 31.3.96 in 8-12 week old birds the owner performed post-mortem examinations, diagnosed histomoniasis (Blackhead) on the basis of liver and caecal lesions and treated with Ronidazole (Ronivet-S^{R}). By the 4.4.96 the mortality rate had reached 100 per day. On 4.4.96 two birds were autopsied at RVL Wagga Wagga and by 6.4.96 (Easter Saturday) a definitive diagnosis of *Pasteurella multocida* septicaemia (and not Histomoniasis) had been established as the cause of the deaths. There was difficulty in obtaining antibiotics over Easter. In the meantime the death rate had increased to 400 per day. In-water medication with Lincospectin^{R} commenced on 7.4.96 and continued until 9.4.96 when supplies were exhausted. Antibiotic sensitivity testing results on 9.4.96 indicated sensitivity to lincomycin-spectinomycin, tetracycline, neomyin and ampicillin. Mortality rate decreased dramatically within 36 hours of implementation of treatment but rapidly increased in the remaining 10-12 week old birds within 24-36 hours of cessation of treatment. Treatment with tetracycline via drinking water was instituted on 12.4.96.

By 9.4.96 there had been approximately 1,900 deaths ex 2,500. Age groups involved: 50 breeders - isolated from other birds, no deaths; hatchlings and poults to 3 weeks - isolated, no deaths; poults 4-5 weeks showed negligible deaths despite being in close contact with 6-7 week poults which showed 100% mortality by 9.4.96; 8-12 week birds (ready for market at 10-12 weeks), first birds affected, approximately 600 dead ex 900 by 12.4.96.

Ongoing quarantine restrictions were adequate. No stressors were identified; the organism may have been introduced in young poults obtained from a large turkey producer in February as these birds were amongst the 10-12 week age group where the outbreak started.

Clinical signs: Affected birds showed slight depression and pale combs and wattles for 12-24 hours prior to death. In the terminal stages birds lay down and died shortly afterwards in a short-lived frenzy of flapping wings.

Gross pathology: 2 adult birds necropsied on 4.4.96 showed good body condition with moderate congestion and cyanosis of wattles and combs. Both also had very fluid crop contents, severe congestion and oedema of the lungs, congested myocardium with occasional epicardial ecchymoses, swollen congested spleen and congested liver. In one of the birds there was also a catarrhal enteritis and fibrinous exudation around the right lung, the latter appeared firmer than normal. In the other bird there were petechial haemorrhages in the pancreas and its spleen was severely enlarged.

Bacteriology: Culture of the liver, spleen and lung of both birds yielded a heavy pure growth of *Pasteurella multocida*.

Histopathology: Moderate, acute, periacinar congestion with many sinusoidal thrombi in the livers and numerous fibrin thrombi with isolated septic emboli in the spleens of both birds. The right lung of one bird showed severe congestion, exudation of fibrin and heterophils and massive numbers of bacterial colonies.

Peracute septicaemic pasteurellosis with DIC was diagnosed in both birds in addition a severe acute fibrinous pneumonia was present in one bird.

Avian Influenza (AI Exclusion: In the 1985 Bendigo AI outbreak *Pasteurella* sp and *Haemophilus* sp infections were diagnosed over a 3 week period prior to AI being confirmed. While the gross pathology, bacteriology and histopathology in the present outbreak were not indicative of a viral infection such as Avian Influenza, in view of the extremely high mortality rate, there was need to exclude involvement of AI.

Follow-up investigation: On 12.4.96 two more birds were submitted for autopsy. These were 10 weeks of age and showed evidence of dehydration, slight splenic enlargement and enlarged congested livers. One bird had fibrinous epicarditis and pericarditis and some small amounts of caseous material in the peritoneal air sacs over the kidneys. One of its lungs showed gross consolidation (firm, dry, honeycomb appearance when cut) involving the whole of the lung and the other lung was severely congested and oedematous (possibly haemorrhagic). There was also a mild airsacculitis. The other bird had a more severe airsacculitis, with some loose caseous material in the larynx. Intestines of both birds appeared normal and there was no evidence of subcutaneous oedema or haemorrhage in the wattles or combs.

Histopathological findings in these birds were similar to the original birds examined with the addition of a more severe, subacute fibrinous pneumonia which in one bird showed the exudate in the parabronchi being walled off by palisaded multinucleated giant cells and numerous septic emboli in the other. It was noted that there were no changes to support a diagnosis of Avian Influenza Virus Infection.

Bacteriological culture of lung from both birds and liver from one again yielded a heavy pure growth of *Pasteurella multocida*.

Virology: Also on 12.4.96 cloacal swabs were collected from 10 birds and submitted in PBGS for virus isolation. Primary, secondary and tertiary inoculation of eggs for Avian Influenza Virus and Newcastle Disease Virus were negative. On 24.4.96, 15 blood samples were collected and submitted for Avian Influenza GDPT. All 15 samples were negative.

Comments:

1. Murphy's Law states that disasters will occur on holiday weekends.
2. Why were 4-5 week old poults unaffected despite being in close contact with birds showing 100% mortality?
3. Is there a need for routine vaccination of turkeys against *Pasteurella multocida*? The current isolate has been sent to AUSVAC (Bendigo) for preparation of a vaccine.
4. Disposal of approximately 10 tonnes of dead birds presented major logistical problems. Economic loss represented approximately $80,000 plus costs of treatment and lost production and market access.
5. Any delay in diagnosis and implementation of effective treatment results in chronic lesions in many surviving birds which quickly succumb if treatment ceases.
6. Strict quarantine is required to prevent introduction of infection. Once present it may be impossible to eradicate.

Contributors to this disease investigation were Mark Sayer (private practitioner), Ian Links (SVRO, Wagga Wagga), Rob Walker (SFVO, Wagga Wagga), Tony Morton (DVO, Wagga Wagga), John Glastonbury (Specialist Pathologist, Wagga Wagga), Rod Reece (Poultry Pathologist, EMAI) and George Arzey (SVO Poultry, EMAI).

Western Australia - David Forshaw

Visit by Dean Percy

Dean Percy is the co-author of "Pathology of Laboratory Rodents and Rabbits" and will be here for the ANZSLAS conference 1-3 October and is giving a Lab Animal Pathology workshop on 4th October. Both the ANZSLAS conference and the workshop should be of interest to pathologists. For more information contact David Pass, Animals Resource Centre, Murdoch Drive, Murdoch, WA 6150. Phone (09) 332 5033.

Disease roundup - Barry Richards, Agriculture Western Australia, South Perth

Western Australia's long dry summer produced the usual nutrition-related diseases in **sheep** including nutritional myopathy caused by selenium and/or vitamin E deficiency. There were also many cases of chronic lupinosis and lupin-associated myopathy, salmonellosis, fluoroacetate poisoning and oxalate nephrosis. Cases of oxalate-induced rumenitis were recognised in sheep grazing "iceplant" (*Mesembryanthemum nodiflorum*). A glomerulonephritis of undetermined cause was diagnosed in ill-thrifty full-mouth ewes.

Cattle diseases encountered included *Haemophilus somnus* pneumonia in calves, fatty liver syndrome (ketosis) in calving cows, ostertagiasis in lactating beef cows and neonatal ataxia in cross-bred calves. For the first time in WA, *Neospora caninum* was diagnosed as the cause of bovine abortion. Ionophore toxicity was the cause of sudden death in calves with acute myocardial necrosis. Lower limb swelling in febrile cattle was seen on two farms but the cause was not elucidated. Congenital cardiomyopathy was seen in Angus calves. Visceral pleural cartilagenous plaques were seen in abattoir specimens from a Brahman-cross steer.

An unusual finding in **goats** was multiple 5-10mm diameter pustules in the skin that were packed with Demodex mites. Goats also died of enterotoxaemia. A post-parturition ill-thrift was seen in goats fed lupin grain and canola meal, associated with allergic enteritis.

In domestic **poultry** a variety of diseases were seen including leucosis, Marek's disease and infectious laryngotracheitis. **Ostrich** fading syndrome occurred again but was not as widespread as in the previous year. Increased surveillance of diseases of **rabbits** identified outbreaks of myxomatosis, hepatic coccidiosis and pasteurella pleuritis. In **kangaroos** the newly described orbivirus disease (viral chorio-retinitis) was diagnosed in blind grey kangaroos and black-gloved wallabies.

Other conditions of interest included "milky flesh" syndrome in **Yellowfin tuna** caused by Kudoa cysts (*Kudoa thyrsites*), polioencephalomalacia in **deer**, and urolithiasis in **alpacas**. A **New World Bot Fly** (*Dermatobia hominis*) larva was identified in a poorly healing forearm lesion of a person who had recently toured South America.

Rabbit Calicivirus arrives in WA - Marc Kabay, Agriculture Western Australia, South Perth.

Rabbit calicivirus was diagnosed in a wild coloured rabbit previously noticed near the homestead area of a station at Rawlinna, 300 km west of the South Australian border. The rabbit was found freshly dead one morning. The station owner had seen a higher than normal number of dead rabbits around the station bore and homestead. There were no obvious clinical signs and no rabbits were sighted sick before death.

The rabbit was in good condition at necropsy. The gross findings were unremarkable. There were focal areas of congestion at the tips of the lung lobes, the kidneys were congested but the liver appeared normal. In contrast on microscopic examination the liver had undergone peracute massive necrosis with karyorrhexis and apoptosis of hepatocytes. Many alveolar capillaries in the lungs contained prominent

thrombi. The kidney was congested with thrombosis of glomeruli and congestion of medullary vessels. Focal thrombi were also present in the splenic sinusoids and lymphoid follicles were depleted. Individual lymphocytes had undergone necrosis and karyorrhexis. Some vessels in the brain also contained fibrin thrombi.

The lesions of peracute hepatic necrosis with disseminated intravascular coagulation are consistent with those reported for rabbit calicivirus. The diagnosis is supported by direct electron microscopy and ELISA.

Chlamydiosis in wild Western Rosellas - David Forshaw, Agriculture Western Australia, Albany

Western Rosellas (*Platycercus icterotis*) were seen sick and dead at a back garden feeder in downtown Denmark, 50km west of Albany. The local wildlife officer suspected some form of poisoning but necropsy examination revealed swollen pale spleens and fibrinous pericarditis in two birds. Histopathology revealed a non-suppurative hepatitis, splenitis and pericarditis. In all lesions cells containing very obvious blue stippled organisms were present with H&E staining. Liver smears were strongly positive in the Imagen immunofluoresence test for Chlamydia.

The gross appearance is unusual in my experience; parrots usually have swollen, congested, mottled livers and spleens. Histologically organisms can be difficult to detect with H&E.

Chlamydiosis has been seen in wild Twenty-Eight parrots (*Barnardius zonarius*) in WA. An Agriculture Protection Board survey showed that 11% of Red-Capped parrots (*Purpureicephalus spurius)* excreted the organism in faeces collected from May to December of 1992 but no birds were detected shedding the organism in the following January - April.

Conserved fodder diseases - Listeriosis and ARGT - David Forshaw, Agriculture Western Australia, Albany

In south-west WA there appears to be a rise in the number of farmers making silage with a resultant increase in cases of listeriosis. Annual Ryegrass Toxicity appeared for the first time in the south coastal region in a herd of cattle fed hay from a ryegrass contaminated oat crop. The cattle first appeared depressed and anorexic, then collapsed and showed intermittent convulsions. Fifteen cattle of various ages died including mature cows and a bull. Histopathology revealed marked vacuolar change in the cytoplasm of hepatocytes, most severe mid zonal. No brain changes were seen. A newly developed ELISA test enabled detection of *Clavibacter toxicus* organisms in samples of the hay.

ARGT due to feeding contaminated hay has been recorded a further seven times during this "season" in WA. Most of these cases have involved only small numbers of animals with horses most commonly affected. With the large amounts of hay being shipped to various drought affected areas across the country this is a disease to look out for.

Colibacillosis in a Major Mitchell Cockatoo - Shane Raidal, Murdoch University

A mature male Major Mitchell cockatoo in poor to moderate body condition was presented for necropsy examination. The body was dehydrated and the tail feathers were soiled with milky-green urates. The liver was enlarged and molded over the surface and throughout the parenchyma with white nodules. Several *Ascaridia* spp. were present in the small intestine. The kidneys were markedly enlarged by pale firm abcesses of varying size. Histologically there was a severe multifocal, necrotising, heterophilic nephritis with abundant intralesional gram negative bacteria. A severe multifocal heterophilic hepatitis was also present. Culture of the kidney yielded a heavy growth of *E. coli* which was sensitive to neomycin, gentamycin, tetracycline, sulphonamide and nitrofuran but resistant to ampicillin.

Heartworm in a ferret - Shane Raidal, Murdoch University

A mature male ferret with a history of chronic respiratory disease non-responsive to antibiotic therapy was presented for necropsy examination. The ferret was in moderate body condition and dehydrated. The intestinal tract and liver appeared moderately congested both grossly and histologically. However, the most significant abnormalities were in the lungs, particularly the caudo-dorsal lobes. There was marked fibrous thickening of the pleura and lung lobes appeared pale and resilient. Other areas were collapsed and congested. Histologically there were marked areas of pulmonary fibrosis and a mixed inflammatory cell infiltration. There was early to mature thrombosis of major pulmonary arteries associated with numerous mature filaroid nematodes which were probably *Dirofilaria* spp.

Diagnosis: Interstitial pneumonia, chronic with thrombosis and infarction associated with intralesional nematode parasites.

Recent reports from Communicable Diseases Intelligence

The Editor gratefully acknowledges permission to reproduce the following two articles from recent issues of Communicable Diseases Intelligence published by AIDS/Communicable Diseases Branch, Department of Health and Family Services, G.P.O. Box 9848, Canberra, A.C.T. 2601.

1. "Possible reservoir host of Equine Morbillivirus identified", CDI **20**(11) p.262 27th May 1996

2. "Human health aspects of a possible Lyssavirus in a black flying fox". CDI **20**(14) p.325 8th July 1996

POSSIBLE RESERVOIR HOST OF EQUINE MORBILLIVIRUS IDENTIFIED

Peter Young, Animal Research Institute, Department of Primary Industries Locked Bag 4, Moorooka, Queensland, 4105.

Investigations by a team from the Queensland Department of Primary Industries (DPI) into the reservoir of equine morbillivirus has produced evidence that a related virus (bat paramyxovirus) is present in two of four *Pteropus* species of fructivorous bats with an antibody prevalence of about 20%. Insufficient samples have been examined to date from the other two species to determine if they also have antibody.

Equine morbillivirus has been associated with two separate incidents involving fatal disease in humans and horses[1,2]. The first incident occurred in August 1994 in Mackay, Queensland. Two horses were infected and died after a severe, acute illness. Transmission apparently occurred to one human who developed recurring encephalitis resulting in death about 12 months later.

The second incident occurred in September 1994 in Brisbane, about 1,000 kilometres south of Mackay. In this incident, 21 horses were infected of which 14 died or were euthanased. Transmission occurred to two humans, one of whom died after a short illness. A paramyxovirus was isolated from lungs of two Brisbane horses. An identical virus was also isolated by the Australian Animal Health Laboratory (AAHL) which was subsequently described as equine morbillivirus[3]. In spite of intensive investigations, no connection has been established between the two incidents.

Work at AAHL has shown that the virus obtained from horses in the Brisbane and Mackay incidents are identical, indicating a common source[3,4].

In the DPI's considerations of possible reservoir hosts, the following criteria were applied to prioritise species for investigation:

- the species should be present in both the Brisbane and Mackay areas;
- the species should be capable of migrating between these areas, and
- contact with horses should be possible.

The two groups of animals which readily fitted this description were birds and bats. Because EMV is a mammalian virus and because transmission of paramyxoviruses from birds to mammals is uncommon, bats were given a higher priority than birds.

In addition to focusing on bats, considerable time and effort has been devoted to serological surveys of domestic animals and wildlife. To date, 5,264 sera from 46 species have been tested, including 263 samples from 34 species of wildlife. None of these animals has shown any indication of antibody to the test antigen, indicating that infection is uncommon.

Examination of a relatively small sample of fruit bats has shown a seroprevalence of the bat virus of about 20% (11 positive of 55 tested). Serology has been carried out using an ELISA (enzyme-linked immunosorbent assay) and confirmed by neutralisation tests at the AAHL.

Speculation about how the bat paramyxovirus might be introduced to other species, including horses and humans, assumes that there is a connection between the two viruses. One possibility is that infection of horses in Brisbane and Mackay may have only occurred after a very unusual event, or that a change in the bat virus resulted in a virus which was more virulent for horses, or perhaps both conditions were necessary.

Our next tasks are to isolate virus from as many species and locations as possible and to describe the natural history of infection in bats. When more is known about how the virus behaves in its natural host, it may be possible to devise testable hypotheses about how infection of other species may occur.

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3. Murray K, Selleck P, Hooper P *et al*. A morbillivirus that caused fatal disease in horses and humans. *Science* 1995; 268:94-97.

4. Hooper PT, Gould A, Mitchell G *et al*. The retrospective diagnosis of a second outbreak of equine morbillivirus infection. Aust. Vet J. 1996 (in press).

HUMAN HEALTH ASPECTS OF A POSSIBLE LYSSAVIRUS A BLACK FLYING FOX

Scott Crerar[1,2], Helen Longbottom[1], John Rooney[3] and Peter Thornber[4]

Introduction

On 24 May 1996 a black flying fox displaying neurological signs was found in Ballina, New South Wales and submitted to the New South Wales Agriculture Wollongbar regional veterinary laboratory for autopsy examination. Histopathologic examination of the brain revealed severe non-suppurative encephalitis. Tissues were examined for evidence of equine morbillivirus (EMV) infection at the Animal Research Institute, Brisbane. Additional fixed tissues were sent to the Australian Animal Health Laboratory (AAHL) at Geelong for EMV and rabies testing. Results were negative for EMV. However, immunoperoxidase

testing on fixed brain tissue was positive for lyssavirus antigen and was subsequently confirmed by immunofluorescence testing. Viral particles consistent with Rhabdovirus morphology were seen on electron microscopic examination of brain tissue. Cytoplasmic inclusions and tubular membranous structures suggestive of Rhabdovirus replication were also visible under electron microscopy in formalin-fixed brain samples. AAHL is currently attempting to isolate the virus using a range of cell cultures and mice inoculations.

The genus *lyssavirus*, family Rhabdovirus, includes classic rabies virus and five other rabies-like serotypes. The latter are Lagos bat virus, Mokola virus, Duvenhage virus and the European bat *lyssaviruses* (EBLV) 1 and 2. These viruses are all antigenically related but distinct. Rabies occurs in numerous countries in Europe, Africa, Asia, North America, Central America and South America, but the other rabies-like *lyssavirus* have been recorded only in Africa and Europe. Australia is currently considered rabies free and no other rabies-like *lyssavirus* infections have been documented in animals or humans.

With the exception of rabies virus, human infections with members of the *lyssavirus* genus are rare. All members of the group, however, except Lagos bat virus, have been shown to infect humans. Duvenhage and EBLV1 and EBLV2 have not been shown to occur in mammals other than bats and humans and are not thought to have a significant role in the spread of rabies-like disease to terrestrial mammals, including wildlife. This is in contrast to the situation in North America, Central America and South America, where the type of rabies found both in bats and in the main terrestrial carriers, raccoons and foxes, is sylvatic classic rabies.

Public health implications of *lyssaviruses* in Australia

In the absence of an isolated virus, investigations to date have indicated that the present *lyssavirus* is not classic rabies, serotype 1, and Australia's rabies-free status is not compromised by this finding. The extremely low health risk posed by rabies-like lyssaviruses combined with the probable isolated nature of this incident in Australia indicates there is not a need to change current public health advice. It is recommended that if people are bitten or scratched by flying foxes or bats, they should immediately clean the wound thoroughly with soap and water. People who are concerned about the wound should seek medical advice.

In 1986 the World Health Organization (WHO) issued general guidelines on bats and rabies which are documented in the eighth report of the WHO Expert Committee on rabies, TRS 824, WHO Geneva, 1992. Recommendations were that persons exposed to a non-rabies *lyssaviruses* infected bat should receive the standard post-exposure rabies treatment recommended by WHO. The same applies to pre-exposure treatment of groups of people at risk of exposure to bats in countries where rabies or *lyssaviruses* are endemic in bat populations. In these countries, no differences are made in post-exposure treatment according to the type of *lyssavirus* involved. At present, there is no indication for specific *lyssavirus* treatment in people bitten or scratched by flying foxes or bats in Australia. The exception would be if the flying fox or bat was known to be infected with a *lyssavirus*.

Additional work is underway at AAHL in an attempt to characterise the specific *lyssavirus* involved. Isolation studies, if successful, will take up to one month to complete. It is unclear at the moment whether *lyssaviruses* are endemic in Australian flying fox colonies, although it is considered unlikely. Nevertheless, a surveillance system to investigate the presence of both EMV and *lyssaviruses* in sick and dead flying foxes is to be established by animal health authorities.

1. Department of Health and Family Services, GPO Box 9848 Canberra, ACT 2601
2. Master of Applied Epidemiology Program, National Centre for Epidemiology and Population Health, Canberra, ACT
3. New South Wales Health Department Sydney, NSW
4. Department of Primary Industries and Energy, Canberra, ACT

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Victorian Veterinary Pathology Services

Veterinary Pathologist

Victorian Veterinary Pathology Services invites applications for a full time staff position in diagnostic pathology at their Veterinary Laboratory at South Yarra, Melbourne. Candidates should have a Veterinary degree registerable in Victoria, at a minimum be a registered veterinary specialist, but preferably be a Diplomate of the ACVP or hold an Australian Fellowship in anatomic pathology. The laboratory has an excellent case mix of laboratory, equine, companion and production animal cases as well as being the exclusive provider of diagnostic services to the Royal Melbourne Zoo and to Healesville Sanctuary. The laboratory is situated conveniently in central Melbourne, close to all major attractions offered by a city of this size. An attractive salary and benefits package is offered and is negotiable, depending on qualifications and experience. Review of applicants will begin immediately and continue until the position is filled.

Position Description. Demonstrated experience in diagnostic pathology with specialist skills in anatomic pathology is a prerequisite. Primary responsibilities are in the service areas of surgical biopsy, cytology and necropsy specimens. A basic understanding of clinical biochemistry and haematology is desirable. The incumbent is expected to have excellent interpersonal skills in order to liaise with veterinary clinicians effectively. The successful candidate is also expected to possess well developed managerial skills and preferably be computer literate in order to assist the Director with overall supervision of the laboratory.

Applications. Applicants should submit a letter of intent, detailed curriculum vitae, and the names of 3 or more references to Dr. Judith Slocombe, Victorian Veterinary Pathology Services, 14 Yarra Street, SouthYarra, Victoria, Australia. 3141. Facsimile: (61) 3-9824-1174.

N A T A

Media Release - October 1996

NATA Launches Veterinary Testing Accreditation Program

In response to demand, Australia's laboratory accreditation authority, NATA, in association with key veterinary professionals, has developed a program for the accreditation of veterinary testing. The program is applicable to private, government and university testing laboratories, and to veterinary practitioners who themselves undertake a range of laboratory tests. The program is designed to add confidence, credibility and value to these testing services.

What is NATA?

NATA, the National Association of Testing Authorities, Australia, began in 1947 as the world's first national laboratory accreditation organisation. It remains the worlds largest and most diversified national laboratory accreditation body and has served as the model for similar bodies in many other countries.

Importantly for international recognition of testing results, NATA has established mutual recognition agreements with many overseas laboratory accreditation and calibration authorities.

In 1996, the Australian Government, with which NATA has a Memorandum of Understanding, again endorsed NATA as the national authority for laboratory accreditation, and has undertaken to inform other governments, relevant international organisations and the laboratory community that NATA is the only nationally recognised laboratory **accreditation body**.

NATA's Veterinary Testing Accreditation Program

The NATA Veterinary Testing Accreditation Program was developed with the assistance of veterinarians representing the Australian Society for Veterinary Pathology, the Australian Veterinary Association, the Pathobiology Chapter of the Australian College of Veterinary Science, the Australian Quarantine and Inspection Service, the Sub-committee on Animal Health and Laboratory Standards, the CSIRO Australian Animal Health Laboratory, the universities' veterinary schools, and small and large private veterinary laboratories.

NATA accreditation is more than just accreditation to ISO Guide 25. Based on its experience, NATA has developed its General Requirements for Registration which fully meets the requirements of ISO Guide 25 but are written in a language meaningful to testing laboratories, small and large.

In addition, NATA has published the Supplementary Requirements for Accreditation: Veterinary Testing, and the Classes of Test Veterinary Testing. These documents, developed in association with the abovementioned veterinarians, tailor the General Requirements to veterinary testing in particular, and provide a list of the types of tests for which veterinary testing laboratories, and veterinary practice laboratories, may seek accreditation.

Finally, NATA has a pool of veterinary specialists to assist NATA in undertaking assessments of specialist veterinary testing laboratories and veterinary practice laboratories.

How Veterinary Laboratories and their Customers Benefit

In summarising the benefits of the Veterinary Testing Accreditation Program, NATA's Project Development Manager, Chris Winston, said "All types of veterinary laboratories will gain added credibility through third-party accreditation by an internationally-respected organisation."

"For the specialist veterinary testing laboratory, accreditation provides confidence that the laboratory's testing has been assessed by an expert team and found to meet, and to continue to meet, internationally recognised criteria for laboratory systems and technical competence".

"For the veterinary practitioner undertaking his or her own testing, accreditation provides confidence that those tests are undertaken competently and that the laboratory, while perhaps smaller than a specialist veterinary laboratory, nevertheless has a laboratory quality system sufficient to meet its needs."

"For the user of accredited veterinary testing services, confidence follows from knowing that tests have been undertaken by a laboratory assessed as capable of providing the needed testing competence and quality".

"Value is added because management, staff and customers have confidence in the accredited laboratory's competence, and the laboratory can advertise its accreditation to the world".

Isn't ISO 9000 Certification Appropriate?

Even though it is also a certifier of Quality Management Systems to the ISO 9000 series, NATA believes that for veterinary testing laboratories, accreditation to NATA's General Requirements for Registration and the Supplementary Requirements for Accreditation: Veterinary Testing (which more than covers all requirements of ISO Guide 25) is indisputably the most appropriate form of independent assessment and recognition.

"Of course," Mr. Winston added, "if laboratories want ISO 9000 certification for their operations, perhaps in addition to laboratory accreditation, NATA also offers that service."

A detailed Information Package on the NATA Veterinary Testing Accreditation Program is available by contacting Agnes Koltai, Veterinary Testing Co-ordinator, or Chris Winston, Project Development Manager, NATA, by one of the following means:

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