AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

Print Post Approved

VETERINARY PATHOLOGY REPORT
Publication No. PP5444059/0003
EDITOR: Cleve Main

Number 55 **June 2000**

PAGE **CONTENTS**

3 Baron-Hay Court, South Perth, Western Australia 6151
Postal Address: Locked Bag 4, Bentley Delivery Service WA 6983

SECRETARIAT

PO Box 114 Walkerville SA 5081
Phone: 088 344 6337 (Amanda Whyte) Fax: 088 3449227

ASVP EXECUTIVE 1997-98

President	Clive Huxtable	School of Veterinary Studies Murdoch University MURDOCH WA 6130	08 9360 2297
Secretary	Cleve Main	Animal Health Laboratories 3 Baron Hay Court SOUTH PERTH WA 6151	08 9368 3351
Treasurer	Jeremy Allen	Animal Health Laboratories 3 Baron Hay Court SOUTH PERTH WA 6151	08 9368 3466
Committee Members			
	Barry Richards	Animal Health Laboratories Albany Highway ALBANY 6330	08 9892 8444
	David Forshaw	Animal Health Laboratories Albany Highway ALBANY 6330	08 9892 8444
	John Jardine	Vetpath Laboratory Services 9 Lyall Street REDCLIFFE WA 6104	08 9277 9847

APPOINTMENTS

Chairman - Registry of Domestic Animal Pathology	Tony Ross
Newsletter Editor	Cleve Main
Continuing Education - NRDAP	Roger Kelly

CONVENOR - SLIDE OF MONTH

Rod Reece	EMAI Private Mail Bag 8 CAMDEN NSW 2570	02 4640 6333

Editorial

Well this is my last issue of the bulletin. The next one will be produced by someone else in another state. Thank you all those who contributed and made my life as editor a little easier. To those of you who read but didn't contribute, I hope you found each issue interesting.

The most important thing on your Executive's agenda in the AGM and conference which is scheduled for June 24/25th at the Esplanade Hotel Fremantle. The registration fee has been set at $80 (same as last year) and will cover day to day expenses. As the attached conference program indicates, we have a full scientific program for you. If you haven't already made a commitment to come to this fair State, bite the bullet and do so.

With this issue of the Vet Path Report, you will find a conference registration form. It's not very complicated, so fill it in and send it with the registration fee to the ASVP Treasurer Dr Jeremy Allen. Not only will it save time on the morning of the conference, but it will give us an idea of how many are coming for catering purposes. The registration form also asks if you intend to go to the conference dinner. Once again this will give me some indication of numbers. Note that the cost of the dinner is not included in the registration fee. We don't know what the dinner will cost, but it will not be expensive. Not unless you buy the most expensive wine and choose the most expensive dish on the menu.

PS Jeremy Allen has asked that I include in this issue a list of seafood toxin analytical facilities and related expertise in Australia and New Zealand. I do so with pleasure - it is included as a separate document.

Cleve Main
Hon Editor

I

PROGRAMME - ASVP Conference 24th & 25th June 2000
Esplanade Hotel - Fremantle WA

Theme: Dermatopathology

Saturday 24th June 2000

Plenary Sessions

Adhesion molecules as Targets of Disease: Drs Mandy Burrows and Mandy O'Hara

9.00-10.30 The Dermoepidermal Function & Disease
This session will discuss the structure and function of the dermoepidermal junction and how that relates to the clinical and histopathological presentation of diseases such as: Bullous pemphigoid, Herpes gestationis, Epidermolysis bullosa, Cicatrial pemphigoid & Linear IgA bullous dermatoses in small and large animals.

10.30-11.00 Morning Tea

11.00-12.30 The Keratinocyte & Disease
This session will discuss the structure and function of the keratinocyte and desmosome and how acquired and inherited disorders of these components of the epidermis lead to the clinical and histopathological presentation of diseases in the Pemphigus group.

12.30-1.30 Lunch

Member Presentations

1.30-1.50 Sharon Bryden - A review of the pathogenesis, clinical and histopathological features of sebaceous adenitis in dogs.

1:50 - 2:05 Judith Nimmo-Wilkie - Mycosis fungoides and paraneoplastic pemphigus in a dog.

2:05 -2:20 Roger Kelly - Porcine dermatitis/nephropathy syndrome: just what IS going on?

2:20-2:35 Jenny Charles - Cutaneous and renal vasculitis in Victorian pigs (dermatitis/nephropathy syndrome)

2:35-2:50 Ron Slocombe - Photoaggravated dermatosis in horses

2:50-3:20 Afternoon Tea

3:20-3:40 David Pass - Avian skin: interpretation of the feather follicle in heath and disease. This session will discuss the functional anatomy of avian skin with particular emphasis on interpretation of the feather follicle in health and disease.

3:40-4:00 Brian Jones - Fish dermatology

4.00 AGM

7.00pm Dinner

Sunday 25th June 2000

Members Presentations

9.00-9:15	Julia Lucas - Eosinophilic Pustular Dermatitis in a Juvenile Pharaoh Hound.
9.15-9:30	Malcolm Lancaster - Familial follicular dysplasia and storage disease in Southdown lambs.
9.30-9:45	Judith Nimmo-Wilkie - Cutaneous nodules associated with acid-fast organisms in long-footed potoroos.
9.45-10:00	John Jardine - Cutaneous neosporosis in the dog.
10.00-10:15	Christina McCowan - Pox viral dermatitis in salt water crocodiles.
10.15 - 10:45	**Morning tea**
10:45-11:00	Julia Lucas - Streptococcus iniae: a cause of death in farmed .Barramundi.
11:00-11:15	David Taylor - Lesions in the mesenteric lymph nodes of sheep.
11:15-11:30	Terry Nichols - Borna Disease virus infection - implications for veterinary pathologists in Australia.
11:30-11:45	Alan Kessel - An outbreak of *Lactococcus garviae* infection in farmed Rainbow trout.
11:45-12:00	Malcolm France - Complex pulmonary disease in gene knockout mice with deficient phagocyte function.
12:00-12:15	John Finnie - Multifocal cerebellar granular layer necrosis: a distinct pathological entity.
12.15-1.30	**Lunch**
1.30-2:00	Tony Ross - - A Wander Through Birds' Brains
2:00-2:15	Fran Stephens - Exophiate sp in the Western Australian dhufish.
2:15-2:30	Brad Chadwick - Pilchard herpesvirus infection
2.30-2:45	Sue Jaensch - Avian pulmonary oxygen toxicity
2:45-3:00	Shane Raidal - Leucocytozoon infection in Nankeen kestrels
3:00	**Afternoon tea**
3:30	Jeremy Allen - McArdles Disease in sheep
3:45	David Taylor - Pathology in Salmonids related to the presence of jellyfish.
4:00	David Forshaw - Glycolic aciduria associated with renal oxalosis in Gilbert's Potoroos
4:15	Phil Nichols - The life history of canine oral papilloma virus infection.
4:30	**Close**

Letter from Clive Huxtable (remember me?)

A message from afar. First of all best wishes to all members from this aging expat. Next - the major issue here at the moment is an expectation of another outbreak of West Nile Virus in the coming summer. Plenty of prominence in the press and naturally lots of political activity. Here at Cornell our biggest problem is dealing with equine necropsies where WNV is a major differential. Other likely differentials are rabies, protozoal myelitis, and EHV1 encephalmyelopathy. There is some risk to the operators, but probably less than with Eastern Encephalitis and only a little more than with Rabies. Our protocol is to remove the head and work with the brain and the first segment of the spinal cord. The rest of the carcass is then incinerated. Tissue from the cord and caudal brainstem go for viral culture and PCR, and half the brain goes into formalin, while the other half goes fresh for rabies immunodiagnosis.

Interesting cases here recently include visceral leishmaniasis in a dog, Pemphigus vulgaris in a dog, Halicephalobus deletrix encephalitis in a foal, Saprolegnia infection in frogs, "spider lamb" disease, acute glomerulonephritis in Finn lambs, and cutaneous B-cell lymphoma with infiltrating eosinophils in two dogs.

Any members planning to wander through upstate NY would be welcome to drop in - but don't come between Dec and March!

Cheers

Clive H

New South Wales - Paul Gill

Acknowledgment of Pathologists in Publications - Malcolm France, University of Sydney

Users of Pathmail will already be familiar with this correspondence but I decided to reproduce the main elements in the Vet Path Report for those who missed out.

From time to time, case reports containing histopathological findings have been published in the Australian Veterinary Journal without any acknowledgement given to a pathologist in most of these instances, co-authorship would not have been warranted. However, a brief acknowledgement would have reassured readers that the pathologist had presumably been consulted about the manuscript and been given the chance to ensure that the pathological findings were accurately reported. Such acknowledgement would have also demonstrated a desirable level of professional courtesy.

When I raised this matter with the AVJ editor, Colin Wilks, his response was very helpful and included the results of a discussion with the Editorial Committee. Among other things, he pointed out that the problem is not confined to pathology and that, despite the Journal's efforts, workers across a broad range of areas sometimes do not receive the acknowledgement they deserve. He has suggested that modifications to the Instructions for Authors and the letter to reviewers would go some way to addressing the problem. This seems a practical approach and I see no need for further action. Readers might be interested in some comments from Pathmail subscribers which I have copied below with their permission.

From Ross McKenzie:

"This is one of those issues that will always be with us and doesn't appear to me to be susceptible to easy solutions. Each of us is responsible to ensure as far as we can that the outcome of the publication process is fair to all parties. The journals can't be expected to work miracles.

Acknowledgements are always a touchy subject, as is deciding who has done enough to deserve authorship. I always try to err on the side of generosity (says he, polishing his halo!). It is not unknown, of course, for pathologists to write papers and completely forget (?) to acknowledge the clinician whose case it started out as, instead of including the clinician in the authorship list, so we pathologists as a group are not totally innocent.

I have a theory that says that, if your parents taught you at a young age to say thank-you and to consider the needs and feelings of others, this problem doesn't arise. By the time you become a veterinarian, it's often too late to break bad habits such as being a tad selfish in your interactions with colleagues. Some people are so self-focused that they don't think of the consequences of ignoring the contributions of others to their work. It was ever thus, and I don't see it changing.

On the other hand, problems of this nature can arise quite innocently from poor communication between the parties early in the publication process - "The first problem of communication is the illusion that it has been achieved." Consider this hypothetical situation: You might have believed that you were to be acknowledged, but you didn't get a clear commitment from the senior author, so it's a bit late to be complaining when your name is in fact missing from the printed paper.

Also, private practitioners, with some exceptions, are not frequent authors and should not be expected to know all the "rules" right down to the last detail, and particularly not the "small" matter of acknowledgements. This is going to be even more of a problem if they decide to publish without talking to the lab about it before or during the writing process - you don't get the chance to stake your claim for recognition at all. They may consider the results of laboratory work as belonging to them and so requiring no acknowledgement at all, particularly if the lab work was paid for. They may think that payment cancels all debts, financial and professional.

The price of recognition in print is eternal vigilance, folks!"

From Jenny Mills:

"I agree that this is an important issue.

It must be extended to cytopathology. For this very reason, and following an unacknowledged publication containing misleading information many years ago, I have added onto our cytopath result forms the words … “Privileged information. Not for unauthorised publication".

Unfortunately it has not stopped the problem completely, but I think it helps. A guideline from the editors is essential. It needs, at least, to allow the pathologist to review the final report."

From John Mackie:

"I agree wholeheartedly with Malcolm's concerns regarding acknowledgements and co-authorships in journal articles.

For me the most important consideration is scientific credibility. It is difficult for the wider scientific community to place much confidence in a paper which has a component of pathology but which does not include a pathologist as an author or in the acknowledgements.

Furthermore, if the pathological findings are central to the paper, I believe a pathologist should be included as an author (just as e.g. a virologist or radiologist should be a co-author if virology or radiology are central to a paper). Payment for the lab work may cancel debts but that isn't sufficient to achieve acceptance of the work by the scientific community.

Professional courtesy is also important, and, as Ross McKenzie rightly points out, we need to ensure we reciprocate that courtesy with our other veterinary and scientific colleagues.

Thanks to Malcolm for pursuing the issue with Colin Wilks. I think it would be appropriate for the AVJ to include a comment in their Guide to Authors and standard letter to referees."

Northern Territory - Anton Janmaat

Leptospirosis in NT dogs - Helen Parkes, Berrimah Veterinary Laboratories (BVL), NT Department of Primary Industry and Fisheries

We have had five cases recently at Berrimah Veterinary Laboratories (BVL) with clinical histories and a range of laboratory test results consistent with acute canine leptospirosis. This is the first time canine leptospirosis has been diagnosed at BVL.

History and clinical signs

Cases included three mature female dogs, one six months old male pup and an 11 weeks old pup. One of the females was the mother of the young pup. Clinical course of disease was about seven days or less. Two of the dogs had initial lameness or limb pain. Most had some or all of the following (based on the histories on submission forms, which are often scanty!): lethargy, anorexia, vomiting and jaundice. Two were noted to have normal temperatures (the others didn't say).

Clinical pathology

Haematology and clinical chemistry were done on four of the dogs at BVL. Three showed mild to moderate anaemia, with little evidence of regeneration, and three had moderate or marked neutrophilia, two with a left shift. All four showed markedly elevated urea and creatinine, with raised phosphorus, amylase and lipase attributable to renal failure. Three of the four also had moderately or markedly raised bilirubin, ALT, AST and ALP. Three had moderately raised CK.

Post-mortem findings

Two of the dogs were examined post-mortem at BVL. Both showed marked jaundice. Both had extensive haemorrhages, involving the subcutis, lungs, thymus (in the young pup) and kidney (subcapsular). Lymph nodes were dark red. One also had petechial and ecchymotic haemorrhages of the gastric and intestinal mucosa, with bloody intestinal contents.

Histopathological changes

Histological examination was done on four of the five dogs. All showed very similar changes. In the liver, there was dissociation and necrosis of hepatocytes, binucleate hepatocytes and biliary stasis. The kidneys showed acute tubular degeneration and mild, interstitial, lymphocytic infiltration in the cortex. Generalised haemorrhages, lymphoid depletion, and mild myocarditis and skeletal muscle degeneration were other changes seen. The brains of two of the dogs were examined and both had mild, diffuse, non-suppurative meningitis.

In three out of the four dogs examined, occasional organisms consistent with leptospires were found in the renal tubules, using a silver stain (Warthin-Starry). Note that all the dogs had been treated with antibiotics prior to death.

Serological testing

Sera from four of the five dogs were sent to the WHO/FAO Collaborating Centre for Reference and Research on Leptospirosis, in Queensland. All four had antibody titres to *Leptospira interrogans* serovar *australis*. Two of these had titres of 1:200, one had a litre of 1:800 and the fourth had a litre of 1:1600. Two of these dogs also had lower titres to various other serovars, including *copenhageni*, and *bulgarica*.

In conclusion

Canine leptospirosis in the NT has not been diagnosed at BVL before this outbreak. Since it appears that transmission probably requires a moist environment, the long wet season this year may have been a contributing factor. It remains to be seen whether the disease disappears with the onset of the dry season (which has finally arrived), and if so, whether it recurs next year.

An unidentified orbivirus associated with encephalitis in horses - Lorna Melville, Berrimah Veterinary Laboratories (BVL), NT Department of Primary Industry and Fisheries

During the past year a number of horse submissions to BVL have remained undiagnosed as to an aetiological agent. Two distinct syndromes have been involved, musculo-skeletal/neurological and acute respiratory.

Two of the horse submissions were from the Katherine area and involved animals euthanased with severe neurological symptoms. The second case was submitted to AAHL for Japanese Encephalitis exclusion and sera from both animals were negative for JE. Flavivirus. Serology performed at BVL showed both horses had titres to Murray Valley Encephalitis (MVE) which did not change with repeat bleeds taken about three days apart.

Histopathology on both horses showed severe diffuse encephalitis. One horse had a non-suppurative encephalitis, with perivascular cuffing, affecting all areas of the brain and cranial cervical cord. Lesions were most severe in the mid brain and cerebrum. The other horse also had marked perivascular cuffing but was more suppurative with multi focal areas of necrosis. Bacteriology on both brains resulted in no significant isolations.

Virus isolation was carried out on brain, spleen and EDTA bloods from both horses using Aedes albopictus (AA) cells followed by passage into mammalian cells. A virus was isolated from the spleen and EDTA blood of one horse and from the spleen of the other horse. All three isolates produced similar CPE. Electron micrographs of the isolates from both spleens showed morphology consistent with an orbivirus.

These viruses cannot be identified despite using a wide panel of antisera to both Australian and Australian and overseas viruses, including African Horse Sickness, at both BVL and AAHL.

Wallal serogroup viruses in black walleroos - Lorna Melville, Berrimah Veterinary Laboratories (BVL), NT Department of Primary Industry and Fisheries

The Territory Wildlife Park near Darwin had four black wallaroos in their macropod collection. Three adults were housed in an outdoor enclosure and the juvenile was housed separately in an indoor area.

In November 1999, the three adults became acutely ill. Symptoms included depression, lethargy, facial oedema and conjunctivitis. Two died within two days and the third recovered after a long illness that required surgical ablation of the scrotum and testicles which had become necrotic. The juvenile remained unaffected.

Blood and fixed and fresh tissues were submitted from one of the dead adults and blood and surgical tissues from the surviving male.

Histopathology on the dead animal showed a severe systemic disease with inflammation in a large range of tissues including the heart, skin, liver, brain, lung and kidney. Intranuclear inclusion bodies were present in the renal tubular epithelial cells and focal lesions of the epidermis. No significant bacteria were isolated from the lung, liver, spleen and kidney.

Virus isolation was carried out on blood and kidneys. A virus was isolated from blood through both Aedes albopictus (AA) cells and mammalian cells. A virus was also isolated from the blood of the surviving black wallaroo.

Another submission from the Territory Wildlife Park a short time later included blood from a black wallaroo/northern wallaroo hybrid. This animal had a chronic skin lesion and a history of a possible viral illness. A virus was also isolated from the blood of this animal through AA cells.

The three viruses were tested with a range of antisera and reacted to the Wallal serogroup. The viruses were sent to AAHL where cross neutralisation testing to the prototype Wallal virus and the Wallal virus isolated from cases of Kangaroo blindness indicated these viruses were different. Sequencing of the three new Wallal viruses indicated 99% and 98% homology with each other, but only 87% homology with prototype Wallal and 83% homology with Kangaroo blindness Wallal.

Electron microscopy at AAHL confirmed the viruses were Wallal. The intranuclear inclusions seen in histological sections were identified under EM but did not contain viruses.

South Australia - Ruth Reuter

Johne's Disease-like lesions in an alpaca due to *Mycobacterium avium subspp avium* - Juliet Lucas, Veterinary Pathology Services, Adelaide

M. avium subsp. *paratuberculosis* (Johne's disease) infection has been described in sheep, cattle and occasionally other ungulates. In Australia in 1995, 10 cases of *M. avium* subsp. *paratuberculosis* infection were reported in alpacas, causing similar lesions to Johne's disease in cattle and sheep (Ridge *et al*). In contrast, *M. avium* subspp. *avium* has not been reported in alpacas, but causes sporadic disease in other species .

M.avium. paratuberculosis infection occurs in young alpacas, often less than 2 years old (Ridge *et al*) The histological lesions are characterised by granulomatous enteritis and granulomatous lymphadenitis particularly involving the mesenteric lymph nodes (Ridge *et al*). There may also be widespread visceral lesions in some cases (Ridge *et al*)

In the case reported here, a 9 month old alpaca presented with profuse watery diarrhoea and rapid weight loss over 10 days. Clinical biochemistry revealed hypoalbuminaemia and a marked elevation in LDH.

At necropsy the carcass was severely emaciated. The mesenteric lymphatics were thickened and the mesenteric, mediastinal and other internal lymph nodes were enlarged. There was an excessive amount of serous peritoneal and pleural fluid. The ileocaecal area of the intestine was within normal limits. These features have been described as characteristic of *M. avium. paratuberculosis* infection in alpacas and are in contrast to sheep and cattle, in which intestinal mucosal thickening is characteristic of Johne's disease.

Histologic examination of the small intestine, liver, and mesenteric and mediastinal lymph nodes revealed a severe granulomatous inflammation with large numbers of acid fast organisms in Ziehl Neelsen (ZN) stained sections. Large numbers of epithelioid macrophages and low numbers of giant cells, lymphocytes and plasma cells diffusely infiltrated the small intestine mucosa. The macrophages had abundant foamy cytoplasm in H&E sections, with occasional Gram negative bacilli. On ZN stain there were large numbers of acid fast bacilli in the cytoplasm of the macrophages.

There were large, multi-focal granulomas throughout the hepatic parenchyma. ZN stains revealed large numbers of acid fast bacilli. The normal architecture of the mediastinal and mesenteric lymph nodes had been obliterated and replaced by necrotic, mineralised debris. Along the margins of the necrotic areas there were numerous epithelioid macrophages which contained large numbers of acid-fast bacilli. Thus the histology in this case is similar to that described in cases of *M avium* subspp. *paratuberculosis* (Ridge *et al*).

Faeces were cultured for *Mycobacterium spp.* and acid-fast bacilli were identified. PCR using genus primers (16S) and species primers (IS900), specific for *M. avium*, subsp. *paratuberculosis* confirmed the bacilli as *Mycobacterium spp*, but tested negative for *M. avium* subsp. *paratuberculosis*. Further testing for Mycobactin dependency, using Herrold's and modified 7H10 medium with and without mycobactin, demonstrated that the isolate was not mycobactin dependent and therefore not consistent with *M.avium* subsp. *paratuberculosis*[1].

In addition to the faecal isolate, DNA was extracted from cut sections of paraffin embedded formalin fixed tissues. Both samples were tested by PCR using primers: IS900, *Mycobacterium* 16S genus, Tuberculosis complex, *M. intracellulare /M avium* complex and IS 1311 *M. avium* complex[1]. All tested tissues reacted with the two *M. avium* specific PCR primers (*M intracellulare /M avium* complex and IS1311 *M. avium* complex), but not with other species specific PCR primers. Thus the results are consistent with me presence of *M. avium subsp avium.*

This case report highlights the importance of culture and PCR in identification and differentiation of *M avium subspp. paratuberculosis* from *M. avium subspp avium* in alpacas, as histological lesions may be indistinguishable.

References

Ridge SE, Harkin JT, Badman RT, Mellor AM, Larsen JWA, 1995, "Johne's disease in alpacas (Llama pacos) in Australia," Australian Veterinary Journal 72: 150-153.

Acknowledgements

1. These tests were performed by Debbie Cousins, Animal Health Laboratories, WA Agriculture, South Perth.

Cheyletiella parasitivorax **infection in a rabbit and suspected zoonoses to owners** - Julia Lucas, Veterinary Pathology Services, Adelaide

An adult, male rabbit was presented to a local veterinary clinic for chronic scratching and dandruff. The rabbit had been in good body condition and the hair coat was near normal, except for an excessive amount of keratin flakes (dandruff). There was no alopecia, erythema or papules in the skin.

Sticky tape preparations from the hair coat in the mid dorsal region contained numerous mites, at different developmental stages, including some eggs. The mites were large, with distinctive palpal claws and chelicera. Dr Ian Carmichael, veterinary parasitologist, confirmed the mite as *Cheyletiella parasitivorax*.

Cheyletiellidae include nine genera infecting birds and mammals[1]. The mite does not burrow or infect hair follicles, but remains superficially in the keratin layer of the epidermis or on the hair shafts[1]. Clinical signs vary from subtle hyperkeratosis (as in this case) to erythema, scale and alopecia[5]. The lifecycle is completed on the host and transmission is predominantly by direct contact between hosts[1].

The owners of the rabbit reported that an adult female (human) and adolescent female (human) in the household had both developed "allergic dermatitis" about the same time the rabbit had been acquired. Both were prescribed topical corticosteroids by their general practitioner. The treatment apparently aggravated the lesion and both elected to cease treatment without further medical consultation. The lesions resolved spontaneously about three weeks later, but have recurred intermittently in both females. On further questioning it was discovered only the two females in the household had direct contact with the rabbit.

It appears that the two humans have developed a transient infection with *Cheyletiella* from direct contact with the rabbit. Transmission of *Cheyletiella parasitivorax* between domestic animals and man has been reported extensively[2,3,4,6,7]. One report cites that patients are typically female, less than 40 years old, and skin eruptions occur predominantly in winter[7].

References

1. Kettle D.S 1995, *Medical and veterinary Entomology*, second edition.
2. Austad R1968 "Cheyletiella parasitivorax. A mite parasitic on animals causing an itching eruption on humans" *Tidsskr Nor Laegeforen* Jan 1;88(1):28-30 (abstract)
3. Bjarke T, Hellgren L, Orstadius K1973 "Cheyletiella parasitivorax dermatitis in man." *Ada Derm Venereol*;53(3):217-23 (abstract)
4. Clark JD, Ah HS1976 "Cheyietiella parasitivorax (Megnin), a parasitic mite causing mange in the domestic rabbit" Journal of Parasitology Feb;62(l): 125 (abstract)
5. Cloyd GG, Moorhead DP 1976 "Facial alopecia in the rabbit associated with Cheyletiella parasitivorax". *Laboratory Animal Science* Oct;26(5):801-3 (abstract)
6. Dal Tio R, Taraglio S, Tomidei M, Vercelli A 1990 "Dermatitis caused by Cheyletiella. Description of 8 cases and review of the literature" *G Ital Dermatol Venereol* Feb;125(l-2):19-24 (abstract)
7. Lee BW 1991 "Cheyletiella dermatitis: a report of fourteen cases." *Cutis* Feb;47(2):111-4(abstract)

Mice profusely passing protein - John Finnie, Veterinary Division, IMVS, Adelaide

A large group of mice (36) were sent from an animal house in Sydney to Adelaide for routine disease surveillance. All animals were clinically normal when despatched. Upon arrival, mice appeared unaffected or were slightly depressed (but who wouldn't be after a long interstate flight in a small box!). At necropsy, pathological changes were confined to the kidneys, which were slightly swollen and paler than normal. Microscopically, in 28/36 mice, nearly all tubular lumina and collecting ducts contained eosinophilic, proteinaceous casts. Glomeruli, at least at the light microscope level, were normal and there were no other significant morphological alterations in these kidneys.

This is the second occasion on which this protein-losing nephropathy has been present in a large number of apparently clinically normal mice sent from this facility. The lesion appears acute, but its pathogenesis is unknown (at least to me!).

13.

Tasmania - Phil Ladds

Department of Primary Industries Water and Environment Mount Pleasant Laboratory Report

C**ases of general interest** - Philip Ladds and Les Gabor

Bovine - Two rare neoplasms, a heart base tumour and a sarcoma (presumed rhabdomyosarcoma) were diagnosed in abattoir specimens from an 8 year-old cow and a 2 year-old steer, respectively. Equivocal striations and filaments were demonstrated in the latter tumour by PTAH staining and electron microscopy. Occasional "false positive" reactors to the ELISA for Johne's Disease continue to occur; no gross or microscopic lesions are found in such reactors and acid-fast organisms are not demonstrated in faeces nor sections.

Ovine - An abattoir survey of gut and mesenteric lymph nodes for ovine Johne's Disease (OJD) has revealed a number of incidental lesions in the nodes including parasitic granulomas, focal granulomas associated with pigment and debris, megakaryocytosis and apparent granulopoiesis in nodes which are otherwise normal, and OJD-like focal granulomas in which acid-fast bacilli cannot be found even with careful searching.

Porcine - Classic Glasser's disease was diagnosed in weaner pigs. Lesions were those of severe fibrinocellular polyserositis and fibrinous meningitis, and *Haemophilus parasuis* was isolated.

Wildlife - Submissions included Chlamydiosis in a Green Rosella (*Platycercus caledonicus*) which had mononuclear infiltrations and elementary bodies in the liver, severe gastro-intestinal parasitism with gastric ulcers (associated with sp.) and possible anaemia in a Little Penguin (*Eudyptula minor*), hepatic coccidiosis in rabbits, and (presumed malicious) organophosphate poisoning in seagulls. A total of 20 birds, including Silver gulls and one Pacific gull were submitted. No consistent lesions were found. Gut contents, kindly examined by the Animal health Laboratory, South Perm were negative for botulinum toxins, but subsequent examination by Analytical Services, Tasmania, revealed the organophosphate, "Mevinphos" at a level of 7.1mg/kg.

Report from Fourth International Abalone Symposium, Capetown, February 2000 - Judith Handlinger

I was invited to attend this conference co-present with Dr Carolyn Freidman from California and Anna Mouton from South Africa in what is believed to be the first international workshops on abalone health, which were attended by nearly 80 delegates, and covered abalone disease findings world-wide.

The health session at the main Symposium presented new disease findings, especially regarding the major epizootic disease "Withering Foot" which has decimated Californian abalone since early 1980s, and has more recently been found in Central America. The cause of Withering Foot has been identified as a new species of the intracellular bacteria group Rickettsia. The other major disease discussed was the sabellid shell worm known to be introduced to California with abalone from South Africa. The possibility that Withering Foot was also introduced to America by this route was raised by South African findings of a similar Rickettsia, which is not pathogenic in South African abalone. (DNA studies are awaited.)

Also of concern were South African findings of paralytic shellfish poisoning in abalone, which as a grazer rather than filter feeder, was not thought to carry a significant risk. Not only has this product implications for human health and closed the industry for a significant period, but the toxin was also shown to affect the survival and recruitment of the abalone.

In all, this provided valuable linkages for consolidation of knowledge of abalone disease, and has resulted in increased awareness and interest in health issues in the local industry, particularly with the lack of background data in abalone disease world wide.

Perspectives from this trip will be presented at the Mollusc Pathology Workshop being held for the first time in conjunction with the University of Tasmania's Fin-fish histology for pathologists (initiated by Fisheries Victoria). In the longer term it is hoped to produce a second CD of abalone pathology, so I invite all and sundry to submit interesting abalone cases and findings for inclusion so this eventuates, because pathology-wise these are still frustratingly subtle critters!

Footnote: The impact of these findings on the many Australian industry delegates, plus the massive expansion in train for abalone aquaculture world-wide, and the development of disease surveillance programs in other countries has since prompted industry interest in several Australian states, including Tasmania, in developing suitable health monitoring programs on a state or possibly national basis.

Pentacapsular myxosporean encephalomyelitis of Striped Trumpeter - David Taylor, Fish Health Unit, DPIWE, Mt Pleasant

Since 1997 a new potentially significant disease has been increasingly recognised in the Striped Trumpeter (*Latris lineata*). This species is currently undergoing development for commercial aquaculture. Affected fish varied from 5-300g in weight. Clinically they exhibit scoliosis, swim on their sides, circle, and are wasted. Affected fish die. Smears of the brain and the spinal cord revealed large numbers of refractile myxosporean spore structures, Diff-Quik and gram stains allowed visualisation of the pentacapsular structure. Smears of the blood and other tissues are normal.

Histologically, the number and maturity of the spores within the brain and spinal cord varies as does the degree of granulomatous host reaction. The distribution of the spores is predominantly adjacent to the ventricles or meninges, involving both grey and white matter, but not uniform. Similarly, spores are present within various spinal nerve tracts. Scattered spores can also be detected within the retina and the optic nerves and cells suggestive of a myxosporean plasmodia have been noted within blood vessels of the choroid. Skeletal muscles adjacent to the spinal cord appear to be undergoing early degenerative change but no spore forms can be identified. Examination of other tissues has been unremarkable.

There are very few pentacapsular myxosporean species reported from fish[1] and none reported to show neurotrophism. A number of different myxosporean species target the brain and spinal cord[2],[3]. Transmission of myxosporea reputedly involves an intermediate oligochaete worm. However, a recent report has suggested that direct transmission is possible[4]. The exact method of entry into this host has yet to be defined. This disease has the potential to severely limit the development of this species for aquaculture.

l. Lom J and Dykova I. (1992). *Myxosporidia (PhylumMyxozoa) In*: Protozoan Parasites of Fish, Developments in Aquaculture and Fisheries Science, Volume 26, p 226, Elsevier, Amsterdam.

2. Egusa S. (1985). *Myxobolus buri* sp. n. (Myxosporea: Bivalvulida) parasitic in the brain of *Seriola quinqueradiata* Temminck et Schlegel. Fish Pathology, 19: 239-234.

3. Langdon JS. (1987). Spinal curvatures and an encephalotrophic Myxosporean *Triangula percae* sp.nov. (Myxozoa: Ortholineidae), enzootic in redfin perch, *Perca fluviatilis* L., in Australia. Journal of Fish Diseases, 10: 425-434.

4. Diamant A. (1997). Fish-to-fish transmission of a marine myxosporean. Diseases of Aquatic Organisms. 30: 99 -105

Victoria - Malcolm Lancaster

Crvptococcosis/Toxoplasmosis in an Echidna - Peter Phillips (Victorian Veterinary Pathology Services)

Fixed tissues and fresh lung from a male Echidna of unknown age were received from Melbourne Zoo. The history was that the animal died unexpectedly while under treatment for presumed chronic allergic conjunctivitis. Histopathology showed patches of necrosis within lungs with surprisingly little inflammation. The alveoli and airways contained vast numbers of cryptococci and there were several schizonts of a sporozoan organism, presumably Toxoplasma, within alveolar walls. Whether these schizonts were intracellular was not determined. There was a severe mononuclear periportal hepatitis with smaller foci of inflammation scattered through hepatic sinusoids, with one sporozoan schizont present in a sinusoid.

Small numbers of Cryptococcus organisms were present in several renal glomeruli. There was widespread membranous glomerulonephritis with occasional scattered foci of necrosis and haemorrhage in the cortex.

Unfortunately we were unable to recover the Cryptococcus from the fresh lung for typing.

It was concluded that the animal was probably immunosuppressed, possibly iatrogenically, and succumbed to Cryptococcus/Toxoplasma infection.

Vascular Hamartoma in the Ear of Border Collie - Judith S. Nimmo Wilkie, Idexx/CVDL, Melbourne.

A male border collie of one-year-old presented with a swollen, congested and oedematous left ear with a necrotic tip. Tissue from the ear showed a diffuse infiltration by abnormal blood vessels, involving the dermis on both sides of the aural cartilage. The abnormal vessels consisted of large dilated vessels with well-developed walls surrounded by proliferations of smaller, capillary-sized vessels and numerous fibroblasts and pericytes. There was necrosis of the ear tip, probably secondary to ischaemia, and a resultant chronic, secondary inflammation. Histologically the lesion is identical with scrotal vascular hamartoma, except for the site. No previous reports of this condition were found although other vascular anomalies have been reported in this breed.

Possible Congenital Tremors and Hypomyelinogenesis in Sharpei Puppies - Judith S. Nimmo-Wilkie, Idexx/CVDL, Melbourne.

Two Sharpei pups from the same litter were euthanased at 3 weeks of age due to nervous signs which include tremors, hypermetria, ataxia and inability to walk forwards. Both pups had very jelly-like brains grossly. One was examined histologically and showed overall pallor with H&E and Luxol-fast blue stain. The white matter of the cerebellar folia was narrow and oligodendrocytes had prominent cytoplasm. The kidney was also submitted for examination but no lesions were noted. The affected pups were much smaller than their litter-mates. Another pup from another litter with the same sire but different dam, also showed similar neurological signs. This pup is still alive and continues to show neurological signs although there has been some improvement.

No previous reports of this condition in this breed were found.

STATE REPRENTATIVES

Queensland	Jim Taylor Toowoomba Vet Lab, QDPI PO Box 102 TOOWOOMBA QLD 4350	 074688 1351
Victoria	Malcolm Lancaster VIAS 475 Mickleham Road ATTWOOD VIC 3049	 03 9217 4200
South Australia	Ruth Reuter VPS PO Box 445 GLENSIDE SA 5065	 08 8372 3700
New South Wales	Paul Gill RVL WOLLONGBAR NSW 2480	 02 6626 1261
Western Australia	David Forshaw Animal Health Laboratories Agriculture WA ALBANY WA 6330	 08 9892 8444
Northern Territory	Anton Janmaat PO Box 990 DARWIN NT 0801	 08 8999 2240
Tasmania	Philip Ladds TAWQDS PO Box 46 KINGS MEADOWS TAS 7249	 03 6336 5216