Print Post Approved

AUSTRALIAN SOCIETY FOR VETERINARY PATHOLOGY

VETERINARY PATHOLOGY REPORT
Publication No. PP544059/0003

EDITOR: CleveMain

Number 47 December 1997

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 3446337 (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 S PERTH WA 6151 08 9368 3351
Treasurer	Jeremy Allen	Animal Health Laboratories 3 Baron Hay Court S PERTH WA 6151 08 9368 3466
Committee Members		
	Barry Richards	Animal Health Laboratories 3 Baron Hay Court S PERTH WA 6151 08 9368 3424
	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
Coordinator - Training Committee	Judith Wilkie

CONVENOR - SLIDE OF MONTH

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

STATE REPRESENTATIVES

Queensland	Bruce Hill Rockhampton Vet Lab, QDPI PO Box 6014 ROCKHAMPTON QLD 4702 07 4936 0211
Victoria	Malcolm Lancaster VIAS 475 Mickleham Road ATTWOODVIC 3049 03 9217 4200
South Australia	RuthReuter 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	Roy Mason TAWQDS PO Box 46 KINGS MEADOWS TAS 7249 03 6336 5216

PRESIDENTIAL REPORT

As I apply fingers to the keyboard for my first report to you as the President of your Society, I must begin by saying how pleased I am to take on this task with the support of such a motivated and competent group of colleagues on the Executive. In picking up the baton from the previous Executive, we acknowledge its efforts and those of all the elected officers and members who worked so hard on tasks like the annual conferences, the training committee and the slide of the month.

To say the least, our discipline faces a period of intense challenge, and I can assure you that your new Executive will do its utmost to advance the interests of veterinary pathology and veterinary pathologists across the nation during the next three years. We need your help to do this, and I encourage you all to communicate with us frequently, to keep us updated on important issues and to let us know if we are not doing the job well enough. The new Executive met for the first time on 12 August and has regularly met each month since. We saw as our first priority, identification of critical issues and the development of strategies for addressing them. It was generally agreed that the current long term outlook for the discipline in Australia is not encouraging, with a fairly large proportion of our experienced people approaching the end of their working lives, coupled with a general running down of facilities and opportunities for bright young people to get the sort of training required. Employment opportunities in the Government sector and the Universities will remain tight, while the further development of the private sector is an unknown at present, and seems unlikely to impact with rapid effect on the area of training. Furthermore, the strength of the discipline within the universities is in general likely to erode.

Given this gloomy forecast, what needs to be done? The answer is political - influential people in the right places need to be convinced that a corps of appropriately trained veterinary pathologists is essential to the national interest, with for instance, a direct bearing on the success of any national disease surveillance scheme. Any ideas you have for ammunition to fire in such a campaign would be welcome.

From my own perspective within a university, it is my conviction that in spite of the diffusion of discipline boundaries, pathology remains at the core of any bio-medical science. It is true that in this increasingly specialised world clinical specialists develop considerable knowledge and understanding of the pathology of their particular area. However, there has to be a well from which they drink and that is the discipline of modern pathology and the specialist pathologists who advance it.

We do have credibility, our expertise is definable, and we have a very important role to play. We need to make clear to certain people the consequences of our decline. Let us all bend our brains to the task of making this happen.

In October I represented the Society at the workshop on quality assurance held at the Elizabeth McArthur Institute, Menangle NSW, under the auspices of SCAHLS. Another member of the executive, Dr Barry Richards also attended (as a representative of SCAHLS). The workshop canvassed the whole issue of quality assurance in veterinary pathology services, with inputs from a wide range of sources, including the human pathology sector. The role and relevance of the Registry of Domestic Animal Pathology also came under scrutiny and it became obvious that in its present form it is not highly valued by many members.

As a result of what was learned at the workshop, Dr Richards and I decided that a proposal for a new "business arm" of the Society should be drafted, aimed at incorporating the Registry and its continuing education activities into a new and broader function to be run by a full-time Coordinator/Director. This new unit would be central to a quality assurance program, and will seek funding from SCAHLS and the AAHC. This proposal is now in its second draft, with comment received from the current Registry Management Committee, and the executive is keen to have it finalised as soon as possible

That is my perspective, but what is yours? I look forward to hearing from you after you have what I trust will be a very merry Xmas and a positive start to the New Year

EDITORIAL

By now you will have read the minutes of the 1997 Brisbane AGM and are aware that your new Executive was instructed to carry out several tasks. One of these was to determine the fate of archival pathological material held at recently closed veterinary laboratories in Victoria. In reply to my letter the Chief Veterinary Officer for Victoria, Dr Andrew Turner has advised that:

"The Victorian law requires that the material and records be retained for at least 7 years. Negotiations have been undertaken with the official Receiver of Centaur to ensure that the relevant material is retained and returned to the Department."

A second task directed to the incoming Executive concerned proposed guidelines for the histopathological diagnosis/exclusion of ovine Johne's disease. SCAHLS is aware of these concerns and has received several drafts containing suggested amendments. When the final draft is available it will be published in the Veterinary Pathology Report.

Committee was also requested to obtain from the Australian Veterinary Association (AVA), requirements for affiliation of the Society with special reference to cost, non-AVA members and non-veterinarians who hold Society membership. The position of the AVA is unchanged; non-veterinary members are permitted "associate" member status with no voting rights or eligibility to hold office. Veterinarians who are not members of the AVA are not entitled to membership status in, or participate in, the affairs of a Special Interest Group (SIG), but may subscribe to publications of the SIG.

The costs of registration are simply those incurred by a prospective SIG in applying for AVA board for recognition and in modifying or developing a Constitution. There are, however, ongoing costs relating to financial reporting and voluntary attendance at policy/administration meetings. No costs are incurred by participating in the AVA annual conference. In fact an SIG is entitled to receive a subsidy for speakers at such Conference and to share in the profit, if any, from conference.

A copy of the reply from the AVA is appended.

The proposition that the ASVP affiliate with the AVA has previously been voted on by the membership in postal ballot, the motion for affiliation being defeated by 37 votes to 33 (refer Minutes AGM May 1995, published Vet Path Report 42, Sept 1995). As a result of a successful motion at the 1997 AGM (moved J. Mackie, seconded P. Ladds, carried 23 for, 8 against), I am required to ask the membership to once again consider the proposition that the Society become a Special Interest Group, again be put up for determination by membership by postal ballot.

Accordingly, enclosed with this report is a ballot paper, an addressed envelope, a copy of the last letter from the AVA and previously circulated paper outlining the advantages and disadvantages of ASVP/AVA affiliation. Please give the matter serious consideration. Postal votes must be with the Secretariat by 15 February 1998.

I need not remind you that only financial members may vote. Circulation of the ASVP report is currently limited to those who have paid their dues for 1997/98 plus those members whose dues are one year overdue. If you are one of those who have not paid up, now is the time do so. Remember, the consensus of opinion from the Brisbane AGM was that members who fail to renew their subscriptions should be allowed one year's grace. Failure to pay dues outstanding for more than one year will result in removal of the offenders name from the membership list.

In the same vein, if you know of anyone who is entitled to receive the ASVP Report and is not receiving it, please ask them to contact Amanda Whyte at the ASVP Secretariat C/- PO Box 114 Walkerville SA 5081 (Ph 088 344 6337).

STATE REPORTS

New South Wales - Paul Gill

News & Activities

Norseq Veterinary Pathology Group Meeting, Paul Gill, Agriculture NSW, Wollongbar

A very successful Norseq meeting was held at Wollongbar on 2 August. The theme was "Protozoan Diseases". Bill Hartley spoke on the pathology of apicomplexan diseases and John Ellis (University of Technology, Sydney) spoke about the molecular phylogeny of Sporozoa. Case presentations included Babesiosis in a mountain ringtail possum (Paul Gill); Microsporidian myositis in an aquarium fish (Michael Rozmanec); Giardiasis in a calf (John Boulton), Spironucleosis in laboratory mice (Roger Kelly); Haemoparasites in Eastern Grey kangaroos (Roger Cook); Human myiasis due to *Dermatobia hominis* (Sandra Howden); Malaria in a lorikeet (Neil Sullivan) and Haemoparasites in Australian native birds (Bill Hartley).

New Facilities for Vet Path at Sydney, Malcolm France, University of Sydney

The pathologists at the Sydney vet school have just completed moving into newly refurbished labs and offices in what used to be the CSIRO McMaster laboratory building. This brings together all the disciplines of veterinary pathology (clin path, micro, parasitology, immunology etc.) under one roof for the first time in many years. One of the final stages in the project was the demolition of the fibro building which had been the 'temporary' home of veterinary pathology for half a century. Despite inevitable financial constraints, the new facilities are a great credit to Alan Husband and the many who contributed to making the most of this opportunity to provide a more functional, concrete and comfortable home for the Department. There will be an opportunity to view these facilities and the new Conference Centre/Post-Graduate Foundation building at the next meeting of the NSW Veterinary Histopathology Group (details below).

NSW Veterinary Histopathology Group Meeting

The next meeting will be on Saturday 8 November at the Sydney Vet School. A tour of the new pathology facilities will begin at 11.30 a.m. followed by lunch at 12.00 noon and case presentations from 1.00 p.m. Further details please contact Malcolm France on 029 351 2023, Fax 02 9351 7348 or email m.france@vetp.usyd.edu.au.

Pathology Reports

Pathology of porcine paramyxoviridae, an emerging disease Tony Ross, Agriculture NSW, Elizabeth MacArthur Agricultural Institute, 02 4640 6312

A new cause of infectious reproductive failure has been identified in a large piggery in coastal NSW recently. Over a 5 month period the piggery experienced a major decline in reproductive performance.

Clinical disease appeared to be confined to pregnant sows. Whilst affected sows remain well, the virus targets the pregnant uterus and is highly teratogenic. Bob Love of Sydney University submitted affected piglets to RVL Menangle. He reported a major decline in farrowing rate and an increase in litters containing stillborn and mummified piglets. The disease showed a classic epidemic pattern moving from pen to pen within a shed, then between sheds, affecting sows of all parities.

In utero death occurs at a range of foetal ages implying that viral infection spreads relatively slowly from foetus to foetus and or that some piglets are lethally affected in utero and others sub-lethally. Some cannot survive independently of the womb and die at birth, whilst others survive and appear to grow normally. Examination of affected litters shows a continuation of mummies, semi-mummified foetuses, stillbirths and live piglets.

Mummified foetuses occur as early as 30 days gestation and often show evidence of arthrogryposis.

Stillborn foetuses also show a high rate of teratogenic defects. Gross reductions in brain and spinal cord size have been observed in over 50% of piglets examined. These lesions are usually accompanied by arthrogryposis.

Defects seen at lower frequencies include undershot jaws, kyphosis, absence of digits and pulmonary hypoplasia.

Excessive amounts of straw coloured fluid, sometimes containing strands of fibrin are present in the body cavities of the majority of stillborn piglets and are pronounced in approximately 25% of cases.

From the size of the cranial and spinal cavities of piglets with major brain and cord defects, it is likely that the CNS had developed normally until it was attacked by the virus. Large areas then became necrotic and were phagocytosed and resorbed leaving variable amounts of brain and cord remnants.

Histological findings in stillborn piglets are most marked in the central nervous system. Lesions vary from degeneration and malacia to necrosis within various parts of the brain and cord. Gliosis and macrophage activity are common at some sites. Intranuclear and intracytoplasmic eosinophilic inclusions are common in several areas of non-suppurative encephalitis and myelitis. Preliminary immunohistochemical studies by Peter Hooper at AAHL have demonstrated that sow serum containing neutralising antibody to the virus specifically binds to neurones and the neuropil in the lesions. Binding favoured cells which contained intranuclear inclusions whilst not actually binding to the inclusions themselves.

Other histopathological changes are mild and are not present in all affected piglets. They may include multifocal non-suppurative meningitis, multifocal non-suppurative myocarditis and occasionally multifocal non-suppurative to suppurative hepatitis.

Partial characterisation of the virus at EMAI by Peter Kirkland and Adrian Philbey suggests that the virus is a paramyxovirus. Further characterisation is being carried out at AAHL. At present the virus appears to be a new member of the Paramyxoviridae family of RNA viruses which include: mumps, measles, distemper, rinderpest, Newcastle disease and bat paramyxovirus.

Specimens for laboratory examination should include serum from sows with affected litters together with around 5 to 10 cohort sera for serum neutralisation tests (SNT). Stillborn piglets should be examined for brain and spinal cord defects and body fluid taken for SNT. Fresh and fixed lung, heart, brain and cord should be submitted for virus isolation together with a wide range of specimens to exclude parvovirus, classic swine fever, encephalomyocarditis virus, Aujeszky's disease and other causes of infectious abortion.

In summary the virus appears to be highly pathogenic for the gravid uterus. It is lethal and teratogenic for the majority of piglets in affected litters and shows particular tropism for the central nervous system.

Respiratory Disease in SPF Mice, Malcolm France, University of Sydney

A shipment of BALB/c mice from a well maintained, SPF barrier facility was introduced to a 'conventional' animal house of uncertain health status. Over the following weeks, the new mice began to lose condition. Post mortem of one mouse revealed enlarged mediastinal lymph nodes and lungs which were only partially collapsed. Histologically there was severe suppurative bronchopneumonia, tracheitis and otitis media. There was also moderate to marked hyperplasia of respiratory epithelium and occasional epithelial syncytium formation. A serum sample was positive for mouse hepatitis virus (MHV) and *Mycoplasma pulmonis* but negative for Sendai virus and pneumonia virus of mice.

The lesions in this mouse appear to be the result of a dual infection with two of the most important pathogens of laboratory mice: a respiratory strain of MHV (hence the syncytia) and *M. pulmonis*, an important pathogen of the upper respiratory tract. While the need to use SPF animals in research is becoming more widely appreciated, there remains some resistance to this trend, particularly in cases where a costly upgrading of facilities and equipment may be required to maintain SPF status. In the case described here, the losses in naïve animals served to draw attention to an endemic disease problem (mycoplasmosis) which had previously been overlooked but which had the potential to confound research results.

Break-down of erysipelas vaccines in pigs, John Glastonbury, Regional Veterinary Laboratory, EMAI

At the RVL Camden (EMAI) during June 1997, we were involved in the investigation of a substantial mortality in growers and finishers in a large intensive unit with 3,000 sows housed in five units. The farm was situated on the south-west slopes of NSW. At the time of submission of samples to the laboratory, 50 of 5,000 pigs had died in three of the units. Vaccines from three different manufacturers had been tried in an effort to stem the losses.

The affected animals were found dead.

Gross pathological findings described by the submitting veterinarian were cyanotic extremities, excessive volumes of straw-coloured pericardial and peritoneal fluids and the presence of strands of fibrin in the peritoneal cavity. There were no classical "diamond skin" lesions.

From various tissues from different animals, we recovered seven isolates of *Erysipelas rhusiopathiae*. These were all serotyped as 2B by the Microbiology Section.

The most interesting histopathological feature was almost diffuse subacute non-suppurative interstitial nephritis in five of seven animals examined. In each instance there was visceral congestion, diffuse pulmonary oedema and septic emboli in the alveolar walls, interstitium of the kidney and splenic red pulp in decreasing order of frequency.

Embolic interstitial nephritis is described as a feature of porcine erysipelas in the literature but in our cases the inflammation was too severe and widespread to ascertain its origin. One theory is that the vaccination had rendered the animals partially immune leading to the immunological reaction in the renal interstitium, and when this was overcome the pigs succumbed to acute septicaemia.

Super Resistant Haemonchus contortus in the northern tablelands of NSW, Stephen Love, State Worm Control Coordinator, NSW Agriculture, Armidale

Why a 'parasitology' article in a pathology publication? Firstly, worms cause disease, and secondly I am a long time member of the ASVP and was a vet. pathologist (1986-1996) at the former Regional Veterinary Laboratory, Armidale. Now for the (bad) news:

A strain of *Haemonchus contortus* resistant to both closantel (Seponver etc.) and the macrocyclic lactones (ivermectin, moxidectin etc group) was discovered by Bruce Chick (Veterinary Health Research laboratory, Armidale) in samples from a sheep farm at Bundarra near Armidale in northern NSW. This strain has serious worm control implications, at least where the control is heavily reliant on managing worms with anthelmintics.

Haemonchus contortus of course is a major parasite in the sheep raising areas of northern NSW. This and other worms have been successfully controlled using the WORMKILL strategic worm control program, introduced in 1984. *Haemonchus contortus* control has relied heavily on the strategic use of closantel, a narrow spectrum anthelmintic with persistent activity against this parasite. However, resistance to closantel appeared in northern NSW in 1987, and has since become somewhat more prevalent. In some localities, as many as one farm in two has closantel resistant *Haemonchus contortus*. (A closantel resistance study is currently underway in northern NSW).

Resistance to macrocyclic lactones (ML) the newest group of broadspectrum anthelmintics, is another emerging problem. ML resistant strains are commonplace in South America, for example, and are "bubbling to the surface" in Australia. In WA there are at least seven farms with confirmed ML resistance in *Ostertagia sp* (B. Besier, pers. comm.). To my knowledge five ML resistant strains have been uncovered in NSW, two *Ostertagia sp* strains in goats (North Coast; Central Slopes), and three *Haemonchus contortus* (all in sheep), one of which is the Bundarra strain resistant to both Closantel and MLs.

Various alternative control options for resistant *Haemonchus contortus* are outlined in the booklet, "Closantel Resistant Barber's Pole Worm in Sheep - Management Options and Control Strategies", produced by the Wormkill Technical Committee (1995) (and available from Rural Lands Protection Boards and NSW Agriculture offices). Obviously the burgeoning anthelmintic resistance problem does not augur well for worm control based only on chemicals. A "multi-strand" or integrated approach needs to become the norm; an approach combining the strategic but reduced use of chemicals with breeding resistant animals (NEMESIS program), grazing management, flock and weaner management and regular monitoring of parasite levels (WORMTESTS) and anthelmintic resistance (DRENCHTEST/ DRENCHRITE). And hopefully in the medium term we can add further "strands": nematophagous fungi ? Vaccines?

Time is running out. We need more "strings to the bow" than just anthelmintics. Ask the farmer at Bundarra.

Experiences with the laboratory diagnosis of ovine Johne's disease, Jeff Marshall, Regional Veterinary Laboratory, Orange

This report describes the gross pathological and histopathological findings we are seeing at the Veterinary Diagnostic Centre Orange in our research and diagnostic activities associated with ovine Johne's disease. I will also try to summarise the scientific literature in these areas and compare them with our experiences at Orange.

- Experimental Infection

Studies of experimental infection help us understand the progression of the disease in the animal and hence the importance of different diagnostic tests during this progression. A number of studies of experimental infections in sheep have been reported (1,3,6,8,7,10,11). These can be summarised as follows:

- to 30 days post infection - no lesions observed, *Mycobacterium paratuberculosis* cannot be isolated.
- to 60 days post infection - Mycobacterium paratuberculosis can be isolated abundantly, infiltration of neutrophils and macrophages into interfollicular areas of Peyer's patches.

- 60 days post infection - ability to isolate Mycobacterium paratuberculosis drops off to almost undetectable levels. Definite granulomatous inflammation is evident in interfollicular areas. Initial granulomas are associated with small lymph vessels in the submucosa.

- 120 days post infection - the development of disease has taken its course and different types of lesions can be observed.

A strong host immune response is thought to be associated with the granuloma formation diminishing in number and size. Those remaining acquire a more circumscribed even fibrotic appearance. If the host is not able to mount an efficient immune response, lesions may progress towards a more lepromatous form, where inflammation spreads to adjacent lamina propria, mycobacteria increase in number and circulating antibodies rise.

A spectrum of intermediate forms between these two.

- Gross Pathology

Lesions are similar to those seen in cattle.

Carcass emaciation and oedema

Diarrhoea in less than 20% of clinically affected sheep. Most have formed pellets.

Thickening and corrugations of the intestinal mucosa, sometimes reddened, showing crevices and a granular appearance. Lesions present mainly in terminal ileum and commonly extend to caecum/colon and jejunum and occasionally the duodenum.

Lymphatic cording and lymphadenopathy. Lymph node lesions mainly present in caudal jejuna! lymph node, and commonly in ileocaecal lymph nodes and other lymph nodes in the mesenteric chain.

Some clinical cases show only minor macroscopic abnormalities. Sheep with no obvious clinical signs may have gross pathological findings which range in severity from no detectable abnormality to severe changes. Histological lesions can be moderate to minimal in the absence of gross pathological changes.

Sometimes see:

- Ascites and hydropericardium.
- Intermandibular oedema.
- Atrophy and necrosis of fat.

We do not see the orange-yellow pigmentation of intestinal mucosa caused by pigmented strains of mycobacteria as reported from Britain (2,4,15).

- Histopathology

A comprehensive classification system of the range of lesions seen by Perez et al., has recently proposed (11,12). In summary;

Category 1 - aggregates of foamy macrophages forming small granuloma in the interfollicular and basal areas of ileal Peyer's patches. Usually no intracellular acid-fast organisms present. Gross lesions not detectable.

Category 2 - more marked lesions in Peyer' s patches with granulomas extending into the adjacent mucosa. Acid-fast organisms usually present. Gross lesions not detectable.

Category 3a - multifocal and large granulomas within the lamina propria, submucosa, serosa of ileum extending to jejunum and draining lymph nodes. Villi are distended, mucosa thickened and acid fast organisms obvious. Gross lesions usually not detectable.

Category 3b - Numerous macrophages and few giant cells spread diffusely in mosaic-like sheets through the lamina propria and submucosa. Villous fusion and marked thickening of the gut. Acid fast organisms can be found packed in the macrophages of the lamina propria but are less numerous in lymphoid tissues of the intestine and draining lymph nodes. Gross lesions detectable. Referred to by some workers as "multibacillary, lepromatous" (4).

Category 3c - Diffuse granulomatous enteritis characterised by marked lymphocytic infiltration of the mucosa with small well defined granulomas and abundant giant cells scattered throughout. Intestinal lymph nodes characterised by granulomas and focal areas of giant cell necrosis. Acid-fast organisms are either sparse or undetectable. Gross lesions detectable. Referred to by some workers as "paucibacillary, tuberculoid"(4).

- Our experiences at the Veterinary Diagnostic Centre, Orange (see references 2,15)

Most sheep that we see which are clinically affected by Johne's disease have histological lesions fitting into the categories 2 (20%), 3a (30%) and 3b(50%). We can, however, see moderate to severe invasion of the lamina propria with epithelioid cells without the presence of acid-fast bacteria. In our experience acid-fast bacteria cannot be found in 50% of the lesions which have a distribution similar to those described by category 2 above. We do not see any lesion resembling that described by category 3c. However we do see sections from a small number of animals (approximately < 2%) in which there is a diffuse infiltration of the lamina propria with lymphocytes and multifocal aggregations of epithelioid macrophages. These are different to what is described for category 3c in that acid-fast organisms are usually abundant and giant cells absent.

- Other Comments

We regularly see aggregations of foamy macrophages containing crystalline material in basal areas of the mucosa and within Peyer's patches in intestinal sections and in cortical and less frequently medullary areas of lymph nodes from sheep not infected with Johne's disease. No acid-fast bacteria can be detected in Ziehl-Neelsen stained sections. The macrophages in these locations can usually be differentiated from those seen in Johne's disease by their cytoplasmic contents which are granular containing crystalline fragments (9). In the Ziehl-Neelsen stained sections these cells tend to stain more intensely. Epithelioid macrophages in these areas in Johne's affected sheep do not stain with this intensity.

Granulomas in hepatic parenchyma and or periportal locations can usually be found in histological sections of the liver from clinically affected sheep. Peribronchiolar granulomas are sometimes detected but are rare. It is also difficult to find acid-fast organisms in these liver and lung lesions.

Tuberculous type lesions with areas of caseous necrosis and calcification of intestine and lymph node have been described (13, 16). We have not seen these lesions in the sheep we have studied. Similar lesions have been absent or have been attributed to parasitic causes in other reports (2,3,12).

- References

1. Angus RW and Gilmore NJL (1971) - Effect of rimino phenazine B663 (G30320) on Mycobacterium johnei infection and re-infection in sheep. II. Pathology, J Comp Pathol **81**:227-236.

2. Carrigan MJ and Seaman JT (1990) -The pathology of Johne’s disease in sheep. Aust Vet J **67**:47-50.

3. Chaverz Gris G, Garcia Martin JF, Aduriz G, Moreno B, Gutierrez M and Badiola JJ (1992) - Preliminary results of the comparative study of early response to M. avium and M. paratuberculosis in experimentally infected lambs. Medicina Veterinari **9**:16-18.

4. Clarke CJ and Little D (1996) - The pathology of ovine paratuberculosis: gross and histological changes in the intestine and other tissues, J Comp Pathol **114**:419-437.

5. Clarke CJ (1997) - The pathology and pathogenesis of paratuberculosis in ruminants and other species. J Comp Pathol **116**:217-261.

6. Gilmore NJL, Angus RW and Mitchell B (1978) - Intestinal infection and host response to oral administration of Mycobacterium johnei in sheep. Veterinary Microbiology **2**:223-235.

7. Juste RA, Garcia Martin JF, Peris B, Ocariz CZ and Badiola JJ (1992) - Experimental infection of vaccinated and non-vaccinated lambs with Mycobacterium paratuberculosis. J Comp Pathol **110**:185-194.

8. Kluge JP, Merkal RS, Monlux WS, Larsen AB, Kopecky KE, Ramsey FK and LehmannRP (1968) - Experimental paratuberculosis in sheep after oral, intratracheal, or intravenous inoculation: lesions and demonstration of aetiological agent. Am J Vet Res **29**:953-962.

9. Ladds PW (1988) - A colour atlas of lymph node pathology in cattle. Boolarong Publications, Townsville.

10. Nisbet DI, Gilmore and Brotherston JG (1962) - Quantitative studies of Mycobacterium johnei in tissues of sheep. III. Intestinal histopathology. J Comp Pathol **72**:80-91.

11. Perez V, Garcia Marin JF and Bodiala JJ (1992) -Pathological features in sheep naturally infected with M. paratuberculosis. Medicina Veterinari **9**:20-22.

12. Perez V, Garcia Marin JF and Bodiala JJ (1996) - Description and classification of different types of lesions associated with natural paratuberculosis infection in sheep. J Comp Pathol **114**:107-122.

13. Rajya BS and Singh CM (1961) - Studies of the pathology of Johne's disease in sheep. III Pathologic changes in sheep with naturally occurring infections. Am J Vet Res **22**:189-203.

14. Reddy KP, Sriraman PK, Gopal Naidu NR and Rama Rao P (1984) - Pathology of Johne's disease in sheep. Indian Vet J **61**:179-184.

15. Seaman JT and Thompson DR (1984) - Johne's disease in sheep. Aust Vet J 61:227-229.

16. Stamp JT and Watt JA (1954) - Johne's disease in sheep. J Comp Pathol **64**:26-38.

Northern Territory - Anton Janmaat

Opportunist Infections of Emu Chicks? Lorna Melville and David Pritchard, Berrimah Veterinary Laboratories, NT Department of Primary Industry and Fisheries

Two one-week old emu chicks were submitted for post mortem after sudden onset of "energy loss", immobility and anorexia over 24 hours ending in death. At necropsy both chicks had extremely large yolk sacs, swollen friable livers with foci of yellow discolouration and multiple small yellow caseous foci in the lungs. These latter were thought to be consistent with *Aspergillus* sp infection. Histologically, multifocal pulmonary pyogranulomas contained masses of filamentous, branching Gram positive bacteria. A heavy growth of *Nocardia* sp. was isolated from lungs of both chicks.

One week later another chick was submitted in lateral recumbency, spastic paralysis of the legs, extreme ventral flexion of the neck, with the head between the legs, and torticollis. At necropsy there was a massive yolk sac again, multiple peritoneal adhesions and the lungs seemed to be replaced by multifocal caseous nodules. Looked like another case of *Nocardia* infection. Microscopically, lungs had multifocal, typical fungal pyogranuloma with abundant hyphae and in the cerebellum one arteriole has a small thrombus containing fungal hyphae. *Aspergillus flavus* was isolated from lung.

Fooled twice by the one outbreak!

These chicks had been hatched in an incubator and held there for a day or two before being put into a small bare, dry earth pen where presumably they acquired their infections from the soil.

Blood Flukes in Hawksbbill Turtles, Lois Small and David Pritchard, Berrimah Veterinary Laboratories, NT Department of Primary Industry and Fisheries

A sea-turtle researcher found, in his observation lagoon, a Hawksbill turtle floating, unable to dive, weak and barely able to swim. Upon capture, the animal was found to be in very poor condition, had multiple "growths" over flippers, around the eyes and on the mouth. The turtle refused food and died about 48 hours after capture.

At necropsy the turtle was emaciated. Autolysis was advanced. The lumps in the skin looked more to be suppurative inflammation than neoplastic (submitter suspected fibropapilloma). A thick fibrino-membranous exudate covered the mucosa of mouth and pharynx. There were numerous small, white nodules about the size and shape of sesame seeds beneath the peritoneal coat and in the muscle coat along the entire length of the intestine. No other pathological changes were obvious through the autolysis.

Microscopically small blood vessels in brain, intestine, thyroid, pharynx, heart, lung, liver, spleen, kidney and adrenal are packed with eggs of trematodes. The spleen is the most heavily affected while in the intestinal lamina propria and the muscularis, there are granulomatous reactions to many of the egg emboli. Ulceration of the pharyngeal mucosa and exudation is also associated with massive egg embolism. The skin lesions are similar with pyogranulomatous reaction. Adult trematodes are present in large blood vessels of the kidney and lung. Also beneath the peritoneal coat of the intestine there are spherical cysts with thick fibrous capsules and a hyaline membrane lining the apparently empty centre; these resemble the cystic intermediate stages of cestodes. What appears to be a scolex is present in one cyst.

Trematodes were dissected from vessels of the kidney and lung and more were recovered from lung, kidney and liver that had been finely sliced then soaked overnight. These were identified as belonging in the family *Spirorchiidae* and another, as yet unidentified digenetic trematode was recovered from the gall bladder.

Trematodes were also recovered from the lumen of the intestine and identified as *Pyelosomum*. The organism in the cystic structures in the intestinal wall has not been identified yet.

More recently another Hawksbill turtle was also submitted, alive but very thin and unwell. It had skin lesions similar to those of the first turtle. Both necropsy and microscopic findings were similar to those of the first, but more and fresher parasitological specimens were collected. Identifications and confirmations are not completed.

We have heard (unconfirmed) reports of sea turtles caught in WA waters also having massive infestations of helminth parasites. Although the life cycles of these parasites is not known, it is surprising to see such overwhelming parasitism in "free-ranging" wild animals.

Septicaemia in a Wallaby, Kevin Fomiatti and David Pritchard, Berrimah Veterinary Laboratories, NT Department of Primary Industry and Fisheries

A dedicated wildlife rehabilitator submitted a 21-week old agile wallaby. The wallaby had been reared on a wildlife milk substitute and had been in apparently good health until suddenly developing diarrhoea for two days and shallow, rapid breathing for a day before dying. Post mortem decomposition was moderate. Lungs did not deflate when the thorax was opened, were oedematous and contained multiple small haemorrhages up to about 3-4 mm diameter. Microscopically, there is pulmonary hyperaemia and oedema with serous effusion into alveoli and respiratory bronchioles and focal haemorrhages. Bacteria abound throughout the tissue, being most abundant in areas of haemorrhage and exudation.

A heavy growth of *Chromobacterium violaceum* was recovered from lung and liver. On McConkey agar and sensitivity plates the isolate produced a stunning, brilliant blue pigment which, on blood agar turned the medium black. *Chromobacterium violaceum* is a soil and water organism of tropical regions. It occasionally causes serious infections of man and animals. This is the first occasion that we have isolated the bacterium.

Melioidosis in an Alpaca, Anton Janmaat, Berrimah Veterinary Laboratories, NT Department of Primary Industry and Fisheries

Nothing unusual about Burkholderia pseudomallei infection (melioidosis) in the Top End but a first for its occurrence in an alpaca - and in fact wiping out the entire NT alpaca population. Alpaca, in the form of a single 7-months old mate, born and bred in South Australia, appeared in the NT in mid-August this year on a hobby farm at Humpty Doo near Darwin and was found dead without prior signs of illness on 17 September at 6.00 p.m.

The animal, which was in good nutritional condition, was necropsied late the following morning which meant that post mortem changes were well advanced. The only abnormal findings, apart from the post mortem changes, were an abscess, 3 x 1cm, along the left side of the neck just behind the angle of the jaw and abscesses in the mediastinal lymph nodes and lungs. The mediastinal abscesses were contained within the lymph nodes and varied in size from 1-2cm to 6cm in diameter. One of the abscesses was firmly attached to the aorta at its bifurcation. The lung abscesses were smaller, from mm to 1cm in size, and were present in a group at the dorsal surface of both diaphragmatic lobes. Smaller abscesses may have been present throughout the lung but were not detectable in the autolytic lungs. The pus in all abscesses was greyish to white and of caseous consistency.

B. pseudomallei was isolated from all samples submitted i.e. neck abscess and two mediastinal lymph nodes. Anecdotal wisdom has it that goats and camels are most susceptible to *B. pseudomallei* infection. On the strength of this case we shall have to put alpaca right up there as well.

Western Australia - David Forshaw

Endosulfan toxicityy in fish, John Creeper, Agriculture Western Australia, South Perth

Fish kills were observed in a river near Kununurra adjacent to irrigated horticultural farms. The deaths were cyclical (approximately every 7 days) and appeared to coincide with excavation work associated with clearing of irrigation channels.

There were no gross pathological lesions of note. Histopathological examination revealed the accumulation of eosinophilic staining globules, 2-5 micron in diameter, within the cytoplasm of renal tubular epithelial cells. There were randomly distributed clusters of necrotic and degenerate hepatocytes together with increased numbers of apoptotic cells within the liver. In addition, the livers of several fish contained similar cytoplasmic globules to that seen in the kidney. Within the brain of one fish there was a focal 20 micron diameter irregular malacic area of rarefied neuropil and containing gitter type macrophages centred upon the thalamus.

Chemical analysis revealed there to be highly toxic levels of Endosulfan® insecticide, and traces of PCB's. A diagnosis of Endosulfan Toxicity was made.

Endosulfan® is well documented as being highly toxic to fish and invertebrates. Histopathological changes described in published accounts of toxicity include toxic necrosis of the liver and an inflammatory infiltrate of eosinophilic granule cells within the meninges and cerebrum. The latter changes were not seen in this particular outbreak.

Flexibacter collumnaris Infection in Barramundi fingerlings, John Creeper, Agriculture WA, South Perth

The deaths of all Barramundi fingerlings (total 100,000) being raised in tanks was investigated. Deaths were evenly spread over a three day period and only occurred during a six hour period b the evening. Moderate numbers of Flexibacter were in association with necrosis and inflammation of branchial epithelial cells.

The history provided by the operator was unsatisfactory, however, it was suggested that reduced oxygenation during the evening period was sufficient to cause mortalities in fish with an already compromised respiratory system.

Bovine Non Suppurative Encephalitis, Cleve Main, Agriculture WA, South Perth

Seven animals in a mob of 1-2 year old steers developed severe nervous signs which progressed to hind limb paralysis. No remarkable features were seen at the post mortem examination. However, examination of the brain of two of these cattle revealed mild to moderate non-suppurative encephalitis characterised by the presence of focal gliosis, vasculitis and mild perivascular cuffing. The lesions were distributed throughout the brain and in the cerebral cortex there were also areas of acute multifocal necrosis with many reactive glial cells. The cattle were suspected to have been suffering from sub acute Infectious Bovine Rhinotracheitis Virus infection but attempts to demonstrate the virus in brain material were not successful. No serum samples were available to check for the presence of viral antibody.

Eperythrozoon ovis infection in hoggets, Cleve Main, Agriculture WA, South Perth

Several cases of *E. ovis* induced anaemia have been diagnosed over the past 12 months. In one such case, 20/300 hoggets died and another 50 were reported to be sick. Losses began after mulesing and over the next 4 weeks died in sporadic fashion. Losses increased when the sheep were mustered for crutching. Histopathological examination of the livers of two necropsied sheep revealed paracentral lesions typical of

hypoxic necrosis. Severe spongiform lesions were also present in many white matter tracts in their brains. On another property, weakness and death in 5/180 in a single mob of recently marked lambs was reported. Again highly characteristic brain and liver lesions were seen during histopathological examination. In both cases the diagnosis was confirmed by positive ELISA tests on serum from affected animals.

Disseminated Algal Granulomatosis in Tree Frogs, Cleve Main, Agriculture WA, South Perth

7/15 tree frogs from the Perth Zoo have died. No remarkable lesions were seen at necropsy, but histopathological examination of tissues submitted to the laboratory revealed overwhelming disseminated algal/fungal? infection.

In virtually every organ examined there were varying sized acute to sub acute granulomas, each containing numerous and highly characteristic forms. These were characterised by numerous, often deeply basophilic encapsulated structures approximately 25µ in diameter containing several segments arranged radially to resemble an orange. In addition there were less numerous hyphae-like structures which were sometimes bulb-like and sometimes finger-like. These organisms are even present in the epidermis of the distal digit of one of the limbs.

The identity of the structures are unknown but are reminiscent of *Prototheca* sp. in sections. They are, however, far more numerous, larger and more densely stained.

Chronic Tubular Nephropathy in Stick Nest Rats, Cleve Main, Agriculture WA, South Perth

Several of the colony of Stick Nest Rats from the Perth Zoo have died and histopathological examination of submitted tissues from four rats has revealed chronic moderate to severe multifocal interstitial nephropathy with significant reduction in the thickness of the cortical mass. Less chronic lesions were characterised by multifocal zones of cortical fibrosis and areas of acute tubular necrosis, pseudodilation and minor areas of attempted epithelial repair. More chronic lesions were represented by narrow radial bands of fibrosis which penetrated the deep cortex. Below these areas many tubules were dilated and contained amorphous debris and proteinaceous material.

Oat poisoning in dairy cows, David Forshaw, Agriculture WA, Albany

Severe scouring and acute malaise with a precipitous drop in milk production affected many cows in a dairy herd which were grazing a mixed pasture of oats, clover and ryegrass. The oats were frost damaged, waterlogged and infected with barley yellow dwarf virus which gave them a gross appearance of being droopy with red tips. After removal from the paddock, some affected cows developed mastitis in all four quarters and many took up to a week to recover and even then were producing less milk than before the incident. Dry stock were able to graze the paddock with only mild scouring of some animals but when the milking cows were re-introduced, they again developed severe diarrhoea and illness within 24 hours. Nitrate poisoning and hypomagnesaemia were ruled out.

Samples of rumen fluid from two sick cows revealed a paucity of protozoa and a methylene blue reduction test indicated poor metabolic activity. Both cows were also hypocalcaemic. No microbial cause of the diarrhoea could be identified.

These findings are consistent with "oat poisoning" as reported by Bruce Adams in Bega (Sydney Post Grad Vade Mecum 20 page 164). Little appears to be known about this syndrome although severe disturbance to the rumen microflora is a consistent finding in the case reports I have looked at. Cases have also been seen in Queensland and NSW. A similar disease in South Africa is attributed to *Dreschlera campanulata* a fungus that grows on oats. No *Dreschlera* was seen on oat samples from our case.

Colonic cryptosporidia in a piglet, David Forshaw, Agriculture WA, Albany

Increased mortality in weaners on an extensive farm was attributed to salmonellosis. While investigating this outbreak, a sucker piglet was submitted for necropsy. Mild gaseous distension of the mid jejunum and coarse fibrous material in the colon were seen. There was no scouring.

Microscopically, there was diffuse necrosis of epithelial cells in crypts and superficially in the colon. Adhering to the lumenal surface of many epithelial cells within crypts, there were large numbers of small round basophilic bodies of variable size but no larger than about 5 microns. These bodies were often surrounded by a narrow clear space. The lamina propria was diffusely infiltrated by polymorphs which were passing into the lumen of the crypts. In the most severely affected areas, there was marked dilation of crypts which contained clumps of cellular debris with squamous metaplasia of the lining epithelium. Smears of colonic scrapings contained round bodies which stained with a fluorescent antibody test for cryptosporidium.

Cryptosporidiosis in pigs must be uncommon because it isn't even mentioned in the all new "Pathology of the Pig". From the limited information I have found, it is usually seen in the small intestine but one case report from Kansas also reports organisms in colonic crypts. The Parasitology Department at Murdoch is interested in receiving samples from cases of cryptosporidiosis in all animals for DNA typing. In the first instance, contact Dr Dieter Palmer, Agriculture WA, South Perth on 08 9368 3674.

References

Kennedy GA *et al* (1977) Cryptosporidiosis in three pigs JAVMA **170**(3): 348-350.
Links IL (1982) Cryptosporidial infection of piglets AVJ **58**: 60.

Neuro-axonal degenerative disease in a young Rottweiler with TSE-like pathology, Clive Huxtable, Murdoch University

An eight month old Rottweiler pup, born and raised in outer suburban Perth, was presented with a two month history of worsening tetraparesis and ataxia. Laryngeal paralysis was profound terminally. The animal was very poorly grown and at necropsy weighed only 12Kg. No littermates were reported to be affected. Apart from the underdevelopment the only significant gross finding was pronounced atrophy of the intrinsic laryngeal muscles.

Histologically there was a spectrum of neuropathologic changes which included scrapie-like vacuolation of neurons in several hindbrain locations, multifocal gliosis, Purkinje cell degeneration and loss, multifocal spongiform lesions which appeared to be axonal in origin, and axonal loss and astrogliosis in dorso-lateral and ventro-medial funiculi of the spinal cord.

The findings were very similar to those recently published in Rottweiler pups from Europe and the US in Vetpath **34**: 296 (1997). In these cases no evidence of prion proteins was detected. Tissue from the Perth case has been sent to AAHL and results are awaited.

Green sea turtle diseases, Shane Raidal andMandy O'Hara, Murdoch University

Recent reports between April to July of an increased number of medium-sized green sea turtles found either beached and lethargic or floating and unable to dive in the Exmouth gulf were investigated. In one situation, 10 turtles were found dying or dead by rangers near Onslow. Department of Conservation and Land Management (CALM) rangers acting on these reports submitted five dead or dying turtles for necropsy examination.

Histological sections from five juvenile green sea turtles demonstrated a severe granulomatous vasculitis with intralesional spirorchid trematode eggs affecting the majority of internal organs, major vessels and meninges. In four cases there were scattered microabscesses containing Gram negative bacteria in affected vessels.

In one turtle from Exmouth which had a severe granulomatous meningitis with microabscess formation, culture of the liver, lung and spleen yielded a heavy pure growth of *Salmonella* sp. (Group 0 Type B). Whereas, culture of liver, kidney and spleen from a second case from this area yielded a heavy mixed growth of *Moraxella* sp. and *Citrobacter* freundii. Bacteriological cultures from a turtle found near Karratha yielded a heavy growth of *E*. coli.

Russ Hobbs identified at least seven different species of Spirorchid cardiovascular flukes in several of the turtles. Several species of cestode were present in the gut of some of the turtles necropsied.

The bacteria isolated from the turtles were probably opportunistic environmental organisms because strains of the bacteria isolated normally found in the marine environment have been known to cause opportunistic disease in other marine species.

Cyclic Haematopoiesis and Trapped Neutrophil Syndrome, Jenny Mills, Murdoch University, Ph 08 9360 2646

Reporting a case of a 14 week old Border Collie pup with cyclic haemopoiesis; it was the runt of litter and coat colour was diluted but not grey. Bone marrow at the time of death showed marked cellularity, but with reduced erythroid series. 12 to 14 day cycles of neutropenia (to 0.39 x 10*9/L) were associated with bouts of infections; the first episode resembling parvovirus infection clinically; the second was associated with swollen joints and skin lumps. The pup was perfectly bright in between episodes. A second pup from the same litter (unbeknown to us at the time) also presented with severe leukopenia and clinical signs of hypertrophic osteopathy. It was not closely monitored and died at six months of age. No necropsy was done.

South Australia - Ruth Renter

News & Activities

Veterinary Micro biologist joins Vetlab/VPS Adelaide, Ruth Reuter, VPS, Adelaide

In October Dr Steven McOrist will be joining VPS Adelaide as veterinary pathologist in charge of microbiology. Steve will be known to many ASVP members as a graduate from Melbourne University. In the last eight years he has been working in the UK as a research fellow, consultant pathologist and teacher. We are looking forward to him joining the group in Adelaide.

Pathology Reports

Lysosomal storage disease in a dog, Ruth Reuter, VPS, Adelaide

An 18 month cross-bred Fox Terrier dog was given his first vaccination against canine distemper, hepatitis, parvovirus and parainfluenza with a combined vaccine. He had been active and playful, with a very good appetite up to this time. The day after vaccination he became ataxic, reluctant to eat and trembled continuously. The owners thought he appeared blind. Over the next six weeks he developed weight loss, appeared timid and afraid of people and other dogs. His temperature was normal. Following the second vaccination his condition deteriorated and he was euthanased at the request of the owners.

Significant post mortem findings were restricted to the brain, which was pale and soft on gross examination. The lateral ventricles were dilated with excess clear cerebrospinal fluid. Histopathology demonstrated cytoplasmic vacuoles typical of a lysosomal storage disease in neurons throughout the brain, Kupffer cells in the liver and macrophages in the spleen. The material in the vacuoles was positive on PAS stains.

The changes were consistent with a storage disease such as GM2 gangliosidosis, associated with deficient activity of B-hexosaminidase A. Although this disease is usually identified in purebred animals as an autosomal recessive condition, it has been reported in mixed breed dogs. Clinical signs usually become noticeable around 1.5 years of age. The method of inheritance in mixed breed dogs has not been defined.

Ovine Johne's disease in South Australia, Ruth Reuter, VPS, Adelaide

Ovine Johne's disease is one of the notifiable diseases which has not been previously identified in South Australia. Early in September blood and tissues from a Coopworth ram were received at VPS/VETLAB following a trace forward from a Victorian sheep stud identified as infected. The ram in question had been on the property in South Australia since November 1996. He had spent six weeks in a flock of 150 stud ewes. The remainder of the time he had been in a ram paddock with 12 other animals. The animal was clinically normal with a body condition of Score 3. On post mortem the only visible abnormality was enlargement of a single mesenteric lymph node. A blood sample taken at this time was positive on the Agar Gel Diffusion Test for ovine Johne's disease. Histopathology on multiple sections of lymph node and intestine revealed epithelioid plaques and giant cells typical of *Mycobacterium paratuberculosis* infection. Numerous acid fast bacilli were present in the sections.

Porcine malignant hyperthermia, John Finnie, Veterinary Services Division, IMVS

An experimental, 2-3 month old, Large White Landrace cross pig being induced with halothane anaesthesia became markedly pyrexic within a few minutes of exposure and the muscles were tightly contracted resembling exaggerated rigor mortis. There was purple discolouration of the head, ears and underline and, to a more variable degree, other cutaneous sites. At necropsy, skeletal muscles and myocardium were very pale and the lungs were red, firmer than normal, wet on cut section and failed to collapse completely.

Microscopically, there was an acute myopathy characterised by segmental regions of hypercontraction with bands across fibres traversing a more irregular course, branching and sometimes discontinuous; diminution of striations in many fibres; occasional myolysis; and myofibres separated by abundant oedema fluid. There was also patchy degeneration of cardiac myocytes and pulmonary congestion and oedema.

The muscle pathology and clinical circumstances were consistent with a diagnosis of malignant hyperthermia (porcine stress syndrome). This disorder may occur naturally or be halothane-induced and a mutation in the gene for the calcium release channel of skeletal muscle sarcoplasmic reticulum (ryanodine receptor) has been identified. The excess calcium ions in myofibres are believed to uncouple oxidative phosphorylation leading to ATPO consumption, accelerated glycolysis and lactic acidosis. Malignant hyperpyrexia also occurs in man with mortality rates approaching 40%.

Tasmania - Roy Mason

PCBs and DDT in Tasmanian Platypuses, Barry Munday, Department of Aquaculture, University of Tasmania

As part of an ongoing project at the University of Tasmania to investigate the epidemiology of ulcerative mycosis of platypus, carcases of animals killed accidentally have been collected and specimens taken for a range of purposes.

In collaboration with Professor Anders Sodergren of the University of Lund, fat samples from nine animals have been analysed for PCBs. None have been found to be free of these persistent pollutants and two from northern Tasmania have had levels in excess of 500 ppb. It will be necessary to analyse more samples before coming to any conclusion about possible relationships between PCB levels and the occurrence of ulcerative mycosis, but it is interesting that the two high values were from animals from the West Tamar where the disease has only been seen in recent years.

We would be very interested to learn of any analytical data for platypuses from mainland areas. Also we would be willing to arrange analysis of fat samples from mainland animals. Please contact Barry Munday at the Department of Aquaculture, University of Tasmania, PO Box 1214, Launceston, Tas 7250, Ph 03 632 3804, e-mail Barry.Munday@utas.edu.au.