President’s Corner

This brief message constitutes the last of my “President Corner” contributions to the Supplement to the JWD. Two years ago I was preparing to attend our 50th Annual Conference, held in Pilanesberg National Park, South Africa. There, I would assume the duties as the 23rd president of WDA. I was, admittedly, a bit naïve as to the magnitude of responsibility assumed by those privileged to represent WDA members as their president!

Nearly two years later, I can say that I, and our Association, have been blessed with a wonderful cadre of hard working, dedicated, officers, Council members, committee chairs and members, business and financial advisors, as well as the WDA membership at large. From dissemination of the landmark Pilanesberg Resolution, the welcoming of our new African Section, the continued refinement of our exceptional Journal, to the conduct of two outstanding annual conferences in Arcata, CA and Saskatoon, SK, there has been constant activity and positive results.

I have been especially grateful for the leadership, guidance, talent, and collegiality demonstrated by your WDA Council members and committee chairs. In addition to the regular meetings of Council at our annual conferences, we have also met, via phone conference, approximately every 4 months, in an effort to more efficiently conduct the business of your Association.

Of particular importance was the action taken by Council, at our meeting in 2002 at Humboldt State University, to support the implementation of significant increases in membership fees. We all recognized both the need for such an initiative, as well as the fact that there were associated risks. Nearly a year later, that action has proven to have been a sound one and of great benefit to the continued fiscal stability of WDA. Our increased income has provided the financial foundation necessary for the hiring of an Executive Manager, the first such hiring to take place in our 52-year history. Additionally, the increased funds will enable WDA to enhance the Journal publication process and engage in other initiatives destined to greatly benefit our members and the Association at large. It is anticipated that by the time you read this message we will have the new Executive Manager on board. That person will be invaluable to Council, and the membership, by providing administrative and operational assistance, as well as ensuring organizational continuity as WDA moves forward with its structured cycling of officers, council members, and committee participants.

It has truly been an honor for me to serve the WDA these past two years, just as it has been my privilege to be a part of this highly professional association over the past 30 years. Our mission has never been more relevant, our challenges never more daunting, and our prospects for the future never more exciting! The visibility of wildlife, and the wildlife/human disease interface, seems to increase on a monthly basis. Most recently, the public has been deluged with stories relating to WNV, CWD, SARS, and monkeypox. What follows is anyone’s guess. What is certain, however, is that the WDA, and its talented and dedicated members, will be involved in discovering, researching, and solving a host of wildlife disease issues for years and years to come!
Again, I offer my sincerest appreciation for the encouragement and support provided to me and our Association during these past two years.
—Paul L. Barrows, WDA President

WDA ACTIVITIES

52nd Annual Meeting of the Wildlife Disease Association. August 11–14, 2003; Saskatoon, Saskatchewan, Canada. By this printing, the 52nd annual scientific meeting of the Wildlife Disease Association will have taken place in Saskatoon, SK on August 11th to 14th, 2003. This international meeting serves as a venue for presentations and posters on all aspects of wild animal diseases world-wide. It is an excellent opportunity for researchers, both professionals and students alike, to present their work, share their knowledge and meet fellow wildlife disease enthusiasts in a friendly and collegial atmosphere. This year, the conference was hosted by the Canadian Cooperative Wildlife Health Centre. Stay tuned for details of the Saskatoon meeting in the October issue of the Supplement.

WDA STUDENT ACTIVITIES

ATTENTION MENTORS AND ADVISORS! Please encourage your students to participate in next year’s student activities! The deadline for scholarships is APRIL 15, 2004. It’s never too early to start preparing. San Diego 2004 is just around the corner! Please see brief descriptions of scholarship and award opportunities below. Specific instructions will be available in upcoming issues of the Supplement.

Wildlife Disease Graduate Student Research Recognition Award DEADLINE: April 15, 2004. This award is given to the student judged to have the best research project in the field of wildlife disease, based on written communication and scientific achievement. The winner receives a plaque and up to $1000 US to cover travel, housing, registration, etc. related to the annual conference. The student will be the featured presenter during the Student Presentation Session at the conference.

Wildlife Disease Association Scholarship DEADLINE: April 15, 2004. This scholarship acknowledges outstanding academic and research accomplishment, commitment, and potential in pursuit of new knowledge in wildlife disease or health. The scholarship has a value of $2000 US and is awarded annually to an outstanding student who is pursuing a master’s or doctoral degree specializing in research on wildlife disease. To be considered, the candidate must have completed a four-year baccalaureate degree. Candidates with an overall grade point average of 3.5 or above in 4.0 system or 80% or better in percentage system will receive priority. The candidate should be committed to leadership, scholarship, and service in the wildlife health profession.

Terry Amundsen Student Presentation Award ABSTRACT DEADLINE: May 1, 2004. This award acknowledges outstanding oral presentation of research findings. Winner receives $250.00 and a plaque. To be considered, the student must give an oral presentation (13–15 min) of his/her topic of choice to the WDA meeting participants in a special session. Upon completion of the presentations, evaluation forms will be handed out to the audience who will be asked to score the presentations.

HAPPENINGS IN THE FIELD

Anti-inflammatory drug implicated in vulture die-off in India. The mystery of the vulture mortalities now appears to be solved. Instead of a disease it is a widely used pharmaceutical that is exceptionally toxic to Asian white-backed vultures. At the Sixth World Conference on Birds of Prey, Dr. Lindsay Oaks of Washington State University, whose work in Pakistan has been supported by the Peregrine Fund, reported on results obtained in May 2003. Like Andrew Cunningham of the Zoological Society of London and the Royal Society for the Protection of Birds, he had been obtaining only negative results in a search for a virus or other disease pathogen. Tests for pesticides, metals, and other poisons continued to be negative. He then made inquiries about what kinds of medicines were being given to local cattle and buffaloes. One of them contained Diclofenac, which is a very widely used pain killer and anti-inflammatory drug for people. In both Pakistan and India there are a number of products containing it that are used by veterinarians in the treatment of sick cattle and buffalo. Tissues of 23 vultures that had died with gout symptoms, which have been characteristic of dying vultures throughout India and in Pakistan, all contained Diclofenac; none was present in tissues of vultures that had died of other causes. Three vultures given very small amounts all died within a short time. In another experiment tissues from a dead vulture were fed to other vultures. All birds died including however, the controls. This was temporarily an awkward
situation but it turns out that buffalo meat fed to all of the vultures contained Diclofenac. Other experiments are in progress. The preparations are used to treat sick cattle and buffaloes. If they die and the carcasses are disposed of in the conventional manner by leaving them out for vultures, the persistence of Diclofenac is evidently sufficient to be taken up by vultures. The doses given to the three vultures that died were 2.5 and 0.25 mg/kg (parts per million). The LD50 would appear to be lower. This morning I did a brief computer search with the keywords Diclofenac, India, and veterinary. There were about 250 responses; it is present in a number of medicines made in India for cattle and buffalo. Dr. Oaks has said that it is not used in America or Europe for livestock; the amounts used by people, however, are immense such that it has been detected in surface waters. Are any pharmaceuticals being given to cattle that might end up in condors? This is a topic we should know something about.

—Bob Risebrough, Budapest, 21 May 2003 Sixth World Conference on Birds of Prey

First recorded outbreak of monkeypox in the United States. On June 7, 2003, public health officials from the Centers for Disease Control and Prevention (CDC) and the states of Wisconsin, Illinois and Indiana reported the first outbreak of human infections with monkeypox virus to be documented in the Western Hemisphere. As of July 2, 2003, a total of 81 cases of monkeypox has been reported by six states to CDC since early May. These are from Wisconsin (39), Indiana (22), Illinois (16), Missouri (2), Kansas (one), and Ohio (one), and include 32 (40 percent) cases laboratory-confirmed at CDC and 49 (60 percent) suspect and probable cases under investigation. The majority of patients with confirmed monkeypox reported exposure to wild or exotic mammals, including prairie dogs; some patients also had contact with other persons with monkeypox virus infection in a household setting. No cases of monkeypox that could be attributed exclusively to person-to-person contact have been confirmed. CDC and state and local health departments continue to investigate cases of monkeypox among persons who had contact with wild or exotic mammalian pets or persons with monkeypox. Traceback investigations have implicated a shipment of animals from Ghana that was imported to Texas on April 9, 2003 as the probable source of introduction of monkeypox virus into the United States (1,2). The shipment contained approximately 800 small mammals of 9 different species, including 6 genera of African rodents. These rodent genera included rope squirrels (Funisciurus sp.), tree squirrels (Heliosciurus sp.), Gambian giant rats (Cricetomys sp.), brushtail porcupines (Atherurus sp.), dormice (Graphiurus sp.), and striped mice (Hybomys sp.). Gambian rats from this shipment were kept in close proximity to prairie dogs at an Illinois animal vendor implicated in the sale of infected prairie dogs. CDC laboratory testing of some animals by using PCR and virus isolation demonstrated that one Gambian giant rat, 3 dormice, and 2 rope squirrels from the April 9 importation were infected with monkeypox virus. Evaluation of other animals associated with the shipment is ongoing. Evidence of infection was found in some animals that had been separated from the rest of the shipment on the day of their arrival into the United States, indicating early and possibly widespread infection among the remaining animals in the shipment. CDC had recommended previously that state health officials place quarantines on commercial facilities or households that had infected animals or received African rodents from the April 9 shipment (1). CDC has issued guidance on the quarantine and euthanasia of all rodents from the shipment, as well as prairie dogs that were exposed to the imported rodents or other animals with illnesses consistent with the case definition for monkeypox. (http://www.cdc.gov/ncidod/monkeypox/quarantineremoval.htm.) CDC and the Food and Drug Administration issued a joint order on June 11, 2003 prohibiting the importation of any African rodent. In addition, the order prohibits the sale and transport within the United States of prairie dogs and 6 genera of African rodents (http://www.cdc.gov/ncidod/monkeypox/pdf/embargo.pdf). To prevent the spread of monkeypox virus into domestic or wild animal populations, the order also prohibits releasing any of these animals into the wild. State and local health departments or departments of agriculture should be consulted for guidance on the safe disposal of animals. The joint order remains in effect regardless of the actions related to the guidance for quarantine and euthanasia of animals of concern. Health-care providers, veterinarians, and public-health officials who suspect monkeypox in animals or humans should report such cases to their state and local health departments. State health departments should report suspect cases to CDC, telephone 770-488-7100. An updated case definition with revised case exclusion criteria is available: http://www.cdc.gov/ncidod/monkeypox/index.htm. Clinical specimens should be submitted for testing after consultation with the state and local health department. Interpretation of laboratory results requires completion of specimen submission forms, which are available at http://www.cdc.gov/ncidod/monkeypox/diagspecimens.htm. Reported by: State and local health departments. Monkeypox investigation team, CDC.

References
—Adapted from ProMED-AHEAD Digest, July 2, 2003 Volume 2003: Number 123
National Wildlife Health Center Quarterly Mortality Report

Avian Cholera in Northern California Wintering Waterfowl. From mid February to mid April 2003, U.S. Fish and Wildlife Service personnel collected carcasses of 4,759 waterfowl at Lower Klamath and Tule Lake National Wildlife Refuges in California on border of Oregon. The total losses are estimated at 9,000. The predominant species affected in order of mortality were Ruddy ducks, Snow geese, Ross’ goose, American wigeon, Northern pintail, White-fronted geese, and American coots. Populations of geese and ducks have been increasing with significant expansion of quality waterfowl habitat on and off the Refuges in California. The impact of avian cholera was greater in the 1980’s when waterfowl populations were lower and less quality habitat was available.

West Nile Virus Returns in 2003. As of April 1, 2003, WNV was detected in birds in Georgia, Louisiana, Mississippi and Florida. In addition, WNV antibodies were detected in resident, non-migratory birds in Mexico, Dominican Republic, El Salvador and Jamaica. WNV was diagnosed in horses in El Salvador. This southern progression of WNV further supports the hypothesis that migratory birds transmit the virus. Given the rapid progression of WNV to 44 states in 2002, it is anticipated that WNV will occur in the 48 continental states and possibly AK and HI during 2003. The 2002 WNV epidemic was the largest ever recorded in the United States. In their final report for 2002, The Centers for Disease Control and Prevention report 4,156 human cases with 284 deaths, almost 15,000 horse cases, and thousands of WNV positive birds. West Nile virus was confirmed in 44 states, the District of Columbia, five Canadian Provinces (Saskatchewan, Manitoba, Ontario, Quebec and Nova Scotia), and northeastern Mexico reported WNV activity in 2002.

High Mortality Threatens the Survival of the Mississippi Gopher Frogs. In March 2003, mortality of gopher frog tadpoles was detected in Mississippi at the only known breeding site for this species in the United States. Gopher frogs live in burrows of gopher tortoises, which have been released in the same area in Mississippi. The September 2002 cohort of gopher frogs has died. The majority of tadpoles from the February breeding cohort have been taken into captivity. A protozoan-like organism is responsible for the larval mortality. During a field investigation by the NWHC, amphibians were collected from three potential release sites for gopher frog tadpoles if there is a successful breeding attempt by the estimated 150 remaining adult gopher frogs in the fall of 2003 or 2004.

Songbird Mortality in Texas. Texas Parks and Wildlife Department reported a die-off of thousands of American Robins and a smaller number of Cedar Waxwings from an area that extended from Dallas to San Antonio to Goliad, Texas. One of the largest areas of mortality was Waco, which reported several hundred dead birds. All diagnostic tests conducted at Texas A & M and other state laboratories were inconclusive and pesticide exposure was ruled out. Speculation as to the cause of death includes alcohol toxicity following ingestion of fermented berries from exotic bushes planted in the area.

Ruddy Ducks Succumb to the cold in Maryland. During January 2003, low temperatures and extensive ice coverage in the Chesapeake Bay may have contributed to the starvation of an estimated 2,000 Ruddy ducks and other wintering waterfowl. Birds, submitted to the Maryland Department of Agriculture Diagnostic Laboratory and the NWHC, were emaciated with empty stomachs and all cultures were negative. Avian cholera was ruled out in these birds.

QUARTERLY WILDLIFE MORTALITY REPORT
January 2003 to March 2003

<table>
<thead>
<tr>
<th>State</th>
<th>Location</th>
<th>Dates</th>
<th>Species</th>
<th>Mortality</th>
<th>Diagnosis</th>
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<td>Palmer Station</td>
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<td>Adelie Penquin</td>
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<td>Open</td>
<td>NW</td>
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<td>Lower Klamath and Tule Lake NWR</td>
<td>02/17/03-04/21/03</td>
<td>Ruddy Duck (Aleutian)</td>
<td>4,759</td>
<td>Avian cholera</td>
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<td>Volusia, Orange, Brevard, Martin, Palm Beach and Broward Counties</td>
<td>03/03/03-ongoing</td>
<td>Northern Gannet</td>
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<td>Emaciation</td>
<td>FL, NW</td>
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<tr>
<td>State</td>
<td>Location</td>
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<td>Species</td>
<td>Mortality</td>
<td>Diagnosis</td>
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<tr>
<td>GA</td>
<td>Lincoln County</td>
<td>12/29/02-01/20/03</td>
<td>Bald Eagle</td>
<td>3 (e)</td>
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<td>01/08/03-ongoing</td>
<td>American Crow, Blue Jay, Common Grackle, Northern Cardinal, American Goldfinch</td>
<td>35 (e)</td>
<td>Viral infection: West Nile</td>
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<td>DeSoto NWR</td>
<td>12/13/02-01/03/03</td>
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<td>Trauma</td>
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<td>Talbot County</td>
<td>01/25/03-03/01/03</td>
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<td>MD, NW</td>
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<td>ME</td>
<td>Villa Gonzalez and Villa de Casas</td>
<td>12/14/02-01/20/03</td>
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<td>MS</td>
<td>Harrison County</td>
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<td>Mississippi Gopher Frog, Southern Leopard Frog, Mallard</td>
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<td>Parasitism: protozoa-like</td>
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<td>NC</td>
<td>Halifax County</td>
<td>02/01/03-04/15/03</td>
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<td>Toxicosis: organophosphorus cmpd.</td>
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<td>Scotts Bluff County</td>
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<td>Hooded Merganser, Common Golden-eye, Green-winged Teal Mallard</td>
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<td>Maxwell NWR</td>
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<td>OK</td>
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<td>Erie County, Presque Isle St. Park</td>
<td>03/15/03-03/19/03</td>
<td>Killdeer, Snapping Turtle</td>
<td>75 (e)</td>
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<td>PR</td>
<td>Isla Mona</td>
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<td>Blount County</td>
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<td>Nueces County</td>
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<td>Oldham County</td>
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<td>37 (e)</td>
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<td>Bailey County</td>
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<td>Surry County</td>
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<td>WA</td>
<td>Yakima County</td>
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<td>Milwaukee County</td>
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<td>Lesser Sculp</td>
<td>250 (e)</td>
<td>Parasitism: trematodiasis</td>
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**Updates/Corrections**

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<tr>
<th>State</th>
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QUARTERLY WILDLIFE MORTALITY REPORT
January 2003 to March 2003
Continued

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<td>Bald Eagle</td>
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<td>Lead poisoning</td>
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</table>

(e) = estimate; * = morbidity, not mortality; Ψ = No specimens received in laboratory setting

New York State Dept. of Environmental Conservation (NY), Southeastern Cooperative Wildlife Disease Study (SCW), USGS National Wildlife Health Center (NW), Wisconsin Dept. of Natural Resources (WI), National Wildlife Refuge (NWR), Texas Parks and Wildlife Commission (TX), North Carolina State University Veterinary School (NCA), Maryland Department of Agriculture Diagnostic Laboratory (MD), Florida Fish and Wildlife Conservation Commission (FL), Washington Department of Fish and Wildlife (WAS), State Diagnostic Laboratories (ST).

Written and compiled by Kathryn Converse/Rex Sohn - Western US, Grace McLaughlin - Eastern US, Christine Lemanski, NWHC. The Quarterly Wildlife Mortality Report is available at http://www.nwhc.usgs.gov. To report mortality or receive information about this report, contact the above NWHC staff, or for Hawaiian Islands contact Thierry Work. Phone: (608) 270-2400, FAX: (608) 270-2415 or e-mail: kathy.converse@usgs.gov. USGS National Wildlife Health Center, 6006 Schroeder Road, Madison, WI 53711.

WDA SECTION NEWS

NEWS FROM EUROPE

European Section

Surveillance of Wildlife Diseases on the European Scale. M. ARTOIS1, T. MÖRNER2, P. DUFF3 and D. SIBARTEE4. In recent decades the movement and translocation of wildlife, including zoo animals, domestic and free-ranging wildlife, has increased worldwide. To reduce the risk of introducing new diseases into new areas and animal populations, significant diseases of wildlife in different parts of the world have been reported annually to a permanent Working Group of the OIE. The OIE, Office International des Epizooties or World Animal Health Organisation, is an intergovernmental international organisation based in Paris, France. Its main duty is to secure international trade in animals and their products by harmonisation of regulations and by informing the OIE Delegate of each Member Country (often the Chief Veterinary Officer of the country) of outbreaks of disease. In 1992, on the initiative of its former Director General, Dr Jean BLANCOU, an ad hoc group of experts on diseases of wildlife was created. This group has sent a questionnaire to all OIE Delegates asking them to report on the main diseases that were occurring in their country.

Animal diseases are listed by the OIE according to their seriousness and contagiousness. Among list A diseases, classical swine fever (CSF) in European wild boar, foot and mouth disease (FMD) in hoofed mammals, and paramyxovirus (PMV1) serological traces were regularly reported. Among list B diseases, bovine tuberculosis (TB) and rabies are also reported frequently in a wide range of species across Europe. The Working Group has also established a ‘Wildlife list’ including diseases that cause, or can cause, serious problems for wildlife conservation. It is noteworthy that in Europe, wild boar CSF and carnivore rabies still cause significant morbidity, as does sarcoptic mange in many species.

The reporting of wildlife diseases in Europe is mainly based on a collection of data carried out by members of the European section of the Wildlife Disease Association (EWDA), through a network of contacts in each country. In 2002 information has been collected from 24 countries. The individual country reports are increasingly detailed with referencing of incidents and comment on significant incidents. Important events this year included the Phocine Distemper virus outbreak in Common Seals in the North and Baltic seas and the mass mortality of Schreiber’s long fingered bat (Miniopterus schreibersi), from an, as yet, unknown cause. Wildlife disease may also be important for preservation of game resources and an example of this was a previously un-reported condition affecting the nervous system and skin of Pyrenean chamois, again of unknown cause.

‘Emerging diseases’ in wildlife, for example, West Nile Fever and Lyme borreliosis are increasingly recognized by statutory authorities across Europe as being of growing importance. Surveillance for these wildlife diseases increases our global knowledge and allows comparisons of the monitoring and control

1 Ecole Nationale Vétérinaire de Lyon, France
2 The national veterinary institute, Uppsala, Sweden
3 Veterinary Laboratories Agency, Merrythought, Penrith, Cumbria, UK
4 Office International des Epizooties, Paris
methods across Europe. The EWDA and this Report both seek to promote that important study, and we thank all contributors and collaborators.

The efficiency of the annual report of the Working Group has been steadily improved. Any problems that remain are associated with a bias in the countries providing data or with difficulties either in reporting sensitive diseases or assessing the efficacy of control methods: FMD and TB are especially difficult in this respect. Most of the data gathered through the reporting system are relayed to the OIE Delegates through the annual report, which is also sent to all contributors.

Acknowledgements: authors wish to express their gratitude to all those who have contributed to the annual EWDA report to OIE. The detailed list of contributors is given in full extent in the final version of the report (available on the web site: http://www.ewda.org/)

Material for publication in News from Europe can include recent wildlife disease outbreaks and new diseases in Europe, short case and meeting reports; job and scholarship announcements. We encourage submissions, and will help with the English language, if required. The deadline for the next issue is August 2003. Please mail, fax or e-mail submissions to, Paul Duff, VLA Penrith, Merrythought, Calthwaite, PENRITH, Cumbria, CA11 9RR, United Kingdom, e-mail p.duff@vla.maff.gsi.gov.uk; Fax +44(0)-1768-885314 /phone +44(0)-1768-885295.

Nordic Section

Meeting of the Nordic Section of the WDA

The 2003 biannual meeting of the Nordic section of WDA was, as tradition demands, held on an island in May this year. This time we were on Hallo, which means shark reef, in the northernmost part of the Baltic Sea and Finland. The Finnish hosts heroically organized a three-day event at rather short notice, gathering about 20 participants from Sweden, Finland and Denmark. Norway was unfortunately not represented, due to their National Day celebrations on May 17th, when it is impossible to get babysitters. But the Norwegians did have a presentation, albeit a virtual one. The scientific program included, as usual, a report from each country on the highlights of wildlife diseases throughout the past year. The other presentations covered various topics such as the trichinosis situation in Finland, predator mortality in Sweden, the ongoing sea-bird mortality in Sweden, the latest phocine distemper outbreak in Denmark, echinococcus research in Finland, and a half-day of reindeer disease and general reindeer biology. The field trip day was clear and sunny after the previous day of rain, and visits were made to a lesser white-fronted goose (Anser erythropus) farm, a local reindeer project aimed at clearing shrubs from shore-lining marshlands, and a bird-watching session with mainly water fowl seen. At the general meeting a partly new board was elected, with Erik Ågren, Sweden, as new president, Henrik Uhlhorn, Sweden, as secretary, Torsten Mörner, Sweden, was re-elected treasurer, and the national representatives were Antti Oksanen for Finland, Hans-Henrik Dietz for Denmark, and Kjell Handeland for Norway. The next meeting of the Nordic section will be held in Sweden, on the island of Öland, one of the best sites in Sweden and the Baltic for watching migratory birds. For further information please contact: Erik Ågren, Department of Wildlife, National Veterinary Institute, 751 89 Uppsala, SWEDEN, FAX +46 18 30 91 62 or E-mail: Erik.Agren@sva.se

—Torsten Mörner, DVM, PhD, Senior Veterinary Officer, Associate Professor, Department of Wildlife, National Veterinary Institute, Uppsala, Sweden, Phone +46 18 67 42 14 or +46 70 5679 352 (where you can leave messages), FAX +46 18 30 91 62, E-mail Torsten.Morner@sva.se

AUSTRALASIAN SECTION

2002/2003 Committee

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Treasurer: Jenny Youl, University Av. Vet Hospital, 66 University Avenue, Durack, NT 0830, Ph (08) 8931-0455, Fax (08) 8931-0466, Jenny.youl@hotmail.com

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State Representatives: Tasmania—Philip Ladds; Western Australia—Phillip Clark; South Australia—Sue Bigwood; Queensland—Vere Nicholson; New South Wales—Julie Barnes; Victoria—Kate Bodley; New Zealand—Joanne Connolly; ACT—Chris Bunn; Northern Territory—Jenny Youl
President’s Message: This year’s WDA meeting will be held in scenic Healesville in Victoria which, at various times in the past, has been the Garden State, On The Move and The Place To Be. In December the latter of the three will definitely be the case. Electronic and hard copies of the conference flyer have been mailed out. The registration fee covers everything except accommodation. The Badger Creek Caravan Park has a host of accommodation options available and should be contacted directly to book your preferred domicile for the week. The theme will be Australian wildlife: a healthy future? Papers that focus on this area are strongly encouraged, but don’t be shy if your amazing discovery doesn’t fit the bill. We encourage all comers to share their knowledge and expertise. Shirley will once again be on hand to perform feats of culinary excellence. Local biologists will provide insights into the area’s unique fauna, hopefully leading to closer collaborative links between all those associated with wildlife health. As we all know health and disease are multi factorial processes involving more than just veterinarians. We hope that this conference will be the first step in bringing all involved groups closer together to improve our understanding of wildlife health (and perhaps indulge in a local wine or two).

Despite the rural feel Healesville is only an hour’s drive from the middle of Melbourne. Those wishing to extend their stay can enjoy the scenery of Kinglake National Park, the Black Spur and Acheron Way. For the more culinary minded there are a host of local wineries and eateries. I encourage everyone to visit for some pre-Christmas stimulation, of the intellectual kind. An omission from the registration form is that the fee for bona fide students is $350, accompanying spouses $300 and kids $150. This will cover all food, Sanctuary admission and assorted field trips.

If you haven’t yet signed up for 2003 it’s never too late to do so. The parent body reports a 78.6% renewal rate. By comparison Jenny informs me that our membership renewals are currently running at a very pathetic 38%!! Without fully paid up members the WDA will simply cease to function. Funds will be exhausted and the organisation will no longer be able to support the Wildlife Pathology Registry, provide money for research projects, subsidise conferences or support the Australian Wildlife Health Network. It is unfair for the 38% to support the 62%. It’s not really all that complicated. When you fill out your form to renew your licence to practise veterinary medicine (for those of you who are vets) at the end of the year you also set $40 aside to go to the WDA. Pretty miserable amount really considering the amount you fork out for registration, AVA membership, movies, nights on the town, etc. Half the year has already been and gone. There’s really no excuse not to renew your WDA registration.

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Australian Wildlife Health Network—Quarterly Report April 2003

The Animal Health Committee has met and endorsed the Network’s approach to general wildlife surveillance. A system complementing the NAHIS will be used. The first teleconference of co-ordinators, assistants and representatives from Animal Health Australia was scheduled for May 20th to discuss modus operandi. Reporting will focus on six disease categories: 1) OIE list diseases; 2) bat viral diseases; 3) mass, or unusual mortality events; 4) Salmonella cases; 5) Arbovirus cases; 6) diseases State/Territory co-ordinators think are unusual or interesting.

Animal Health Committee has also requested that the Network be involved in developing a protocol for wildlife sample submission. The protocol will be based on those currently used by the Regional Veterinary Laboratories for livestock sample submission.

Free ranging wild animals

Mortalities of threadfin leather jacket fish (Artropleps filicauda), Fraser Island, Queensland. Two incidents reported over a six-day period in early March. The first involving up to an estimated 300,000 animals washed up along the entire east coast of Fraser Island, the second an estimated 100,000 animals centred on the north-eastern beaches of the island. Cause of death unknown.

Deaths of up to 30 eastern grey kangaroos (Macropus giganteus) at Yarrambat (near Yan Yean), Victoria over a two-week period: under investigation.

Reports of an emerging proliferative skin disease in Tasmanian devils (Sarcophilus harrisii) in Tasmania. A structured investigation has commenced as a model for improving Tasmania’s capacity for prevention, preparedness, and response to emerging infectious disease threats in wildlife.

Investigation into deaths of koalas (Phascolarctos cinereus) (n = 26) over a five week period at Raymond Island (SSE of Bairnsdale, Victoria). No further mortalities. Investigation stood down. Cause of death unknown.

Australian Bat Lyssavirus Category 3 exclusions (where a person had been bitten or scratched) (n = 3, NSW), grey-headed flying-fox (Pteropus poliocephalus; n = 1, NSW), black flying-fox (Pteropus alecto; n = 2, NSW), eastern forest bat

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Captive wild animals

An acute mortality event (n = 6) in a private aviary, of mixed psittacine species, in South Australia currently under investigation. Hepatic intranuclear inclusions observed in tissues from one individual. Samples were sent overseas to rule out Pacheco’s disease. Preliminary results by DNA hybridisation: negative.

The Network is interested in receiving reports of wildlife incidents, and definitive diagnoses of causes of death in wildlife in Australia. A list of the “ten least wanted” diseases of wildlife, to be based on Biosecurity Australia’s Import Risk Analyses is being prepared for comment.

—Contributed by: Chris Bunn, Office of the Chief Veterinary Officer, AFFA, and Rupe Woods, AWHN Coordinator. The Network acknowledges those who submitted information to the Network and to Dr Karrie Rose (Australian Registry of Wildlife Health) who allowed access to the Registry database.

Mortalities, interesting cases, and oil spills

The following are some interesting cases that have passed through the Registry recently. These reports originate from free-ranging animals, and native fauna held in a variety of zoos, fauna parks and private collections.

December 2002

NZ Fur seal—wild—NSW—emaciated fur seal, update from previous report. The fur seal was found to have a very large gastric lymph node with extensive caseous necrosis and foci of mineralisation. Although mycobacteriosis was suspected on gross post mortem examination, histopathology revealed disseminated fungal infection. The fungus identified within the organs had a very unusual morphology and failed to sporulate in culture. Two mycology reference labs were unable to identify the fungus. PCR identified the fungus as Aspergillus fumigatus (3014.1).

A veterinary practitioner contacted the Registry to ask if it was possible to use the chicken Newcastle Disease Virus to protect snakes from Ophidian Paramyxovirus infection. The veterinarian was advised to contact NSW Agriculture, since Ophidian Paramyxovirus is considered to be exotic to Australia. We subsequently examined one death adder thought to be infected with the virus, and assisted NSW Agriculture in the investigation by reviewing numerous suspect cases. Several anaerobic bacteria were identified within the lung tissue of the adder and no viruses were isolated in culture. Thus, the snake was most likely suffering from a bacterial infection in the respiratory tract (3200.1). None of the snakes examined had respiratory lesions characteristic for Ophidian Paramyxovirus infection, such as marked epithelial hyperplasia, formation of syncytia, and cytoplasmic or intranuclear inclusion bodies in respiratory epithelium. However, we may have received snakes that died after an initial outbreak that was reported to have occurred in October. NSW Agriculture is pursuing serological studies to further rule out the presence of Ophidian Paramyxovirus in the captive collection (3200.1).

Green turtle—NSW, NPWS—Two green sea turtles being cared for at Sydney Aquarium from the outbreak of coccidiosis in October. Euthanasia elected due to ongoing neurological dysfunction. Each animal had severe lesions in the brain, thyroid glands, kidneys, and intestinal tracts associated with the single celled parasite Caryospora cheloniae. Through the investigation of this outbreak we have better characterised the disease caused by this parasite and have identified a megaloschizont stage of the lifecycle. (3226.2, 3227.2). During and shortly after the epizootic, a total of 13 green turtles, and four hawksbill turtles (Eretmochelys imbricata) were subject to gross and microscopic post mortem examination. Eleven of the subadult and adult green turtles had systemic coccidiosis. Affected turtles ranged between 28.4 and 105 kg, with straight carapace lengths ranging between 615 and 940 mm. These animals were feeding on sea grass beds in estuaries along coastal NSW, particularly in Port Stephens. Concurrently, a mon bentwing bat (Miniopterus schreibersii; n = 1, ACT), yellow-bellied heathtail bat (Saccolaimus flaviventris; n = 1, NSW). Category 2 investigations (species not reported; n = 3, NSW) all negative.

Long-nosed bandicoot—captive, NSW—Multiple lung abscesses—atypical mycobacteriosis (3235.1). (Qld reference lab—Mycobacterium sp. Slow Grower.

Sealy-breasted lorikeet—captive, NSW—chronic hepatitis with many intracellular bacteria. E. coli isolated in pure culture—quite unusual for this bacterium to cause chronic liver disease. (3201.1).

Bar-tailed Godwit—suspected tick toxicity (3236.1)

Brush-tail possum—wild, NSW—burned in bushfires. Multiple ulcers and areas of avascular necrosis of the skin, marked dehydration, and secondary bacterial infection (Salmonella sp.) (3237.1).
Eastern long-neck turtle—VIC—died due to dog attack, but had a severe, underlying inflammatory disease in the large intestine (multiple extensive granulomata throughout the colon wall (3329.1).

Little forest bat—VIC—euthanased due to trauma. Incidental finding of mites in the sinus cavity—reported to be common (3212.1).

Rainbow boas—SA—two small boas confiscated by customs and euthanased. One snake had ulcers associated with single celled parasites in the intestinal tract. This animal also had some unusual inclusion bodies in the kidney. We have seen several boas and pythons with large nuclei in the renal tubular epithelium. Some of these nuclei have peripheralised chromatin and contain amorphophilic “polymy-like” inclusion bodies. A DNA hybridization test to detect avian polyoma-virus was negative and unfortunately there are usually too few inclusions to chase with electron microscopy.

January 2003

Bilby—captive, NSW—euthanased due to weight loss, dental attrition, the presence of a large, infected ulcer in the groin region containing the bacteria Morganella morgani and recent neurological deficits. The bilby had a systemic protozoal infection, which is morphologically consistent with sarcocystosis. Immunohistochemistry would be required to further confirm the identity of the organism. Most notably were protozoal zoites escaping from cysts within cardiac myocytes, and necrosis within the adrenal gland associated with large numbers of zoites. Smaller numbers of zoites were evident within small foci of necrosis and non-suppurative inflammation in the liver and pancreas (3317.1).

Short-beaked echidna—rehabilitation, NSW—Euthanasia due to ongoing weight loss and marked bilateral conjunctivitis that had resulted in both eyes prolapsing. The animal’s weight loss was a result of a combined infection with systemic coccidiosis and Salmonella bovismorbiﬁcans. We are noticing a trend, that echidnas dying with systemic coccidiosis have concurrent Salmonella septicaemia. This animal had coccidial gametocytes and oocysts within the conjunctiva, in addition to zoites in many of the parenchymatous organs (3260.1).

New Zealand fur seal—wild animal that had been attacked by a shark, and then had been in quarantine. Biopsies from raised plaques in the oral cavity were morphologically consistent with papillomas (warts). The lesions may have resulted from the presence of a papilloma virus, or closely related virus. Papilloma virus has been reported to cause similar lesions in cetaceans and pinnipeds, but not NZ fur seals. Attempts will be made to conﬁrm the presence of this virus. Papilloma viruses do not grow well in culture and immunohistochemistry will be required to conﬁrm the diagnosis. Papilloma virus infections could be transmissible to other pinnipeds, but infections are usually self-limiting, and generally only occur in immunosuppressed animals (3254.1). The lesions were debrided on several occasions in the hopes that the procedure would act similar to an autogenous vaccine.

Green and golden bell frog—captive, NSW, bacterial infection in the braincase and sinuses, Mycobacterium sp., and probably other bacteria. (3253.1).

Green turtle—NSW, NPWS—young turtle markedly constipated due to the presence of large strings of plastic throughout the large intestine (3320.1).

Centralian Carpet Python—NSW—Euthanased due to chronic respiratory illness and the identiﬁcation of acid-fast bacteria in a lung wash. The animal had many granulomata throughout the lung, and smaller granulomata throughout the liver and kidney, caused by Mycobacterium abscessus (3261.1).

February 2003

Green and golden bell frog—captive, NSW—euthanased after being found thin and with a kinky back. The frog had multiple granulomata along the spinal ganglia and in the liver—Stenotrophomonas maltophilia (liver and spine lesions), Enterobacter aerogenes (liver only). Rare acid fast bacilli (Mycobacterium sp.) were identiﬁed in ZN stained sections of the granulomata, but the organism did not grow in culture (3334.1).

Metallic starling—captive, NSW—severe dietary iron overload, E. coli infection around the liver, and enteritis associated with bacteria and yeast infections. (3356.1).

Feathertail glider—captive, NSW—Animal had fungal sinusitis most likely caused by a strain of Cryptococcus sp., and yeast within periodontal lesions, most likely Candida sp. (3299.1).

Tammar Wallabies—captive, NSW—Outbreak of subacute to chronic sinusitis associated with Bordetella bronchiseptica. Two of the wallabies examined had evidence of concurrent meningitis with migrating nematodes (probably Angiostrongylus cantonensis). The wallabies had marked sinus congestion, and neutrophilic sinusits and tracheitis. One wallaby had necrotising sinusits that extended through the hard palate, down to a necrotising pharyngeal lesion. This animal had terminal systemic E. coli infection. Although Bordetella bronchiseptica was isolated within the lungs of some animals, this bacterium did not seem to spread systemically. Many of the animals examined had been ill for some time and had mobilised their fat stores. These animals were quite thin and had hepatic lipidosis. (3330.1, 3353.1–3353.4).

Common wombat—NSW, NPWS—Carcase remains—one of many wombats thought to have been run over maliciously on the border of a national park and then buried (3333.1).

Gippsland water dragon—NPWS conﬁscation—admitted emaciated, dehydrated and with a severe bacterial skin infection (3329.1)

Bluetongue lizard—NPWS conﬁscation—ruptured stomach after gorging on mealworms (3407.1)
Kakariki—captive, SA—euthanased due to the loss of several birds in the aviary after introduction of a group of Kakariki. The bird had inclusions in the Bursa of Fabricius that were consistent with Psittacine Beak and Feather disease. There were also many eosinophilic intranuclear inclusion bodies in hepatocytes, suggestive of a herpes virus or adenovirus infection. Due to the history and the fact that the bird was an exotic species, Pacheco’s disease virus (an exotic herpes virus) had to be included in the differential diagnoses, NSW Agriculture and the SA agriculture departments were notified. Samples from this bird were sent to a specialist avian diagnostic laboratory in the USA, where DNA hybridisation was undertaken, but neither Pacheco’s disease virus nor Psittacine Beak and Feather Disease were identified. DNA hybridization was selected rather than PCR, since formalin fixation modifies DNA and may make it unsuitable for PCR (3338.1).

Australian Magpie—wild, NSW—released from a rehabilitation facility in January, but was brought back lame and unable to fly in February. Radiographic examination revealed bony proliferation along several long bones, where there did not appear to be any fractures. Euthanasia was elected, and post mortem examination revealed multifocal bony proliferation along the endosteme and periosteum of the long bones, a nodular bony lesion along the dorsal aspect of the sternum, and proliferation of the compact bone of the skull. The histologic lesions were highly suggestive of fluorine toxicity. Several similar cases are lodged in the Registry, as are a number of young magpies with rickets. Fluorine toxicity can cause large bony lesions in adult animals, and rickets in young animals (where it binds to the bone substrate instead of calcium). Since fluorine toxicity can affect the development of bone and teeth in humans, EPA has agreed to conduct toxicological tests on the bone from this animal (3326.1).

Red-winged parrot—wild, NT—severe skin wounds—bacterial infection & candidiasis of the skin, in conjunction with Psittacine Beak and Feather inclusions (3283.1).

Green tree snake—NT—cataract, squamous cell carcinoma of the oviduct (very unusual) (3305.1).

Diamond Python—captive, SA—Euthanasia due to neurological disease. The snake had small numbers of basophilic smudgy intracytoplasmic inclusions in cells within the brain—consistent with a diagnosis of Inclusion Body Disease of Boids (3343.1).

Agile Wallaby—wild, NT—Biopsy of skin from a very crusty and ulcerative skin lesion in a young female wallaby. Sarcoptiform mites were identified in the skin scraping. Histologically the lesions are very similar to those of birds with Cnemidocoptes sp. infestations, and wombats with Sarcoptes sp. infestation. Mites have been forwarded to Dr. Ian Beveridge—University of Melbourne for identification (3399.1).

Agile Wallaby—wild, NT—hand raised young, male wallaby with skin disease, emaciation, blindness and then death. The animal had a non-suppurative colitis and skin lesions as described in the animal above (3277.1).

Black-headed pythons (2)—captive, NSW—multisystemic bacterial infections—splenitis and hepatitis (3394.1, 3395.1).

Green tree frog—captive, SA—euthanased due to an eye infection. The frog had multisystemic granulomata associated with the fungus Mucor sp. (3339.1).

Black faced cuckoo shrike—SA—subacute granulomatous enteritis and hepatitis. The lesions were suggestive of yersiniosis, but salmonellosis and other bacterial infections could not be ruled out without bacterial culture (3347.1).

March 2003

Koala—captive, NSW—died after several weeks suffering from a large ulcer on the tongue. Although a viral infection was suspected based on biopsies, no virus was isolated from the lesion. This is the second koala that we have seen that has died with severe, non-healing oral/pharyngeal ulcers. Has anyone seen anything similar? (2265.5).

Grey Nurse Shark—Wild, NSW Fisheries—found dead by divers 24 hours after being caught and tagged. The shark had acute foci of haemorrhage and inflammation in numerous tissues, suggestive of systemic bacterial infection, and it had a very large fishing hook that penetrated the oesophagus, coelomic cavity and lodged in the pericardium. The hook related lesions were very acute and it seems likely that the animal was caught twice within a short period of time, between the hooking and the subsequent manual catch and tagging. There was some concern regarding whether the tagging or the hook caused the shark’s death. Foci of inflammation along the coelomic cavity were suggestive of bacterial spread from the fish hook site. The hook type found in the oesophagus is illegal, but only in some locations (3466.1).

Australian Fur Seal—captive, NSW—aged animal, died with severe constipation and a very large hard faecolith obstructing the large intestine and devitalising the mucosa. Terminal constipation is not uncommon in aged, captive pinnipeds. This animal had a thyroid cyst and bilateral thyroid adenomas, thus, its thyroid function was questionable. The animal also had haemosiderosis and fairly high concentrations of hepatic mercury (Liver Iron Levels—18.8 nmol/kg wet weight, Liver mercury 220.00 (mg/kg FW), liver moisture (% as received 69.50). It is uncertain whether heavy metal concentrations or abnormal thyroid function contributed to altered intestinal motility (3465.1).

Superb Lyrebird—wild, NSW—hit by a car and sustained an eye injury. Euthanased after several
weeks in rehabilitation due to severe debility. The bird had a contusion of the brain, and a ruptured lens, detached retina and secondary inflammation in the eye. The bird had marked fibrous coelomitis and air sacculitis with *Providencia stuartii*, and *Pseudomonas aeruginosa*. It is possible that steroid administration to alleviate inflammation in the eye increased the bird’s susceptibility to infection (3406.1).

**Sea Turtles**—Brains from 22 turtles that stranded in Coffs Harbour throughout 2002 were examined for evidence of systemic coccidiosis, as seen in the October outbreak of mortality in Port Stephens. Although two large green turtles had severe bacterial meningitis none of the animals had coccidiosis (3402.1–3402.22).

**Red-collared lorikeet**—wild, NT—euthanased due to several lumps in skin, predominantly over the head and neck. The bird had multiple intra-dermal cysts filled with large numbers of mites. The case is very similar to a Musk Lorikeet from SA, where the mites were identified as *Harpyrynchus rossellinus*. Mites from this lorikeet will be submitted to Dr. Ian Beveridge, University of Melbourne, for identification.

—Karrie Rose, Veterinary & Quarantine Centre, PO Box 20, Mosman, NSW, 2088, 02-9978-4749 phone, 02-9978-4516 fax

**Selected Laboratory Reports from Massey University June–Dec 2002**

**Nematodes in native birds.** Metazoan parasites are not recognised as a serious threat to the health of free living birds since the adults of most species have adapted to carry mild parasite burdens. Heavy parasite burdens are usually only seen in naïve individuals or birds that have other concurrent diseases. However, two recent cases of visceral larval migrans in wild birds have shown that migrating parasites can sometimes cause serious impairment and even death and have highlighted the need to investigate the parasite status of native species more fully.

**Visceral larval migrans in a kiwi.** A young male kiwi (*Apteryx aupteryx*) was found lying freshly dead on a vehicle track in bush on the east coast of the North Island. It was in very good body condition but had severe traumatic injuries to the neck, back and pelvis with extensive subcutaneous haemorrhage and penetrating wounds which were consistent with predation by a dog. Histology of the brain revealed a large parasite granuloma located in a dorso-medial white matter tract in the brain stem immediately beneath the cerebellum. It contained a central area of necrosis in which several cross sections of nematode larvae were embedded. A second granuloma was present in the cerebellar white matter. The left ventricular myocardium and the gizzard mucosa also contained parasite granulomas. A worm count revealed: Gizzard: 90 cestodes, 30 nematodes, Small intestine: 2080 cestodes, Caecum: 800 Heterakis sp. At this stage it is not possible to accurately identify the migrating nematodes but there was no histological evidence of immunosuppression or other disease processes in body tissues. It seems highly likely that the lesions in the heart and brain had a significant effect on the animal’s fitness and behaviour which could well have predisposed it to predation.

**Visceral larval migrans in a saddleback.** A free living North Island saddleback (*Phil esturnus carunculatus* and *carunculatus*) from a sanctuary near an urban area was found depressed, hunched up and sluggish with ruffled feathers and an enlarged abdomen. The bird was captured but rapidly developed difficulty in breathing and died that evening despite symptomatic treatment. The bird was in average to good body condition but on opening the coelom a severe chronic fibrino-purulent peritonitis was observed. Covering the serosal surface of the small intestine was a layer of granulation tissue which merged with thick areas of exudate surrounding the jejunal wall. A cross section of a large migrating nematode was found in the jejunal muscle and serosa surrounded by an active inflammatory response. Other granulomas containing nematode remnants were present elsewhere in the intestinal wall. The peritonitis was likely to have arisen following migration of the nematode through the jejunal wall into the abdominal cavity. An acute bacterial bronchopneumonia was also present and this was probably the ultimate cause of death.

**Mycotic dermatitis in reptiles.** A young tuatara (*Sphenodon punctatus*) and a striped skink (*Oligosoma striatus*) were kept in separate outdoor enclosures in a captive breeding facility with others of their own species. Both died within a few days of each other after a spell of cold weather. The skin of the tuatara exhibited multiple small (up to 3mm in diameter) brown plaques, particularly under the left axilla and along its right side. The skink had several transverse erosive skin lesions on its ventral body. One (5 × 3 mm) in the mid-thorax, one (4 × 1 mm) on the left abdomen, two (2 × 2 mm) mid pelvis and three (2 × 2 mm) mid tail. Microscopically, the tuatara skin showed extensive proliferation of branching septate fungi throughout the epidermis, dermis and subcutaneous tissues plus mixed Gram-positive cocci and...
Gram-negative rods. The skin of the skink showed extensive loss of epithelium and deep ulcerative lesions that extended into the underlying muscle. A large number of inflammatory cells were present in the subcutaneous tissues and these were associated with numerous invading fungal hyphae (non-septate and branching). Proliferating fungal hyphae were also present in the gastric lumen and there was very early focal invasion of the gastric mucosa. Unfortunately, the fungal organisms were not cultured but they appeared morphologically to be of different types in each individual. These skin lesions were quite different to the “black spot” lesions that have been described in New Zealand geckos. These manifest as a general blackening between the scales, are seen only in winter and are caused by *Cladosporium* sp. The current lesions more closely resemble those caused by *Paecilomyces* which cause ulcerative skin lesions that may progress to mycotic pneumonia and septicaemia. It is believed that environmental factors such as cold weather and high humidity may contribute to a reduction in the immune response of affected animals and allow the skin infections to become overwhelming.

**References**

McCall PG. Studies on “Black Spot”, a skin disease of New Zealand Geckos. BSc (Hons) Thesis, Massey University, 1981.


**Necrotising stomatitis in Yellow-eyed Penguins.** Increased mortality in nestlings has occurred in yellow-eyed penguin (*Megadyptes antipodes*) breeding colonies this season and a number of dead and undersized chicks have been recovered. Several of the affected chicks examined have had a large amount of caesaeous exudate in their mouth. On closer examination of the oral cavity yellow/brown diptheritic membranes were found and these were particularly severe beneath the tongue, in the buccal cavities and pharynx. Histology showed widespread loss of the oral mucosa and replacement with a densely cellular exudate containing large numbers of Gram positive pleomorphic coco-bacilli which, on culture were identified as *Corynebacterium* sp. The possibility that this was a primary bacterial infection seemed unlikely and an extensive search for viral inclusion bodies in the remaining oral epithelium produced no definite evidence of infection. Electron microscopy of affected tissue is in progress but we also need to investigate the pathogenicity of the diptheria-like organism isolated.

**Meningo-encephalitis in black stilts.** Over the past two years there have been occasional cases of meningo-encephalitis diagnosed in juvenile black stilts (*Himantopus novaezelandiae*) raised in captive breeding institutes. Besides having the usual sick bird signs (depressed, fluffed up etc.) affected birds have often exhibited CNS signs including walking backwards intermittently, shaking the bill from left to right, head tilt and circling. Some cases have responded to antibiotics and fluid therapy but others have died within 1–2 days. No gross lesions have been visible at necropsy but localised areas of acute inflammation were seen in the cerebral cortex histologically. Many heterophils were seen migrating from capillaries into the neuropil and larger blood vessels in the affected areas were cuffed with a mixture of mononuclear cells and heterophils. In the more severe cases, areas of capillary proliferation and focal inflammatory necrosis were evident. The organisms recovered from the brain have been usually Gram negative coco-bacilli and in some cases there has been a history of skin wounds inflicted by brooder mates suggesting these may be the source of infection.

—Maurice Alley, Brett Gartrell

**Aspergillosis in Hector’s dolphins.** In July 2001 a juvenile male South Island Hector’s dolphin (*Cephalorhynchus hectori hectori*) was found recently dead on an east coast beach. On post mortem it had severe disseminated granulomatous pneumonia. *Aspergillus fumigatus* was cultured from the lung. In June 2003 a juvenile male North Island Hector’s (also known as Maui’s dolphin, *C. hectori maui*) was found on a west Auckland beach. Granulomatous pneumonia was also found at post mortem but the actual cause of death was haemothorax as a result of haemorrhage from the left lung. Other findings in this dolphin included dolphin pox characterised by tattoo lesions, intracytoplasmic Bollinger body-like inclusions, and pox virions on TEM. Sarcocysts were also present in the myocardium as an incidental finding but not previously reported for this species. There was also severe hepatic scarring with portal fibrosis and biliary hyperplasia associated with intraresional trematode eggs and adult trematodes within bile duct lumens. We have observed hepatic trematodiasis previously but rarely with such severe fibrosis. Aspergillosis has been recorded in captive dolphins previously as have other opportunistic fungal and bacterial infections. However, aspergillosis is relatively rare in free-living dolphins. Published cases from the northern hemisphere have been seen in bottlenose (*Tursiops truncatus*) and striped (*Stenella coeruleoalba*) dolphins with concurrent morbillivirus infection causing immunosuppression (Domingo et al., 1992; Lip-scomb et al., 1994). In the former, 22 of 42 morbilliviral antigen positive dolphins had concurrent fungal infection while 3 of 18 antigen-positive striped dolphins had fungal pneumonia. Obviously we will have to rule out this virus and although viral culture on the first Hector’s case was negative, results on the second are pending. Immunohistochemistry for dolphin morbillivirus is being carried out on tissues from
both animals by Dr. Seamus Kennedy, Veterinary Research Laboratories, Belfast, N. Ireland. No clinical cases of dolphin morbillivirus infection have been recorded in the New Zealand/Australian region, however, there is a high rate of seropositivity in long-finned pilot whales (Globicephala melas) in NZ waters (Van Bressem et al., 2001), a situation similar to what occurs in the North Atlantic (Duignan et al., 1995). Other more nebulous causes of immunosuppression will also have to be considered such as social stress (both were pubertal males), time of year, nutritional stress etc. Whatever the underlying cause, the consequences for the species could be serious. There are approximately 6000 to 7000 South Island Hector’s remaining but only an estimated 75 (CI 48–130) of the North Island subspecies (Ferreira and Roberts, 2003). The latter are already on the brink of extinction and disease combined with incidental deaths as a result of fisheries bycatch could tip the balance.

References
Ferreira S.M. and C.C. Roberts. 2003. Distribution and abundance of Maui’s dolphins (Cephalorhynchus hectori maui) along the North Island west coast, New Zealand. DOC Science Internal Series 93. Published by DOC, Wellington.

OIE WORKING GROUP ON WILDLIFE DISEASES REPORT 2002

The OIE Working Group on Wildlife Diseases met in Paris at the OIE Central Bureau from February 17 to 19, 2003. This is a summary of the Australasian content of the report:

Preliminary results of an investigation into unusually high Shy albatross (Thalassarche cauta) chick mortality at Albatross Island, Bass Strait, Tasmania (40.375°S, 144.656°E) suggested mortalities were due to a poxvirus (histology and clinical signs). Serological tests on sera from 37 live chicks were negative for avian influenza and infectious bursal disease, however, a single serum sample was positive for Newcastle disease virus (NDV). This is likely to represent prior infection with an endemic strain of NDV.

There were 3 serological positives (ELISA) for avian influenza virus in Silver gulls (Larus novaehollandiae) in Sydney, Australia (n = 10).

Wildlife List Diseases

In Australia a recent study found histological evidence of Angiostrongylus cantonensis infection in 16 of 86 flying foxes (Pteropus spp). In 10 of the 16 flying foxes the parasites were definitively identified as A. cantonensis 5th stage larvae. The 16 flying foxes were negative for lyssaviruses by direct fluorescent antibody test.

In March 2002 there were serological positives for Japanese encephalitis virus in sentinel pigs on Badu Island, Torres Strait.

New Zealand reported Psittacine pox infection in aviary-kept eastern rosellas (Platycercus eximius). This disease has the potential to infect native populations of highly endangered parrots, and thus is of significant conservation concern.

Taiwan continues to have mortalities in endangered black-faced spoonbills (Platalea minor) due to botulism. This disease could threaten the species.

There was an outbreak of neurological disease in endangered green sea turtles (Chelonia mydas) due to systemic coccidiosis in Australia. Turtles were found stranded on beaches in northern New South Wales in mid-October.

In Hong Kong melioidosis occurs frequently in certain locations and kills many animals and people in other parts of Asia. It is infrequently diagnosed and reported because of difficulties in diagnosing the bacteria, Burkholderia pseudomallei.

A large number of acid fast bacteria were seen in the faeces or various tissues/aspirates from Australian native wildlife including (for 2002): scarlet chested parrot (Neophema splendida), budgerigar (Melopsittacus undulatus), whiteheaded pigeon (Columba leucomela), rainbow fish (species not
stated), diamond dove (Geopelia cuneata), rose crowned fruit dove (Ptilinopus regina), gouldian finch (Erythrura gouldiae), zebra finches (Taeniopygia guttata), long nosed bandicoot (Perameles nasuta).

**Human Nipah virus infection** was confirmed from India. In Siliguri, North Bengal, 6 samples were confirmed positive by the Centres for Disease Control. There were also some unconfirmed cases of the viral disease from the Chandpurthana Meherpur district.

Australia reported 64 different isolates of *Salmonella* spp. from Australian native species in 2002. There were 1633 records for native animal *Salmonella* isolates since 1983 in the National Enteric Pathogen Surveillance Scheme databases as of 29.1.03.

During the 2001/02 breeding season of New Zealand sea lion (Phocarctos hookeri) in the subantarctic Auckland Islands, there were 30% fewer pups born and mortality in the 2 months post partum was 3 times greater than the normal rate for this time of year. The principle cause was thought to be systemic bacterial infection with *Klebsiella pneumonias*.

Six of 34 young flying foxes (Pteropus poliocephalus) being maintained in a pre-release creche in Australia died within a 36 hour period. Three additional flying foxes in the creche concurrently developed respiratory illness and dysphagia. There was no known exposure to toxins. There were no significant bacterial and viral agents identified. All flying foxes were tested for Australian bat lyssavirus and Hendravirus and were negative.

The Ministry of Agriculture of India reported on a project on wildlife disease including reports from individual animals and from outbreaks of disease collected in mammals between 1995 and 2001, and in birds between 1997 and 2002. The pertinent results were as follows: rabies in tiger (*Panthera tigris*), anthrax in lyena (*Hyaena hyaena*), black bear (*Ursus thibetanus*), and Crown pigeon (*Goura victoria*); tuberculosiis in cheetal (*Axis axis*), hog deer (*Axis porcinus*), nilgai (*Boselaphus tragocamelus*), and Rhesus macaque (*Macaca mulatta*); pasteurellosis in deer, *ox* and Newcastle disease in pigeon. There were many other diseases listed, but they were of non specific aetiology, for example fibrinonecrotic myositis in leopard.

A “new” undiagnosed frog mortality syndrome possibly associated with *amphibian mucormycosis* is currently causing mortality of frogs in Queensland, Australia. Mucormycosis (infection with *Mucor amphibiourum*) has been found in cane toads (*Bufo marinus*), green tree frogs (*Litoria caerulea*), white lipped frogs (*Litoria infrafrenata*), and striped marsh frogs (*Limnodynastes peronii*) in the wild in Australia. 0.7% of cane toads in one survey in Queensland, NSW and Northern Territory were infected. This disease is also associated with significant morbidity in platypus (*Ornithorhynchus anatinus*) in Australia.

In Nepal, there was mortality in langurs (*Presbytis entellus*) of unknown aetiology in Bardiya National Park in 2002. Poisoning cases were reported in nilgai (*Boselaphus tragocamelus*) and rhinoceros (*Rhinoceros unicornis*). The latter were laboratory confirmed. Concern was expressed about FMD, PPR and Swine Fever in wildlife in Nepal. There were epidemics of human Japanese encephalitis reported from in Banke, Bardiya, Kailali and Kanchanpur, where there is a large population of wild boar, egrets and herons.

Systemic protozoal infection in the highly endangered South Island saddleback (*Philesturnus carunculatus carunculatus*) was diagnosed on Motuara Island, New Zealand. Sarcocystosis was seen in endangered southern lesser short tailed bats (*Mystacina tuberculata tuberculata*), on Codfish Island, New Zealand.

Wild (n = 7) Australian king parrots (*Alisterus scapularis*) in Victoria were found to have *spironucleosis* (Spironucleus-like protozoa formerly *Hexamita* sp.). The disease is believed to be responsible for a syndrome of wasting and diarrhoea in wild king parrots (*A. scapularis*) between 1984 and 2000 in eastern Australia.

—Stephanie A. Haigh (Member, OIE Working Group on Wildlife Diseases—Antipodes and South East Asia)

**Reminiscences of Barry L. Munday**

It was with particular sadness that I learned of our loss of Barry Munday, . . . and it wasn’t helped by the stark realisation that I still owed him a beer for being unable to meet with him the last time he was in Canberra! Our acquaintance goes back to 28th November 1967 when I identified some filarial nematodes from Tasmanian possums which he had sent to Professor John Sprent at the University of Queens-land. Thus commenced decades of correspondence and the uninterrupted movement of parasite specimens from Launceston to Brisbane and later to Canberra. Well do I remember meeting Jack Arundel at a conference in Armidale in Jan 1970 and Jack commenting that he was very pleased with the thesis that he was carrying in his briefcase at the time . . . the work of one Barry Munday. Then in 1972 Barry commenced his efforts to drum up support for an Australasian Section of the Wildlife Disease Association. By April 1973 he had the required 10 supporters and was able to forward a petition to the parent body requesting formation of an Australasian Section. He was successful and at the request of the Parent Body
in the USA undertook the role of inaugural Chairman until we held our first scientific and annual general meetings in conjunction with an ANZAAS meeting at the Australian National University in Canberra in January 1975. To his credit, the Australasian Section of the WDA has never looked back and remains a strong and forceful body of interested and active scientists to this day. For that effort and for his enormous contribution to wildlife disease studies and education in Australia Barry was awarded a trip to Uppsala, presented a keynote address on wildlife disease in Australia and accepted the Distinguished Service Award of the Wildlife Disease Association parent body in July 1985. Or, as Barry put it to me in a letter seeking some slides for his talk, “As you well know, some very convincing liars have managed to organise a trip to Sweden for me.” This was followed by life membership in the Australasian Section. We owe Barry a tremendous debt of gratitude for his untiring efforts at liaison and co-operation with so many of us over more than four decades. My pile of correspondence, almost all handwritten, is more than 30 mm deep, many of the Dept of Agriculture, Mt Pleasant Laboratories pages having been splashed with formalin during the writing and still bearing hints of that odorous chemical. And then when he moved to the University of Tasmania, his incredulity that the secretary/typist had difficulty deciphering his scrawl!

On 30th July 1973, still having never met face to face, he wrote, “As you say, once again we seem fated not to meet. I sincerely hope our first meeting is not in a geriatric ward!” We finally met 7 years after our first correspondence when Barry, Fay and Philip came to Canberra 19–26 January 1975 for the inaugural meeting of the Australasian Section of the WDA. Barry would often stay with my wife and me during his one or two day trips to Canberra on “Veterinary/Quarantine/Vertebrate Pest Control” business. He always arrived with something in hand, a good SA wine or a coloured fleece for spinning, though on one occasion we missed out on a forequarter of venison which he had lined up during a potential stay in May 1979. Unfortunately, I’d promised the family a week in Cairns & Townsville in conjunction with a WDA meeting which Barry couldn’t attend because of his Canberra commitments. And he was a great host himself. How many of us have stayed with Barry and Faye and some or all of the children over the years, enjoyed a beer, good wine and great meals while we were visiting only briefly or on collecting trips of several weeks duration . . . and who could forget Barry’s slow roasted Tasmanian potoroo garnished with bacon! Barry Munday’s name shall always remain associated with the science of wildlife disease in Australia.

—Dave Spratt

WDA SECTION CHAIRS AND CONTACT INFORMATION

African Section. For information regarding the African Section, contact Elizabeth Wamba, Kenya Wildlife Service, P.O. Box 40241, Nairobi, Kenya. Telephone: 254-2-504180; Fax: 254-2-505866; email: ewamba@yahoo.com

Australasian Section. For information regarding the Australasian Section, contact Peter Holz, Healesville Sanctuary, P.O. Box 248, Healesville, Victoria 3777 Australia. Telephone: 61 3 5957 2864; fax: 61 3 5957 2870; email: pholz@zoo.org.au

European Section. For information regarding the European Section, contact Marc Artois, ENVL, Unite Pathologie infectieuse, BP33, 69280 Marcy l’Etoil, France, Telephone: 33-487-87-27-74, email: m.artois@fvet-lyon.fr

Nordic Section. For information regarding the Nordic Section, contact Hans-Henrik Dietz, Danish Veterinary Laboratory, Dept. of Fur Animal and Wildlife, Diseases, 2 Hangovej, DK-8200 Aarhus N., Denmark, Telephone: +45-89-37 24 17, FAX: +45-89-37 24 0, email: hhdl@svs.dk

Wildlife Veterinarian Section. Terry Kreeger, Wyoming Game and Fish Department, 2362 Highway 34, Wheatland, WY 82201, USA, Telephone: (307) 322-2571, FAX: (307) 322-5630, email: tekreege@wyoing.com

TRAINING/EDUCATIONAL OPPORTUNITIES

Post-Doctoral Training in Zoo Animal Pathology. The Smithsonian National Zoological Park, Washington, DC has an anticipated 3-year traineeship in pathology available July 1, 2004. The annual stipend is approximately $26,650 plus some benefits. Time in training may be applied to ACVP eligibility. Training will emphasize gross and microscopic diagnoses of case material originating from the zoo’s collection with opportunities to conduct pathologic investigations of diseases in a wide variety of zoo animals. The zoo has a strong program in clinical and comparative medicine. Members of the veterinary staff have affiliations with the Armed Forces Institute of Pathology and maintain academic appointments at Johns Hopkins and George Washington Universities, and the Uniformed Services University of the Health Sciences where participation in comparative pathology training and formal course work are avail-
able. Applicants must have a DVM or equivalent degree from an accredited veterinary college and should send academic transcripts, 3 letters of recommendation, and a resume with a short narrative of prior pathology experience and goals by December 30, 2003 to Ms. Nancy Huddy, Department of Pathology, Smithsonian National Zoological Park, 3001 Connecticut Ave. NW, Washington, DC 20008.

This program is provided through the Friends of the National Zoo (FONZ) and is subject to funds availability; it is not a federal position. Equal Opportunity Employer.

Training Available in Fish Diagnostics, Inspections, and Laboratory Methods. The US Fish and Wildlife Service Fish Health Centers provide laboratory and field examination services to the National Fish Hatcheries. Our main emphasis is to assist the hatcheries in producing quality fish that will contribute to the enhancement and restoration of aquatic ecosystems. At the Olympia and Idaho Fish Health Centers, the work may involve travel to field sites to perform diagnostic examinations and collect samples that are then evaluated in our laboratories. Routine testing procedures include bacteriology (biochemical, ELISA, and PCR methods), virology (cell culture, serological, and PCR methods), parasitology (microscopic and PCR methods), histology, and clinical chemistry. Training may be arranged for one day or several weeks at one or both of these laboratories depending on the interests and availability of the individual. In general, most broodstock inspections are performed from September through November, juvenile inspections are performed from January through April, and wild fish surveys are conducted from March through September. Routine diagnostic examinations are performed year round and special projects are conducted as time and necessity permit. For more information, please contact Joy Evered DVM, at the Olympia Fish Health Center; email joy@evered@fws.gov or Marilyn Blair DVM, at the Idaho Fish Health Center; email marilyn.blair@fws.gov.

Sr. Veterinary Student Preceptorship in Avian and Conservation Medicine. A four to six-week preceptorship in Avian and Conservation Medicine is being offered to a senior-year veterinary student by the International Crane Foundation (ICF) in Baraboo, Wisconsin. The preceptor will train with the Veterinary Services Unit of the Conservation Services Department in all phases of the clinical practice, but have opportunities for interaction with the Crane Conservation Department to learn captive propagation, husbandry and management of this unique family of birds. The preceptor can expect to gain practical experience in crane capture, transport, anesthesia, preventive medicine, disease surveillance and the contribution of veterinary medicine to crane conservation including field project support and professional consultations. Preceptors are encouraged to complete and report on a research or laboratory project during their stay. Opportunities for visiting the University of Wisconsin School of Veterinary Medicine and the National Wildlife Health Center in Madison, WI will be made available to interested preceptors. No stipend is available for this position; however, on-site housing in the ICF Guesthouse will be provided depending on availability at the time the preceptorship is scheduled. Applicants should send a cover letter, curriculum vitae or resume and one letter of recommendation from a faculty member of their home institution to: Barry Hartup, Director of Veterinary Services, International Crane Foundation, E-11376 Shady Lane Road, Baraboo, WI 53913, email hartup@savingcranes.org. Please view our website at www.savingcranes.org.

Directory of Post-Graduate Educational Opportunities in Zoo and Wildlife Medicine. The World Association of Wildlife Veterinarians has recently produced a Directory of Post-Graduate Educational Opportunities in Zoo and Wildlife Medicine. The Directory covers opportunities in over fifty countries and is a must for veterinary students or graduates interested in furthering their careers in the field of wildlife medicine. For further information, please contact the Secretary of the WAWV at: F.Scullion@zoo.co.uk

MEETING ANNOUNCEMENTS

53rd Annual Meeting of the Wildlife Disease Association. August 9–14, 2003; Saskatoon, Saskatchewan, Canada. Please see details of the meeting under “WDA Activities” in this issue.

10th Annual Conference of The Wildlife Society. September 6–10, 2003; Burlington, Vermont. The meeting will include symposia, workshops, contributed papers and posters on topics within the theme of Excellence in Wildlife Stewardship through Science and Education. Deadline for submission of abstracts was February 14, 2003. Instructions for preparing and submitting abstracts can be found at www.wildlife.org under ‘conferences’.

The 21st Annual Congress of the European Society of Veterinary Pathology. The 21st Annual Congress of the European Society of Veterinary Pathology will be held jointly with a meeting of the British Society of Toxicological Pathology in Dublin, Ireland from 10–13 September 2003. The Congress venue is Trinity College Dublin, which is located in the city centre and within walking distance of many of
Dublin’s most famous landmarks. From there you will be able to enjoy the rich cultural heritage made famous by Joyce and Wilde. The scientific programme will comprise offered oral and poster presentations, invited keynote lectures and symposia. Dublin is easily accessible with international air and sea links to continental Europe, Britain, North America and other regions. For details, contact Congress Secretariat, Ovation Group, 1 Clarinda Park North, Dun Laoghaire, Co Dublin, Ireland. TEL: +353 1 2802641; FAX: +353 1 2805405; E-MAIL: esvp@ovation.ie
—Dr. Seamus Kennedy, Veterinary Sciences Division, Belfast BT4 3SD, Telephone +44 (0)28 9052 5701, Fax +44 (0) 28 9052 5767, E-mail seamus.kennedy@dardni.gov.uk

First International Zoo Keepers Conference. Rijn, Netherlands, Oct 2nd 2003, for 9 days.

American Association of Zoo Veterinarians Annual Conference. October 5–9, 2003; Hyatt Regency, Minneapolis, Minnesota. The American Association of Zoo Veterinarians will hold its 2003 annual conference in Minneapolis, MN in conjunction with ARAV and NAG. Program sessions include Nutrition, Pharmacology (Nutriceuticals and phytochemicals), Conservation Medicine, AZA Programs, SSP/TAG Veterinary Advisor Updates, Emerging Diseases, Pathology, Advances in Technology and Diagnostics Testing, Case Reports and Practice Tips, Avian, Aquatics and Marine Mammals, Hoofstock, Primates, Carnivores and Small Mammals, Hospital Administration and Leadership, and Poster Session. For additional conference information, visit our website www.aazv.org, or contact Dr. Nadine Lamberski, Program Chair, San Diego Wild Animal Park, nlamberski@sandiegozoo.org.


NZVA Wildlife Society Conference. Dec 4th to 7th 2003, Stewart Island, NZ. Details to be published in the next issue of Kokako or may be obtained by contacting Katie Hicks, convenor, at katie.vet@hotmail.com


Sixth Conference of the European section of WDA. The European section of the Wildlife Disease Association will hold its sixth conference in Uppsala, Sweden in September 2004. The theme of the conference will be “Disease interactions between predators and prey animals”. For further information please contact Dolores Gavier-Widén (Dolores.Gavier-Widen@sva.se) or Torsten Mörner (Torsten.Morner@sva.se), Department of Wildlife, National Veterinary Institute, SE 751 89 Uppsala, Sweden, FAX: +46 18 30 91 62.

The 5th World Congress of Herpetology will be held in Cape Town, South Africa, in November 2005. More information at: http://www.adobe.com/products/acrobat/readstep2.html. If for some reason you can’t open the file then visit the Herpetological Association of Africa site (www.wits.ac.za/haa). It has a link to where you can down-load the file and will also be updating information on the conference as it becomes available. We will also be setting up links to all of the above on the SRARNZ website soon (http://www.vuw.ac.nz/srarnz/).

Note from the Editor:

Please send meeting announcements, diagnostic riddles, position and grant announcements, miscellaneous items, etc. for the Supplement to the Journal of Wildlife Diseases to Pauline Nol, USGS/National Wildlife Health Center, 6006 Schroeder Rd., Madison, WI, 53711. Phone: (608) 270-2489 Email: pauline.nol@usgs.gov. Files in WordPerfect or Microsoft Word sent electronically or via disk are preferred, though submissions in any form are welcome!! The deadline for submission of articles for the next issue (October 2003, JWD Vol. 39, No. 4) is September 1, 2003.