Wildlife Health in a Shrinking World:
ECOLOGY, MANAGEMENT AND CONSERVATION

WILDLIFE DISEASE ASSOCIATION INTERNATIONAL CONFERENCE

June 26 - July 1, 2005
Cairns, Queensland, Australia

Cover Image and Page Motives by Kim Hauselberger

Kim Hauselberger, a talented PhD student based at James Cook University in Townsville, Australia, is currently studying chytridiomycosis in microhylid frogs (contact Kim.Hauselberger@jcu.edu.au).

The Southern Cassowary (*Casuarius casuarius johnsonii*) is an endangered flightless bird of the Wet Tropics rainforests of northern Australia, which grows up to two metres tall. The males incubate the eggs and raise the chicks. These huge birds are the only animals capable of distributing seeds of more than seventy species of rainforest trees whose fruit is too large for any other forest dwelling animal to eat and relocate. In the Wet Tropics of Australia the cassowary plays this role which is accomplished by entire guilds of animals elsewhere. Latest estimates suggest the total Australian population of the Southern cassowary numbers only between 1,200 and 1,500 adults. Some of their many problems include loss and fragmentation of habitat through clearing for residential settlement and agricultural expansion.

See also Annabelle Olsson's abstract, 'The impact of disease on free living cassowary populations in far north Queensland', on page 199.

WDA
Wildlife Disease Association
Founded in 1951

We wish to acknowledge the valuable support from the following sponsors:

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Ecology of Introduced Diseases Session ..... Damien Joly

Environmental Drivers Session ..... Raina Plowright, Hume Field

Lyssavirus Session ..... Charles Rupprecht, Robert McLean

Chytridiomycosis Forum ..... Rick Speare

Activities ..... Sam Young, Annabelle Olsson, Lee Berger, Lee Skerratt, Scott Cashins, Keith McDonald

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WDA 2005 CONFERENCE PROGRAM OVERVIEW

Sunday, June 26

Preliminary Meetings		
8.00am – 12.00pm	Editorial Board Meeting, Journal of Wildlife Disease	(Lockhart Room)
12.00pm – 1.00pm	Lunch	
1.00pm – 5.00pm	Wildlife Disease Association Council Meeting	(Lockhart Room)
4.00pm – 10.00pm	**Conference Registration**	(Reception then Lockhart Verandah)
7.00pm – 10.00pm	**Conference Welcome Reception** *Australian Wildlife Health Network and Australian Registry of Wildlife Health Website Launches*	(Lockhart Verandah)

Posters
Posters will be on display from Monday morning through to Friday afternoon in the Lockhart Room and Lockhart Verandahs. Authors will be available at morning and afternoon tea breaks on Thursday, June 30 to answer questions.

Presentation Schedule
All papers will be presented in the Lockhart Room, apart from the sessions on "Ungulates" and "Endangered and Captive Animals" on Thursday afternoon that are in the Palmerston Room upstairs.

Monday, June 27

7.00am – 8.00am	Registration and Information Desk open	
8.00am – 8.05am	Welcome and Introduction	
8.05am – 8.30am	Opening Plenary	
8.30am – 2.00pm	**Session 1: Ecology of Introduced Wildlife Diseases**	
10.00am – 10.30am	Morning Tea	
12.00pm – 1.00pm	Lunch (Homestead Restaurant)	
2.00pm – 5.30pm	**Student Presentations**	
3.00pm – 3.30pm	Afternoon Tea	
3.05pm – 3.30pm	Documentary by student Meir Sussman, 'Coral Disease in the Republic of the Marshall Islands'	(Lockhart Room)

Evening Activities
Please sign up at board located in Foyer at morning tea. Information available at boards.

6.00pm – 8.30pm
'Reef Teach'
Take 6.00pm shuttle bus from Main Colonial Club Resort Reception

OR

7.00pm – 9.30pm
Frog Spotlighting
Meet at Conference Centre Reception

Tuesday, June 28

Silent Auction held all day in Palmerston Room (upstairs) during tea breaks.

7.00am- 8.00am	Prayer meeting in gazebo in Conference Centre gardens
7.30am – 8.00am	Registration and Information Desk open
8.00am – 8.30am	Introduction and Carlton Herman Fund Speaker
8.30am – 10.00am	**Session 2:** **Environmental Drivers of Emerging Infectious Diseases**
10.00am – 10.30am	Morning Tea and Silent Auction (Palmerston Room)
10.30am – 12.00pm	**Session 3: West Nile Virus**
12.00pm – 1.00pm	Lunch (Homestead Restaurant) and Silent Auction
1.00pm – 5.45pm	**Student Presentations**
3.15pm – 3.45pm	Afternoon Tea and Silent Auction (last chance to bid!)
5.45pm	Collection of Auction Items

Professional Meetings

7.30pm – 8.30pm	*WDA Parent Body Annual General Meeting ..... (Lockhart Room);* ***OR*** *WDA Australasian Section Annual General Meeting ..... (Palmerston Room)*
8.30pm – 9.00pm	*WDA Parent Body and Australasian Section Combined Meeting .....(Lockhart Room)*

Evening Activities

Evening activities will be held concurrently with Professional Meetings. Please sign up at board located in Foyer at morning tea. Information available at boards.

6.00pm – 8.30pm *'Reef Teach'* *Take 6.00pm shuttle bus from Main Colonial Club Resort Reception*	***OR***	*7.00pm – 9.30pm* *Frog Spotlighting* *Meet at Conference Centre Reception*

Wednesday, June 29

7.30am – 8.00am	Registration and Information Desk open

Conference Field Trips: Meet at Conference Centre Reception.

8.10am – 5.00pm	Outer Great Barrier Reef trip with Reef Magic Cruises.
8.30am – 5.30pm	Rainforest Adventure trip. Option to stay in the rainforest for Rainforest Mammal Spotlighting (7.30pm – 10.30 pm). Sign up on boards in foyer on Monday.

Optional Evening Activities

6.00pm – 8.30pm *'Reef Teach'* *Take 6.00pm shuttle bus from Main Colonial Club Resort Reception*	***OR***	*7.30pm – 9.30pm* *Chytridiomycosis Forum* *Lockhart Room*

Thursday, June 30

7.30am – 8.00am	Registration and Information Desk open
8.00am – 11.30am	**Session 4: Management of Wildlife Diseases**
10.00am – 10.30am	Morning Tea and Poster Session
11.30am – 12.15pm	**Session 5: Monkeypox**
12.15pm – 1.15pm	Lunch (Homestead Restaurant)
Concurrent Sessions	
1.15pm – 2.45pm	**Session 6: Lyssavirus Emergence and Management**
OR	
1.15pm – 2.45pm	**Session 7: Diseases of Ungulates (Palmerston Room)**
2.45pm – 3.30pm	Afternoon Tea and Poster Session
3.30pm – 5.00pm	**Session 8: Miscellaneous Tools and Techniques**
OR	
3.30pm – 5.00pm	**Session 9: Endangered and Captive Animals (Palmerston Room)**

Conference Dinner at Hartley's Crocodile Adventures

5.15pm	*Pick up from Conference Centre Reception (35 minute coach trip to sanctuary)*
6.00pm – 7.40pm	*Pre-dinner drinks and tour of wetlands*
7.40pm – 10.00pm	*Dinner and Awards*

Friday, July 1

7.30am – 8.00am	Registration and Information Desk open
8.00am – 10.15am	**Session 10: Health of Marine Ecosystems**
10.15am – 10.45am	Morning Tea
10.45am – 12.15pm	**Session 11: Wildlife Health in the Tropics**
12.15pm – 1.15pm	Lunch (Homestead Restaurant)
1.15pm – 2.00pm	**Session 12: Marsupials**
2.00pm – 3.00pm	**Session 13: Birds and Reptiles**
3.00pm – 3.30pm	Afternoon Tea
3.30pm - 4.15pm	**Session 14: Lagomorphs**
4.15pm - 5.00pm	**Session 15: General**
5.05pm	**Close of Conference**

Option Evening Activities

6.00pm – 8.30pm *'Reef Teach'* *Take 6.00pm shuttle bus from* *Main Colonial Club Resort Reception*	***OR***	*7.00pm – 9.30pm* *Rainforest Mammal Spotlighting* *Atherton Tablelands* *Sign up in foyer by Thursday morning tea.* *Meet at Conference Centre Reception*

WDA 2005 DETAILED CONFERENCE PROGRAM

POSTERS

Posters will be on display Monday morning to Friday afternoon on the Lockhart Verandahs and the back of the Lockhart Room. Authors will be available at morning and afternoon tea breaks on Thursday, June 30 to answer questions.

PRESENTATION SCHEDULE

All papers will be presented in the Lockhart Room apart from the sessions on 'Ungulates' and 'Endangered and Captive Animals', which will be held in the Palmerston Room upstairs.

REGISTRATION AND INFORMATION DESK OPENING TIMES (CONFERENCE CENTRE FOYER)

Sunday	4.00pm - 10.00pm
Monday	7.00am - 8.00am, and Morning, Lunch and Afternoon Tea Breaks
Tuesday, Thursday and Friday	7.30am - 8.00am, and Morning, Lunch and Afternoon Tea Breaks
Wednesday	7.30am - 8.00am

Sunday, June 26

Preliminary Meetings

8.00am – 12.00pm	**Editorial Board Meeting, Journal of Wildlife Disease**
12.00pm – 1.00pm	Lunch
1.00pm – 5.00pm	**Wildlife Disease Association Council Meeting**
4.00pm – 10.00pm	Conference Registration
7.00pm – 10.00pm	Conference Welcome Reception Australian Wildlife Health Network and Australian Registry of Wildlife Health Website Launches

Monday, June 27

7.00am – 8.00am **Registration and Information Desk open (and during Morning and Afternoon Tea breaks, and Lunch)**

8.00am **Welcome and Introduction (Lee Berger)**

8.04am **Plenary (Chair: Lee Skerratt)**
Introduction (Lee Skerratt)

1) OVERVIEW OF WILDLIFE HEALTH IN AUSTRALASIA – AN ASSESSMENT OF THE ECOLOGY, MANAGEMENT, AND THE IMPACT ON CONSERVATION, OF INTRODUCED DISEASES (22 min presentation, 3 min questions)
David M. Spratt

8.30am **The Ecology of Introduced Wildlife Diseases (Chair: Damien Joly)**
Introduction (Damien Joly, 5 min)

Invited Speakers:

2) PATHOGEN POLLUTION: UNDERSTANDING THE CAUSES AND IMPACT OF INTRODUCED WILDLIFE DISEASES (17 min presentation, 3 min questions)
Peter Daszak

3) A GLOBAL HISTORICAL PERSPECTIVE ON DISEASE INTRODUCTIONS (17 min presentation, 3 min questions)
Frederick A. Leighton

4) EMERGING EPIDEMIOLOGICAL PATTERNS OF RABBIT HAEMORRHAGIC DISEASE IN AUSTRALIA (12 min presentation, 3 min questions)
Gregory Mutze

5) ECOLOGY OF AVIAN MALARIA IN NATIVE HAWAIIAN FOREST BIRDS (12 min presentation, 3 min questions)
Michael D. Samuel

6) A COMMUNITY-BASED APPROACH TO UNDERSTANDING AND DETECTING 'INTRODUCED' OR 'EMERGING' DISEASES IN THE CANADIAN NORTH (12 min presentation, 3 min questions)
Susan J. Kutz

10.00am **Morning Tea**

10.30am **The Ecology of Introduced Wildlife Diseases (continues)**

7) ECOLOGY OF PLAGUE AS AN INTRODUCED DISEASE AND ITS IMPACT ON THE CONSERVATION OF THREATENED AND ENDANGERED SPECIES (12 min presentation, 3 min questions)
Tonie E. Rocke

8) CHYTRIDIOMYCOSIS AND AMPHIBIAN DECLINES (12 min presentation, 3 min questions)
Rick Speare

9) EMERGING INFECTIOUS DISEASES: THE PILCHARD EPISODE (12 min presentation, 3 min questions)
Alex Hyatt

10) BOVINE TUBERCULOSIS IN NEW ZEALAND WILDLIFE (12 min presentation, 3 min questions)
Maurice Alley

Contributed papers for EID Symposium:

11) HYBRIDISATION INCREASES RESILIENCE TO NOVEL DISEASE CHALLENGES (12 min presentation, 3 min questions)
Daniel M. Tompkins

12) THE INTRODUCTION OF SARCOPTIC MANGE IN THE RED FOX (*VULPES VULPES*) POPULATION IN SWEDEN AND ITS ECOLOGICAL CONSEQUENCES – THIRTY YEARS OF EXPERIENCE (12 min presentation, 3 min questions)
Torsten Mörner

12.00pm **Lunch (Homestead Restaurant)**

1.00pm **The Ecology of Introduced Wildlife Diseases (continues)**

13) AN EPIDEMIOLOGICAL MODEL OF CANINE DISTEMPER VIRUS SPILLOVER FROM DOMESTIC DOGS TO JAGUARS IN THE BOLIVIAN ISOSO (12 min presentation, 3 min questions)
Christine V. Fiorello

14) SIMULATING THE POTENTIAL CONSEQUENCES OF INTRODUCED CANINE DISTEMPER VIRUS FOR AMUR TIGER POPULATION DYNAMICS (12 min presentation, 3 min questions)
Damien O. Joly

15) RESEARCH INTO THE TASMANIAN DEVIL FACIAL TUMOUR DISEASE (DFTD): A PROGRESS REPORT (12 min presentation, 3 min questions)
Stephen B. Pyecroft

16) EPIDEMIOLOGICAL FEATURES OF A NEW DISEASE IN THE TASMANIAN DEVIL (*SARCOPHILUS HARRISII*) (12 min presentation, 3 min questions)
Clare E. Hawkins

2.00pm **Student Presentations**
(Chair: Todd Cornish)

17) AN OUTBREAK OF TYPE C BOTULISM IN HERRING GULLS (*LARUS ARGENTATUS*) IN SOUTHEASTERN SWEDEN (12 min presentation, 3 min questions)
Aleksija Neimanis

18) REEF COMMUNITY ECOLOGY AND RISK FACTORS FOR THE CORAL DISEASE, YELLOW BAND SYNDROME, IN THE MEXICAN CARIBBEAN (12 min presentation, 3 min questions)
Susanne H. Sokolow

19) CROSS-SHELF PATTERNS IN THE PREVALENCE OF CORAL DISEASE ON THE GREAT BARRIER REEF (12 min presentation, 3 min questions)
Cathie A. Page

20) ISOLATION AND IDENTIFICATION OF THE CAUSATIVE AGENT FOR A WHITE SYNDROME CORAL EPIZOOTIC IN THE MARSHALL ISLANDS (12 min presentation, 3 min questions)
Meir Sussman

3.00pm **Afternoon Tea**

3.05pm **Documentary by Meir Sussman**
'Coral Disease in the Republic of the Marshall Islands'
(showing in Lockhart Room)

3.30pm **Student Presentations (continues)**

21) A REVIEW OF THE 1988 AND 2002 PHOCINE DISTEMPER EPIDEMICS IN THE EUROPEAN HARBOUR SEAL POPULATION (12 min presentation, 3 min questions)
Caroline Millins

22) RISK FACTORS ASSOCIATED WITH INFECTION WITH PATHOGENIC AND ANIMICROBIAL RESISTANT GASTROINTESTINAL BACTERIA IN NORTHERN ELEPHANT SEALS (*MIROUNGA ANGUSTIROSTRIS*) (12 min presentation, 3 min questions)
Robyn Stoddard

23) POXVIRUS INFECTIONS IN NORTH AMERICAN PINNIPEDS (12 min presentation, 3 min questions)
Hendrik H. Nollens

24) DISEASE ECOLOGY OF HENDRA VIRUS: EPIDEMIOLOGICAL MODELING TO TEST THEORIES FOR EMERGENCE (12 min presentation, 3 min questions)
Raina Plowright

25) HENDRA VIRUS INVESTIGATIONS IN TWO NORTH QUEENSLAND FLYING FOX COLONIES FOLLOWING EQUINE AND HUMAN INFECTIONS (12 min presentation, 3 min questions)
Andrew Breed

26) EFFECTS OF CLIMATE WARMING ON THE EPIZOOTIOLOGY OF NORTHERN HOST-PARASITE SYSTEMS (12 min presentation, 3 min questions)
Emily J. Jenkins

27) VARIABLE SUSCEPTIBILITY TO CHYTRIDIOMYCOSIS IN ANURANS (12 min presentation, 3 min questions)
Nicole Kenyon

END OF STUDENT SESSION

28) TECHNIQUES FOR DETECTING CHYTRIDIOMYCOSIS IN WILD FROGS: COMPARING HISTOLOGICAL WITH REAL-TIME TAQMAN PCR (12 min presentation, 3 min questions)
Jean-Marc Hero

5.30pm **Day One Finish**

Optional Evening Activities

Please sign up at board in reception at morning tea. Information is available at boards.

"Reef Teach"
Shuttle bus leaves **main Colonial Club Resort Reception** at 6.00pm. The talk is from 6.15pm – 8.30pm in town. There will be no return bus.

OR

Frog Spotlighting near Cairns (limited to 15 people)
Bus leaves **Colonial Club Conference Centre Reception** at 7.00pm. Return around 9.30pm.

Tuesday, June 28

Silent Auction all day in Palmerston Room (upstairs).

7.00am **Prayer meeting in gazebo in Conference Centre gardens.**

7.30am – 8.00am **Registration and Information Desk open (and during Morning and Afternoon Tea breaks, and Lunch)**

8.00am **Plenary (Chair: Anne Fairbrother)**
Introduction (Anne Fairbrother)

CARLTON HERMAN FUND INVITED SPEAKER:

29) HOW DO PARASITES INFLUENCE THE ECOLOGY OF THEIR HOST POPULATIONS? STUDIES ON RED GROUSE AND THEIR NEMATODE, *TRICHOSTRONGYLUS TENUIS* (22 min presentation, 3 min questions)
Peter Hudson

8.27am **Environmental Drivers of Emerging Infectious Diseases (Chairs: Raina Plowright and Hume Field)**
Introduction (Raina Plowright, 3 min)

Invited Speakers:

30) SEASONALITY, CLIMATE CHANGE AND PARASITE TRANSMISSION (12 min presentation, 3 min questions)
Andy Dobson

31) ANTHROPOGENIC FOREST CHANGE AND EMERGING TICK-BORNE DISEASES (12 min presentation, 3 min questions)
Janet Foley

32) THE EMERGENCE OF NIPAH AND HENDRA VIRUS IN AUSTRALIA, MALAYSIA AND BANGLADESH (12 min presentation, 3 min questions)
Peter Daszak

33) THE EMERGENCE OF SARS – PRECIPITATING FACTORS (12 min presentation, 3 min questions)
Hume Field

34) SOCIAL STRUCTURING, VIRULENCE AND HOT SPOTS (12 min presentation, 3 min questions)
Peter Hudson

35) TOWARDS AN UNDERSTANDING OF CORAL DISEASES ON THE GREAT BARRIER REEF AND POTENTIAL LINKS TO ELEVATED TEMPERATURES (12 min presentation, 3 min questions)
Bette Willis

10.00am **Morning Tea**
Silent Auction (Palmerston Room)

10.30am **Environmental Drivers of Emerging Infectious Diseases**
West Nile Virus (Chair: Bob McLean)

36) UTILITY OF CLIFF SWALLOWS AND THEIR PARASITES FOR WEST NILE VIRUS SURVEILLANCE (12 min presentation, 3 min questions)
Larry Clark

37) IMPLEMENTATION OF A NATIONAL SURVEILLANCE PROGRAMME FOR WEST NILE VIRUS IN DEAD WILD BIRDS: CANADA 2000-2004 (12 min presentation, 3 min questions)
Ian K. Barker

38) PATHOLOGY AND EFFECTS OF WEST NILE VIRUS ON CALIFORNIA'S ENDEMIC BIRD, THE YELLOW-BILLED MAGPIE (12 min presentation, 3 min questions)
Holly Ernest

39) DECREASED AVERAGE DAILY TEMPERATURES AS A CAUSE OF REDUCED WEST NILE VIRUS AVIAN MORTALITY IN WYOMING DURING 2004 (12 min presentation, 3 min questions)
Terry Creekmore

40) PROTECTING THE WILDLIFE AND PEOPLE OF HAWAII FROM WEST NILE VIRUS WITH AN INTEGRATED PREVENTION, SURVEILLANCE AND ERADICATION RESPONSE PLAN (12 min presentation, 3 min questions)
Jeff Burgett

41) MODELING WEST NILE VIRUS TRANSMISSION IN THE SOUTHWESTERN UNITED STATES: HABITAT, VECTORS, AND HOST DISTRIBUTIONAL PATTERNS ALONG THE LOWER COLORADO RIVER CORRIDOR MIMIC POTENTIAL WEST NILE VIRUS INFLUENCES ON NEOTROPICAL MIGRANT BIRDS THROUGHOUT THE SOUTHWEST (12 min presentation, 3 min questions)
Charles van Riper III

12.00pm **Lunch (Homestead Restaurant)**
Silent Auction (Palmerston Room)

1.00pm **Student Presentations**
(Chair: David Schultz)

42) KOALA RETROVIRUS (KoRV), THE LINK TO DISEASE AND ITS PLACE IN KOALA ECOLOGY (12 min presentation, 3 min questions)
Rachael Tarlinton

43) SEROLOGIC SURVEY FOR SELECTED DISEASE AGENTS IN URBAN BRUSHTAIL POSSUMS (*TRICHOSURUS VULPECULA*) FROM SYDNEY (12 min presentation, 3 min questions)
Jutta Eymann

44) URBAN POSSUMS IN TARONGA ZOO AND SURROUNDS: IMPLICATIONS FOR DISEASE TRANSMISSION BY A 'NATIVE PEST' (12 min presentation, 3 min questions)
Nichola Hill

45) ECTOPARASITE BURDENS AND PELAGE CONDITION IN MOUNTAIN BRUSHTAIL POSSUMS (*TRICHOSURUS CUNNINGHAMI*) (12 min presentation, 3 min questions)
Jasmin Hufschmid

46) ECTOPARASITES AND THEIR IMPACT ON HEALTH AND SURVIVAL OF ENDANGERED AUSTRALIAN ANIMALS (12 min presentation, 3 min questions)
Inger-Marie Vilcins

47) CRYPTOCOCCAL INFECTIONS IN CAPTIVE GILBERT'S (*POTOROUS GILBERTII*) AND LONG NOSED (*POTOROUS TRIDACTYLUS*) POTOROOS (12 min presentation, 3 min questions)
Rebecca J.Vaughan

48) PATHOLOGY AND SERODIAGNOSIS OF HYDATID DISEASE IN MACROPODS (12 min presentation, 3 min questions)
T. S. Barnes

49) IMMUNOMODULATORY COMPOUNDS IN MARSUPIAL MILK (12 min presentation, 3 min questions)
Janice L. Joss

50) ISOLATION, PURIFICATION AND CHARACTERISATION OF NEUTROPHIL PROTEINS FROM THE TAMMAR WALLABY (12 min presentation, 3 min questions)
Kiran S. Ambatipudi

3.15pm **Afternoon Tea**
Silent Auction (Palmerston Room)
Last chance to bid!

3.45 pm **Student presentations (continues)**
(Chair: Ro McFarlane)

51) GENETIC STRUCTURE AND VARIATION OF RACCOONS (*PROCYON LOTOR*) IN THE EASTERN UNITED STATES: INSIGHT INTO ORAL RABIES VACCINATION (ORV) PLANNING (12 min presentation, 3 min questions)
Serena A. Reeder

52) IDENTIFYING ECOLOGICAL FACTORS AFFECTING POPULATIONS OF RESERVOIR HOSTS OF LEPTOSPIROSIS: IMPLICATIONS FOR MANAGEMENT (12 min presentation, 3 min questions)
Dario F. Rivera

53) *CONTRACAECUM* SPECIES (NEMATODA: ANISAKIDAE) FROM THE AUSTRALIAN PELICAN: MORPHOLOGICAL CHARACTERISATION AND THE DEFINITION OF GENETIC MARKERS FOR ELUCIDATING THEIR TAXONOMY AND ECOLOGY (12 min presentation, 3 min questions)
Shokoofeh Shamsi

54) BLOOD PARASITE PREVALENCE AND INFECTION INTENSITY IN *EGERNIA STOKESII* (REPTILIA: SCINCIDAE) IN RELATION TO TRANSMISSION TYPE AND SOCIAL STRUCTURE OF HOST POPULATIONS (12 min presentation, 3 min questions)
Stephanie S. Godfrey

55) RED-EARED SLIDERS (*TRACHEMYS SCRIPTA ELEGANS*) AS A MODEL OF RANAVIRUS INFECTIONS IN CHELONIANS (12 min presentation, 3 min questions)
April J. Johnson

56) RECOMBINANT VACCINE FOR PSITTACINE BEAK AND FEATHER DISEASE (12 min presentation, 3 min questions)
Nicolai Bonne

57) DEVELOPMENT OF AN ELISA FOR THE DETECTION OF INTERFERON-GAMMA AS A DIAGNOSTIC TOOL FOR TUBERCULOSIS IN BLACK RHINOCEROS (*DICEROS BICORNIS*) AND WHITE RHINOCEROS (*CERATOTHERIUM SIMUM*) (12 min presentation, 3 min questions)
Darshana Morar

58) VISITING T. REX ON PATHOLOGY ROUNDS; CASE REPORTS ON THE LOWER JAWS OF THE LARGEST PREDATORY DINOSAUR (12 min presentation, 3 min questions)
Ewan D. S. Wolff

5.45pm	**Day Two Finish**
5.45pm	**Collect Auction items**
7.30pm	**Professional Meetings (Concurrent)** Parent Body AGM (1 hr) (Lockhart room) OR Australasian Section AGM (1 hr) (Palmerston room upstairs)
8.30pm	**Parent Body and Australasian Section Combined Meeting (30 min) (Lockhart room)**

OR

Optional Evening Activities

Please sign up at board in reception by morning tea. Information is available at boards.

"Reef Teach"
Shuttle bus leaves **main Colonial Club Resort Reception** at 6.00pm. The talk is from 6.15pm – 8.30pm in town. There will be no return bus.

OR

Frog Spotlighting near Cairns (limited to 15 people)
Bus leaves **Colonial Club Conference Centre Reception** at 7.00pm. Return around 9.30pm.

Wednesday, June 29

7.30am – 8.00am **Registration and Information Desk open**

Field Trips to Reef and Rainforest

Meet at the Colonial Club Conference Centre Reception.

8.10am Pick up for Reef Trip. Return 5.00pm.

8.30am Pick up for Rainforest Trip. Return 5.30pm.
Option to stay in the rainforest for Rainforest Mammal Spotlighting on Atherton Tablelands (7.30pm – 10.30 pm).

PLEASE SIGN UP FOR SPOTLIGHTING AT BOARD IN RECEPTION BY MORNING TEA ON MONDAY. INFORMATION IS AVAILABLE AT BOARDS.

Optional Evening Activities

"Reef Teach"
Shuttle bus leaves **main Colonial Club Resort Reception** at 6.00pm. The talk is from 6.15pm – 8.30pm in town. There will be no return bus. Please sign up at board in reception by morning tea. Information is available at boards.

OR

7.30pm – 9.30pm
Chytridiomycosis Forum (Moderator: Rick Speare) (Lockhart Room)
Update on some of the latest discoveries (short presentations) followed by an informal discussion of key issues and areas for possible collaboration.

Thursday 30 June

7.30am – 8.00am **Registration and Information Desk open (and during Morning and Afternoon Tea breaks, and Lunch)**

Poster Session Authors available Morning and Afternoon tea breaks.
Posters are on display Monday morning to Friday afternoon on the Lockhart Verandahs and at the back of the Lockhart Room.

8.00am **Management of Wildlife Diseases**
(Chair: Tim Portas)

59) PROSPECTS FOR PREVENTION, DETECTION, AND MANAGEMENT OF EMERGING DISEASES (12 min presentation, 3 min questions)
Christopher Bunn

60) EMERGING WILDLIFE DISEASES: A MANAGER'S DILEMMA (12 min presentation, 3 min questions)
Bruce Morrison

61) PATHOLOGY OF NATIVE AUSTRALIAN WILDLIFE SPECIES – WHO'S RESPONSIBILITY? (12 min presentation, 3 min questions)
Philip Ladds

62) THE AUSTRALIAN WILDLIFE HEALTH NETWORK AND WHIS - THE WILDLIFE HEALTH INFORMATION SYSTEM (12 min presentation, 3 min questions)
Rupert Woods

63) A WILDLIFE DISEASE SURVEILLANCE STRATEGY FOR NEW ZEALAND (12 min presentation, 3 min questions)
Susan Cork

64) CONSERVATION MEDICINE IN A THREATENED ECOREGION: ADDRESSING ECOLOGICAL HEALTH IN THE CALIFORNIAS (12 min presentation, 3 min questions)
Alonso Aguirre

65) MANAGEMENT OF SEMI-FREE RANGING POPULATION OF ARABIAN ORYX IN UNITED ARAB EMIRATES (12 min presentation, 3 min questions)
D Hoy

66) EVALUATION OF CARCASS DISPOSAL TECHNIQUES AFTER AN ANTHRAX OUTBREAK IN FREE-RANGING BISON IN NORTHERN CANADA (12 min presentation, 3 min questions)
Brett Elkin

10.00am **Morning Tea and Poster Session**

10.30am **Management of Wildlife Diseases (continues)**

67) FEASIBILITY OF BAIT DELIVERING A PSEUDO-RABIES DISEASE VACCINE TO WILD FERAL PIGS (12 min presentation, 3 min questions)
Steven J. Lapidge

68) FORMULATION OF LIVE BCG VACCINE FOR DELIVERY TO WILDLIFE SPECIES (12 min presentation, 3 min questions)
Mark Chambers

69) HEALTH ASSESSMENT OF GEOFFROYS' CATS (*ONCIFELIS GEOFFROYI)* IN ARGENTINA (12 min presentation, 3 min questions)
Marcela Uhart

70) DEVELOPMENT OF A HEALTH SCREENING PROTOCOL FOR CAPTIVE BRED LITTLE PENGUINS (*EUDYPTULA MINOR)* PRIOR TO TRANSLOCATION TO A WILD POPULATION (12 min presentation, 3 min questions)
Frances Hulst

Monkeypox (Chair: Tonie Rocke)

71) MONKEYPOX ZOONOTIC ASSOCIATIONS: U.S. 2003 OUTBREAK (12 min presentation, 3 min questions)
Russell Regnery

72) EVIDENCE OF EXPOSURE OF WILD RODENTS TO ORTHOPOXVIRUSES IN WEST AFRICA: A MONKEYPOX INVESTIGATION IN GHANA (12 min presentation, 3 min questions)
Jeff Root

73) THE MOLECULAR PHYLOGENETICS OF POTENTIAL MONKEYPOX RESERVOIRS (12 min presentation, 3 min questions)
Darin S. Carroll

12.15pm **Lunch (Homestead Restaurant)**

1.15pm **Concurrent Session: Lockhart Room**
Lyssavirus Emergence and Management
(Chairs: Charles Rupprecht and Bob McLean)

74) POTENTIAL APPLICATION OF LESSONS LEARNED FROM ORAL RABIES VACCINATION IN THE UNITED STATES (12 min presentation, 3 min questions)
Dennis Slate

75) RESEARCH METHODS DEVELOPMENT TO ENHANCE WILDLIFE RABIES CONTROL STRATEGIES IN THE UNITED STATES (12 min presentation, 3 min questions)
Robert G. McLean

76) PARENTERAL VACCINATION OF ETHIOPIAN WOLVES (*CANIS SIMENSIS*) TO CONTROL AN OUTBREAK OF RABIES IN BALE MOUNTAINS NATIONAL PARK (12 min presentation, 3 min questions)
Darryn L. Knobel

77) LYSSAVIRUSES FLOURISH AMONGST A BEVY OF BATS (12 min presentation, 3 min questions)
Charles Rupprecht

78) AUSTRALIAN BAT LYSSAVIRUS: OBSERVATIONS OF NATURAL AND EXPERIMENTAL INFECTION IN BATS (12 min presentation, 3 min questions)
Janine Barrett

79) THE EMERGENCE OF AUSTRALIAN BAT LYSSAVIRUS – A REVIEW OF CURRENT HYPOTHESES (12 min presentation, 3 min questions)
Hume Field

1.15pm **Concurrent Session: Palmerston Room**
Diseases of Ungulates (Chair: Scott Wright)

80) HEMORRHAGIC DISEASE IN WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*) IN POPULATONS DERIVED FROM NATIVE AND INTRODUCED ANIMALS (12 min presentation, 3 min questions)
David Stallknecht

81) BOVINE TUBERCULOSIS IN MICHIGAN WILDLIFE (12 min presentation, 3 min questions)
Janet B. Payeur

82) EXPERIMENTAL INFECTION OF ROCKY MOUNTAIN BIGHORN SHEEP (*OVIS CANADENSIS CANADENSIS*) WITH *BRUCELLA OVIS*. (12 min presentation, 3 min questions)
Matt McCollum

83) COULD WOLVES CONTROL CHRONIC WASTING DISEASE? (12 min presentation, 3 min questions)
Margaret A. Wild

84) PESTIVIRUS OF CHAMOIS (*RUPICAPRA* SPP.) IN EUROPE: AN EMERGING INFECTIOUS DISEASE? (12 min presentation, 3 min questions)
Ignasi Marco

85) CAPTURE AND ANAESTHESIA PROTOCOL OF ARABIAN ORYX AND ITS EFFECT ON BLOOD PARAMETERS IN CLIMATE CONDITION OF MIDDLE EAST (12 min presentation, 3 min questions)
L. Molnar

2.45pm **Afternoon Tea and Poster Session**

3.30pm **Concurrent Session: Lockhart Room**
Miscellaneous Tools and Techniques
(Chair: Margaret Wild)

86) ESTIMATING DISEASE INCIDENCE FROM AGE-SPECIFIC PREVALENCE DATA IN THE PRESENCE OF SIGNIFICANT DISEASE-INDUCED MORTALITY AND SPATIAL AGGREGATION (12 min presentation, 3 min questions)
Peter Caley

87) A NOVEL MOLECULAR-ECOLOGY APPROACH TO ASCERTAINING EMIGRATION / IMMIGRATION AND POTENTIAL DISEASE SPREAD IN FERAL PIGS (12 min presentation, 3 min questions)
Brendan Cowled

88) USE OF INFRARED THERMOGRAPHY TO DETECT RABIES INFECTION IN RACCOONS (*PROCYON LOTOR)* (12 min presentation, 3 min questions)
Mike R. Dunbar

89) USE OF SATELLITE TELEMETRY TO STUDY THE MOVEMENT OF THE MALAYAN FLYING FOX (*PTEROPUS VAMPYRUS*): IMPLICATIONS FOR CONSERVATION AND PUBLIC HEALTH (12 min presentation, 3 min questions)
Craig S. Smith

90) DEVELOPMENT OF NEW IMMUNOLOGICAL DETECTION METHODS FOR USE IN THE DUCK (12 min presentation, 3 min questions)
Karen Vickery

91) EVALUATION OF CHEMICAL IMMOBILIZATION AGENTS FOR ANAESTHETIZING AMERICAN BEAVER (*CASTOR CANADENSIS)* (12 min presentation, 3 min questions)
Thomas J. DeLiberto

3.30pm **Concurrent Session: Palmerston Room**
Endangered and Captive Animals
(Chair: Wendy Blanshard)

92) DISEASE IN THE ENDANGERED MAURITIAN PINK PIGEON *COLUMBA MAYERI* (12 min presentation, 3 min questions)
Nancy Bunbury

93) HEMATOLOGIC AND SERUM BIOCHEMISTRY VALUES FOR FREE-RANGING NORTHERN HAIRY-NOSED WOMBATS (*LASIORHINUS KREFFTII*) (12 min presentation, 3 min questions)
Tim Portas

94) COMPARISON OF ANTI-PHOSPHOLIPID ANTIBODIES BETWEEN WILD AND CAPTIVE BLACK RHINOCEROS (*DICEROS BICORNIS*): IMPLICATIONS FOR HEALTH AND REPATRIATION (12 min presentation, 3 min questions)
Ray L. Ball

95) VETERINARY DIAGNOSTIC EVALUATIONS OF GIANT PANDAS WITH CLINICAL AND SUBCLINICAL DISEASE AT THE CHENGDU RESEARCH BASE FOR GIANT PANDA BREEDING, CHINA (12 min presentation, 3 min questions)
Kati Loeffler

96) AN OUTBREAK OF *BORDETELLA BRONCHISEPTICA* INFECTION IN A CAPTIVE COLONY OF TAMMAR WALLABIES (*MACROPUS EUGENII*) (12 min presentation, 3 min questions)
Catherine A. Herbert

97) *MYCOBACTERIUM TUBERCULOSIS* INFECTION IN ELEPHANTS (*ELEPHAS MAXIMUS* and *LOXODONTA AFRICANA*) IN SWEDEN (12 min presentation, 3 min questions)
Dolores Gavier-Widen

5.00pm **Day Four Finish**

Evening Activities

Conference Dinner – Hartley's Crocodile Adventures

5.15pm	Pick up from Colonial Club Conference Centre Reception
6.00pm – 7.40pm	Pre-dinner drinks and tour of wetlands and sanctuary
7.40pm – 10.00pm	Dinner and awards

OR

"Reef Teach"
Shuttle bus leaves **main Colonial Club Resort Reception** at 6.00pm. The talk is from 6.15pm – 8.30pm in town. There will be no return bus.

Friday, July 1

7.30am – 8.00am **Registration and Information Desk open (and during Morning and Afternoon Tea breaks, and Lunch)**

8.00am **Health of Marine Ecosystems**
(Chair: Janine Samuel)

98) RECENT DISCOVERY OF BRUCELLA INFECTION IN NEW ZEALAND HECTOR'S DOLPHINS (*CEPHALORHYNCHUS HECTORI HECTORI*) – A POTENTIAL CAUSE OF LOW FECUNDITY AND HIGH PERINATAL MORTALITY (12 min presentation, 3 min questions)
Pádraig J. Duignan

99) NEW VARIANT OF *BRUCELLA* FROM WHALES INHABITING WESTERN NORTH PACIFIC (12 min presentation, 3 min questions)
Tadashi Maruyama

100) THE ST. LAWRENCE ESTUARY BELUGA: A TALE OF A WHALE (12 min presentation, 3 min questions)
Lena N. Measures

101) IMPACT OF GLOBAL WARMING ON THE NESTING OF MARINE TURTLES IN THE UNITED ARAB EMIRATES (12 min presentation, 3 min questions)
Vijaya Kumar

102) SUB-LETHAL AND LONG-TERM EFFECTS OF EXPOSURE TO DOMOIC ACID IN STRANDED CALIFORNIA SEA LIONS (12 min presentation, 3 min questions)
Frances M. D. Gulland

103) SEROLOGICAL EVIDENCE OF INFLUENZA A VIRUS INFECTION IN SEALS INHABITING RUSSIA AND IN CETACEANS IN THE NORTH PACIFIC AND ANTARCTIC OCEAN (12 min presentation, 3 min questions)
Kazue Ohishi

104) AN IMPROVED CELL CULTURE METHOD FOR THE ISOLATION AND STUDY OF MARINE MAMMAL DISTEMPER VIRUSES **(**12 min presentation, 3 min questions)
Ole Nielsen

105) EFFECTIVE, FIELD-BASED INHALATION ANAESTHESIA FOR CRABEATER SEALS (*LOBODON CARCINOPHAGUS*) (12 min presentation, 3 min questions)
Julie Barnes

106) DETECTION OF PATHOGENIC PROTOZOA IN MARINE ECOSYSTEMS USING MUSSELS (*MYTILUS* SPP.) AS BIOINDICATORS (12 min presentation, 3 min questions)
Patricia Conrad

10.15am **Morning Tea**

10.45am **Wildlife Health in the Tropics**
(Chair: Joanne Connolly)

107) THE CONSEQUENCES OF HAEMOGREGARINE INFECTION IN A TROPICAL SNAKE (12 min presentation, 3 min questions)
Cathy M. Shilton

108) THE IMPACT OF DISEASE ON FREE LIVING CASSOWARY POPULATIONS IN FAR NORTH QUEENSLAND (12 min presentation, 3 min questions)
Annabelle Olsson

109) CAUSES OF DEATH FOR MOUNTAIN GORILLAS (*GORILLA BERINGEI BERINGEI* AND *G. B.* UNDECIDED) FROM 1968-2004: AN AID TO CONSERVATION PROGRAMS (12 min presentation, 3 min questions)
Felicia B. Nutter

110) AN OUTBREAK OF POXVIRUS INFECTION IN TWO GROUPS OF MOUNTAIN GORILLAS (*GORILLA BERINGEI BERINGEI*) (12 min presentation, 3 min questions)
Christopher A. Whittier

111) IMMOBILIZATION AND ANESTHESIA OF AMERICAN ALLIGATORS (*ALLIGATOR MISSISSIPPIENSIS*): A BRIEF REVIEW AND UPDATE (12 min presentation, 3 min questions)
Darryl Heard

112) UNRECOGNISED AMPHIBIAN DISEASE PROBLEMS IN FAR NORTH QUEENSLAND, AUSTRALIA (12 min presentation, 3 min questions)
Deborah Pergolotti

12.15pm **Lunch (Homestead Restaurant)**

1.15pm **Marsupials (Chair: Pam Whiteley)**

113) *CRYPTOSPORIDIUM* IN MARSUPIALS: OCCURRENCE, INFECTION PATTERNS, GENETIC CHARACTERISATION AND PUBLIC HEATH IMPACTS (12 min presentation, 3 min questions)
Michelle Power

114) EPIDEMIOLOGY AND HOST SPECIFICITY OF *CRYPTOSPORIDIUM* IN EASTERN GREY KANGAROOS (12 min presentation, 3 min questions)
Corina Radu

115) INVESTIGATION OF CUTANEOUS PAPILLOMATOSIS AND OCULAR CHLAMYDIALES INFECTION AFFECTING ENDANGERED WESTERN BARRED BANDICOOTS (*PERAMELES BOUGAINVILLE*) IN THE WILD (12 min presentation, 3 min questions)
Mandy O'Hara

2.00pm **Reptiles and Birds (Chair: Pam Whiteley)**

116) INVESTIGATIONS INTO SNAKE VIRAL DISEASES IN AUSTRALIA (12 min presentation, 3 min questions)
Tony Ross

117) OPHIDIAN PARAMYXOVIRUS AND INCLUSION BODY DISEASE OF BOID SNAKES: EXPERIENCES FROM THE UNITED STATES (12 min presentation, 3 min questions)
Elliott Jacobson

118) PSITTACID HERPESVIRUS (PsHV-1) IN AUSTRALIA (12 min presentation, 3 min questions)
Larry Vogelnest

119) PERSISTENCE OF ANTIBODIES TO WEST NILE VIRUS IN NATURALLY INFECTED ROCK PIGEONS (*COLUMBA LIVIA*) (12 min presentation, 1 min questions)
Samantha E. J. Gibbs

3.00pm **Afternoon Tea**

3.30pm **Lagomorphs (Chair: Sue Bigwood)**

120) THE IMPACT OF OVINE NEMATODES ON THE EUROPEAN HARE (*LEPUS EUROPAEUS*) (12 min presentation, 3 min questions)
Philip Stott

121) EVOLUTIONARY ASPECTS OF EUROPEAN BROWN HARE SYNDROME VIRUS (EBHSV) (12 min presentation, 3 min questions)
Kai Frölich

122) EPIDEMIOLOGICAL AND MOLECULAR EVIDENCE FOR THE FIRST BIOLOGICAL VECTOR AND RESERVOIR HOST OF MYXOMA VIRUS IN THE UK (12 min presentation, 3 min questions)
Dawn Wilkinson

4.15pm **General (Chair: Sue Bigwood)**

123) *SALMONELLA* SPP. IN CALIFORNIA WILDLIFE: A COMPARISON WITH INVERTEBRATE, DOMESTIC ANIMAL, AND HUMAN ISOLATES (12 min presentation, 3 min questions)
Woutrina Miller

124) AVIAN INFLUENZA VIRUS: A NEW PATHOGEN FOR WILD AND DOMESTIC FELIDS (12 min presentation, 3 min questions)
Thijs Kuiken

125) USE OF ANTICOAGULANT RODENTICIDES AND WILDLIFE POISONING: FROM THE BEST TO THE WORST (12 min presentation, 3 min questions)
Philippe Berny

5.05pm **Close of Conference**

Optional Evening Activities

6.15pm – 10.30pm **Rainforest Mammal Spotlighting on Atherton Tablelands**
Please sign up for spotlighting at board in reception by morning tea on Thursday. Information is available at boards. Meet at Conference Centre Reception.

OR

Visit Cairns Frog Hospital By arrangement with Deborah Pergolotti

OR

"Reef Teach"
Shuttle bus leaves **main Colonial Club Resort Reception** at 6.00pm. The talk is from 6.15pm – 8.30pm in town. There will be no return bus.

LIST OF POSTERS

Non-Student Posters

126) PENTASTOMIASIS AND SPARGANOSIS IN A BANDED MONGOOSE (*MUNGOS MUNGO*)
Erik Ågren

127) ANTIBODY FREQUENCY, PREVALENCE AND POTENTIAL IMPACT OF *COXIELLA*, *SALMONELLA*, AND *CHLAMYDOPHILA* INFECTIONS ON CHAMOIS (*RUPICAPRA RUPICAPRA)*
Marc Artois

128) REVIEW: RE-EMERGING PARASITES AND INVASIVE SPECIES ISSUES IN JAPAN
Mitsuhiko Asakawa

129) RESEARCH AND EDUCATIONAL ACTIVITIES OF THE WILD ANIMAL MEDICAL CENTER IN RAKUNO GAKUEN UNIVERSITY, JAPAN
Mitsuhiko Asakawa

130) TYPE E BOTULISM IN WILD BIRDS ON THE LOWER GREAT LAKES: A CONSEQUENCE OF INVADING ALIEN SPECIES?
Ian Barker

131) EFFECTS OF *CLOSTRIDIUM BOTULINUM* TYPE E NEUROTOXIN IN GREAT LAKES FISH: IMPLICATIONS FOR TRANSMISSION OF AVIAN BOTULISM TO FISH-EATING BIRDS
Ian Barker

132) BASELINE PHYSIOLOGICAL PARAMETERS FOR TASMANIAN DEVILS
Jemma Bergfeld

133) FIELD LABORATORY DIAGNOSIS OF *BACILLUS ANTHRACIS* INFECTION FROM SWAB AND INSECT SAMPLES COLLECTED FROM DEAD BISON
Gary Carter

134) COMPARISON OF STANDARD SEROLOGICAL TESTS TO THE FLUORESCENCE POLARIZATION ASSAY FOR THE DETECTION OF SERUM ANTIBODIES TO *BRUCELLA ABORTUS* IN MONTANA ELK (*CERVUS ELAPUS*)
P. Ryan Clarke

135) SUSCEPTIBILITY OF GREATER SAGE GROUSE TO EXPERIMENTAL INFECTION WITH WEST NILE VIRUS
Larry Clark

136) CAPTIVE VERTEBRATE MANAGEMENT BY DISTANCE EDUCATION THROUGH CHARLES STURT UNIVERSITY
Joanne Connolly

137) SALMONELLA SPECIES IN PINNIPEDS IN NEW ZEALAND
Joanne H. Connolly

138) *LIBYOSTRONGYLUS* SP. IN OSTRICHES: ASSESSING DISEASE RISKS TO KIWI
Susan Cork

139) ORAL DELIVERY MICROBIAL VACCINES FOR WILDLIFE
Martin L. Cross

140) THE UNITED STATES DEPARTMENT OF AGRICULTURE, ANIMAL AND PLANT HEALTH INSPECTION SERVICE, WILDLIFE SERVICES' NATIONAL WILDLIFE DISEASE SURVEILLANCE AND EMERGENCY RESPONSE SYSTEM
Thomas J. DeLiberto

141) FIELD LABORATORY DIAGNOSIS OF *BACILLUS ANTHRACIS* INFECTION FROM TISSUE AND BODY FLUID SAMPLES FROM DEAD BISON AND A MOOSE IN THE WOOD BUFFALO NATIONAL PARK
William Dorman

142) EXPRESSION OF IMMUNOLOGICAL MOLECULES IN THE DEVELOPING IMMUNE TISSUES OF THE TAMMAR WALLABY (*MACROPUS EUGENII*)
Rebecca L. Carman

143) IMPACT OF *KLEBSIELLA PNEUMONIAE* EPIDEMICS ON NEW ZEALAND SEA LION RECRUITMENT
Pádraig J. Duignan

144) PATTERNS OF GENETIC RELATEDNESS AMONG WHITE-TAILED DEER FROM A CHRONIC WASTING DISEASE ENDEMIC AREA
Holly Ernest

145) PATCH: A SPATIALLY EXPLICIT WILDLIFE POPULATION MODEL FOR ASSESSING RISKS OF PESTICIDES TO SONGBIRDS
Anne Fairbrother

146) NON-NATIVE ANIMAL PATHOGENS AS INDICATORS OF ECOSYSTEM CONDITION
Sylvia M. Fallon

147) HEAVY METAL POISONING AS A REASON FOR PRESENTATION IN WILD AUSTRALIAN PARROTS
Anne Fowler

148) LACK OF PASSIVE TRANSFER IN A NEONATAL RIGHT WHALE CALF (*EUBALENA GLACIALIS*)
Kendal E. Harr

149) EXPERIMENTAL CROSS-SPECIES TRANSMISSION OF TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES (TSEs) AT THE NATIONAL ANIMAL DISEASE CENTER (NADC), AMES, IOWA, USA: AN UPDATE
Amir N. Hamir

150) EFFECTS OF TEMPERATURE AND DURATION OF SAMPLE STORAGE ON HAEMATOLOGICAL CHARACTERISTICS OF WESTERN GREY KANGAROOS (*MACROPUS FULIGINOSUS*)
Lisa Hulme-Moir

151) ADVERSE EFFECT OF SELECTED XENOBIOTIC ON CHOLINESTERASE ACTIVITY IN *RANA CYANOPHLYCTIS* BRAIN, LIVER AND KIDNEY
Muhammad Zaheer Khan

152) DEVIL FACIAL TUMOUR DISEASE (DFTD) IN TASMANIAN DEVILS (*SARCOPHILUS HARRISII*) – THE GROSS, HISTOLOGICAL, ULTRASTRUCTURAL AND IMMUNOHISTOCHEMICAL CHARACTERISTICS OF THE NEOPLASM
R Loh

153) RELATIONSHIP BETWEEN INTER-TIDAL VEGETATION AND RAINFALL IN ABU DHABI, THE UNITED ARAB EMIRATES
R. A. Loughland

154) TICK PARALYSIS AND CLEFT PALATE SYNDROME IN SPECTACLED FLYING FOXES
Jenny Maclean

155) FIELD SURVEY: SOUTH AFRICAN CLAWED FROGS (*XENOPUS LAEVIS*) IN GOLDEN GATE PARK, SAN FRANCISCO, CA
Emily K. Matz

156) SEROSURVEY FOR ANTIBODIES TO WEST NILE VIRUS IN WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*) FROM IOWA (1999-2003)
Robert McLean

157) INFECTIVITY OF *CRYPTOSPORIDIUM* OOCYSTS FROM *MACROPUS GIGANTEUS* (EASTERN GREY KANGAROO) IN HUMAN GUT EPITHELIAL CELLS
Cushla Metcalfe

158) PRESENCE OF ENDOVIRAL (EV) GENES OF AVIAN LEUKOSIS AND SARCOMA VIRUSES IN SOME WILD SPECIES OF BIRDS
T.R. Mohanty

159) DETECTION OF ANTI- *TOXOPLASMA GONDII* ANTIBODIES IN CAPTIVE AND WILD TOOTHED WHALES BY SEROLOGICAL METHODS FOR DIAGNOSIS OF *TOXOPLASMA GONDII* INFECTION
Koichi Murata

160) SEROLOGIC EVIDENCE OF DISTEMPER, BRUCELLOSIS, AND TOXOPLASMOSIS CIRCULATING IN STELLER SEAL LIONS IN THE NORTH WESTERN PACIFIC OCEAN
Ole Nielsen

161) CLEFT PALATE IN A NEONATAL VIRUNGA MOUNTAIN GORILLA (*GORILLA BERINGEI BERINGEI*)
Felicia B. Nutter

162) MANGE CAUSED BY *PANGORILLALGES GORILLAE* IN THREE VIRUNGA MOUNTAIN GORILLAS (*GORILLA BERINGEI BERINGEI*)
Felicia B. Nutter

163) THE USE OF A TADPOLE SURVEY METHOD COMBINED WITH A CHYTRID-SPECIFIC POLYMERASE CHAIN REACTION TEST TO DECTECT CHYTRIDIOMYCOSIS INFECTION IN FREE-RANGING TASMANIAN AMPHIBIAN POPULATIONS
David L. Obendorf

164) CLINICAL COCCIDIOSIS DUE TO *CARYOSPORA* (APICOMPLEXA) IN TAWNY FROGMOUTHS, *PODARGUS STRIGOIDES* (CAPRIMULGIFORMES) IN AUSTRALIA
Peter O'Donoghue

165) HAEMATOLOGY AND SERUM BIOCHEMISTRY OF THREE NATIVE AUSTRALIAN DESERT MURIDS
Julie M. Old

166) INCIDENCE OF CRYPTOCOCCAL DISEASE AT PERTH ZOO - AN EPIDEMIOLOGICAL INVESTIGATION
Karen L. Payne

167) SEQUENCING OF THE HEPATITIS B VIRUS IN A SILVERY GIBBON (*HYLOBATES MOLOCH*) AT PERTH ZOO
Karen L. Payne

168) CYTOGENETIC SUPPORT OF THE ALLOGRAFT THEORY OF TRANSMISSION OF DEVIL FACIAL TUMOUR DISEASE (DFTD) IN TASMANIAN DEVILS *(SARCOPHILUS HARRISII)*
Anne Maree Pearse

169) SEROLOGICAL EVIDENCE OF EXPOSURE OF WILD MAMMALS TO FLAVIVIRUSES IN THE CENTRAL AND EASTERN UNITED STATES
J. Jeffrey Root

170) MANAGEMENT OF EPISTAXIS IN A TIGER
A. B. Shrivastav

171) THE IMPACT OF PHYTOESTROGENIC INFERTILITY ON THE EUROPEAN HARE (*LEPUS EUROPAEUS*)
Philip Stott

172) AVIAN MALARIA IN NEW ZEALAND: HAWAII ALL OVER AGAIN?
Daniel M. Tompkins

173) USE OF TRICLABENDAZOLE/LEVAMISOLE MEDICATED FEED PELLETS IN THE CONTROL OF NATURALLY INDUCED LARGE AMERICAN LIVER FLUKE (*FASCIOLOIDES MAGNA*) INFECTION IN FREE-RANGING RED DEER (*CERVUS ELAPHUS ELAPHUS*) IN CROATIA
Ivan Vickovic

174) SEROPREVALENCE OF INFECTIOUS AGENTS IN FREE LIVING MOUNTAIN GORILLAS (*GORILLA BERINGEI* SSP)
Christopher Whittier

175) EQUINE HERPESVIRUS 9 (EHV-9)-INDUCED ENCEPHALITIS IN ANIMALS; PATHOLOGY AND EXPERIMENTAL INFECTIONS
Tokuma Yanai

Student Posters

176) ISOLATION AND IDENTIFICATION OF *TRYPANOSOMA* SPECIES IN GILBERT'S POTOROO (*POTOROUS GILBERTII*)
Jill Austen

177) CONSERVING OUR NATIVE CARNIVORES: THE APPLICATION OF MOLECULAR GENETICS TO THE CONSERVATION MANAGEMENT OF QUOLLS
Maria J. Cardoso

178) INVESTIGATIONS INTO THE DEVELOPING GUT MICROFLORA OF THE TAMMAR WALLABY
Kim Ly Chhour

179) URBAN INDUCED INFLUENCES ON THE HEALTH OF FRAGMENTED POPULATIONS OF *ISOODON OBESULUS*
Matthew G. Dowle

180) SEROPREVALENCE OF PATHOGENS IN AN ISLAND POPULATION OF WILD OCELOTS (*LEOPARDUS PARDALIS*)
Samuel P. Franklin

181) INTERACTION OF MULTIPLE PATHOGENS AND HABITAT ALTERATION ON CALIFORNIA RED-LEGGED FROGS (*RANA AURORA DRAYTONII)* ALONG THE CENTRAL CALIFORNIA COAST
Valentine Hemingway

182) PLASMA BIOCHEMISTRY AND HEMATOLOGICAL VALUES FOR WILD-CAUGHT FLYING FOXES (*PTEROPUS GIGANTEUS*) IN INDIA
Amanda B. McLaughlin

183) PORPHYRIA OBSERVATIONS ON THE CANEFIELD RAT (*RATTUS SORDIDUS*)
Dario F. Rivera

184) PRESENCE OF *ANGIOSTRONGYLUS* SPECIES IN POPULATIONS OF *RATTUS RATTUS* AND *RATTUS FUSCIPES* IN COASTAL FORESTS OF NSW, AUSTRALIA
Vicki Stokes

185) SPATIAL PATTERNS OF THE AMPHIBIAN PATHOGEN, *BATRACHOCHYTRIUM DENDROBATIDIS* IN EUROPE
Susan Walker

186) HAEMATOLOGY OF HEALTHY AND MORBID SOUTHERN BROWN BANDICOOTS (*ISOODON OBESULUS*)
R. M. Wicks

187) REAL-TIME PCR DETECTION OF *CAMPYLOBACTER* IN WILD MOUNTAIN GORILLAS (*GORILLA BERINGEI BERINGEI*)
Christopher A. Whittier

PRESENTATION ABSTRACT - PLENARY

1) OVERVIEW OF WILDLIFE HEALTH IN AUSTRALASIA – AN ASSESSMENT OF THE ECOLOGY, MANAGEMENT, AND THE IMPACT ON CONSERVATION, OF INTRODUCED DISEASES

David M. Spratt

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The Australasian zoogeographic region is represented by those countries east of the Wallace and Weber lines where the fauna of the Australasian and Oriental regions meet. Australasia differs in many ecological features, something best appreciated through an understanding of the formation of Australasia and the consequences from geological, botanical and zoological viewpoints. From the time of separation of Australasia from Antarctica in the early Tertiary about 96 million years ago (mya), until the collision of the Australian and Asian plates in the Miocene, about 10 mya, plants and animals were isolated and diversified over a period of at least 50 million years. The marsupials in particular occupied most of the major habitats available in Australia and New Guinea, although never colonising New Zealand.

As a consequence of these historical events, the evolution of organisms causing disease in wildlife progressed in isolation as well. Psittacine (circoviral) beak and feather disease is the most significant disease of wild and captive psittacine birds in Australia and has seriously affected the captive breeding program of the endangered orange-bellied parrot. Wallal and possibly Warrego virus cause chorioretinitis in kangaroos, resulting in blindness and significant mortalities often over wide geographic areas. Disease outbreaks usually are associated with prior flooding and large populations of blood-feeding insects, as the waters from the inland rivers gradually evaporate. Biting midges (*Culicoides* spp.) have been detected carrying these viruses during a disease outbreak, although their true role as vectors remains undetermined. Epizootic haematopoietic necrosis virus, the first virus isolated from finfish in Australia, is an indiscriminate pathogen. It lacks host specificity, is an important disease in recreational and farmed fisheries, and an ecosystem management threat. Pilchard herpes virus has caused collateral damage in piscivorous birds. Chlamydiosis caused by *Chlamydophila pneumoniae* and *C. pecorum* is common and may cause respiratory disease, proliferative conjunctivitis and chronic fibrotic disease of the urogenital tract of koalas, frequently resulting in infertility and death. A neoplasia, devil facial tumour disease, is currently devastating populations of the Tasmanian devil. Lumpy jaw is a commonly occurring disease whenever large congregations of macropodids occur due to conditions in captivity or in nature during floods or droughts.

The short-necked or western swamp tortoise was re-discovered in the early 1960s but captive breeding programs of this critically endangered species have been hampered by a necrotising dermatitis caused by an unidentified species of *Pseudomonas*. Mortalities attributable to the fungus, *Mucor amphibiorum*, occurred in frogs, which had been introduced as a food-source (tadpoles) in a new, purpose-built enclosure. Fortunately, there was no illness nor wide-spread mortalities in the tortoises, as occurs in free-ranging platypuses in northern Tasmania infected with this fungus.

Lyssa virus, Hendra virus and Menangle virus, all associated with flying foxes, have emerged in Australia in the past decade and been responsible for deaths in humans, horses and pigs. Knowledge of many of these diseases has accumulated relatively recently. With the exception of duck hunting, which is now almost completely suppressed, game hunting is not a recreational activity and wildlife management tool in Australia, as occurs particularly in the northern hemisphere and parts of Africa. Consequently, there is no hunting lobby, dollars for

wildlife disease research have been scarce and there has been no central body to which die-offs were reported.

The arrival of the old endemic rodents 5–8 mya, the new endemic rodents about 1 mya, aboriginal people about 40,000-50,000 years ago, the dingo about 3,500 years ago and especially European man little more than 200 years ago has played havoc with the Australian environment and the ecosystems therein. As a consequence, Australia has the dubious honour of having one of the richest assemblages of introduced animals in the world. Foxes, rabbits, deer, water buffalo, pigs, camels, goats, cats, goldfish, carp, sparrows and starlings prospered. Having escaped the constraints imposed upon them by their original ecological environments and associated parasites and pathogens, a number of these immigrants are now some of our most serious pests, the European rabbit being the classic example. However, introduced disease has been used as a tool to control this immigrant pest. Australia boasts the only two successes in biological control of a vertebrate pest in the world. Myxoma virus, introduced in 1950, reduced rabbit numbers by an estimated 99%. Subsequently, three years after the introduction of rabbit calicivirus to inland areas of the continent in 1995, numbers remained at 10-15% of their former levels, enabling significant regeneration of pastures and native plant species.

Australia stands in marked contrast to East Africa where domestic animals acquired a number of diseases from wildlife. In Australia, wildlife have acquired some diseases (particularly parasitic) of domestic animals, with drastic effects on populations and a substantial impact on the conservation of some of Australia's unique fauna. *Toxoplasma gondii, Sarcoptes scabiei, Echinococcus granulosus* and *Fasciola hepatica* infection have been acquired from domestic animals and are significant pathogens in a number of marsupial and murid species. Larval plerocercoids of *Spirometra erinacei* occur in free-ranging dasyurid marsupials, echidnas and platypuses. *Leptospira interrogans* serovar *hardjo* also occurs in wild platypuses. The chytrid fungus, *Batrachochytrium dendrobatidis*, has played a significant role in the extinction of one and the demise of many stream-dwelling amphibian species, especially in the montane rainforests of Queensland. The air-sac mite, *Sternostoma tracheacolum*, probably introduced to Australia in aviary birds, causes a bronchopneumonia in the endangered Gouldian finch and may now be suppressing the return of the finch to its former status.

Until recently, Australia and Oceania were considered the only regions of the world where species of *Leishmania* did not occur. No longer is this the case; skin lesions have been observed on the ears, tails and limbs of red kangaroos in a fauna park near Darwin. The causative organism belongs to the genus *Leishmania* but could not be assigned to a known species and its origin is unknown. The Australian brushtail possum has proven to be the most serious "disease" in New Zealand. Indigenous forests in that country are seriously compromised due to the browsing of these introduced pests, which also play a significant role in the epidemiology of bovine tuberculosis.

The future of wildlife health in Australasia rests with the continued development of both the Australian Registry of Wildlife Health and its role as an invaluable wildlife health resource, and of the national Australian Wildlife Health Network.

PRESENTATION ABSTRACTS (BY SESSION)

SESSION 1:
THE ECOLOGY OF INTRODUCED WILDLIFE DISEASES

2) PATHOGEN POLLUTION: UNDERSTANDING THE CAUSES AND IMPACT OF INTRODUCED WILDLIFE DISEASES

Peter Daszak[1], A. Marm. Kilpatrick[1], Nikkita Patel[1], Jonathan H. Epstein[1], Lisa Schloegel[1], Kate Jones[2] and Andrew A. Cunningham[2]

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For decades, anthropogenic environmental changes such as habitat loss, overexploitation, chemical pollution, and more recently climate change have been considered as the major threats to biodiversity conservation. The introduction of non-native animal and plant species to new geographic areas (e.g. rats and cats to islands) has also been highlighted as a significant threat by the World Conservation Union (IUCN). However, the involvement of parasites in the human-mediated loss or modification of biodiversity has only recently been recognised. We have termed this specific form of anthropogenic threat "Pathogen Pollution" - the anthropogenic movement of parasites outside their natural geographic or host-species range. There are several ways that pathogen pollution can occur, but in each case anthropogenic change results in a parasite crossing an evolutionary boundary such as geographic or ecological separation. The effects of such pollution may be obvious and severe, such as the loss of Hawaiian bird species due to malaria and avian pox, or the rapid and marked decline of ungulate populations in Africa following the introduction of rinderpest. In some cases these effects are more subtle, long-term, and complex as millennia of endemic host-parasite co-evolution are rapidly disrupted, e.g. the continued loss of prairie dog (*Cynomys ludovicianus*) populations due to plague, introduced 150 years previously. Hence, the outcomes of many pathogen pollution incidents are potentially far-reaching and hard to predict. Our work on emerging wildlife diseases demonstrates three important points:

- Broad trends underlying the spread of wildlife diseases can be used to predict future introductions;
- Host ecological traits can provide a way of estimating the relative impact of pathogen pollution on different species; and
- Acknowledging this insidious form of ecological change inflicted by man on the environment is the first step to taking preventative or ameliorative action.

3) A GLOBAL HISTORICAL PERSPECTIVE ON DISEASE INTRODUCTIONS

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Over the past 50 years, a crescendo of insightful books and papers on global ecological and human history has provided a framework for beginning to understand the astounding expansion in the number and diversity of disease issues that beset humankind and the global environment today. This ecological history is a new view of our past, still absent from school texts, religious philosophies, and day-to-day political decisions. It explains, in broad outline, our current perilous state and offers a basis for choosing among the many possible societal paths into the future.

Disease introduction, like disease itself, results from a convergence of multiple causal factors. For both, Koch's postulates no longer apply and are replaced by a complex interplay among the epidemiological trinity of host, agent and environment. Thus, a disease may be introduced, and appear for a first time when a new pathogen is introduced into a population or community of susceptible hosts. That pathogen may be new by virtue of transportation or by virtue of in situ genotypic and/or phenotypic change. The introduction of new, susceptible hosts to the ecosystem of the pathogen can have the same effect as can in situ change in a host's susceptibility to an already sympatric pathogen. Perturbation of an ecosystem through changes in environmental conditions can, and often does, alter relationships among hosts and pathogens and result in new expressions and changing patterns of disease.

These mechanisms of disease introduction have operated in the biosphere for hundreds of millions of years. By and large, parasitism and disease, their introductions and emergences, appear to have been beneficial to the biosphere adding to ecosystem stability and resilience, complicating community dynamics with new layers of trade-offs and consequences, and perhaps even driving the evolution of sexual reproduction itself as a means by which multicellular hosts could generate genetic variation rapidly enough to evolve defences against prokaryotic pathogens. Animal migrations, environmental disturbances, and evolutionary changes in hosts and pathogens have been a feature of life on earth from the beginning.

The causes of the striking surge in intensity of the processes and events we refer to as 'disease introduction' and 'disease emergence,' which we now are witnessing, is woven into the cultural evolution of the human race beginning about 8,000-10,000 years ago, and the gradual development of agriculture. Agriculture caused human populations for the first time to reside permanently in one place, to co-opt land for their exclusive use, to increase in number and density, and to live closely and constantly with large numbers of animals. These conditions favoured the adaptation of animal pathogens to humans and probably are the origin of diseases like measles, an adapted form of rinderpest virus perhaps, small pox from related animal pox viruses, and influenza A, made a human pathogen by passage through domestic birds and pigs. Equivalent opportunities for exchange and adaptation of pathogens also occurred among the domesticated animals themselves, and between these and wild animals. Agriculture also produced surplus food, fuelling larger and denser human and animal populations capable of maintaining new acute pathogens of high virulence. It also made possible social elites and their armies, and a rapid evolution of technologists and technology, leading to faster and ever more distant transportation, trade, war and colonisation. Acute, high virulence pathogens for the first time could survive long distance journeys between susceptible populations, and large-scale epidemics entered recorded history. Bubonic plague

swept through Europe in the 6th and the 14th centuries, killing hundreds of millions of people. Small pox and other virulent pathogens endemic to the Old World were introduced to the New World through contact with Europeans in the 15th Century and killed 80 to 95% of the total population of 80-100 million people in a few decades. Aboriginal Australians, though fewer in number, suffered this same fate two centuries later. Disease introduction was the primary engine of European colonial dominance of the past 400 years. During this same period, and for the same reasons, disease introduction affected animal populations around the world, from the extinction of the native rats of Christmas Island to the devastating serial introductions of rinderpest by invading armies in Europe and across the whole of sub-Saharan Africa with their overwhelming effects on human food supply and social institutions, and on wild animal populations. Avian influenza in South-East Asia, BSE, Chronic Wasting Disease, West Nile virus in North America, tuberculosis in Kruger National Park in South Africa, and perhaps the epidemic of facial tumours in Tasmanian Devils are some current episodes in this long-playing series.

At the start of the 21st Century, disease introduction has become a major threat to the biological security of humankind. Our current population of 6.3 billion people is projected to grow to 8-12 billion over the next 50 years. Our global food supply is totally dependent on vast monocultures of a handful of domesticated plant and animal species. We have never been more vulnerable to disease. Historically, each cycle of technological gain in food and energy production over the past 7,000 years has immediately been matched and exceeded by human population growth and resource consumption. Today we are pursuing all of the activities that favour disease introduction and emergence at unprecedented and accelerating rates and scales. As a consequence, we and our fellow residents of the biosphere are suffering new diseases on an exponentially rising trajectory. Our challenge as disease specialists is to understand disease dynamics sufficiently to manipulate proximate causes and reduce their impacts on ourselves and on the ecosystems that sustain us. The larger challenge, however, is to address the ultimate cause of our biological insecurity, which is too many people collectively consuming more resources than the earth can sustain. Our peril is one of scale, not of kind. During the next 100 years, the global limits of sun and soil will impose a crest and decline in the earth's human population. Thomas Malthus proposed, in his Essay on the Principle of Population of 1798, that such population decline can be achieved only through war, famine and disease. However, in a revision of his Essay in 1803, Malthus offered the alternative hypothesis that human populations could achieve sustainable self-regulation through "moral restraint." Thus far, history has fully supported Malthus' original hypothesis. As both world citizens and students of diseases, we must use all of our knowledge and persuasive powers to prove his second hypothesis also to be correct.

4) EMERGING EPIDEMIOLOGICAL PATTERNS OF RABBIT HAEMORRHAGIC DISEASE IN AUSTRALIA

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In arid inland Australia, Rabbit Haemorrhagic Disease (RHD) reduced most rabbit populations by at least 80%, and populations remain low eight years after its initial spread. Disease activity in arid areas generally begins a month or two after the commencement of breeding in autumn or winter, declines in late spring and ceases to be apparent in summer. RHD severely suppresses rabbit numbers during the main breeding season. However, compensatory recruitment of late-born young, protected by maternal antibodies until the disease becomes inactive at the end of spring (also the end of breeding) allows the observed rabbit abundance to increase during summer, albeit to lower levels than pre-RHD. Maternal antibodies are lost during summer and the population becomes susceptible to further RHD outbreaks in the following autumn/winter. The seasonal pattern of population change observed now is almost the inverse of the former pattern because before RHD, recruitment into rabbit populations occurred during winter/early-spring and they declined in late-spring/summer due to malnutrition and myxomatosis.

In higher-rainfall temperate agricultural regions, RHD has had a smaller and more variable impact on rabbit abundance. Five factors are identified as contributing to why RHD has affected rabbit populations less in these areas:

- Higher productivity allows rabbit populations to quickly recover from losses to RHD;
- Different seasonal timing of RHD outbreaks allows high survival of juveniles;
- Another related calicivirus may protect against RHD;
- Persistence and transmission of RHD virus may be poor;
- Possible negative interactions between RHD and myxomatosis.

5) ECOLOGY OF AVIAN MALARIA IN NATIVE HAWAIIAN FOREST BIRDS

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The introduction of mosquitoes and avian malaria to Hawaii and subsequent decline of endemic honeycreepers provides a premier example of the effect of introduced disease on susceptible island populations. Malaria is believed one of the primary factors responsible for native bird disappearance at lower elevations and the restriction of highly susceptible species to upper elevation forests. Outbreaks of malaria in native Hawaiian bird populations depend on environmental conditions which drive mosquito abundance and disease transmission, and the relative abundance of susceptible birds in the population. Based on field investigations, laboratory studies, computer modelling, analysis of malaria prevalence, and climate patterns we evaluated the dynamics of avian malaria infection in apapane in mid-elevation forests on the island of Hawaii. Field investigations suggest that evolution in disease resistance has occurred in low-elevation amakihi populations. Our research provides a linkage between annual climate variation (especially rainfall), mosquito abundance, epizootic disease events, and host impacts. These linkages help explain annual cycles and elevational gradients in the dynamics of avian malaria. Restoration of Hawaiian forest bird communities will depend on management strategies based on the dynamics of malaria at the landscape and evolutionary scales.

6) A COMMUNITY BASED APPROACH TO UNDERSTANDING AND DETECTING 'INTRODUCED' OR 'EMERGING' DISEASES IN THE CANADIAN NORTH

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The Canadian North is experiencing substantial environmental disturbances driven primarily by an unprecedented rate of climate change and increased exploration, and development of non-renewable resources. Resulting habitat alterations have already influenced and will continue to shape the life history patterns, distribution, and abundance of both wildlife species and their pathogens. For example, recent climate warming in the central Canadian Arctic has caused a switch in the life cycle of the lungworm *Umingmakstrongylus pallikuukensis* from a primarily 2-year cycle to a single-year cycle with significant consequences for the health of its musk ox host. Climate warming is expected to relax constraints on other infectious agents (and their vectors) and allow their invasion to higher latitudes and altitudes. Concomitantly, the constantly expanding human footprint is opening the wilderness to people and their domestic animals, which increases the risk of anthropogenic introduction of novel infectious agents. This rapid rate of climate and other environmental changes in the north threatens to disrupt the balance of these 'systems on the edge', with significant biological implications for the ecosystem. There are also potential adverse medical and sociological consequences for the people who depend on wildlife for subsistence, cultural, and economic purposes. The challenge for wildlife managers lies in establishing meaningful baselines for wildlife diseases followed by detecting and long-term monitoring of these diseases over a vast (3.6 million km^2) and sparsely populated (ca. 100,000 people) region. To address this challenge we have established an ongoing program to promote community-based monitoring of wildlife health in the Sahtu region of the west-central Northwest Territories.

There are four major components to this program:

1. Youth education - to encourage knowledge about wildlife health and interest in careers in wildlife management, conservation, and veterinary science for school-age learners.
2. Information exchange - with experienced harvesters (> 25 years of harvesting) and elders to record their observations on wildlife diseases in the past and the present, as well as to identify any concerns they may have regarding wildlife health.
3. Training of 'Wildlife Health Monitors' – to train experienced harvesters to collect specific biological samples from caribou and moose that they harvest for subsistence. Samples are analysed for various pathogens and contaminants, and subsets of samples are archived for future analyses as needed. These samples serve to establish baselines and will be used for long-term monitoring and surveillance of wildlife health
4. Graduate student involvement - to promote experiential education and cross-cultural learning. Additionally, graduate student participation is a critical component of maintaining continuity and promoting expansion of community-based programs over the long term.

All four components together serve to increase overall awareness of wildlife health in a changing environment, increase community-capacity, and promote ongoing building and

maintenance of linkages between northern communities and university/agency-based scientists. The Wildlife Health Monitor program and harvester and elder consultations help guide scientific research programs directed at understanding, predicting, and mitigating potential impacts of climate and other environmental changes on wildlife health in the north.

This 'Community-Based Monitoring of Wildlife Health' program has been running for three years and has gained tremendous support from the Sahtu Divisional Education Council, the Sahtu Renewable Resources Board (regional wildlife co-management board) and the five Renewable Resource Councils (community-based wildlife organisations). Although this program is still in the early stages, it promises to be an efficient way for scientists and communities to work together to monitor wildlife health over a large geographic region. Our success to date seems to stem from mutual trust and respect, a two-way exchange of information regarding scientific findings and harvester observations, and the commitment by our multi-agency team to annually visit each community to listen to and speak with youth, Wildlife Health Monitors, elders, and Renewable Resource Councils.

7) ECOLOGY OF PLAGUE AS AN INTRODUCED DISEASE AND ITS IMPACT ON THE CONSERVATION OF THREATENED AND ENDANGERED SPECIES

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The plague bacterium, *Yersinia pestis,* was introduced into U.S. seaports in the early 1900s via infected rodents and quickly spread into native rodents throughout the western states where it now recurs regularly in some wildlife populations. As a relatively recent introduction to the continent, most rodents and lagomorphs in North America are highly susceptible to the disease, and for some species, the disease is a serious conservation issue. More than half of the species of North American rodents of conservation concern reside within the range of plague outbreaks in western North America, including several species of prairie dogs (*Cynomys* spp.). The disease has extirpated prairie dogs in some areas of North America and often causes local extinctions and population reductions followed by partial recovery. The black-tailed prairie dog (*Cynomys ludovicianus*), once the most abundant mammal in North America, has declined significantly, and sylvatic plague was specifically identified as the most serious threat to the continued existence of this species throughout much of its range. Prairie dogs are considered a "keystone" species, serving a critical role in maintaining the biotic diversity and integrity of the western grasslands that stretch from southern Canada to Northern Mexico. Many animals use prairie dogs as a food resource, including badgers, canine predators, hawks, and owls, but the species most dependent on prairie dogs is the endangered black-footed ferret (*Mustela nigripes*). Because black-footed ferrets rely almost exclusively on prairie dogs for food and on prairie dog burrows for shelter, their management and recovery is tightly linked to prairie dog survival and management. Plague in prairie dog towns significantly impacts black-footed ferret survival by destroying their primary prey base. In addition, the black-footed ferret is also highly susceptible to the plague bacterium, suffering high mortality rates upon infection.

The epizootiology of plague in prairie dog colonies is complex. In some species (i.e. *C. leucurus*) plague is enzootic, while in others (*C. ludovicianus, C. gunnisoni*), the disease is epizootic with high mortality rates. The reasons for this are unknown, but the most important risk factors are presumed to be the susceptibility and sociability of the host species, the density and distribution of colonies, and the species and density of fleas present. Other factors hypothesised to play a role in the occurrence of plague epizootics in prairie dogs are the presence of other rodent species (and their associated fleas) that may harbour the disease between epizootics and the degree of inter-specific contact between those reservoirs and prairie dogs. Presumed to be primarily a flea-borne disease, recent evidence suggests that direct contact or pneumonic transmission may also play a crucial role in plague outbreaks in prairie dog colonies. Because of its wide host range, its pathogenicity in numerous species and its persistence over time, plague is an excellent case study of the direct and indirect impact of introduced disease at a community level.

8) CHYTRIDIOMYCOSIS AND AMPHIBIAN DECLINES

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Chytridiomycosis, an emerging pandemic infectious disease of amphibians, is capable of causing high mortality in susceptible amphibians in particular environments, even resulting in extinction of species. The aetiological agent, *Batrachochytrium dendrobatidis* (Bd), the amphibian chytrid fungus, is unusual in being a primary pathogen, often with very high virulence. Bd is an aquatic fungus with motile zoospores. How Bd kills infected frogs is still being investigated.

Epidemiological and molecular evidence indicates that Bd falls into that category of emerging infectious diseases (EID) of pathogens that have escaped from a source location owing to human activities. Epidemiological evidence suggests that Bd emerged from Africa, possibly in the late 1930s, when the trade in the African clawed frog, *Xenopus laevis*, was increased to meet the demand for human pregnancy tests. The scenario appears to be that Bd left Africa using *Xenopus* and established in North America possibly about the early 1960s. From North America Bd appears to have spread through natural movement on the ground and through humans moving infected amphibians and water. Bd is now moved around the world in amphibians in the pet trade, the scientific trade and the food trade, particularly by the American bullfrog, *Rana catesbeiana*. The host specificity of Bd is low. Current records show that Bd occurs in 20 countries globally infecting 143 species from 43 genera, 19 families and 2 orders, suggesting Bd may be capable of infecting most species of amphibians. The impact of infection varies by species along a continuum with some species of amphibians being highly susceptible while others, such as *X. laevis* and *R. catesbeiana*, can be infected, but rarely show signs of disease. In the wild the impact of infection with Bd is an interplay between pathogen, host, and environment, with low temperature increasing mortality. Salinity may also have a protective effect.

When Bd first enters the amphibian population in a region or country, it spreads through the environment, often first detected as an epidemic wave of amphibian mortalities. This pattern has been particularly well documented retrospectively in coastal Queensland and prospectively in Panama. However, it is important to realize that Bd can progress geographically irrespective of clinical effects and mapping location of Bd through mortality uses the outcome of the pathogen-host-environment interaction rather than the location of the pathogen per se. After invading an area, Bd becomes endemic (in epidemiological terms), and the pattern of disease manifestations can change, typically decreasing in severity. A pattern of sporadic deaths, usually with seasonal peaks associated with lower temperatures seems typical. Some amphibians in these locations appear to eliminate chytridiomycosis as temperatures rise. Prevalences of chytridiomycosis in these endemic situations range from 2% to 25% with peaks in autumn and winter. Chytridiomycosis is the most likely cause of the enigmatic amphibian declines highlighted by Stuart et al (2004). Bd has the capability of exerting a strong and rapid selection for innate resistance in amphibian hosts similar to the pattern seen for naïve populations of humans after exposure to measles virus. Even in humans this selection for innate resistance can occur within a generation or two.

One hypothesis proposes that frogs are irrelevant to the existence of Bd since it can grow and survive without their presence, a very hazardous situation for any host. Current research is attempting to clarify where Bd lives in complex natural environments, what conditions favour

its survival and growth, the role of amphibians in the epidemiology of chytridiomycosis, and whether environmental modification can tip the Bd-amphibian balance in favour of the host.

Stuart, S. N. *et al*. 2004. Status and trends of amphibian declines and extinctions worldwide. *Science* 306**:** 1783.

9) EMERGING INFECTIOUS DISEASES: THE PILCHARD EPISODE

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During the past decade many new diseases have emerged from the environment and into society where there have been impacts on human and/or veterinary health, trade and the 'health' of the environment. In nearly all cases the emergence can be attributed to environmental perturbations via some aspect of human behaviour. Examples of such environmental perturbations can include altered habitat (changes in the number of vector breeding sites and/or host reservoirs), niche invasions (interspecies host-transfers), changes in biodiversity, human-induced genetic changes of disease vectors or pathogens (e.g. mosquito resistance, emergence of disease resistant strains of microbes), and environmental contamination of infectious agents (e.g. dissemination of microbes into water bodies).

In 1995 a large-scale epizootic occurred in the Australasian pilchard *Sardinops sagax*. It has been suggested that this epizootic may have resulted from 'contamination' of the southern Australian coastline with an exotic virus. The deaths occurred along 5000 km of the Australian coastline and 500 km of the New Zealand coastline. Affected fish died within a few minutes of clinical signs of respiratory distress and death was associated with hypoxaemia and hypercapnia. Significant lesions were confined to the gills and comprised acute to subacute inflammation followed by epithelial hypertrophy and hyperplasia. The lesions were initially focal but progressed to become generalised over about 4 days. An exhaustive ultrastructural examination of gills resulted in the identification of an unidentified herpesvirus, the virus was not found in gills of unaffected pilchards. This large-scale mortality event was followed by a similar event in 1998/9. Though the origin of the herpesvirus may never be established, there are several implications for aquaculture and indeed the indiscriminate importation and release of organisms into the environment. In relation to aquaculture, the first is that the practice of feeding large tonnages of imported frozen fish as feed is an activity which could present a high quarantine risk. The second is that novel (previously unknown) viruses exist in the 'environment' and upon spillover to new hosts can cause new diseases that present threats to aquaculture activities. The final implication involves dealing with new and emerging diseases. For the rapid identification of causative agents involved in epizootics a stringent co-ordination of scientific disciplines is of primary importance.

10) BOVINE TUBERCULOSIS IN NEW ZEALAND WILDLIFE

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Bovine tuberculosis came to New Zealand from Europe with the first shipments of cattle nearly 200 years ago, but it is only in the last 30 years that the infection has spread to involve New Zealand wildlife. Over this time, introduced wild mammals such as brush-tailed possums, ferrets and deer have become the maintenance hosts for *Mycobacterium bovis* infection and eradication of tuberculosis from these species in the future will be a major animal health challenge since many of them inhabit inaccessible parts of the country. Infection rates in possums are low (<2%) and the disease occurs in localized "hot spots" making detection difficult. For this reason the presence of the disease can be efficiently monitored by surveying scavenger animals such as ferrets and pigs.

Reinfection of farmed cattle and deer following herd eradication programs occurs particularly on bush margins where there is access to infected wildlife. Infection frequently occurs through the smelling and licking of terminally ill possums which may emerge onto pasture during daylight hours and attract the attention of inquisitive cattle and deer. Wild pigs, cats, hedgehogs and goats may act as amplifier hosts, and stoats, dogs, hares and rabbits are occasionally reported as dead-end hosts. Although most species are infected via the respiratory tract, scavenging animals may be infected via the oral route and skin infection from wounds and scratches is also likely to occur in possums, cats and possibly ferrets.

The lesions observed in the affected species are variable and apart from cattle, none exhibit the characteristic features of classical tuberculous granuloma formation. Advanced lesions are no longer seen in farmed animals and culled reactors usually only have small pinpoint lesions at the periphery of retropharyngeal nodes or tonsils. These lesions contain very few acid-fast organisms. Infected possums however, may have large soft caseous lesions in axillary and inguinal nodes or lungs. These contain large numbers of acid-fast organisms and show no evidence of calcification or fibrosis. The most common routes of excretion in infected possums are by aerosols and discharging subcutaneous sinuses which may drain from infected lymph nodes. In scavenging animals such as ferrets, the lesions are mainly in mesenteric and retropharyngeal lymph nodes and are not grossly spectacular but consist mainly of areas of necrosis and aggregations of macrophages. Feral pigs may also develop *M. bovis* infection and lesions are also found mainly in lymph nodes of the head indicating infection by the oral route.

Each year large amounts of money are spent on possum control in critical areas throughout New Zealand and large research resources are also devoted to developing better methods of poisoning, trapping and biological control of these vertebrate pests as part of the national strategy for control of bovine tuberculosis.

11) HYBRIDISATION INCREASES RESILIENCE TO NOVEL DISEASE CHALLENGES

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Given the increasing threat of 'emerging infectious diseases' (EIDs) and the vulnerability of small populations to such novel challenges, a degree of hybridisation may improve the viability of endangered species. To investigate this hypothesis we quantified three immune parameters in wild populations of hybridising parakeets on the Chatham Islands, New Zealand (Pacific Ocean 44^{o} South, 176^{o} West). We show that measures of immune function (relating to both innate and acquired immunity) are markedly higher in the cosmopolitan Red-crowned parakeet *(Cyanoramphus novaezelandiae)* than in the island endemic (IUCN rank 'Endangered C2 a(ii)) Forbes' parakeet *(C. forbesi)*. Furthermore, we show that measures of immune function are also higher in Forbes' x Red-crowned parakeet hybrids than in the Forbes' parakeet. Even those individuals with few red-crowned characteristics, arising from introgression into the Forbes' population, have significantly higher measures. This study reveals a new management option for hybridising species of conservation concern, where an increase in resilience to novel disease challenges could outweigh some loss of genetic integrity.

12) THE INTRODUCTION OF SARCOPTIC MANGE IN THE RED FOX *(VULPES VULPES)* POPULATION IN SWEDEN AND ITS ECOLOGICAL CONSEQUENCES – 30 YEARS OF EXPERIENCE

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Between the years 1945 and 1972 around 35,000 wild animals were examined in Sweden in the Wildlife Health Monitoring program. During this period *Sarcoptes scabiei* was not found in any wild animal investigated. The only *S. scabiei* infections in the country were present in pigs and humans. In 1972 a single case of sarcoptic mange was observed in a red fox (*Vulpes vulpes*). Thereafter, starting in 1975 a large epizootic of sarcoptic mange occurred in Sweden. The disease was most likely introduced into Sweden with foxes migrating over the ice from Finland where the disease was present before it came to Sweden. It spread very rapidly in fox populations in forest areas, while it took longer for the disease to establish in agricultural areas. Mange spread to all parts of mainland Sweden but was not transmitted to the islands of Öland or Gotland. The first case of sarcoptic mange on Öland occurred in 2004, while the wildlife of Gotland still seems to be free of the disease. Sarcoptic mange has, during the 30 years since its introduction, regularly been observed in other species like arctic foxes (*Alopex lagopus*), lynx (*Lynx lynx*), pine martens (*Martes martes*), wolves (*Canis lupus*) and domestic dogs. During the first decade of the epidemic isolated cases were observed in domestic cats, in one mountain hare (*Lepus timidus*) and one horse. However, presently more and more cases are seen in cats.

The mortality rate among foxes was generally very high, in some areas probably up to 80 per cent. The annual hunting bag for red foxes in Sweden dropped from 80,000 foxes in 1976 to around 20,000 in 1985. Since 1987 the annual figure has increased and was in the year 2000 around 60,000 foxes. The reduction of the fox population had a great impact on population dynamics of many animal species such as roe deer (*Capreolus capreolus*), hares (*Lepus* spp.), grouse and other small game, which all increased considerably in most parts of Sweden. The epizootiological picture has changed during the last 30 years and today only sporadic outbreaks occur throughout the country. There is evidence that some foxes today survive the infection, develop immunity and the mange lesions eventually heal.

An important consequence of the epidemic of *S. scabiei* var *vulpes* was the establishment of the infection in the previous naïve domestic dog population in Sweden. Today sarcoptic mange is very prevalent –one of the most common skin disease of the dogs in the country.

We believe that the infection is transmitted both directly as well as indirectly, including transmission by fomites. There are empirical indications that infections from infected red foxes to dogs are more virulent than the transmission between dogs.

13) AN EPIDEMIOLOGICAL MODEL OF CANINE DISTEMPER VIRUS SPILLOVER FROM DOMESTIC DOGS TO JAGUARS IN THE BOLIVIAN ISOSO

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As disease-mediated population declines become a more urgent concern of conservation biologists, new tools are required to explore the dynamics of pathogens in wildlife populations. Epidemiological models represent one method of gaining insight into potential disease risks faced by threatened species. Disease spillover from domestic to wild carnivore populations may occur where the disease is present in the domestic population, the wild species is susceptible, and there is an ecological mechanism for disease transmission between the populations. These conditions are in place in the Bolivian Isoso, an indigenous territory adjacent to the Kaa-Iya del Gran Chaco National Park. This protected area, established in 1995, supports one of the largest populations of jaguars (*Panthera onca*) known. Domestic dogs are abundant in Isoseño communities bordering the park. Canine distemper virus is endemic in the domestic dogs of the Isoso, and seroconversion of ocelots (CDV) and canids has been documented here. An S-I-R, or susceptible–infectious–recovered, model was constructed to investigate the behaviour of CDV in the domestic dog and jaguar populations. We used serologic and demographic data for dogs, and camera-trapping data for jaguars, to estimate population size, turnover, ranging patterns, and contact rate. Radiotelemetry and camera-trapping data for small carnivores were also incorporated. Results indicate that jaguar density is too low to support the maintenance of a directly transmitted pathogen such as CDV. However, if one assumes equal susceptibility to the virus, the density of all carnivores combined is adequate to permit an epidemic. The model suggests that CDV is a risk for wild carnivore populations in this habitat.

14) SIMULATING THE POTENTIAL CONSEQUENCES OF INTRODUCED CANINE DISTEMPER VIRUS FOR AMUR TIGER POPULATION DYNAMICS

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Canine distemper virus (CDV) is one of the most common viral diseases of domestic dogs, and consequently forms a threat to conservation of free-ranging carnivores worldwide. In January 2004, a female Amur tiger (*Panthera tigris altaica*) died due to infection with CDV in the Russian Far East. Domestic dogs in the area show widespread serological evidence of exposure to CDV, consequently it is presumed that the tiger contracted the virus during contact with a domestic dog. Experience with high rates of mortality due to CDV in captive populations of tigers have raised concern about the effects CDV may have on this threatened population which numbers less than 500 individuals. We used a stochastic, individual-based simulation model to investigate the potential for CDV to affect Amur tiger population dynamics. Depending on assumptions about tiger-tiger contact, degree of contact with domestic dogs, and increases in mortality rate due to infection with CDV, a variety of outcomes are possible for Amur population dynamics ranging from almost no impact to large declines in population dynamics. We discuss these results in the context of the broader issue of disease transmission among domestic and free-ranging carnivores.

15) RESEARCH INTO THE TASMANIAN DEVIL FACIAL TUMOUR DISEASE (DFTD): A PROGRESS REPORT

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A devastating disease in the form of aggressive neoplasms is affecting wild populations of Tasmanian Devil (*Sarcophilus harrisii*). Investigation is under way on multiple fronts including pathology, clinical blood biochemistry, cytogenetics, immunology, endocrinology and molecular biology.

The key focus is to gain an understanding of the disease and provide informed key directional outputs into managing wild devil populations (*S. harrisii*). This presentation is aimed at providing an overview of hypotheses, strategies, outcomes from the research work already undertaken and future directions.

16) EPIDEMIOLOGICAL FEATURES OF A NEW DISEASE IN THE TASMANIAN DEVIL *(SARCOPHILUS HARRISII)*

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We describe the epidemiological features of Devil Facial Tumour Disease (DFTD), a cancerous disease in the wild Tasmanian devil (*Sarcophilus harrisii*). First observed in 1996, the cause and transmission agent of DFTD remain unclear. We report on the timing of its formal identification, its distribution and population impact. DFTD has been confirmed in devils from 35 sites across eastern, central and southern Tasmania. In two intensively monitored sites, DFTD has been associated with dramatic population declines. Prevalence appears to vary widely, but the disease occurs almost exclusively in adult devils: none of 52 devils with histologically confirmed DFTD were immature. In conjunction with several pathological investigations, surveys across Tasmania are being used to monitor changes in distribution, prevalence and population size, and to identify any associations between prevalence and population density or habitat features which may indicate dynamics and transmission agent.

TERRY AMUNDSEN STUDENT AWARDS

SESSION ONE

17) AN OUTBREAK OF TYPE C BOTULISM IN HERRING GULLS (*LARUS ARGENTATUS*) IN SOUTHEASTERN SWEDEN

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Since 2000, over 12 000 seabirds have died from an undetermined cause on islands and the coastal mainland of Blekinge province in southeastern Sweden. Mortality occurs annually and typically peaks in June and July. Herring gulls (*Larus argentatus*) represent the majority of affected birds. In June 2004, 24 affected herring gulls, collected from islands up to 50 km apart, were examined clinically, killed humanely and examined by necropsy. All birds showed similar neurologic signs ranging from mild incoordination and weakness to severe flaccid paralysis of legs and wings. With the exception of the most severely debilitated birds, all gulls were alert and responsive and attempted to defend themselves or escape when approached. Post-mortem and histopathologic examination, complete blood count and serum biochemistry analyses revealed no significant abnormalities. Serology for avian influenza virus (n=5) and avian paramyxovirus-1 (n=5) were negative. Virus isolation from pooled tissues (n=6) was also negative. No pathogenic bacteria were cultured from liver samples (n=20). Serum from 11 of 16 (69%) gulls tested positive for type C botulism in mouse bioassays. Clinical signs, test results and the absence of gross and histologic lesions all support avian botulism type C as the proximate cause of disease. A large-scale botulism outbreak is unprecedented in this area. The source of the toxin and the epidemiology of this outbreak are poorly understood. Affected gulls may be sentinels of environmental change in the Baltic Sea and further investigation into the source of botulism and the initiating conditions in this epidemic is needed.

18) REEF COMMUNITY ECOLOGY AND RISK FACTORS FOR THE CORAL DISEASE, YELLOW BAND SYNDROME, IN THE MEXICAN CARIBBEAN

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Coral reefs throughout the world are in decline and Caribbean reef ecosystems are among the most severely degraded. The definitive causes of the declines are not clear, but contributing factors likely include global climate change, environmental degradation, nutrient overloads, over-fishing, physical damage due to hurricanes and boat anchors, and increased incidence of disease. In the majority of coral diseases, the roles of potential pathogens, vectors, reservoirs, and risk factors are completely unknown, and prospects for intervention and management are elusive. In this paper, we explore the associations between a poorly characterised coral disease, Caribbean yellow band syndrome (YBS), and potential risk factors including host (coral colony) size, the surrounding coral reef community, and the abiotic environment. A total of forty transects sampled from among three sites in a 40km reef tract in the eastern Yucatan Peninsula, Mexico, were assessed for coral health status through direct examination by SCUBA and snorkel techniques in the summers of 2003-2004. Simultaneously, environmental parameters, including depth, water temperature, water quality (nitrate, nitrite, ammonia, soluble reactive phosphate, pH, and salinity), and coral community composition were recorded. Prevalence of YBS among *Montastrea* spp. coral for the total study period ranged from 0% on some transects to 73.3% on others, with an average overall prevalence of 21.3% ± 18.7%. Statistically significant risk factors for disease included: host colony size ($p<0.001$) and abundance of *Halimeda* spp. algae ($p<0.05$). No associations were detected between disease prevalence and any water quality parameters or community parameters including host (*Montastrea* spp.) density, presence or absence of any particular scleractinian coral species, or percent cover of live coral, dead coral, macro-algae, or sand. However, the coral community did change in measurable ways along biotic and abiotic gradients. Specifically, *Montastrea* spp. density and total species richness increased significantly with depth ($p<0.001$ and $p<0.05$, respectively), and live coral cover was positively associated with total species richness ($p<0.05$). This research demonstrates a methodology by which, through investigating environmental, community, and host risk factors for disease, we can slowly unravel the web of causation for a complex disease system. This type of ecological and epidemiological research offers a promising step towards identifying targets for coral disease management, even before we have the benefit of understanding specific disease pathogenesis.

19) CROSS-SHELF PATTERNS IN THE PREVALENCE OF CORAL DISEASE ON THE GREAT BARRIER REEF

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The emergence of novel coral disease types and an increase in the prevalence of coral diseases in a number of reef regions over the last few decades has been unprecedented in geological history. Increases in prevalence and disease types have been most pronounced in the wider Caribbean where coral disease has contributed significantly to the widespread degradation of coral reefs. In the Caribbean, increases in both the number of diseases and their incidence have been linked to increases in anthropogenic pollution including elevated levels of nutrients, inputs of sewage and increases in the African dust reaching the Caribbean during dust storms. Prevalence of black band disease (BBD), in particular, has been linked to elevated nutrient concentrations in both experimental and correlative field studies. BBD is commonly found on reefs of the Great Barrier Reef (GBR); however the role that anthropogenic pollution and elevated nutrients play in influencing prevalence of coral disease on the GBR is unclear.

The main source of pollution affecting reefs of the GBR is inputs of sediment and nutrients into the GBR lagoon via rivers draining from adjacent catchments. Intensive clearing and agriculture in these catchments have resulted in significant increases in suspended particular matter and nutrients inputs into the GBR lagoon. Nutrient discharges from rivers have reportedly increased at least four-fold in the central GBR over the last century. Variation in the extent of nutrient discharge between latitudinal sectors of the GBR as a result of variation in levels of clearing and agriculture in adjacent coastal catchments provides a useful means for comparing disease prevalence between high and low nutrient environments. In addition, terrigenous inputs of nutrients and sediment do not affect inner, mid and outer-shelf reefs equally, resulting in a gradient of declining nutrients and terrestrial inputs from inner- to outer-shelf reefs. To examine the role of terrestrial inputs of sediment, nutrients and other forms of anthropogenic pollution in determining levels of coral disease on the GBR, surveys of coral disease were completed in the summer of 2003/04 in each of the three cross-shelf positions in two latitudinal sectors of the GBR.

We found significant spatial variation in the prevalence of coral disease between latitudinal sectors and also between cross-shelf positions on the GBR, however coral disease prevalence was not positively correlated with levels of terrestrial inputs or anthropogenic pollution. Surprisingly, coral disease prevalence was highest in the more “pristine” Cooktown/Lizard Island latitudinal sector compared to the more intensively cleared and utilised Townsville sector. Also, coral disease prevalence was highest on mid-shelf reefs, which experience intermediate levels of terrestrial inputs or anthropogenic pollution, in both sectors. BBD showed similar spatial variation in prevalence. Disease types affecting corals on the GBR were widely distributed with over 75% of disease types present on over 50% of reefs surveyed. The widespread distribution of disease types throughout the GBR indicate that conditions supporting pathogen growth are ubiquitous throughout the GBR, however factors contributing to spatial variation in disease prevalence remain unclear. Disease appears to be an integral part of coral communities of the GBR, however further studies of factors contributing to spatial variability in coral disease prevalence are required to assess factors promoting coral disease on the GBR.

20) ISOLATION AND IDENTIFICATION OF THE CAUSATIVE AGENT FOR A WHITE SYNDROME CORAL EPIZOOTIC IN THE MARSHALL ISLANDS

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The study of coral disease is a novel science. The first coral disease was identified in 1973, and up to date, 29 coral diseases have been identified and characterised. Only one pathogen for a scleractinian coral disease, namely *Aurantimonas coralicida*, causing White Plague II has so far been verified by fulfilling Koch's postulates.

This study attempted to isolate and identify the causative agent for a coral epizootic currently affecting tabular colonies from the family *Acropora* in Majuro Atoll, the Republic of the Marshall Islands.

Infected corals (1-2 m in diameter) exhibit a white band of newly exposed skeleton which rapidly progresses causing further tissue loss and mortality. Bacteria were isolated from infected corals and used to infect healthy colonies. A bacterium, which caused rapid tissue loss in laboratory experiments, has been taxonomically identified by partial sequencing of its 16SrDNA gene. The isolates partial 16SrDNA gene sequence was found to be 99% identical with the 16SrDNA gene sequence of *Vibrio corallilyticus*, a known coral pathogen, which has been shown to cause microbial coral bleaching. When inoculated with 10^6 bacteria per ml in two separate infection experiments, 93% and 99% of corals exhibited tissue loss, compared with no tissue loss in control tanks. Mortality of infected corals occurred rapidly, within 24 hours, leaving only bare skeleton behind. The adhesion of bacteria to coral tissue was measured by plate counts from infected tanks vs. control tanks. Bacterial presence on coral tissue was quantified by plate counts of crushed infected corals vs. controls. 50% of the inoculated bacteria adhered to corals within the first 8 hours post-infection. By that time, 8.3 $^{+/-}$ 1.9 x 10^4 CFU were counted in 1 gram of infected coral tissue compared with 5.7 $^{+/-}$ 1.7 x 10^3 CFU in 1 gram of non-infected controls.

The re-isolation of *V. corallilyticus* from infected colonies fulfilled the last of Koch's postulates. A separate experiment has shown that successful laboratory infections occur only at concentrations higher than 10^5 bacteria/ml. This high concentration may determine the band appearance of this disease and the means by which it is transmitted.

21) A REVIEW OF THE 1988 AND 2002 PHOCINE DISTEMPER EPIDEMICS IN THE EUROPEAN HARBOUR SEAL POPULATION

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Mass mortality events involving marine mammal populations caused by morbilliviruses have been recognised since 1988. In April 1988 a mass mortality event involving thousands of harbour seals (*Phoca vitulina*) began in Northern European waters. Pathological findings in seals included acute interstitial pneumonia and lymphoid depletion, and a morbillivirus, phocine distemper virus (PDV), was isolated from infected tissues. Mortality from the 1988 PDV epidemic was estimated at 25% of the total European harbour seal population. The duration of the epidemic was 9 months and no evidence of further infection was detected until 2002 when a second epidemic occurred. An estimated 26% of the harbour seal population died. Almost identical gross pathological, serological and immunohistochemical findings were recorded in seals, and the virus showed 97% identity with the virus isolated in 2002. The first cases in each epidemic were recorded on Anholt Island in Danish waters, and the duration and the spread of the epidemic were similar in 1988 and 2002. Preliminary population models simulating the long-term effect of repeated epidemics have predicted that the stochastic growth rate could be reduced by 50%, although these models do not account the effects that immunity and genetic selection for more resistant individuals may have.

Many fundamental questions remain to be answered including the origin of the outbreak, why Anholt Island was the location of the initial cases in both epidemics, regional differences in levels of mortality and the means of spread of the virus within the harbour seal population. Proposed theories as to the origin of the infection are that overfishing or climate change may have resulted in a more southerly migration of Arctic seal species, in which PDV is endemic, and the introduction of PDV into the naïve harbour seal population. Differential mortality may be associated with variation in genetic resistance between subpopulations or may be linked to differences in the environmental levels of immunosuppressive pollutants such as organochlorines. Grey seals (*Halichoerus grypus*), which are also endemic to European waters, appear highly resistant to PDV and may act as vectors spreading PDV between harbour seal colonies.

22) RISK FACTORS ASSOCIATED WITH INFECTION WITH PATHOGENIC AND ANIMICROBIAL RESISTANT GASTROINTESTINAL BACTERIA IN NORTHERN ELEPHANT SEALS (*MIROUNGA ANGUSTIROSTRIS*)

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Although the ocean is relied upon to dilute the wastewater from run-off and sewage treatment outfalls, humans and marine mammals are still exposed to infectious agents through marine exposure. In humans, swimming in natural sources of water is a risk factor for infection with *Campylobacter* spp. Protozoal parasites, such as *Sarcocystis neurona* and *Toxoplasma gondii*, whose only known definitive hosts are terrestrial mammals, are infecting marine mammal species. A limited number of microbiological surveys of marine mammals have isolated zoonotic bacteria, with some of the bacterial strains showing resistance to antimicrobials. The source of these bacteria in marine mammals is unknown, but there is increasing concern over the potential roles of freshwater run-off and sewage outfalls as sources. This study uses northern elephant seals (*Mirounga angustirostris*) as a sentinel species to investigate risk factors associated with infection of marine mammals with zoonotic enteric bacteria.

Campylobacter spp., *Salmonella* spp., and *Escherichia coli* were isolated from juvenile wild northern elephant seals at three rookeries in California, and from seals presenting for rehabilitation at The Marine Mammal Center (TMMC) in the months of February-June in 2003 and 2004. Rectal swabs were collected from the seals, selective culture techniques were used, and isolates were identified through standard identification techniques. Antimicrobial sensitivities on *E. coli* were obtained by broth microdilution.

In 2003 we found a prevalence of *Salmonella* spp. in free-ranging seals of 4.5% (n=66) and in stranded seals of 37.3% (n=102); prevalence of *Campylobacter* spp. in free-ranging seals was 18.2% (n=66) and in stranded seals was 49.5% (n=101). Similar prevalences were found in 2004: in free-ranging seals (n=99) *Salmonella* spp. prevalence was 0% and in stranded seals (n=94) was 37.2%; *Campylobacter* spp. prevalence was 9.1% in stranded seals (n=94) and 46.8% in free-ranging seals. Antimicrobial resistance was present in *E. coli* from 4.5% of free ranging seals. However, in stranded seals, 43% of animals (n=100) that were stranded had *E. coli* which showed some antimicrobial resistance, with 7% of animals having *E. coli* resistant to three or more antimicrobials. The prevalence of zoonotic intestinal bacteria and antimicrobial resistant *E. coli* was thus higher in stranded seals compared to free-ranging seals. One hypothesis to explain this difference is that seals are acquiring zoonotic bacteria and antimicrobial resistance from terrestrial sources. The results of our investigation to determine whether freshwater outflow, sewage outfall, and human population density as risk factors for infection with *Salmonella* spp., *Campylobacter* spp., and antimicrobial resistant *E. coli* using logistic regression will be discussed.

23) POXVIRUS INFECTIONS IN NORTH AMERICAN PINNIPEDS

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Cutaneous pox-like lesions are a common complication in the rehabilitation of stranded pinnipeds. However, the identity and taxonomy of pinniped poxviruses are largely unknown. The epidemiology of the disease in rehabilitation centers and in the wild has not been studied and the prevalence of the disease in wild pinniped populations is unknown. During a recent poxvirus outbreak in California sea lions (*Zalophus californianus*) at The Marine Mammal Center in California, poxvirus nodules were collected from a sea lion that died following arrival. All samples were processed for routine histology, polymerase chain reaction (PCR) and virus isolation. Histological changes were consistent with those of a poxvirus infection with eosinophilic, intracytoplasmic inclusions present in the *s. granulosum* of the epithelium. A poxvirus belonging to the genus *Parapoxvirus* was isolated from a tissue homogenate of a skin nodule following inoculation of early passage California sea lion kidney cells. The assignment of the sea lion poxvirus to the genus *Parapoxvirus* was confirmed via electron microscopy and via partial sequencing of the genomic region encoding the viral envelope protein p42K. Analogue sequences were also obtained from pox-like lesions of other California sea lions and other pinniped species of the east and west coast of North America. Thus far, two independent virus strains have been detected in California sea lions. A third strain was identified in harbor seals (*Phoca vitulina*) from the northeastern United States. All detected pinniped poxviruses belong to the genus *Parapoxvirus*. Comparative partial sequence analysis revealed that the pinniped poxviruses are significantly different from all other parapoxviruses. Next, the newly isolated poxvirus was used to develop an ELISA as a tool to study the epidemiology of poxvirus infections of California sea lions in rehabilitation centres and in the wild. To validate the ELISA, 54 serial serum samples from 26 affected sea lions, collected opportunistically before disease, during the acute stage, and during the convalescent stage of the disease, were analysed. A rise in circulating anti-poxviral antibodies was observed in all sea lions (n = 8) for which a convalescent serum sample (≥1 day post first clinical signs) was available. In two sea lions, the rise in circulating antibodies could not be detected up to respectively 21 and 24 days after hospitalisation, but had occurred by respectively 58 and 73 days after hospitalisation. All serum samples collected prior to clinical disease (N = 21, mean days before FCS = 32 ± 24) were already considered positive. Additionally, 161 serum samples were collected at various time points from 74 unaffected California sea lions. A rise in circulating anti-poxviral antibodies was observed in 11 (15%) of these unaffected sea lions (mean increase = 3.1 ± 1.2, min 1.7, max 5.0) compared to samples collected within 6 days of admission. Of these 11 sea lions, three were maintained at the rehabilitation centre for 31-33 days after the rise in circulating antibodies was detected and no pox lesions were observed. Finally, to determine prevalence of antibodies to poxviruses in wild sea lion populations, serum samples were collected and analysed from 761 wild sea lions distributed across all age categories. Overall, anti-poxviral antibodies were detected in 93.4% of the sera. The highest antibody prevalence (98.0%) was observed in 2-4 mo-old-pups, whereas the lowest antibody prevalence (84.3%) was observed in adult males ($p < 0.05$). From these studies, we can conclude that three, but likely several more, parapoxviruses affect North American pinnipeds. The presence of circulating anti-poxviral

antibodies does not correlate with protection and re-infection may therefore occur. Exposure to the virus did, in some cases, occur during hospitalisation, and subclinical poxvirus infections do take place. It is unknown whether subclinically infected animals also shed virus, which would complicate the management of poxvirus outbreaks in rehabilitation centres. We can further conclude that poxviruses are endemic in wild populations and that introduction of poxviruses into the wild is therefore not of concern.

24) DISEASE ECOLOGY OF HENDRA VIRUS: EPIDEMIOLOGICAL MODELING TO TEST THEORIES FOR EMERGENCE

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Hendra virus (HeV) is an emerging paramyxovirus that is fatal to horses and humans. The flying fox is the only known reservoir of HeV and antibodies are consistently detected in all four Australian mainland species (*Pteropus poliocephalus, P. alecto, P. conspicillatus, P. scapulatus*). Hendra virus has emerged from flying foxes into horses five times; twice in 1994, once in 1999 and twice in 2004. All five outbreaks were temporally clustered in the latter half of the year. Subsequent transmission to humans occurred in three of the five outbreaks with a 50% fatality rate.

In this paper we use mathematical models and computer simulations to explore the hypothesis that periodic fluctuations in the dynamics of HeV could explain the temporal patterns in emergence and the seroprevalence patterns known in the four species. We first explore viral strategies for maintenance of HeV in homogenous populations of flying foxes and conclude that simple SIR (susceptible-infectious-recovered) dynamics are unlikely to account for the persistence of HeV. We outline alternative mechanisms for viral persistence and suggest how we can test these theories with data. One of these theories, metapopulation dynamics, is explored in detail. Metapopulation theory suggests that sub-structured populations with asynchronous dynamics facilitate global viral persistence even if the virus goes extinct in local populations. We build mathematical models to show that metapopulation dynamics could drive epidemic patterns in HeV, resulting in a periodic high incidence of infection which would increase the probability of emergence. For all our models we identify the parameter values that could drive the observed dynamics.

25) HENDRA VIRUS INVESTIGATIONS IN TWO NORTH QUEENSLAND FLYING FOX COLONIES FOLLOWING EQUINE AND HUMAN INFECTIONS

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In October 2004 a horse in the Cairns area died with clinical and post-mortem signs suggestive of Hendra virus infection. While infection in the horse was not confirmed, the attending veterinarian subsequently developed a febrile illness and serology revealed recent exposure to Hendra virus. Six weeks later, a second horse, in the Townsville area (approximately 350km south of Cairns), died of acute respiratory disease with Hendra virus the confirmed aetiology.

Hendra virus is a novel zoonotic paramyxovirus (genus *Henipavirus*) first described in 1994 after the death of 14 horses and their trainer in the suburb of Hendra in Brisbane, Australia. Flying foxes (genus *Pteropus*) are the putative reservoir host of Hendra virus, although the mode of transmission to horses has yet to be established. Previous surveillance has shown a 40-50% seroprevalence in *P. alecto* and *P. conspicillatus*, two of the three mainland *Pteropus* species in north Queensland. In the Cairns area, *P. conspicillatus* (the spectacled flying fox) is the predominant species, while in the Townsville area, *P. alecto* (the black flying fox) predominates. *P. scapulatus* (the little red flying fox) is a nomadic species seasonally found in both areas.

We surveyed the closest known flying fox colony to each of the two equine spillover locations in December 2004 and again in January 2005, to establish their infection and serologic status. We hypothesised that an increased incidence of infection in north Queensland flying fox populations may have been associated with the spatially and temporally clustered spillover to horses. The December surveillance employed a non-invasive sampling method utilising plastic sheeting placed below flying fox roost trees to collect pooled urine and/or faecal samples for screening by Taqman^{TM} realtime PCR and possible viral isolation. A total of 65 urine and/or faecal swabs were collected from a predominantly *P. conspicillatus* colony in the Cairns area, and 80 urine and/or faecal swabs were taken from a predominantly *P. alecto* colony in the Townsville area. Taqman^{TM} PCR failed to detect Hendra viral RNA in any sample from either location. The January surveillance entailed the capture of flying foxes, and the collection of individual blood, urine and saliva samples for Taqman PCR and for serologic screening by serum neutralisation test. Flying foxes were captured for sampling using mist nets, anaesthetised using isoflurane and oxygen and released post sampling. A total of 79 *P. conspicillatus* was caught and sampled at the Cairns roost site sampled in December, and 64 *P. alecto* and 1 *P. scapulatus* were caught and sampled at the Townsville site. The (currently pending) results of the January survey and its relevance to Hendra virus epidemiology will be presented and discussed. It is proposed to seasonally monitor the colonies over the next two years with the objective of better understanding the dynamics of Hendra virus infection in flying foxes and the mode of transmission to horses.

26) EFFECTS OF CLIMATE WARMING ON THE EPIZOOTIOLOGY OF HOST-PARASITE SYSTEMS IN NORTHERN NORTH AMERICA

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Protostrongylid nematode parasites have indirect life cycles where first-stage larvae (L1) are shed in the faeces of the mammalian definitive host, invade a gastropod intermediate host, and develop to infective third-stage larvae (L3) at a temperature-dependent rate. We describe seasonal patterns in development and transmission of two protostrongylids, *Parelaphostrongylus odocoilei* and *Protostrongylus stilesi*, in a population of Dall's sheep in the Mackenzie Mountains, Northwest Territories, Canada, (65ºN; 128ºW). From March 2000 to March 2003, shedding of L1 of both parasites in faecal samples from Dall's sheep peaked in spring and reached a trough in late summer. At a field site in the Mackenzie Mountains, L1 of *P. odocoilei* did not develop to L3 in gastropods experimentally infected in mid-July in 2002, with summer temperatures 0.5ºC cooler than normal, but did in 2003, with normal summer temperatures (as compared to the baseline since 1948). We used parameters determined in the laboratory and a field-validated model for protostrongylid development to describe historical trends and predict the potential effects of climate warming on development of *P. odocoilei* and *P. stilesi*. Based on a middle-of-the-road climate change scenario (CGCM2 A21 - SRES), this region of the Mackenzie Mountains will experience increases in annual temperatures of 1.2ºC by 2020, 3.3ºC by 2050, and 4.9ºC by 2080. Using soil surface temperatures recorded at the field site in 2003 as a baseline, L3 of both protostrongylids would be first available as much as 1, 2, and 3 weeks earlier in the year by the 2020's, 2050's, and 2080's, and L1 could start development correspondingly later in the year and still reach L3. These observations and predictions suggest that even small increases in temperature could lead to significant extension of the "growing season" of protostrongylid parasites, and therefore amplification in endemic regions. In addition, range expansion of definitive hosts, possibly associated with climate change, may result in introduction of *P. odocoilei* in naïve populations of Dall's sheep in the High Arctic ranges. Based on hourly air temperatures recorded at two of these sites (Inuvik, 68ºN and 133ºW, 1961-2004; and Ivvavik, 69ºN and 140ºW, 1996-2004), if introduced, L1 of *P. odocoilei* could have developed to L3 in each year. Therefore, temperature-dependent larval development is not currently limiting range expansion of this parasite. By combining long-term data sets with epizootiological models, we can describe and predict development rates for protostrongylid parasites across their geographic range, and when these pathogens are most likely to cause disease in wildlife hosts.

27) VARIABLE SUSCEPTIBILITY TO CHYTRIDIOMYCOSIS IN ANURANS

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Terrestrial ectotherms experience repeated changes in body temperature and water balance as they modulate their internal environment to conform to their physiological needs through behavioural changes and microhabitat selection. Since their internal environment is less stable than that of endotherms, investigating the host-disease interactions of ectotherms in tightly controlled laboratory environments is likely to lead to a distorted picture of those interactions. In controlled environments, chytridiomycosis (an EID of amphibians caused by the skin fungus *Batrachochytrium dendrobatidis*) is fatal to many Australian frog species including *Litoria caerulea*, *L. chloris* and *Mixophyes faxciolatus*. Chytridiomycosis occurs in these species in the field but is not known to have caused population declines. Recent experimental studies have established that elevated body temperatures similar to those experienced by basking frogs can clear individuals of the disease. The discrepancy between laboratory and field results may thus be due to selection of microenvironments by frogs in the field. We have designed a laboratory environment that allows frogs to select a range of thermal and humidity microenvironments during disease studies. The results we have obtained so far indicate that *L. caerulea*, the Australian tree frog, regularly chooses warm (35-45°C) microenvironments. These temperatures are high enough to eliminate established infections. Preliminary results also suggest that infected frogs behave differently from non-infected frogs, and may select warmer microenvironments. We suggest that future work on the host-disease interactions involving terrestrial ectotherms should allow for the additional complexity introduced by their lower degree of thermal and chemical homeostasis.

END OF STUDENT SESSION ONE

28) TECHNIQUES FOR DETECTING CHYTIDIOMYCOSIS IN WILD FROGS: COMPARING HISTOLOGICAL WITH REAL-TIME TAQMAN PCR

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An accurate, non-invasive technique for detecting chytridiomycosis is urgently needed to determine the current geographical distribution of the disease, and its prevalence in wild amphibian populations. Diagnosis to date has relied largely on histological methods, which are time-consuming, require a toe-clip, and yield many false negatives. Here we evaluate the reliability of a recently devised, rapid, non-invasive, Taqman PCR assay. We sampled 104 wild *Mixophyes iteratus* by both a skin swab for use in the Taqman PCR analysis, and a toe-clip for examination by histological methods. The Taqman PCR assay was nearly three times more sensitive than was histology, detecting chytridiomycosis infection in at least 19.1% of frogs (histology detected infection in no more than 6.7% of frogs). We conclude that the swabbing/Taqman PCR technique is the more reliable means of detecting chytridiomycosis in wild amphibians, and that it precludes the need for toe-clipping as a means of sampling for the presence of the disease in future surveys.

PLENARY
CARLTON HERMAN FUND
INVITED SPEAKER

29) HOW DO PARASITES INFLUENCE THE ECOLOGY OF THEIR HOST POPULATIONS? STUDIES ON RED GROUSE AND THEIR NEMATODE, *TRICHOSTRONGYLUS TENUIS*

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We examined the impact of the parasitic nematode *Trichostrongylus tenuis* on the population dynamics of their host, the red grouse in Northern England over a period of 25 years by undertaking detailed monitoring, field experiments and modelling. Individual level experiments show that parasites reduce the reproductive output of their hosts and when these findings are incorporated into a host-parasite model they account for the cyclic oscillations observed in the wild. The model predicts that treating 20% of the population is sufficient to stop the oscillations and field experiments undertaken at the population level confirm this prediction leading us to suppose that parasites play an important role in destabilising populations. This system is perhaps somewhat special in that there is little acquired immunity although there is evidence of parasites causing population cycles in other systems. Grouse populations also exhibit spatial synchrony between populations and we present evidence to suppose that large-scale weather events operate to cause this synchrony by affecting parasite transmission.

SESSION 2:
ENVIRONMENTAL DRIVERS OF EMERGING INFECTIOUS DISEASES

30) SEASONALITY, CLIMATE CHANGE AND PARASITE TRANSMISSION

Andy Dobson

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Most attention in the climate change debate has focused upon the changes in mean annual temperature. This is short sighted; most species live in a seasonal environment and seasonal environments will likely change dramatically in response to even small changes in mean temperature. In this talk I will describe how models for water-borne (cholera) and vector-borne (WNV, malaria) pathogens respond to seasonal forcing. In particular, I will first focus on comparing how changes in seasonality effect both the duration and magnitude of pathogen outbreaks. I will then illustrate how interactions between host immunity and seasonal variation in transmission rate can produce complex long-term patterns of host and parasite dynamics. I'll conclude by suggesting that conserving biodiversity is one of the key things we can do to buffer changes in pathogen transmission in a world where changes in expected patterns of seasonal climate will become the norm.

31) ANTHROPOGENIC FOREST CHANGE AND EMERGING TICK-BORNE DISEASE

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Granulocytic anaplasmosis (GA) is a potentially fatal disease caused by *Anaplasma phagocytophilum* characterized by fever, muscle pain, organ failure, and occasionally death [5]. Lyme disease (LD), caused by *Borrelia burgdorferi,* is usually mild, but can cause arthritis, neurological and cardiac dysfunction, and, in dogs, fatal nephritis [12, 3]. Tick-borne encephalitis (TBE) can cause a devastating neurological disease with headache, paralysis, seizures, confusion, and death. Each of these diseases is transmitted throughout the Holarctic by closely related *Ixodes* spp. ticks and maintained in nature by rodents. Our focus is the interaction of forest degradation and emergence of tick-borne disease around the Pacific Rim.

LD, TBE, and GA have all recently emerged in Europe (all three)[1] and the eastern US (LD and GA), with emergence directly linked to changes in human activity, including reforestation of abandoned farmland, allowing for an increase of white-tailed deer populations, rodents, and subsequently *I. scapularis* [13]. Increased winter temperatures and rainfall associated with global warming are predicted to further increase tick abundance and distribution in the US [4]. In Sweden, warming and increased deer numbers due to reduced predation were associated with increased *I. ricinus* numbers and LD incidence [9]. Ongoing climate changes are considered consistent with observed changes in TBE over the last several decades [10].

Peoples around the Pacific have utilized forests for food, shelter, cultural activities, and recreation for centuries. Recent extensive deforestation in the northwestern US and Asia has left only relictual old growth forests throughout areas that were previously old-growth coast redwood in North America and larch forests in the Inner Mongolia Autonomous Region in Northeastern China. In California, old-growth coast redwood occupies less than 4% of its former range, as most groves were converted by commercial uses, urban development, and other anthropogenic sources of destruction. Extensive Daurian larch forests occurred previously in moist regions in the Hinggan Mountains but are now being lost to forestry, agriculture, and human habitation.

LD, TBE, and GA are all emerging in Inner Mongolia in areas of intensified forestry and land rehabilitation [2]. While not clearly emerging in California, LD and GA remain important threats and are likely to change as forest changes continue. We are investigating human perceptions of old-growth forest and risk of tick-borne disease, attempting to understand how forest change modulates the host, vector and disease agent factors of tick-borne disease, and developing synthetic predictive models to capture important characteristics of anthropenically influenced tick-borne disease emergence. In order to develop synthetic theory, we incorporate methods to evaluate tick density, community diversity, host community regulation, and pathogen transmission parameters in old growth and paired second-growth forests around the Pacific Rim.

Because of intensifying forestry and recreational forest use, one mechanism for disease emergence is increased human-tick interactions, which is evaluated in our research by questionnaires. A second important object of our research is the role of changing tick abundance, which is linked with rodent abundance. This is better understood in California than in Asia, where surveillance and research is only now beginning. Case reports of GA in California continue to originate from areas near old-growth redwood, such as in Santa Cruz and southern Humboldt County, but anecdotal reports suggest that redwood provides poor

habitat for ticks that transmit disease [6,8]. GIS-based investigation of GA in coyotes documented that rainfall and habitats with oak and hardwood represented the most important landscape risk factors for infection [7]. The increase in second growth has led to dramatic increases in humid microhabitats for ticks, which are typical in deciduous woodlands, dense brush, along ecotones, or in areas with sufficient shade and moisture. Ongoing research systematically characterizes vegetation communities, indices of richness and diversity, and presence of keystone species, as well as tick diversity and abundance (through flagging, dragging, and collections from hosts).

Rodent abundance and diversity are critical for disease and tick maintenance. In California, the dusky-footed woodrat is a key player in LD and GA ecology and thus logically could facilitate disease emergence in communities where this rodent is abundant. Woodrats often are associated with oak and are common in second-growth forests [11]. Quantitative assessment of rodent communities is ongoing in paired old and second-growth forests in each region of interest. Finally, disease assessment in hosts and ticks is accomplished through PCR and serology. These data are synthesized in predictive models which allow for assessment of likely changes in disease dynamics due to environmental perturbation. Future goals include assessment of disease as a function of different management practices including balanced sustainable forestry, ecosystem protection, and management of human and wildlife risks.

[1] Blanco, J. R., and J. A. Oteo. 2002. Human granulocytic ehrlichiosis in Europe. CID 8:763-72.
[2] Cao, W. C., Q. M. Zhao, P. H. Zhang, H. et al. 2003. Prevalence of *Anaplasma phagocytophila* and *Borrelia burgdorferi* in ticks from northeastern China. Am. J. Trop. Med. Hyg. 68:547-50.
[3] Dambach, D. M., C. A. Smith, R. M. Lewis et al. 1997. Morphologic, immunohistochemical, and ultrastructural characterization of a distinctive renal lesion in dogs putatively associated with *Borrelia burgdorferi* infection: 49 cases (1987-1992). Vet. Path. 34:85-96.
[4] Estrada-Pena, A. 2002. Increasing habitat suitability in the United States for the tick that transmits Lyme disease: a remote sensing approach. Environ Health Perspect 110:635-40.
[5] Foley, J. 2000. Human ehrlichiosis: a review of clinical disease and epidemiology for the physician. Infectious Disease in Clinical Practice 9:93-98.
[6] Foley, J., L. Crawford-Miksza, J. Dumler et al. 1999. Two cases of human granulocytic ehrlichiosis in northern California. Clinical Infectious Disease 29:388-392.
[7] Foley, J., E. Queen, B. Sacks, and P. Foley. In press. GIS-facilitated spatial epidemiology of tick-borne diseases in coyotes (*Canis latrans*) in northern and coastal California. CIMID.
[8] Gewirtz, A. S., P. J. Cornbleet, D. J. Vugia et al. 1996. Human granulocytic ehrlichiosis: report of a case in northern California. Clinical Infectious Disease 23:653-4.
[9] Jensen, P. M., H. Hansen, and F. Frandsen. 2000. Spatial risk assessment for Lyme borreliosis in Denmark. Scand J Infect Dis 32:545-50.
[10] Randolph, S. 2001. The shifting landscape of tick-borne zoonoses: tick-borne encephalitis and Lyme borreliosis in Europe. Phil. Trans. Royal Soc. London, Series B 356:1045-1056.
[11] Sakai, H., and B. Noon. 1993. Dusky-footed woodrat abundance in different-aged forests in northwestern California. Journal of Wildlife Management 57:373-382.
[12] Shadick, N. A., C. B. Phillips, E. L. Logigian et al. 1994. The long-term clinical outcomes of Lyme disease. A population-based retrospective cohort study. Ann. Int. Medicine 121:560-7.
[13] Stafford, K. C., 3rd, M. L. Cartter, L. A. Magnarelli et al. 1998. Temporal correlations between tick abundance and prevalence of ticks infected with *Borrelia burgdorferi* and increasing incidence of Lyme disease. Journal of Clinical Microbiology 36:1240-4.

32) THE EMERGENCE OF NIPAH AND HENDRA VIRUS IN AUSTRALIA, MALAYSIA AND BANGLADESH

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In the past decade, three new paramyxoviruses have emerged as significant threats to human health in Australia and South Asia. All three appear to have *Pteropus* spp. fruit bat (flying fox) reservoir hosts and, in most cases, have domestic animal amplifier hosts. Hendra virus emerged in 1994, 1995, 1999 and 2004 in Australia, causing the death of 16 horses (the amplifier host species) and two of four people who became infected. Nipah virus emerged in 1999 in Malaysia in a large outbreak infecting 263 people and killing 103 of them. Nipah virus has since been implicated in outbreaks in people in Siliguri, West Bengal in 2002 and confirmed in four outbreaks in Bangladesh between 2001 and 2004, with another case cluster in February 2005. These viruses belong to a new genus, Henipavirus, and along with Menangle virus, Australian fruit bat lyssavirus and Tioman virus, constitute a group of newly discovered viruses from *Pteropus* bats. In this talk, I will review the latest research into the reasons behind the emergence of Henipaviruses from fruit bats in the last decade. New data from Malaysia suggests that the intensity of pig production in some farms played a crucial role in allowing viral spill-over to humans and that the virus is present in fruit bats throughout the region. In Australia, epidemiological and experimental data have provided new information on the dynamics of Hendra virus in fruit bat populations and the risk of spillover. Finally, data suggest that in Bangladesh, Nipah virus has emerged directly from fruit bats to humans, without amplifier hosts, and is able to be transmitted from human to human. The repeated emergence of these high case fatality viruses and the complexity of their transmission dynamics provide an important case study for combining ecological and veterinary medical approaches to emerging diseases.

33) THE EMERGENCE OF SUDDEN ACUTE RESPIRATORY SYNDROME (SARS) – PRECIPITATING FACTORS

Hume Field

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Knowledge of the origin of emerging agents and an understanding of the factors associated with emergence are fundamental to managing the risk of subsequent spillovers and associated disease outbreaks. Sudden Acute Respiratory Syndrome (SARS) was first reported in February 2003 in China. By the end of June 2003, 8437 cases (813 fatal) had been reported in 29 countries worldwide. An increasing amount of evidence suggests that wildlife markets in southern China were the source of the outbreak. Firstly, epidemiologic studies in Guangdong province, where the outbreak originated, found that early cases were more likely to work in the food industry and to live close to markets, yet found no positive association with proximity to domestic livestock or farms. Secondly, surveillance of wildlife in a market in southern China identified a SARS-like virus in the faeces of two of seven species surveyed. Thirdly, a serologic survey of humans working in the market showed a significantly higher prevalence of antibodies neutralising SARS CoV in (asymptomatic) wildlife traders and animal slaughterers than in market and community controls.

Minimising the risk of re-introduction of SARS to the human population requires an understanding of the reservoir and the ecology of infection in the reservoir. Note that a reservoir may be a discrete population *or* an ecological community in which a pathogen maintains and from which it spills. It is also important to recognise that, for any pathogen, multiple reservoirs are possible. An understanding of these concepts is necessary to grasp the complexity of identifying the source of an emerging infectious disease such as SARS, the identification of risk factors for spillover and exposure, and the formulation of risk management strategies.

While a SARS (or SARS-like) virus infecting animals in wildlife markets is the probable origin of the human outbreak, it is implausible that the cycle of infection in wildlife is limited to markets. Indeed, the emerging picture is of a virus able to infect a wide range of hosts, suggesting a complex ecology. A causal model with interacting natural, market, human, and peri-human animal components is discussed. Where a wildlife commodity constitutes a component of the reservoir, an understanding of what drives the trade in wildlife - a complex mix of environmental, economic, social and cultural factors - is fundamental.

But identification of the reservoir of SARS is only the first step. Minimising the risk of re-introduction to the human population requires an understanding of the ecology of infection in the reservoir, including the temporal and spatial dynamics of infection dynamics the reservoir, and risk factors for infection in the reservoir. This requires a systematic approach that includes prevalence studies, longitudinal studies, and modelling.
In a situation where the wildlife reservoir is a trade commodity, an extension of understanding the ecology of the reservoir is an understanding of the trade. We know that a wholesale and retail structure exists in the wildlife trade in southern China, with multiple wholesalers providing multiple retailers at a city level. We know that some wildlife are farmed and some wild-caught.

1. What about the marketing structure?
2. Are there dealers who buy and sell from both sources?
3. How much farm-to-farm trading (eg replacement breeding stock) is there?
4. Do farms periodically augment their stock from the wild?

Understanding of the trade is critical to its effective management. Directly related to the above is an understanding of what drives the wildlife trade – a complex mix of economic, social and cultural factors. Wildlife is expensive to purchase, and there is evidence that demand and consumption have increased in recent years as economic conditions in China have improved. Why do Chinese people eat wildlife? Typically for perceived health benefits. For example, the masked palm civet (*Paguma larvata*), the putative origin of the human SARS outbreak, was historically eaten in winter when fresh fruit was often unavailable. People believed that eating the animal (known colloquially as the *fruit fox* or *flower fox* because of its dietary preferences) provided the same health benefits as eating the fruit. In the markets, wild-caught *P. larvata* attract a price premium, because people believe it is more health

34) SOCIAL STRUCTURING, VIRULENCE AND HOT SPOTS

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Rabbit Hemorrhagic Disease emerged suddenly as a virulent and highly infectious disease in a group of domesticated rabbits being air transported to China in 1984. Subsequently, the disease caused massive mortality in domestic and wild rabbits throughout Eurasia and Australia. This paper examines a series of questions relating to why and how the disease emerged.

First: *Will the disease have a major impact on rabbit populations in all countries?* We undertook a large scale sera survey in the United Kingdom and found that most populations had already been exposed to the virus. Serial sampling indicated that an epidemic may pass through the population without massive mortality. Moreover, sera samples collected before the emergence of the disease also found evidence of previous exposure leading us to surmise that an avirulent form of the disease had been circulating. However, sequencing of the capsid gene found no evidence of a clear virulent or avirulent strain.

Second: *What conditions and evolutionary selective pressures may cause the rapid evolution and emergence of a highly virulent disease*?" Using a generic, individual-based SIR model we show theoretically that large, stable shifts in virulence may occur in pathogen populations due to a bi-stability in evolutionarily stable virulence caused by the contact/social structure of the host population. We postulate recombination coupled with changes in the social structure of rabbit populations may have initiated the rapid evolution of a virulent strain and caused the observed pandemic.

35) TOWARDS AN UNDERSTANDING OF CORAL DISEASES ON THE GREAT BARRIER REEF AND POTENTIAL LINKS TO ELEVATED TEMPERATURES

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Diseases of coral reef organisms have been escalating in the past few decades, particularly in the Caribbean, but little is known about factors affecting coral disease prevalence either regionally or globally. A targeted research project recently funded by the Global Environmental Facility (GEF) seeks to understand the impacts of localized stress and compounding effects of climate change on coral disease globally. As the Australian arm of the WB/GEF Working Group on Coral Disease, we have commenced baseline surveys of coral disease in the northern, central and southern sectors of the Great Barrier Reef (GBR) Marine Park as a first step in understanding the epidemiology of coral disease in the region. We recognise seven disease states that affect scleractinian corals and one that affects gorgonians. Disease prevalence (combined for all disease states) ranged from 7.2 ±1.06% to 10.7 ±0.76% for scleractinian corals and up to 16.6 ±4.5% for gorgonians in January 2003. Comparisons of disease prevalence between the GBR and other reef regions will be presented. Overall, white syndrome (WS) and skeletal eroding band (SEB) were the two most common disease states on the GBR. Black band disease (BBD), other cyanobacterial syndromes, brown band (BrB) and tumors were also present on all reefs. Disease prevalence increased dramatically between winter and summer surveys, increasing by fifteen-fold in acroporids, twelve-fold in faviids and doubling in pocilloporids in the northern sector. In particular, the number of cases of WS, SEB and BBD was greatest in the austral summer, suggesting a link between higher temperatures and disease incidence. Other evidence of the links between temperature and disease prevalence will be discussed. Given 1) putative links between disease outbreaks and both elevated temperatures and deteriorating water quality, and 2) that current trends in global climate change and intensity of human-related activities predict escalating levels of stress for reef corals, studies such as this one on the Great Barrier Reef are important for establishing global baselines against which to judge whether background levels of coral disease are increasing.

SESSION 3:
WEST NILE VIRUS

36) UTILITY OF CLIFF SWALLOWS AND THEIR PARASITES FOR WEST NILE VIRUS SURVEILLANCE

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We report early seasonal activity of West Nile virus (WNV) infection in cliff swallow nestlings (*Petrochelidon pyrrhonota*) and detection of WNV in over-wintering cimicid swallow bugs (*Oeciacus vicarious*) from the Fort Collins, Colorado area using TaqMan® reverse transcriptase-PCR. The timing of modal WNV prevalence in nestling cliff swallows predated the modal human case reports in the Fort Collins area by 5 weeks. WNV activity in nestlings corresponded spatially to case reports of viral infection in humans. The presence of WNV in overwintering cimicid bugs suggests a mechanism whereby the efficiency of early season amplification is achieved prior to a regional and general amplification via a bird-mosquito cycle. Winter surveillance of WNV infected cimicid bugs may provide spatial guidance for mosquito larvicidal programs, and early season surveillance of nestling cliff swallows may provide spatial guidance for mosquito adulticidal programs.

37) IMPLEMENTATION OF A NATIONAL SURVEILLANCE PROGRAMME FOR WEST NILE VIRUS IN DEAD WILD BIRDS: CANADA 2000-2004

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Following the 1999 emergence of West Nile Virus (WNV) infection in New York, Health Canada convened a national WNV Working Group to determine the risk management responses to be initiated in Canada in 2000. Among other actions, surveillance for WNV in wild birds was to be established in at-risk regions, with the objective of determining the geographical and temporal distribution of WNV to alert the public health system of a proximate threat.

Funded by Health Canada, with provincial contributions to support specimen accession and communications, the Canadian Cooperative Wildlife Health Centre (CCWHC) has coordinated surveillance for WNV in dead birds since 2000. This required CCWHC to establish a communications network with provincial and often local public health agencies, which had to assume the unfamiliar role of participants in enhanced passive surveillance for disease in wildlife. To date, 12 of Canada's 13 provinces and territories have participated, encompassing over 90 regional and local public health authorities, spanning over 5500 km coast-to-coast.

The programme has 3 major components: specimen accession; diagnosis; communication of results. Public awareness of the programme and specimen accession is the responsibility of provincial or local public health authorities, depending on the degree of decentralization of public health activity in the province. The public reports dead birds, in some provinces to a central toll-free telephone number, in others by a call to the local public health authority. Carcasses generally are picked up by the provincial or local public health agency, or by the provincial wildlife agency, depending on the province. Carcasses are submitted by courier daily to regional laboratories for diagnosis. These are either collaborating provincial veterinary diagnostic laboratories in British Columbia, Alberta, Manitoba and Newfoundland, or the CCWHC regional centres in Saskatchewan, Ontario, Québec and Atlantic Canada.

Diagnostic modalities have evolved over the course of the programme. In 2000, when there was no high throughput RT-PCR WNV diagnostic capability in Canada, it was based on histopathology seeking lymphoplasmacytic inflammatory lesions, in which case tissue sections were submitted to the Canadian Food Inspection Agency, National Centre for Foreign Animal Disease, Winnipeg MB for WNV immunohistochemistry. In 2001 and 2002, frozen brain and kidney samples collected at provincial/CCWHC laboratories were couriered daily to the National Microbiology Laboratory of Health Canada in Winnipeg for Taqman® real-time RT-PCR. In 2002, VecTest®, a wicking immunochromatographic antigen-capture dipstick assay, was validated for use on oropharyngeal swabs in American crows, and it was adopted as the front-line WNV diagnostic tool in 2003 and 2004. The VecTest obviates the necessity to dissect carcasses, thereby improving case throughput, decreasing labour costs, and increasing operator safety. It is "low technology", and produces a result within 15

minutes, at a cost comparable to RT-PCR.

The CCWHC database is the core of the case tracking and test reporting system. It has evolved from a Paradox-based system requiring transcription of paper records, which initially were summarized in Excel files and emailed weekly by the regional laboratories for collation in the database on the server at CCWHC Headquarters in Saskatoon. Since 2002 a MySQL-based system has been in place, with direct on-line data entry locally into the central server. Data are updated and summary tables are produced daily, and passworded access is available as needed to several hundred public health personnel across Canada. Adoption of decentralized VecTest diagnosis at the regional laboratories and database upgrades has markedly reduced the interval from specimen pick-up in the field to transmission of a report from the lab. This took weeks to months in 2000, a median of 21 days in 2002, but a median of only 4 days in 2004, including courier time from the field and holding some specimens over weekends prior to shipment.

Dead bird surveillance data, including georeferencing fields (GPS, address, postal code, regional georeferencing systems, and alternative geographical information) are transmitted daily by CCWHC Headquarters to the Health Canada medical geography centre. There they are geo-referenced using a hierarchical procedure, beginning with GPS coordinates and cascading down to street address, regional systems, and postal codes. These data are verified and used to produce daily national and regional summary maps, including test results by species with denominators in each public health jurisdiction, and point data are also used to update interactive internet-based mapping applications.

Public transparency of the surveillance situation is policy. Following a 2-day embargo to permit affected public health agencies to respond if necessary to changes in the surveillance situation, maps and data updated daily to the level of local health region are posted on the national Health Canada WNV Surveillance web site, for perusal by the public and the press.

In 2000, when surveillance involved Saskatchewan eastward to Newfoundland, a cumbersome, insensitive diagnostic system with no surge capacity was swamped when WNV was diagnosed in New York adjacent to the Ontario border. However, WNV was not detected in any of 210 Ontario crows examined among the over 2200 birds submitted nationally. In 2001, when over 3800 birds were accessioned nationally, from Saskatchewan east, WNV was first detected in Ontario, in early August. In 2002 avian surveillance was extended westward to British Columbia, and a total of over 3500 birds were accessioned nationally. WNV activity occurred in Saskatchewan and Manitoba, was intense in Ontario and Quebec, and was detected in late summer in Nova Scotia. In 2003, surveillance continued across Canada. WNV activity was intense in Alberta, Saskatchewan and Manitoba, and was detected at lower levels in Ontario, Québec, New Brunswick and Nova Scotia, with over 12,000 birds accessioned nationally. In 2004, when over 6400 birds were accessioned, WNV activity continued at relatively low intensity in Alberta, Saskatchewan, Manitoba, Ontario and Quebec, but was not detected in Atlantic Canada or British Columbia.

WNV consistently has been detected in dead birds in an area prior to the occurrence of locally acquired human cases, and the programme is considered a valuable component of the Canadian response to WNV.

38) PATHOLOGY AND EFFECTS OF WEST NILE VIRUS ON CALIFORNIA'S ENDEMIC BIRD, THE YELLOW-BILLED MAGPIE

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The Yellow-billed Magpie (YBMA) is a unique endemic corvid with a species range limited primarily to the Great Central Valley and sections of the Central Coast Ranges of California (USA). The species has experienced reduction in numbers and contraction of its historical range due to bounty hunting and loss of habitat. YBMA belongs to the corvid family which includes crows and their sister species, the Black-billed Magpie, both of which experienced high mortality due to West Nile Virus (WNV). As the WNV epidemic front approached California from the east, YBMA were hypothesised to be sensitive to WNV and experience high mortality. That prediction held true. Beginning summer 2004, the first recording of WNV in YBMA was documented by the California Department of Health Services WNV Surveillance Program. Over 300 of hundreds of dead YBMA submitted to the Dead Bird Surveillance Program in 2004 tested positive to WNV by RT-PCR (California Department of Health Services data). As part of a larger study to evaluate the impacts that WNV may have on YBMA population and species health, the goal of this study was to describe pathology caused by WNV in this special endemic California species.

Forty-two carcasses of YBMA were collected May-September 2004 and examined histopathologically at the California Animal Health and Food Safety Laboratory. Thirty-nine of these carcasses were from the California Department of Health Service Dead Bird West Nile Virus Surveillance Program and 3 were supplied courtesy of U.S. Department of Agriculture APHIS Wildlife Services and Sacramento Municipal Airport System (birds that had been killed for airport public safety reasons). The following tissues were collected and sectioned for histopathology examination: brain, heart, kidney, lung, oesophagus, spleen, lever, gizzard, proventriculus, intestine, pancreas, and in some cases, ovary, testis, and additional tissues. Kidney samples were collected for WNV testing by reverse transcriptase-PCR, which was performed at the University of California Center for Vectorborne Diseases. Muscle was collected for genetic analysis.

Of 39 YBMA carcasses from the DHS Dead Bird WNV Surveillance Program, 25 (61%) tested positive and 16 (39%) tested negative for West Nile Virus by RT-PCR. All 3 of the YBMA presenting from the airport tested negative. WNV positive (WNV+) birds often showed striking necrotic lesions with vasculitis in heart, brain, spleen, liver, gastrointestinal tract, and kidney.

Magpies collected through the Dead Bird Surveillance Program that tested WNV negative (WNV-) had surprising levels of disease, but typically lacked the necrosis and vasculitis signature of WNV. Kidney pathology (usually interstitial nephritis) was observed in both WNV- (56% with kidney disease) and WNV+ (44% with kidney disease) magpies. Pneumonia was observed in 5 out of 16 (31%) WNV- magpies, while 3 out of 25 WNV+ magpies displayed pneumonic lesions – WNV+ birds more commonly had necrotic lesions in

lungs. Intestinal worms (cestodes and nematodes) were found in 5 out of 16 WNV- magpies; about half of WNV+ magpies had intestinal nematodes and/or cestodes.

Mortality and pathology observed in 2004 is likely the beginning of a mortality wave that will sweep over the species in the coming few years. Although Yellow-billed Magpies are not yet listed as threatened or endangered on state or federal lists, they are listed as an Audubon species of concern. Their species range had diminished even before WNV due to loss of oak grassland savannah habitat and persecution as agricultural "pests". Current work includes analysis of genetic diversity and species population structure that existed before the WNV epizootic in YBMA and other California native birds, for future comparison with the post-WNV landscape. The pathology findings presented here will help guide future research, management and conservation efforts by providing data to a growing literature that indicate the severity of WNV infection in free-ranging bird species.

39) DECREASED AVERAGE DAILY TEMPERATURES AS A CAUSE OF REDUCED WEST NILE VIRUS AVIAN MORTALITY IN WYOMING DURING 2004

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Dead bird surveillance for West Nile virus (WNV) was initiated in Wyoming in 2001 and the virus first was detected in the state during 2002. In 2003, 555 dead birds from 74 species were tested for WNV using RT-PCR and IHC. Of these, 182 birds from 36 species were WNV positive. In contrast, of the 303 birds tested during the 2004 surveillance season only 23 birds from 11 species tested positive for WNV. While factors such as reporting bias, increased mosquito control, and acquired immunity in bird populations may have contributed to the dramatic decline we concluded that reduced WNV avian mortality was due primarily to a substantial decrease in the average daily temperatures between the 2003 and 2004 transmission seasons. Temperatures for July and August 2004 were approximately 9 and 13 degrees cooler respectively, than for the same time period in 2003. Cooler temperatures contribute to longer developmental times for mosquito larvae and protracted extrinsic incubation of the virus in the mosquito vector.

40) PROTECTING THE WILDLIFE AND PEOPLE OF HAWAII FROM WEST NILE VIRUS WITH AN INTEGRATED PREVENTION, SURVEILLANCE AND ERADICATION RESPONSE PLAN

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West Nile virus (WNV), an Old World flavivirus, has spread across North America and into the neotropics since its introduction in New York in 1999. With a significant mortality rate in many birds and some mammals, the NY99 strain of WNV is an imminent threat to 32 endemic bird species (18 endangered), two endemic endangered mammals, and the people of the Hawai'ian islands. Having evolved in isolation from flaviviruses and insect vectors, the native bird and mammal fauna lacks immunity to WNV, and an epizootic is likely to lead to population declines and multiple extinctions. The potential human morbidity and mortality from WNV has enabled a coalition of interest groups and agencies to gain support for a unique, proactive effort to keep Hawai'i free of WNV and safeguard both wildlife and public health in the Pacific islands.

Pathway management efforts have focused on controlling importation of bird hosts and mosquito vectors, but have been of mixed success due to legal and economic constraints. Vector density at many likely points of entry has been reduced, but the reduction is difficult to maintain, and its effect on the potential initiation of an epizootic is unknown. Eradication of any incipient outbreak is the goal of the WNV response plan. Eradication requires both an efficient surveillance system and a response system able to suppress disease transmission for an extended period. Costs, advantages, and limitations of available surveillance tools were analysed to determine the optimum program. Actual performance of these tools will be compared to predictions. Aerial adulticiding, essential to eradication of incipient WNV, is a costly measure with politically challenging features. Whether Hawai'i can remain free of WNV is a political, economic and biological question with Pacific-wide ramifications.

41) MODELING WEST NILE VIRUS TRANSMISSION IN THE SOUTHWESTERN UNITED STATES: HABITAT, VECTORS, AND HOST DISTRIBUTIONAL PATTERNS ALONG THE LOWER COLORADO RIVER CORRIDOR MIMIC POTENTIAL WEST NILE VIRUS INFLUENCES ON NEOTROPICAL MIGRANT BIRDS THROUGHOUT THE SOUTHWEST

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We examined the distribution of irrigated land, riparian vegetation, streams and drainages, mosquito vectors and bird distribution patterns throughout the states of Arizona and New Mexico in order to assess differential potentials of West Nile Virus (WNV) transmission throughout the southwestern United States. We weighted each factor and then created a Geographic Information System (GIS) model that predicted a weighted risk of WNV occurring in each state of the region. We further refined the model to examine the more susceptible corvid (i.e., crow, raven, jay) species throughout Arizona and New Mexico. All GIS data layers were then combined with human population patterns, and regions of the state were assigned WNV human risk potentials ranging from one to five. Success of this modeling will be discussed in light of the first year (2004) of WNV in Arizona.

To further refine our model, we also examined foraging ecology of spring and fall migrant birds in native and introduced vegetation habitat patches along the Lower Colorado River corridor, in an effort to predict future risks of WNV infections. Study areas were located on the Rio Hardy and Rio Colorado rivers in Sonora, Mexico, Cibola and Bill Williams River National Wildlife Refuges in Arizona. From our census and mist-net capture data, we found that migrant species' arrival and departure dates differed, but were more predictable during the spring migration period. Plant species abundance and phenology patterns dramatically influenced location of avian foraging. Hence, vector access to different bird species in a vegetation patch may be important factor in future West Nile Virus transmission to migrant birds throughout the southwest.

We found that avian species partitioned foraging habitat, thus potentially impacting WNV transmission. Lucy's Warblers and Western Kingbirds preferred the highest vegetation strata, while Yellow Warblers and Warbling Vireos occurred primarily in the middle foliage regions. Wilson's and Townsend's Warblers were observed most often in the lower third of the vertical vegetation strata, while White-crowned Sparrows and Crystal Thrashers preferred the lowest vegetation strata. It thus appears that species, structure, plant phenology, and abundance of vegetation, coupled with bird responses to their food prey base, all appear to play a role in determining micro-site influences on migrating bird susceptibility to WNV infection along the Colorado River and other areas of the southwestern United States.

TERRY AMUNDSEN STUDENT AWARDS

SESSION TWO

42) KOALA RETROVIRUS (KoRV): THE LINK TO DISEASE AND ITS PLACE IN KOALA ECOLOGY

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Koala retrovirus is a recently described virus of koalas (Hanger et al 2000) that has been linked to neoplasia and immunosuppression in its host (Tarlinton et al 2005). While retroviruses are known to cause disease in a range of species including FeLV in cats, KoRV is unusual in that it is an endogenous (inherited retrovirus) that is highly active. Although endogenous retroviruses are very common, with up to 8% of the human genome being retroviral material, they have usually been associated with their hosts for millions of years and are largely inactive. Recent work has demonstrated that KoRV is present in all animals in SE QLD as would be expected for an endogenous virus but that it is not present in animals from Kangaroo Island, a population that has been isolated since the 1920's. Animals in Victoria have a mixed KoRV status. The evidence available indicates that KoRV free populations have a much lower incidence of neoplasia and infectious diseases such as chlamydiosis. In addition KoRV is very closely related to the exogenous (horizontally transmitted) Gibbon Ape Leukaemia Virus (GaLV). The presence of such similar viruses in very disparate species indicates a recent host species jump, probably within the last 100 years. KoRV appears to be still spreading through the koala population and work in progress is focused on identifying KoRV naive populations and characterizing the impact of the virus on these animals.

43) SEROLOGIC SURVEY FOR SELECTED DISEASE AGENTS IN URBAN BRUSHTAIL POSSUMS (TRICHOSURUS VULPECULA) FROM SYDNEY

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Brushtail possums *(T. vulpecula)* have readily adapted to urban areas by utilising human resources for both food and habitat. Their close proximity to humans and their domestic animals may increase the exposure of disease to urban possum populations. Our seroprevalence study focused on diseases that can occur between humans, pets and possums such as toxoplasmosis, neosporosis and leptospirosis. *T. vulpecula* is known to be susceptible for toxoplasmosis and leptospirosis and the main purpose of the present study was to investigate the occurrence of antibodies against these diseases in urban possums.

Serum samples were obtained from 199 adult possums originating from 4 different suburbs and from Scotland Island, a densely populated island by both humans and possums. All sites were located in the North Shore Area of Sydney. The study started in 2003 and included live trapping of possums in 33 householders' backyards. Trapped possums were microchipped, sexed, aged, weighed and body condition was assessed. These populations were monitored 3 times over the last 1.5 years and if individuals were recaptured, they were bled again to allow monitoring over the sampling period.

Serology for the different *Leptospira*, serovars, was carried out using the microscopic agglutination test (MAT) at Queensland Health Scientific Services. The MAT starting dilution was 1:50. Antibodies were found in 17 (8.5%) of the 199 possums, with titres of 1:50 in 3, 1:100 in 4, 1:200 in 2, 1:400 in 2, 1:800 in 1, 1:1600 in 2, and 1:3200 in 3. The main agent was identified as serovar Hardjo, with 2 possums demonstrating exposure to serovar Ballum. This serovar appears to be new to Australia but is endemic to New Zealand. Seroprevalence varied from 0 to 11.8% in the different suburbs, and no positivity was found in the samples from Scotland Island. Seroprevalence for leptospirosis was slightly related with age, with older possums seeming to be more affected. Seroprevalence did not show a significant distinction in relation to gender. Seroprevalence was connected to 6 backyards only, in one case seropositive possums continuously originated from 2 backyards in close proximity over the sampling period. This supports New Zealand research findings that possums are unlikely to contract leptospirosis through a contaminated environment alone. Transmission also probably occurs as a result of affiliative or sexual behaviour. The present study demonstrated for the first time the presence of antibodies to leptospirosis in urban brushtail possums. The results for toxoplasmosis and neosporosis will be available at date of conference.

44) URBAN POSSUMS IN TARONGA ZOO & SURROUNDS: IMPLICATIONS FOR DISEASE TRANSMISSION BY A 'NATIVE PEST'

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The common brushtail (*Trichosurus vulpecula*) and common ringtail (*Pseudocheirus peregrinus*) possums are among the few native mammals to thrive in urban Australia. These arboreal marsupials have adapted to human settlement inhabiting roof cavities, nesting in parks and foraging on garden plantings and domestic food scraps. Within the protected zoo environment possum populations flourish and are often responsible for damage to botanical estates and theft of food supplies intended for exhibit animals. These behaviours have earned possums the reputation of a 'pest' and have divided wildlife agencies and community members over finding a solution. Despite the continuing debate over the management of nuisance possums, important health implications arising from their co-occupancy with humans, domestic pets, wildlife and captive animal collections in metropolitan areas have not been addressed. In light of recent studies from both Australia and New Zealand implicating wild possums as reservoirs for helminth, bacterial and protozoan infections, the potential role of urban possums as vectors for zoonotic disease needs to be investigated.

This study aims to examine the health, population biology and behavioural ecology of possums inhabiting Taronga Zoo and surrounding urban areas. The zoo environment presents a unique scenario in which the possums have unrestricted mobility and the capability to interact with an array of exotic and native animals. A longitudinal assessment of the possum population will be achieved by carrying out monthly trapping sessions over 2 years. Measurements such as weight, age, body condition and reproductive status will be recorded and samples including blood and serum, ectoparasites and faeces shall be collected. Upon analysis this data will provide an insight into the long-term health and prevalence of disease amongst the resident population. To provide a comparison with the urban possums at the zoo an additional population of wild possums at Jenolan Caves Conservation Reserve will also be trapped and monitored. The home range of zoo possums will be tracked using radio-telemetry so that the degree of spatial overlap between possums, captive animal collections and adjacent neighbourhoods can be determined. Video analysis will be used to assess opportunities for disease exchange arising from the frequent interaction of exhibit animals and possums that are attracted into enclosures at night.

It is hoped the findings of this study will contribute to a balanced management strategy for possums that considers not only human perceptions of this animal as a 'pest' but the real consequences of disease transmission in an urban setting. As natural landscapes become increasingly 'urbanised' the critical relationship between native animals and humans needs to be resolved to achieve the duel outcomes of conservation and public health safety.

45) ECTOPARASITE BURDENS AND PELAGE CONDITION IN MOUNTAIN BRUSHTAIL POSSUMS (*TRICHOSURUS CUNNINGHAMI*)

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A wild population of mountain brushtail possums (*Trichosurus cunninghami*) with a high prevalence of pelage damage affecting the rump was monitored for an association between ectoparasite burden and pelage condition. Ectoparasite burden and pelage condition were also compared to general health parameters, skin histopathology and habitat differences. Seasonal data was collected by sampling in July, late September, February and late April. Ectoparasites were counted and identified to species level. *Trichosurolaelaps dixoa, Atellana papilio* and *Haemolaelaps penelope* were the most commonly collected mites. Ticks identified included *Ixodes tasmani, I. trichosuri* and *I. cornuatus,* and one *Acanthopsylla spp.* was found. Mean mite numbers per animal were extremely variable within the population, and were not related to pelage condition. Seasonal differences in tick prevalence were detected and fleas were detected only in a very small number of cases. Mean ectoparasite numbers per animal varied between habitat types and greater mean numbers of mites were found to be associated with the lower density habitat. Pelage damage was more common in the habitat with the higher population density. Additionally, pelage condition of the rump varied seasonally. There was no apparent association between general health parameters and ectoparasite numbers or pelage condition. Skin histopathology of the rump revealed inflammatory changes in a number of possums with and without pelage damage and with varying ectoparasite burdens. Damage to the pelage of the rump in this population of mountain brushtail possums seems to be influenced by season, gender and habitat. However, based on the data collected so far, there is no obvious correlation between pelage condition of the rump and ectoparasite loads, general health parameters or inflammation of the skin.

46) ECTOPARASITES AND THEIR IMPACT ON HEALTH AND SURVIVAL OF ENDANGERED AUSTRALIAN ANIMALS

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True ectoparasites such as ticks, mites (Acarina), fleas and lice (insects) are defined as external parasites that spend at least one stage of their life cycle on a host. In animals, ectoparasites are the leading cause of vector-borne disease. Their impact on the agricultural industry results in massive economic losses through animal mortality, control costs, quarantine and transportation restrictions and production losses. In wild species, they pose a threat to endangered populations already under stress by increasing human encroachment, habitat destruction and fragmentation, and competition with and predation by introduced animals. With stress playing a large role in parasite susceptibility, exposure to ectoparasites from introduced species carrying novel infectious agents is now becoming an increasing threat to these already vulnerable populations.

Since the identification of the agent *Borrelia burgdorferi* that causes Lyme disease in 1982, an increasing number of new and re-emerging ectoparasite-borne diseases have been described throughout the world. These include tick-borne bacterial and protozoal diseases, such as Rickettsiae (Queensland tick typhus), Ehrlichiae (canine ehrlichiosis) and several reptile-borne haemogregarines. Ectoparasites also play a role in the transmission of several viral infections such as Tick-borne encephalitis and Louping Ill, which can be transported over large distances by migratory bird species. Novel flea and louse-borne infections have not been as numerous. There has, however, been a re-emergence of past agents such as louse-borne trench fever and typhus in homeless and displaced populations and flea-borne diseases, including murine typhus and sylvatic plague, as a result of insecticide resistance. Many animal species have been found to act as asymptomatic reservoirs of human pathogens however the implications of most infections in animals are largely unknown.

To date, human and livestock diseases have received more coverage in regards to the documented rise of ectoparasite-borne agents. Unfortunately, the impact of such agents on Australian animal populations, many of which are known reservoirs, has not yet been thoroughly investigated. As the importance of ectoparasites as vectors in disease transmission becomes better understood, so too does the role of past liberal animal trade practices, responsible for the much of the Australian feral animal population, as well as the current international trading (such as the domestic pet trade) in introducing novel vectors and their pathogens. A more thorough understanding of the role ectoparasites play in disease transmission and health of Australian animals is vital for long-term management success of rare and endangered species.

47) CRYPTOCOCCAL INFECTIONS IN CAPTIVE GILBERT'S (*POTOROUS GILBERTII*) & LONG NOSED (*POTOROUS TRIDACTYLUS*) POTOROOS

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Mortalities associated with cryptococcocosis have been recorded in both the critically endangered Gilbert's Potoroo and in the analogue species, the Long-nosed Potoroo. In mammals, inhalation of *Cryptococcus* from the environment usually results in subclinical disease that is rapidly resolved in the healthy animal. However, clinical disease may be seen in the immunologically compromised animal (*C.neoformans*), or when the dose or pathogenicity of the organism is overwhelming (*C.gattii*). Common presentations of the disease in mammals include nasal disease, pneumonia, and disseminated disease including meningitis.

A post mortem diagnosis of cryptococcal meningoencephalitis was made in a captive born male Gilbert's potoroo housed at the CALM Two People's Bay Nature Reserve near Albany, Western Australia. This potoroo was euthanased due to progressive illness that was non-responsive to treatment. The causative agent was found to be *Cryptococcus neoformans* var. *grubii,* which was presumed to have been spread via faecal contamination from Bronzewing pigeons.

A female Long-nosed potoroo, resident of the Perth Zoo colony, initially presented with non-specific signs of hind limb weakness, muscle atrophy, weight loss, and inappetence. CBC and serum biochemistry were inconclusive, and Toxoplasma serology was negative. There was little response to antibiotic therapy. In the ensuing weeks, this animal developed a progressive head tilt, circling, recumbency and visual deficits. Blood taken for LCAT analysis showed serological evidence of cryptococcosis. An aggressive systemic oral antifungal therapy regime was subsequently commenced using fluconazole and amphotericin B.

Although there was an initial improvement in neurological signs and appetite, the animal did not continue to recover, and was eventually euthanased. Post mortem evidence of cryptococcal meningoencephalitis and optic neuritis was found, and *Cryptococcus gattii* was cultured from cerebral tissue. This species can be associated with certain *Eucalyptus* spp. trees, which are offered to the potoroos as browse.

Cryptococcosis represents a potential threat to the survival of the critically endangered Gilberts potoroo. There are reports of outbreaks of cryptococcosis in other captive species, caused by provision of contaminated substrate; therefore, avenues for transmission such as the provision of heavily contaminated eucalypt browse and access to bird droppings may need to be considered in captive management protocols. Early diagnosis and treatment of disease may lead to improved survival rates.

48) PATHOLOGY AND SERODIAGNOSIS OF HYDATID DISEASE IN MACROPODS

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Echinococcus granulosus, the cause of hydatid disease, was introduced to Australia at the time of European settlement. The disease is now widespread in native macropods which act as intermediate hosts for the parasite with dingoes being the most significant definitive host. Some studies have looked at prevalence rates in small numbers of different macropod species in various areas in Australia but no large scale studies have been undertaken. Little is known of the natural progression of the disease in these species. Currently no reliable serodiagnostic test exists for non-human intermediate hosts.

In this study hydatid cysts and serum from infected animals were collected from commercially harvested macropods taken on 21 properties near Roma, southeast Queensland, over a 7 month period (June-Dec 2004).

Prevalence rates on properties ranged from 0-9.1%. No association was found between prevalence of the disease on properties and their orientation to the dingo fence. Further analysis will be undertaken to determine if physical geographic features or presence of other livestock are correlated with prevalence rates.

1971 animals were harvested. 49 were infected (2.5%). Prevalence rates varied among species and between sexes: eastern grey kangaroo (*Macropus giganteus*) males 2.1% (24/1154), eastern grey females 4.4% (24/544), wallaroo (*M. robustus*) males 0.8% (1/129), wallaroo females 0% (0/2), red kangaroo (*M. rufus*) males 0% (0/89), red females 0% (0/34). The mean age of infected animals was 7.5 years (+/-3.2). Using a multilevel model to account for clustering by property, on univariate analysis, females were significantly more likely to be infected than males ($p = 0.004$). Females were approximately 2.7 times as likely to be infected (odds ratio 2.74, 95% confidence interval 1.37, 5.50).

All 49 infected animals had one or more lung cysts. Three animals also had cysts in the pleural cavity and one animal had a liver cyst. Cyst numbers varied from 1-14 (mean 3.1 +/-3.2), cyst size from 0.5x0.5x0.5 to 15x10x10cm. Forty-three animals had fertile cysts demonstrated by the presence of protoscolices, and in 13 animals lesions showed degenerative changes (caseation was present in lesions from 11 of these animals calcification of lesions was seen in three animals). Histopathological examination of at least one cyst from each animal was undertaken to confirm the presence of the laminated membrane, the diagnostic feature for hydatid cysts. Variation in host reaction will be described.

A western blot test, developed for use in human sera, detecting antibodies to *EpC1* protein of *E. granulosus* (Li et al., 2003) was modified and optimised aiming to develop a serological test for the diagnosis of hydatid disease in macropods. Results using the confirmed positive samples above and negative samples from captive bred animals and post-mortem negative wild animals will be presented. The test may be suitable in screening small populations of endangered macropods to determine their infection status. Results from the test will be correlated with cyst size, cyst volume and signs of cyst degeneration to determine whether these factors are reflected in the antibody response.

Li, J., Zhang, W.B., Wilson, M., Ito, A., McManus, D.P., 2003, A novel recombinant antigen for immunodiagnosis of human cystic echinococcosis. Journal of Infectious Diseases 188, 1951-1960.

49) IMMUNOMODULATORY COMPOUNDS IN MARSUPIAL MILK

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Marsupials have short gestation periods compared to eutherian mammals, where young are born at an extremely early stage of development and lack a functional immune system. The young develop *extra uteri* within the pouch and the long lactation period that follows birth is critical to the pouch young by providing nutrients and maternally derived immunological protection.

There are three different types of milk produced during this period of lactation, the colostrum (pre-milk), early milk and late milk. It is hypothesised that marsupial milk contains proteins and peptides that have non-specific defence roles with possibilities for discovery of probiotics and immune stimulants/regulators. By using the tammar wallaby (*Macropus eugenii*) as a model marsupial and collecting milk at different lactation periods, it is proposed to identify the immunogenic proteins and peptides during the different stages and to detect other novel proteins involved in the immune response.

By identifying and characterising these proteins using proteomic techniques such as 2D gels, HPLC and MS, we can provide specific details on the structure and function of proteins involved in non-specific defence roles and have the potential to be used as antimicrobials for other species of marsupials. This can have future applications in managing disease in wild and captive populations of marsupials with possibilities for discovery of probiotics and immune stimulants/regulators.

50) ISOLATION, PURIFICATION AND CHARACTERISATION OF NEUTROPHIL PROTEINS FROM THE TAMMAR WALLABY

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Naturally occurring antimicrobial polypeptides play a major role in innate and adaptive immunity. Among the structured antimicrobial polypeptides, cathelicidins and defensins contribute significantly to host defence against the invasion of microorganisms in animals, humans, insects and plants. In Eutherian mammals, neutrophils play a role in immunity by secreting the peptides stored in the granules. In this study, a systematic analysis of the proteomic components of marsupial granulocytes using 2-D gel electrophoresis and mass spectrometry techniques is being carried out to investigate proteins from different cellular compartments, including granular proteins. The first part of the project involves the isolation and identification of proteins from neutrophil granules from the tammar wallaby. The identification of neutrophil proteins was carried out using the Mascot search engine, relying on identity with previously sequenced proteins in the NCBInr database and high probability scores. Secondly, functional studies will be carried out on HPLC fractions to investigate their antimicrobial activities and determine their role in the first line of defence of the host.

51) GENETIC STRUCTURE AND VARIATION OF RACCOONS (*PROCYON LOTOR*) IN THE EASTERN UNITED STATES: INSIGHT INTO ORAL RABIES VACCINATION (ORV) PLANNING

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Raccoons are mesocarnivores that have been associated with rabies virus in the United States since 1947. The raccoon rabies virus variant spread progressively from Florida into the southeastern states and was transported in the late 1970s to the mid-Atlantic region. It has since spread throughout the northeastern states, and the first raccoon to human transmission occurred in Virginia in 2003. Although an oral rabies vaccination barrier is being constructed in an attempt to halt the westward expansion of raccoon rabies virus, it is important to examine the population dynamics of the host species to make predictions about virus spread and susceptibility. The purpose of this study is to examine the population genetic structure of rabid and normal raccoons in the eastern United States and to determine the extent of gene flow among populations. Several hundred raccoon were sampled along the eastern seaboard, and sequences have been generated for an approximately 500 bp portion of the mitochondrial DNA control region. Levels of genetic variation and phylogenetic relationships of the raccoons were analysed using traditional population genetics methods, e.g., F-statistics with and without pre-assigned geographic subgroups, etc. In addition, we used data from a new focus of raccoon rabies identified in Ohio in 2004 to examine trends at a more localized level. Although results indicate that gene flow does occur among populations, high levels of population subdivision were identified. In addition, we found evidence of nonrandom mating or mating between closely related individuals in the raccoon populations. It appears that genetic structure data may prove useful for future ORV planning.

52) IDENTIFYING ECOLOGICAL FACTORS AFFECTING POPULATIONS OF RESERVOIR HOSTS OF LEPTOSPIROSIS: IMPLICATIONS FOR MANAGEMENT

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In Australia, there is a high occupational risk to contracting leptospirosis among the banana farm workers in the Innisfail and Tully region of north Queensland. Standardised-line trapping was conducted on banana and adjacent key habitats (sugarcane, rainforest and grassland) on farms (Farm I and Farm T) located in two major landscapes of the region during the September 2002 dry season and the March 2003 wet season. Farm I was located in an area with mostly rainforest and Farm T was located in an area with mostly sugarcane fields. Kidney samples from a total of 330 mammals were tested for renal colonisation to determine carrier status. Serum samples of a total of 612 mammals were tested for leptospiral antibodies to determine past and present infections. Since leptospires are shed through the urine of infected host animals, the relative abundance of carriers was used as a quantitative measure of the risk of infection of leptospirosis.

R. rattus, *M. burtoni* and *M. cervinipes* did not show any sign of infection, as determined by either demonstration of leptospires or antibody titres of 1:50 or more. *R. fuscipes*, *R. sordidus*, *R. leucopus*, *M. domesticus, U. caudimaculatus* and *P. nasuta* were renally infected and serologically positive to one or more serovars. *H. chrysogaster* and *I. macrourus* were only serologically positive, and no leptospires were demonstrated in the kidney cultures. Five serovars were isolated from the kidney samples: Australis; Celledoni; Arborea (from Ballum serogroup); Kremastos; and an unknown (serologically identified from the Tarassovi serogroup). Eleven serovars were detected serologically: Australis; Celledoni; Ballum; Kremastos; Tarassovi; Pomona; Robinsoni; Zanoni; Hardjo; Canicola; and Cynopteri.

The sample sizes of the relative abundance of carriers were large enough for statistical analyses for only *R. fuscipes* and *R. sordidus* carrying *Leptospira interrogans* serovar Australis. Both rats exclusively harboured Australis and accounted for 92% of that serovar isolations. The mean relative abundance of carrier *R. fuscipes* was significantly ($P = 0.0196$) higher in Innisfail than Tully. The overall mean relative abundance of carriers differed significantly between the habitats ($P = 0.0353$). The differences that were significant were between sugarcane [(mean±s.e.) 1.103±0.286] and banana (0.051±0.237) and grassland (0.0±0.387). In Farm I, the differences that were significant were between sugarcane (2.21±0.45) and rainforest (1.00±0.35), banana (0.15±0.28) and grassland (0.0±0.50). In Farm T, carriers were found only in rainforest.

The mean relative abundance of carrier *R. sordidus* did not differ significantly between the habitats, the seasons and the sites, however, there was an effect of site ($P = 0.0599$) by which Innisfail was somewhat higher than Tully.

These results indicate a higher risk of infection in Farm I than Farm T, and a higher risk of infection in the sugarcane and rainforest habitats. The implications of these results for managing of these carriers suggest that control efforts should be implemented in the sugarcane and rainforest, particularly in farms with mostly rainforest (Farm I). However, banana workers perform their activities in the banana; consequently, this habitat is where the potential risk of infection occurs. Management strategies should include the banana habitat. Additionally, the population ecology of each potential carrier highlights site-, season- and

habitat-specific times to implement control measures. Non-infected populations of susceptible species also need to be controlled to prevent epizootics or re-infection of a serovar into a population.

Further research is required to critically determine movements of carriers between habitats, relationship between prevalence and population abundance, and vertical transmission dynamics within and between species. This knowledge will be useful for further developing effective strategies for managing leptospirosis. Manipulative experiments may be used to critically determine if infection will decrease with decreasing numbers of carriers.

53) *CONTRACAECUM* SPECIES (*NEMATODA: ANISAKIDAE*) FROM THE AUSTRALIAN PELICAN: MORPHOLOGICAL CHARACTERISATION AND THE DEFINITION OF GENETIC MARKERS FOR ELUCIDATING THEIR TAXONOMY AND ECOLOGY

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Parasitic nematodes of the genus *Contracaecum* (family: Anisakidae) often occur in large numbers in the stomach of the Australian pelican (*Pelecanus conspicillatus*) and may cause clinical signs. However, very little is known about these parasites, particularly their taxonomy, biology, ecology and the pathogenicity in this host. Using classical and molecular approaches, *Contracaecum* from this species of pelican from different parts of Australia were studied. Microscopic study of adults revealed *Contracaecum bancrofti* and *C. clellandi* as well as larval stages of an unknown species of *Contracaecum*. The two known species were re-described (because of limitations of the previous descriptions) and the other was described morphologically. There was some overlaps in the total body length, intestinal caecum, ventricular appendix, oesophagus length and distance between vulva and the anterior end between *C. bancrofti* and *C. clellandi* adults, indicating that these characters could not be used reliably for the specific identification of females. Larvae could not been identified specifically due to a lack of development of informative characters such as the spicules. To overcome this limitation, a molecular approach, based on the mutation scanning and sequence analysis of the first and second internal transcribed spacer regions (ITS-1 and ITS-2) of ribosomal DNA was utilised. While no sequence variation in the ITS-1 and ITS-2 was detected among multiple individuals of each of the taxonomic units (species) (n=26 for *C. bancrofti*, n=14 for *C. clellandi,* and n=3 for the larval *Contracaecum* spp.), sequence differences of 13-25% and 33-48% were recorded for ITS-1 and ITS-2, respectively, among them. Consequently, genetic markers were defined in the ITS-1 and ITS-2 for the identification of *C. bancrofti*, *C. clellandi* and *Contracaecum* sp. and their differentiation. Defining these genetic markers now makes it possible to commence studying the (indirect) life cycles and the ecology of these parasites by specifically identifying larval stages in different species of fish (consumed by pelicans) and matching them with the adult nematodes found in the bird host. Thus far, *Contracaecum* type I from mullet (*Mugil cephalus*) have been defined as *C. clellandi*, indicating that this fish species is involved in its transmission to the pelican. This example demonstrates clearly the usefulness of the molecular approach for investigating these and other species of the Anisakidae.

54) BLOOD PARASITE PREVALENCE AND INFECTION INTENSITY IN *EGERNIA STOKESII* (REPTILIA: SCINCIDAE) IN RELATION TO TRANSMISSION TYPE AND SOCIAL STRUCTURE OF HOST POPULATIONS

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Disease transmission within natural populations may be influenced by transmission type, host density and population structure. While models of parasite transmission typically assume a homogenous host density, aspects of a species ecology and behaviour may create spatially structured host populations. The gidgee skink (*Egernia stokesii*) lives in stable social groups of between 2 and 17 related group members in rocky outcrops in the southern Flinders Ranges, South Australia. *E. stokesii* is host to three protozoan blood parasites (Apicomplexa) that are transmitted by mobile (*Plasmodium*) and immobile (*Schellackia* and *Hemolivia*) vectors. This study investigated blood parasite prevalence and infection intensity within social groups in relation to vector mobility and the social, spatial and genetic structure of host populations. The social structure of seven populations of *E. stokesii* was identified using recapture records of lizards from surveys conducted during 2003/2004. Blood samples were collected once from each lizard over this period to measure blood parasite infection status. Within groups, parasite prevalence and intensity was independent of group size and relatedness levels. The prevalence of mobile, vector-borne parasites (*Plasmodium*) within social groups was normally distributed, reflecting random transmission among social groups. In contrast, immobile, vector-borne parasites (*Schellackia* and *Hemolivia*) reflected an aggregated distribution, where high (0.75 – 1) and low (0 – 0.25) levels of prevalence were more common than expected under random transmission. Lizards belonging to social groups that overlapped with other groups, had higher blood parasite diversity and overall infection intensity compared with those belonging to isolated social groups. These patterns indicate that disease transmission within socially structured populations is influenced by vector mobility and host dispersal between groups.

55) RED-EARED SLIDERS (*TRACHEMYS SCRIPTA ELEGANS*) AS A MODEL OF RANAVIRUS INFECTIONS IN CHELONIANS

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Iridoviruses, of the genus *Ranavirus*, have recently been demonstrated to be emerging pathogens of wild and captive chelonians in the United States (DeVoe et al, 2004, Johnson et al, 2004). However, experimental transmission studies have not been previously performed to confirm that *Ranavirus* is the causative agent of associated histological changes in infected chelonians. Two experimental transmission studies were performed with an iridovirus isolated from a Burmese star tortoise (Johnson et al, 2004). In the first study, we evaluated box turtles and red-eared sliders as models for iridoviral infection in chelonians. While iridovirus infections have been previously reported in box turtles (DeVoe et al, 2004, Johnson et al, 2004), there are no reports of the susceptibility of red-eared sliders. Following inoculation (orally and intramuscularly), turtles were euthanased and necropsied. Histologic findings were similar between both species, indicating that red-eared sliders can serve as an appropriate experimental model for iridovirus infection. In the next study, ten adult red-eared sliders were assigned into two experimental groups (I and II) of four turtles each and one control group (III) of two turtles. Turtles in Group I were infected with 10^5 $TCID_{50}$ of iridovirus orally, and turtles in Group II were infected with the same amount of virus intramuscularly. One Group III turtle was mock infected with uninfected cell lysate orally while the other was mock infected intramuscularly. Experimentally infected turtles were either euthanased because of clinical signs of illness that developed following inoculation or were euthanased one-month post infection. Turtles infected orally showed minimal histological changes and multiple tissues were negative on PCR for iridovirus at the time of necropsy. Three of four intramuscularly infected turtles showed significant histologic changes consistent with those seen in naturally infected cases. Multiple tissues of these three turtles were positive on PCR for iridovirus. The results of this transmission study confirmed that iridovirus can be pathogenic to chelonians.

De Voe, R., Geissler, K., Elmore, S., Rotstein, D., Lewbart, G., Guy, J. 2004. Ranavirus-associated morbidity and mortality in a group of captive eastern box turtles (*Terrapene carolina carolina*). J Zoo Wildlf Med. 35(4):534-535.

Johnson, A.J., Wellehan, J.F.X., Pessier, A.P., Norton, T.M., Belzer, W.R., Brooks, J.W., Wagner, R., Stedman, N.L., Spratt, J., Jacobson, E.R. 2004. Iridovirus infections of turtles and tortoises. Proceedings of the Wildlife Disease Association, San Diego, CA.

56) RECOMBINANT VACCINE FOR PSITTACINE BEAK AND FEATHER DISEASE

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Full length recombinant beak and feather disease virus (BFDV) capsid protein was expressed using the bacterial expression system PinPointT. The full length recombinant protein reacted with sera from naturally immune cockatoo and chicken experimentally inoculated with BFDV. Reactivity proved antigenic epitopes of the BFDV had been conserved in the recombinant protein. The full length recombinant BFDV capsid protein induced an antibody response in two inoculated sheep. Antibodies raised against the recombinant capsid in the sheep recognised native viral inclusions in skin sections from a chronically infected cockatoo by immunohistochemistry. These results confirmed that antigenic epitopes of the BFDV had been conserved in the recombinant protein and are involved in the generation of an antibody response. Haemagglutination inhibition assay demonstrated that some psittacine birds vaccinated with the recombinant protein in conjunction with Freund's incomplete adjuvant produced antibodies that inhibited the haemagglutinating activity of BDFV. This is the first reported evidence of the potential value of a recombinant protein in vaccination and protection of psittacine species against the detrimental effects of BFDV and its future application for preservation of Australian Psittaciforme biodiversity.

57) DEVELOPMENT OF AN ELISA FOR THE DETECTION OF INTERFERON-GAMMA AS A DIAGNOSTIC TOOL FOR TUBERCULOSIS IN BLACK RHINOCEROS (*DICEROS BICORNIS*) AND WHITE RHINOCEROS (*CERATOTHERIUM SIMUM*)

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Bovine Tuberculosis (BTB) is believed to have entered the Kruger National Park (KNP) in the 1960's and was first diagnosed in July 1990 in an African buffalo (*Syncerus caffer*). Since then, in addition to buffalo, BTB has been found in at least 14 other mammalian species including kudu (*Tragelaphus strepsiceros*), baboon (*Cynocephalus papio*) and lion (*Panthera leo*). This has raised concern about the spillover into other potentially susceptible species like rhinoceros, jeopardising breeding and reallocation projects. Practical and reliable procedures to diagnose BTB in black rhinoceros (*Diceros bicornis*) and white rhinoceros (*Ceratotherium simum*) need to be developed. Skin testing as a diagnostic method for BTB in pachyderms has important practical limitations. More, intrinsic values of the test, i.e. sensitivity and specificity, are unknown. In cattle the bovine Interferon-gamma (IFNγ) assay is used as a routine diagnostic test for BTB. As a first step towards an *in vitro* diagnostic test for BTB in rhinoceros, a capture ELISA for the detection of rhinoceros IFNγ (RhIFNγ) was developed. The RhIFNγ was cloned, sequenced, expressed and purified. Subsequently two hybridoma cell-lines were established producing monoclonal antibodies (MoAbs) specific to recombinant RhIFNγ (rRhIFN-gamma). In parallel polyclonal anti-rRhIFNγ antibodies were produced in chicken eggs. Specific binding of the two MoAbs to rRhIFN-gamma was demonstrated in an indirect ELISA. In the development of a capture ELISA the two MoAbs were independently used for the capture of rRhIFNγ and the chicken antibodies anti-rRhIFNγ in the detection step. Both assays were shown to detect rRhIFNγ. Subsequently both systems were shown to detect native RhIFNγ in tissue culture supernatant, obtained after stimulation of purified rhinoceros lymphocytes with Concavalin A (Con A).

The RhIFNγ ELISA established now will enable further development of a whole blood assay that will be instrumental in diagnosis of BTB in rhinoceros.

58) VISITING *T. REX* ON PATHOLOGY ROUNDS: CASE REPORTS ON THE LOWER JAWS OF THE LARGEST PREDATORY DINOSAUR

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At four meters in height and 10 meters in length with jaws a meter long, tyrannosaurids would not have born a strong resemblance to their evolutionary cousins, the crocodilians and ratites. But for pathology purposes, the Tyrannosauridae may provide an interesting link between these two disparate groups. In 2001, numerous oval resorptive lesions were described by E. Rega in the mandible of a recently discovered *Tyrannosaurus rex* housed at the Field Museum in Chicago. These were diagnosed as having an actinomycotic aetiology on the basis of roughened fenestral boundaries, x-rays and CT-scan data. The unique occurrence of this infection in tyrannosaurids relative to other dinosaurian groups, and scattered reports of other cranial abnormalities prompted this study. The results indicate that abnormalities were quite common. Of the fifty-six individuals examined, one fourth of the mandibles displayed some form of anomaly.

Of all the extinct groups that are phylogenetically close to the modern crocodilians and the basal avian ratites and galliformes, the tyrannosauridae (*Tyrannosaurus*, *Albertosaurus*, *Daspletosaurus*, *Gorgosaurus*) provide a good basis for comparison. They retain many primitive characteristics but lie close to the evolutionary split between dinosaurs and birds. Tyrannosaurids have a massive dentary containing eleven to fifteen teeth that thickens rostrally reaching its maximum width at the mandibular symphysis. The dentary is overlain by a sabre-like supradentary dorsal to the Meckelian groove and articulates medially with the triangular splenial and caudally with the tall, gracile surangular-angular complex and crescentic pre-articular bones. The lower jaw differs from that of crocodilians because of the extensive development of the post-dentary series. In comparison to tyrannosaurids, the early toothed birds of the Mesozoic, *Hesperornis* and *Ichthyornis*, had an elongated dentary, a splenial incorporated lateral to the dentary, and reduction of mandibular profile. The ratites have significant variation in the curvature and elongation of the bill and rhamphotheca with the arrangement of the post-dentary bones appearing similar to that of the Anseriformes.

Of the one hundred-sixteen elements examined, the majority of abnormalities occur in the surangular and dentary. Part of this result can be explained by under-representation of other mandibular elements in collections, but the trend continues in the eight out of twelve specimens where all bones are present. Five of seventeen dentaries examined have gashes on the labial aspect of the dentary and sixteen of nineteen surangulars observed have resorptive fenestrae. The abnormalities observed on the dentaries are diagnosed as tooth traces from post-mortem scavenging, an isolated intraspecific wound, and idiopathic periosteal reactive ridges, which are observed on some specimens to display cranial curvature and overlap between the lingual and labial aspects.

The resorptive fenestrae on the surangular are mostly random in distribution with the exception of several cases. The abnormal fenestrae are semi-ovate and ringed by normal periosteum ranging from two to six centimetres in thickness and are generally proximal to other abnormal fenestrae. In some specimens post-burial fragmentation of the bone has obscured a portion of these fenestrae, making them appear to be embayed regions on the bone's surface. The tentative differentials for this condition are individual variation, developmental abnormality, bite trauma, osteomyelitis, fibriscess, idiopathic neoplasia and metastatic carcinoma. Diagnosis of this condition is awaiting confirmation from radiologic

imaging. Of these differentials, individual variation, bite trauma, and metastatic carcinoma may be eliminated as options on the basis of high frequency, incorrect penetration order and pattern, and confinement of the condition to a limited region.

The recurrence of an abnormal fenestra located non-randomly just ventral to the surangular fenestra was recorded for several cases. During the course of investigation, a potential developmental sequence of this anomaly was discovered, with the initial phase mimicking a depression fracture, an intermediate phase consisting of a bowl shaped lytic-lesion on the labial aspect with a small foramen communicating to the lingual aspect, and a third phase in which the fenestra appears but varies in diameter. Preliminary histological analysis of a specimen in the second phase, revealed extensive remodelling of the bone tissue and reduction of bone density. X-rays and CT scans of the region showed similar density trends. The aetiology of this feature is unknown, although pressure from an overlying fibriscess or neoplasia may be theorised.

Isolated cases of noteworthy anomalies include fracture callusing of the pre-articular, an apparent drainage sinus on the ventral aspect of the dentary, and muscle scarring on the dentary and angular. Additionally, two pre-articulars exhibit similar neoplasias located cranially on the lingual aspect. Differentials are pending radiologic examination, but the growths bear a superficial resemblance to osteomas seen in some avian species.

The preliminary results of this survey indicate that the Tyrannosauridae had mandibular abnormalities that more closely resemble those found in crocodilians and not those in ratites. With future research on ratite oral pathology, perhaps more connections may be found between these dinosaurian tyrants and their avian descendants.

END OF STUDENT SESSION TWO

SESSION 4:
MANAGEMENT OF WILDLIFE DISEASES

59) PROSPECTS FOR PREVENTION, DETECTION, AND MANAGEMENT OF EMERGING DISEASES

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Biosecurity Australia develops and reviews quarantine policies to protect Australia's agricultural industries and environment from exotic pests and diseases. The process to develop a new quarantine policy, where no policy exists, is called an import risk analysis (IRA), which is undertaken by a team of scientists and technical specialists.

Biosecurity Australia is active in the development of international quarantine standards and helps to develop quarantine expertise in our region.

To improve the capability of recognizing emerging wildlife diseases better national surveillance systems are needed as well as better integration and sharing of information. More and more there is a need for meaningful collaboration between organisations and government departments.

In 2002 the Australian Wildlife Health Network (AWHN) became a reality. The aim of the AWHN is to promote and facilitate collaborative links in the investigation and management of wildlife health surveillance and diagnostic information.

While some progress has been made with the management of emerging wildlife diseases with the production of generic plans, the issues are complex. One major issue is that the veterinary services normally responsible for animal disease management are usually within departments of agriculture, while authority for wildlife are located elsewhere in the government sector.

60) EMERGING WILDLIFE DISEASES: A MANAGER'S DILEMMA

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Since the beginning of formal training for wildlife management in Universities across the United States, students have been taught to focus on building up populations of animals for the benefit of the species and for human use. Throughout the history of wildlife management, the impact of disease organisms on wildlife populations has received little attention except in exceptional cases. The globalisation of travel, increase in interest in exotic pets and more sophisticated techniques for diagnosis has advanced the spectre of zoonotic diseases impacting wildlife populations and potentially infecting humans or livestock to the forefront in all fields of wildlife, agricultural and human health circles. Wildlife managers, some with decades of experience, are facing a new paradigm on how to manage populations that have been or may be impacted by zoonotic diseases. This shift in strategy has caused a dilemma for those who have spent a career trying to increase populations and provide a harvestable surplus for the enjoyment of the public. Additionally, the general public has long been told that more is better, while disease management may require a reduction or elimination of certain populations of treasured wildlife species. Today's wildlife manager must balance public perceptions, political demands, human and livestock health concerns, and species viability with the need to monitor, control, and, in some cases, attempt to eliminate zoonotic diseases in wildlife populations for the benefit of all concerned.

61) PATHOLOGY OF NATIVE AUSTRALIAN WILDLIFE SPECIES – WHO'S RESPONSIBILITY?

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Whereas for commercial livestock and companion animals funding for pathological examination is normally available from owners, this is clearly not so with wildlife species which therefore too often are neglected, and unfortunately "fall through the cracks" when such examination is needed to establish or confirm a diagnosis of illness or fatal disease.

During recent years the need to better examine wildlife found sick or dead has been increasingly recognized in consequence to emerging zoonotic diseases such as morbillivirus, Nipah virus or West Nile virus infections. Additionally there is now greater awareness of loss or degradation of the habitat of many wild species, and the impact this has on "stress" and disease, such as with *Chlamydophila* spp. infection in the koala. Furthermore, it is probably now being better understood and appreciated, that even in the absence of overt disease, examination of wildlife can provide a valuable and perhaps unique index of environmental change; heavy metal and pesticide residue levels in marine mammals are appropriate examples. Importantly, some pathological examination must accompany toxicology if any injurious effects of such toxins are to be revealed.

In spite of this heightened community awareness of wildlife disease and its significance there is a need to strengthen procedures that will ensure that adequate, ongoing and appropriate pathological examination of wildlife species will be pursued.

In Australia, the amount and type of wildlife pathology conducted have in recent years been influenced by contraction of government veterinary laboratory services and a shift of much pathology to the private sector. In addition to these laboratories, pathological examination of wildlife - project-related or for primary diagnosis - is carried out within the veterinary schools, at the Australian Registry of Wildlife Health based at Taronga Zoo, and at the Australian Animal Health Laboratory at Geelong.

In this presentation the extent to which these organizations collectively provide a satisfactory, nation-wide coverage of wildlife pathology, will be considered and suggestions made as to how such coverage might be improved.

62) THE AUSTRALIAN WILDLIFE HEALTH NETWORK AND WHIS - THE WILDLIFE HEALTH INFORMATION SYSTEM

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The Australian Wildlife Health Network (AWHN) is a National initiative of the Commonwealth Government and is managed under the Wildlife Exotic Disease Preparedness Program (Australian Department of Agriculture, Fisheries and Forestry). Its mission is to promote and facilitate collaborative links in the investigation and management of wildlife health in support of human and animal health, biodiversity and trade. Core business is wildlife disease surveillance. Key strategic objectives of its surveillance and investigation program are to:

1. provide and operate a national database of wildlife health information, which includes historical disease incident reports and;

2. provide and operate an interactive Website, which can be used for reporting and accessing Australian wildlife health information.

Staged development is being used to deliver a simple data capture system for wildlife health information around Australia (the wildlife health information system or WHIS). The system is designed to function in close-to-real time using a website with a series of purpose built data entry screens linked to a simple back-end MS Access database platform.

The first stages of WHIS have recently gone on-line. The system, its strengths, weaknesses and how data are accessed, is demonstrated.

WHIS functions as an integral part of a new, nationally integrated wildlife health system for Australia. Though currently data poor, it will grow over time to become a significant resource for those requiring information on diseases with wildlife as part of their ecology. Access to this type of information will improve management of issues relating to trade, human health and conservation that involve consideration of diseases with wildlife as part of their ecology.

63) A WILDLIFE DISEASE SURVEILLANCE STRATEGY FOR NEW ZEALAND

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The EpiCentre at Massey University will complete a 2-year project to develop a national strategy for wildlife disease surveillance in New Zealand, by the end of June 2005. This work has been conducted under contract to the Ministry of Agriculture and Forestry (MAF) Biosecurity New Zealand on behalf of an interagency team comprising MAF, Department of Conservation (DOC), and Ministry of Health (MoH), in response to an increasing awareness of the need to strengthen wildlife disease surveillance in New Zealand.

The scope of this project defines wildlife as "terrestrial vertebrates native to New Zealand, feral terrestrial vertebrates introduced to New Zealand, marine mammals, freshwater fish, native macro-invertebrates, and invertebrate vectors of disease". The review covers diseases that affect wildlife species, plus diseases of wildlife that may infect humans and/or managed populations such as livestock and domestic pets.

Key components of the project include: a review of current wildlife disease surveillance in New Zealand, developing methodology for prioritising wildlife diseases for surveillance purposes, implementing a pilot project to collect surveillance information from wildlife species, developing a strategy for wildlife disease surveillance, including data collection and information management.

A database of diseases in wildlife was established during the course of the project. Given the disparate nature of wildlife disease information, this provides an important resource which maintains wildlife disease information in one location. The process of gathering the disease information enabled us to identify key sources of information on wildlife diseases in New Zealand, which provided an important basis to developing an information management strategy to support wildlife disease surveillance.

We interviewed a large number of individuals and organisations involved in wildlife management and wildlife disease expertise, which enabled us to identify the networks through which wildlife disease information is generated for each of the taxonomic groups of wildlife. This information will form an important part of developing the wildlife disease surveillance strategy by identifying systems that are working well, as well as identifying important gaps, which need to be addressed to ensure that we have an understanding of the baseline disease status of wildlife species, and that unusual morbidity/mortality events are recognised and investigated.

Key components of the disease surveillance strategy will include:

- obtaining information on the baseline disease status of each order;
- investigating unusual morbidity and mortality incidents; and
- targeting surveillance activities for priority pathogens.

The strategy will identify systems that need to be in place, plus the steps needed to develop existing systems so that they meet those recommended in the strategy. This will include factors such as the capacity to detect morbidity & mortality events in the different wildlife

orders, the awareness to report these and have them investigated, the expertise to diagnose diseases, plus systems to promote further investigation of new pathogens to determine their significance and their epidemiology in New Zealand.

An information strategy to support wildlife disease surveillance will include:

- a system for storing and updating wildlife disease information, and identifying potential for links to current information systems together with any associated issues of information compatibility; and
- recommendations for an automated, or semi-automated, system to allow access and retrieval of information from a variety of sources, including information on wildlife species, wildlife disease, plus expertise, resources and facilities information relating to wildlife surveillance;

We have developed a methodology for prioritising wildlife diseases for surveillance, which is based on the OIE approach for risk analysis to support international trade of animals and animal products. Rapid risk analyses were conducted on a total of 79 exotic and endemic pathogens, which comprised a release assessment to evaluate the probability of entry to New Zealand, an exposure and a consequence assessment within the major population sectors: wildlife, livestock, humans and companion animals. A semi-quantitative approach was used to score each pathogen within each of these categories. We evaluated various approaches to using the scores to identify priority pathogens which would justify the allocation of resource to implementing a targeted surveillance system to detect their introduction to New Zealand.

The pilot project focused on conducting surveillance for avian influenza virus and paramyxoviruses in migratory birds in New Zealand. The aim of this project was to evaluate approaches to on-going surveillance in a wildlife species. Given the logistical challenges of capturing and sampling migratory birds, we focused on evaluating the methods for sampling endemic waders and waterfowl that are in the same environment as birds that migrate to New Zealand. We compared sampling faecal deposits in the environment as an alternative to capturing birds and collecting cloacal swabs, as a less invasive technique that may be more feasible to implement on a repeated basis in the future.

The wildlife disease surveillance strategy aims to incorporate the information and experience generated from the project activities described above to produce a set of recommendations that build on the existing infrastructure to detect and investigate wildlife morbidity and mortality events in New Zealand, identifying additional resources, training and support material to make this more effective where necessary.

64) CONSERVATION MEDICINE IN A THREATENED ECOREGION: ADDRESSING ECOLOGICAL HEALTH IN THE CALIFORNIAS

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The *Escalera Ecológica* Initiative is proposed as a mega-scale linkage of terrestrial and marine protected areas throughout the Californias, providing a bi-national ecological staircase and an organizing theme for galvanizing regional wilderness preservation. This initiative is needed as a linkage for the regional wildlands and to countervail the *Escalera Nautica* mega-tourism project. The Escalera Ecológica Initiative will bring greater attention to these reserves, and draw attention to the fact that some have real protection and some do not. The initiative will focus on the interconnectedness between wildlife, plants, and people of our region and our common heritage, health, and prosperity. Many of these reserve areas remain largely in pristine condition, and contain great diversity of geological, biological, and cultural resources. We need greater collaboration, communication, and cooperation among the separate regional and local agencies, NGOs, researchers, and residents as the direct threats to our wildlands escalate. The Escalera Ecológica Initiative will provide an organizing concept and a networking opportunity for communicating the need to preserve the wildlands of this region and promote rigorous research and conservation actions.

We begun with international and national partners to evaluate the health of marine vertebrates, primarily California sea lions, sea turtles and brown pelicans, and determine if we can link their health status to specific ecosystems along the Californias. We are using these species as sentinel species (SS) of marine ecological health. Sentinel species serve as indicators of their environment and may reflect the quality of health in marine ecosystems. The single species approach may provide a series of "snap shots" of environmental changes to determine if animal, human or ecosystem health may be affected. Marine vertebrates are good integrators of changes over space and time and represent excellent sentinels of ecosystem health. By moving in and out of infected/polluted areas, they can spread pathogens and contaminants geographically as well as throughout the food chain. The "utility" of the SS selected should consider its value and relevance to decision makers, conservationists, local communities and to society at large. The SS concept is a proactive approach in the development of an "*Escalera Ecologica*". We will be able to measure the health impacts in this fragile ecoregion by providing an "early warning" system for emerging diseases and environmental contaminants and proactively monitoring the course of disease related activities requiring prevention, remediation or control as the "*Escalera Nautica*" may develop. It will require a suite of sentinel species (sea turtles, sea birds and marine mammals) including different trophic levels, ecological roles, taxa and different spatial/temporal scales and conservation medicine teams. Sentinel species are the proverbial "canaries in the mineshaft" clearly indicating the health and condition of this threatened ecoregion. We have identified a number of critical research needs and opportunities for transdisciplinary international collaboration with a proactive vision that could help advance the use of marine vertebrates as SS to monitor the health of the Californias ecoregion and in turn the "*Escalera Ecologica*".

65) MANAGEMENT OF SEMI-FREE RANGING POPULATION OF ARABIAN ORYX IN UNITED ARAB EMIRATES

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Authors are summarizing their experience with establishing and managing of two areas where Arabian oryx (Oryx leucoryx) were kept in conditions similar to wild free-ranging environment. After enormous conservation effort the world population of Arabian oryx gets stabilized and serves as an excellent example to save an almost extinct species. United Arab Emirates holds probably the largest part of the world population. The first ideas were to manage the surplus animals from different existing and rapidly expanding collections as well as to create as much as possible genetically diverse population living with minimum human interference.

In 1998 the first stage of relocation started with the establishing of a new semi-free ranging population of an Arabian oryx (*Oryx leucoryx*) to the premises of Al Maha Desert Resort. The selected area was predominantly used as camel grazing in the past with relatively abundant natural vegetation represented by acacia "salam" tree (*Acacia tortilis*), ghaf tree (*Prosopis cineraria*) and sidr tree (*Ziziphis spina-christi*) providing enough shade for the introduced oryx. Large sand dunes as well as hard gravel plains dominate the features of the area. A total 72 animals were introduced into the dessert ecosystem represented by 25 km^2 of fenced enclosure. Animals arrived as donations from different private and governmental establishments with no history of infectious diseases in the past. They were kept in the holding pens for 1 month to acclimatize, adapt to the new feed and monitor any signs of disease or capture myopathy. To improve the body condition of the herd, alfalfa pellets and good quality hay with mineral supplements were provided before the release from holding pens, as well as during the first years. All animals were dewormed and vaccinated with polyvalent clostridium vaccine at the day of arrival. Quarantine and health screening protocol were introduced (Woodford, 2001). Serology survey for brucellosis and tuberculosis was conducted prior the release, all with negative results. Six feeding spots and two water pools were built around the places with abundant vegetation to provide food and water for the animals. Large numbers of mineralised salt licks were placed near the water sources. Once released our aim was to observe the herd with as little interference as possible. Wounded or sick animals were removed from the population. All cases of mortalities were collected and examined at the closest veterinary pathology department. We have experienced strong winds and shifting dunes, which leave large gaps under the fence from where the oryx can escape. We solved the problem by employing a full time bull-dozer to level the sand around the perimeter of the fence. We observed that the oryx were forming 2 large groups, which started to split immediately after the first calving period. A few adults died due to ingestion of different foreign objects, such as plastic bags, ropes, irrigation pipes, and the remains of old camel farms in the area. Later the most common causes of mortality were the traumatic injuries of males due to fighting. The fights for position in the social hierarchy took place predominantly around the feeding spots. Their distribution into remote places reduced the contact time and consequently the chance for the conflict between individual groups. The population reached numbers of 220 individuals in 2004 and the resort area expanded by additional 120 km^2. The herds of oryx in the other area of Ajban farm in Abu Dhabi were kept in far less extensive conditions. We have had 4 different breeding herds in pens of approximately 10 hectares in size. In these smaller enclosures we experienced far higher incidences of fighting between males. We had to form a separate bachelor herd. To keep a

varied gene pool we have rotated the males every 2 years between the breeding herds and bachelor herd. Translocation of individuals was successfully carried out by darting the animals with different mixtures. Most frequently the combination of 1 mg/kg xylazine (Anased) and 1mg/kg ketamine (Ketavet), reversed with 1.5 ml atipamezole (Antisedan) i.v./pro toto was used. This anaesthesia procedure was introduced after recurrent cases of capture myopathy and mortalities due to hyperthermia using etorphine (Immobilon), mainly in hot summer months (Molnar *et al.,* 2002). The most suitable combination was the medetomidine-ketamine-butorphanol mixture 1.5-2 ml+0.8-1 ml+ 0.2 ml/animal i.m. (Domitor-Ketavet-Torbugesic), that causes minimum influence on blood and acid–base parameter with very rapid onset (Molnarova *et al*., 2004). During the first 4 years the population has constantly growing, reaching more than 200 individuals at the moment. Food supplementation with hay was important during summer months to prevent extensive damage on the vegetation and severe deterioration of body conditions in young, not adopted animals.

The project showed that Arabian oryx could easily establish itself into natural conditions, even after spending a long time in captive conditions. There are minimum health risk factors influencing the population if separated from domestic livestock. The biggest challenge for free ranging reintroduction in conditions of United Arab Emirates will be to find suitable environment, large enough to provide sufficient diverse vegetation for shading and grazing. To consider around 850 kg dry matter food requirement per animal per year it will be difficult to find suitable abundant vegetation for the large groups. Respectively, permanent food supplementation will be necessary to avoid extensive overgrazing. A united management guidelines accepted by all parties involved in Arabian oryx breeding would be essential for the future to coordinate the diversity of the population.

Molnar, L., McKinny, P. 2002. Immobilization of Arabian ungulates in Middle East climate conditions. Proceedings of 4th scientific meeting European association of Zoo and Wildlife Veterinarians (EAZWV) 56-58

Molnarova, M., Molnar, L., Halanova, M., Halan, M., Hoy, D.2004. Effect of anaesthesia and translocation during hot climate condition on acid-base and other blood parameters of Arabian oryx.WDA Africa and Middle East 3rd Annual Meeting

Woodford, M.W. 2001. Quarantine and health screening protocol for wildlife prior to translocation and release into wild. OIE –IUCN, Care for Wild International and European Association of Zoo and Wildlife Veterinarians (EAZWV)

66) EVALUATION OF CARCASS DISPOSAL TECHNIQUES AFTER AN ANTHRAX OUTBREAK IN FREE-RANGING BISON IN NORTHERN CANADA

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Following an outbreak of anthrax in a free-ranging bison (*Bison bison*) herd in the Hook Lake region of the Northwest Territories, the infected carcasses were treated with various combinations of incineration with either wood or coal, and application of a 7.5% solution of formaldehyde. Before and after each treatment, surface environmental samples and tissue swabs were collected from around the carcass site and screened for the presence of viable *Bacillus anthracis* spores via selective culture and confirmatory assays in order to quantitatively evaluate the effectiveness of treatment. Three hundred forty environmental samples and 74 swabs were collected from around seven carcass sites. Viable *B. anthracis* spores were recovered from 115 (33.8%) of the environmental samples and from 29 (39.2%) of the swabs. The culture data indicated the importance of ensuring adequate airflow underneath a carcass either by elevating the carcass or applying solid fuel below the carcass to facilitate complete incineration. Empirical observations suggested that coal is more effective than wood for carcass incineration as it burns hotter and longer. Culture data also indicated that the combination of carcass incineration followed by directed decontamination with formaldehyde was the best approach to reducing anthrax spore contamination at a remote field site.

67) FEASIBILITY OF BAIT DELIVERING A PSEUDO-RABIES DISEASE VACCINE TO WILD FERAL PIGS

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Feral pigs, *Sus scrofa*, are an introduced pest species in numerous countries, including Australia, New Zealand, America and the United Kingdom. In the United States, wild swine are estimated to number around 1 million individuals, with the species continuing to increase their range. Concurrently, increased prevalence of pseudo-rabies (Aujeszky's Disease) and *Brucella suis* has been reported. Both these diseases are of economic importance to domestic pig producers and wildlife in the USA. Although Aujeszky's Disease is currently exotic to Australia, it could also significantly impact on domestic piggeries in this country should it be accidentally introduced, with the millions of feral pigs in Australia potential acting as a vector for the disease. Vaccines to combat pseudo-rabies diseases are available, however a suitable commercial delivery vehicle has been lacking. The advent of PIGOUT® for lethal pig control in Australia may solve this problem through providing an attractive and target specific vehicle in which encapsulated vaccines could be orally delivered. Pen and field trials are currently underway in Australia and the USA. This paper will present the results of recent trials, detail further trials, and outline the potential of PIGOUT® for large-scale pseudo-rabies disease vaccinations in the future. It will also outline other potential uses for this technology.

68) FORMULATION OF LIVE BCG VACCINE FOR DELIVERY TO WILDLIFE SPECIES

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Tuberculosis caused by *Mycobacterium bovis* affects a wide variety of animal species, threatening economies and biodiversity in many countries worldwide; as well as presenting a source of zoonotic infection. Whilst control programmes based on a test and slaughter strategy have been effective in controlling bovine tuberculosis in some countries, in others the infection persists in cattle populations in the face of control due to maintenance of *M. bovis* within a wildlife reservoir. In other settings it is either impractical or uneconomical to introduce or maintain such control measures, and large-scale culling of endangered or protected species is frequently unacceptable and unsustainable. Into this context, vaccination against *M. bovis* is an attractive and potentially cost-effective option. However, there are a number of obstacles to the introduction of vaccination. These include identification of a suitable vaccine that is both safe and efficacious, as well as the means to deliver the vaccine to the target species. As such, the delivery of BCG (Bacillus Calmette-Guerin) vaccine in bait for oral consumption provides the most realistic approach in the short to medium term. BCG, a live attenuated form of *M. bovis*, has a history of safety and efficacy in a variety of species but it is generally accepted that the vaccine must be administered as a live preparation to be fully efficacious. This presents the first challenge: to incorporate live BCG vaccine in a formulation able to: 1) protect the vaccine from degradation in stomach acid; 2) release the vaccine in the lower gastrointestinal tract for uptake by the gut-associated immune system; and 3) be compatible with a palatable bait and stable in the environment before consumption. We have begun to address these issues by evaluating a variety of enteric formulations, including pH-sensitive polymers, polysaccharide matrices, calcium alginate, and gelatine. Use of the latter two materials has allowed us to formulate a live BCG vaccine that remains viable in simulated gastric fluid for up to three hours, whilst being released at elevated pH. We shall present these data together with the persistence of the vaccine at different temperatures and preliminary evaluation of the efficacy of BCG-gelatine vaccine in a guinea pig aerosol *M. bovis* challenge model.

69) HEALTH ASSESSMENT OF GEOFFROY'S CATS (*ONCIFELIS GEOFFROYI*) IN ARGENTINA

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During two projects on wild felid ecology and conservation 15 Geoffroys' cats (*Oncifelis geoffroyi*) were sampled for health evaluations at two different protected areas in Argentina. Captures were conducted in 2000 at Campos del Tuyu Wildlife Reserve and at Parque Nacional Lihue Calel in years 2002 and 2003. Five males and 10 females were fitted with radio collars after receiving a complete physical exam and blood and fecal samples were collected. Serology for selected infectious agents was performed, including feline leukemia virus (FLV), infectious peritonitis (FIP), feline immunodeficiency virus (FIV), feline panleukopenia (FP), canine distemper virus (CDV), feline callicivirus (FCV), feline herpesvirus (FHV), rabies (R), leptospirosis (Lep), toxoplasmosis (To) and dirofilariasis (Di). All animals were negative to FLV, FIV, FHV, R and Lep (serovars Pomona, Hardjo, Icterohemorrhagiae, Grippotyphosa). Antibodies to FCV were found in 93 % of tested animals, while 67% were positive for To, 33% for CDV, 13 % for FIP, 6.6% for FP and 6.6% for Di. Adult parasites recovered from necropsied animals and eggs in fresh feces revealed the presence of various nematode families, including: Ascarididae. Trichuridae, Capillariideae, Rictulariidae, Spiruridae and Ancylostomatidae; cestodes from families Taenidae and Anaplocephaliidae and oocists of Eimeriidae. Many of these findings are reported for the first time in free-ranging O. geoffroyi. During the last year of the study, six animals were found dead and complete necropsies were performed. Gross observations revealed severe emaciation, with varying degrees of parasite infestation. Histopathology confirmed degenerative changes in several organs which were compatible with progressive inanition and death by starvation. There was no evidence of concurrent bacterial or viral infections. The death of these individuals was directly related to a marked decrease in prey species in the study area, as determined by radio-tracking of animals and prey abundance studies. Our results suggest exposure (recent or past) to common domestic carnivore diseases. These findings highlight the need for integrating the effects of diseases, environmental changes and contact with domestic animals in the planning of conservation efforts for wild free-ranging felines in Argentina.

70) DEVELOPMENT OF A HEALTH SCREENING PROTOCOL FOR CAPTIVE BRED LITTLE PENGUINS (*EUDYPTULA MINOR)* PRIOR TO TRANSLOCATION TO A WILD POPULATION

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Little Penguins are fairly common in the waters of Southern Australia, mainly breeding on offshore islands. The last known breeding population of Little Penguins on the mainland of NSW (North Sydney Harbour) was listed in 1997 as endangered on Schedule 1 of the NSW Threatened Species Conservation Act 1995.

Translocation of captive bred fledglings from an established breeding colony at Taronga Zoo was proposed as part of a wider recovery program conducted by the Department of Environment and Conservation.

A health screening protocol was developed in order to minimise the risks of transmission of potential pathogens to the wild population, and to ensure that the survival of translocated penguins was not compromised. The protocol was based on a review of known diseases of Little Penguins in the wild and in captivity together with necropsy results from the captive population at Taronga Zoo, and from wild penguins from the Sydney area.

The screening protocol requires that each bird undergo a physical examination within seven days of release. Blood is collected for full blood count, biochemistry panel, DNA sexing and serum storage. A cloacal swab is submitted for microbiological culture, and a faecal sample for parasitological examination. The bird is weighed and permanently identified by subcutaneous microchip transponder insertion.

Translocation of individual fledglings is dependent on the results of health screening, disease status of the rest of the captive colony and continuing isolation of the penguin collection from other birds within the Zoo.

SESSION 5:
MONKEY POX

71) MONKEYPOX ZOONOTIC ASSOCIATIONS: U.S. 2003 OUTBREAK

Christina. Hutson, Kemba Lee, Jason Abel, Victoria Olson, Michael Dillon, Kevin Karem, Zachary Braden, Darin Carroll, Joel Montgomery, Paul Spurlock, Mary Reynolds, Inger Damon, and Russell Regnery

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A 2003 U.S. outbreak of human monkeypox was associated with exposure to captive prairie dogs (PD) (*Cynomys* spp.). The PD had been exposed to rodents, imported from Ghana, at an Illinois animal distributor. Animals (both dead and alive), with possible epidemiologic links to the outbreak, were analyzed for evidence of monkeypox virus (MPV).

Examples of at least three of the African species imported from Ghana were confirmed as MPV infected; these included, dormice (*Graphiurus* spp.), pouched rats (*Cricetomys* spp.), and rope squirrels (*Funisciurus pyrropus*). 14 out of 20 PD submitted for evaluation were MPV PCR-positive, and 10 of 11 of these were isolate positive. Multiple PD were shown to have high viral loads. MPV transmission from PD to another North American rodent species was suggested by isolation of MPV from a captive ground hog (*Marmota monax*) living in a household that contained an infected PD. Other species with evidence of MPV DNA and associated with the Illinois distributor, but not originally from the Ghanaian shipment, included two species of South American opossums, a hedgehog, and a jerboa.

Serologic evidence of orthopoxvirus-specific IgG was found in 13 of 18 Gambian rats, although only 2 of 18 of this species was either MPV DNA or isolate-positive at the time of sampling, suggestive of transient infections. 7 of 37 dormice tested were found to be MPV DNA-positive. MPV PCR-positive ocular and oral swabs were obtained from two dormice (without obvious lesions) one of which died one month later, at which time the tissues were isolate positive, and the other was euthanized approximately 6 months later at which time tissues were MPV DNA-positive (only).

These results provide a new appreciation for the potential for novel host species to participate in MPV transmission to humans, lead towards a better understanding of potential African MPV reservoir species, and suggest that future introductions of MPV outside of Africa may pose threats to non-African mammalian species.

72) EVIDENCE OF EXPOSURE OF WILD RODENTS TO ORTHOPOXVIRUSES IN WEST AFRICA: A MONKEYPOX INVESTIGATION IN GHANA

Mary Reynolds[1], Darin Carroll[1], Richard Suu-Ire[2], Victoria Olson[1], Mubarak Osei Kwasi[3], Anna Likos[1], Jack Galley[4], Jeff Root[5], Joel Montgomery[1], Zach Braden[1], Jason Abel[1], Cody Clemmons[1], Kevin Karem[1], Russell Regnery[1]and Inger Damon[1]

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In April 2003, monkeypox virus was introduced into the United States *via* a shipment of wild-caught African rodents collected in the West African nation of Ghana. To better understand the source population for the 2003 U.S. introduction of monkeypox virus, we investigated rodent populations at locations from which the animals implicated in the outbreak had been collected, surveying for evidence of orthopoxvirus infection in a diverse rodent assemblage. Gambian rats (*Cricetomys* spp.) and several species of rope (*Funisciurus* spp.) and sun (*Heliosciurus* spp.) squirrels were collected from a forested/agricultural region of the country and dormice (*Graphiurus* spp.) and other small rodents were collected from a savannah-type habitat near the Volta Region. Animals with elevated levels of antibodies reactive with orthopoxvirus antigens and animals positive for orthopoxvirus DNA were found in both regions. Two animals with pox-like lesions were also identified.

73) THE MOLECULAR PHYLOGENETICS OF POTENTIAL MONKEYPOX RESERVOIRS

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Beginning in June 2003 CDC began receiving reports of human rash illness, subsequently diagnosed as monkeypox, among persons having had contact with prairie dogs (*Cynomys* spp.) which originated from an Illinois pet distributor. Several African rodent species, including potential monkeypox reservoirs intended for sale as pets, had been co-housed with these prairie dogs. The African rodents were traced to a shipment from Accra, Ghana containing 812 individuals including, Gambian rats (*Cricetomys* spp.), rope squirrels (*Funisciurus pyrropus*), sun squirrels (*Heliosciurus gambianus*), striped mice (*Lemniscomys spp.*), brush-tailed porcupines (*Atherurus africanus*) and African dormice (*Graphiurus spp*). Multiple rodent species from the African shipment and the Illinois facility had evidence of infection with monkeypox, and many of these had not previously been linked to monkeypox infections. The presence of virus and antibody in multiple species exported from Ghana, suggests either an active epizootic involving many species in the wild, or active transmission while animals were kept at a common holding facility, or during their transport to the US.

In order to identify the specific organism(s) responsible for the US outbreak, a follow-up investigation to Ghana was conducted in 2004. This expedition yielded a controlled collection of the implicated species of African rodents. The poorly understood taxonomy of these groups has complicated the exact identification of potential reservoirs of human infection. To resolve this we have obtained tissue samples from a wide geographic representation of individual mammal specimens assigned to these taxa and which are currently housed in various natural history museums. The genetic characterisation of these previously collected specimens, together with our specimen collection associated with the original shipment, are resolving long standing taxonomic complications of the potential host species, providing insight into the overall genotypic diversity of these species, and facilitating the identification of specific mammalian genotypes which co-occur with human monkeypox disease.

SESSION 6:
LYSSAVIRUS EMERGENCE AND MANAGEMENT

74) POTENTIAL APPLICATION OF LESSONS LEARNED FROM ORAL RABIES VACCINATION IN THE UNITED STATES

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Oral rabies vaccination (ORV) represents a potential method to be integrated with more conventional approaches to suppress and eliminate new variants of rabies that could emerge from host shifts in the virus or translocations. Recent experiences from coordinated ORV in the eastern U.S. and Texas may serve as applied operational models for the preparation of contingency action plans to address these potential rabies control challenges in Australia or elsewhere. In the U.S., formation of a multi-disciplinary team of expertise from key state, federal, county, and municipal agencies responsible for public health, agriculture, and wildlife management remains central to ORV decision making. This Rabies Management Team provides critical input on environmental compliance, communications planning, assessment of strategies, contingency action planning, ORV evaluation, economic analyses on the impacts of rabies, laboratory support and surveillance, vaccine and bait development, air and ground baiting capability and contracting, and research prioritisation. The formation of a similar team would be recommended to consider the likelihood of emergence of terrestrial variants of the rabies virus in naive areas; the potential impacts to public and animal health, including rare or endangered wildlife species; economic impacts; and cost-feasible strategic actions that could be taken to eliminate the virus. Contingency action planning is an integral component of the National ORV plan in the US, and has recently been adapted to try to meet critical needs in Ohio, Massachusetts, New York, Tennessee and Alabama. Contingency actions typically rely on enhanced rabies surveillance, trap-vaccinate-release (TVR), and ORV, often at higher bait densities, to contain outbreaks where ORV zones are not already in place or where ORV zones may have been compromised. The success of contingency actions in the context of larger scale ORV to contain and eliminate specific variant of the rabies virus continues to be evaluated in the U.S., as well as Canada. ORV is expensive, but as an adjunct to conventional rabies control, it represents a method that may be integrated in a strategic fashion to potentially eliminate emerging strains of the virus or the introduction of terrestrial rabies through translocations. We discuss ORV in the U.S. and some of the management challenges we face for perspective in considering actions that may be appropriate to contain and eliminate emerging rabies variants.

75) RESEARCH METHODS DEVELOPMENT TO ENHANCE WILDLIFE RABIES CONTROL STRATEGIES IN THE UNITED STATES

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Oral rabies vaccination using Vaccinia-Rabies recombinant (V-RG) vaccine-laden baits designed to target raccoons in the eastern and southeastern United States and coyotes and grey foxes in south and west-central Texas are used operationally to control specific strains of wildlife rabies. Research is conducted at the National Wildlife Research Center (NWRC) to develop methods to enhance the control strategies for the various host species. Investigations of the biology and population densities of the target species are being conducted to determine the optimum number and distribution of oral baits to achieve effective control and host movement patterns are being measured to evaluate if landscape features can serve as barriers to the spread of rabies. Skunk rabies occurs throughout a broad area of the central U.S. and California, but no oral vaccine is currently effective for this species. New vaccines are being developed; therefore, research on the development of acceptable baits for skunks is being conducted by NWRC scientists and we have found new bait designs and formulations to use in skunks and are working on better baits to use in raccoons for delivery of the oral V-RG vaccine. Field studies by NWRC scientists are presently underway to evaluate the skunk baits in 5 states to select optimum oral baits for the eventual delivery of vaccine. Further studies on the biosafety of the V-RG vaccine to non-target species are being conducted in experimental pen trials and during field vaccination programs. An 18-month experimental study to evaluate the long-term efficacy of oral V-RG vaccine for raccoons is being conducted at NWRC. The study will measure the development and duration of antibody following single and multiple vaccination doses and will determine if the antibody is protective against a rabies challenge at 6, 12, and 18 months. These data will be useful in evaluating post vaccination results following operational vaccination programs and assist in evaluating long-term vaccine efficacy and effectiveness in rabies control programs.

76) PARENTERAL VACCINATION OF ETHIOPIAN WOLVES (*CANIS SIMENSIS*) TO CONTROL AN OUTBREAK OF RABIES IN BALE MOUNTAINS NATIONAL PARK

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The last two decades have seen a number of infectious disease epidemics in wildlife populations. Ecologists and wildlife managers have begun to acknowledge the role played by pathogens and the potential threats they pose to endangered populations. However, conservationists remain ill-equipped to manage infectious disease outbreaks, through lack of knowledge, few existing practical strategies to provide guidance, or a lack of funding. There is a clear need to incorporate disease risks into conservation policy and programme management. We describe an outbreak of rabies in Ethiopian wolves in the Bale Mountains National Park, and the management steps taken to control the disease. The existence of a well-established monitoring programme in several sub-populations of wolves within the park afforded the opportunity to apply a quasi-experimental design to the intervention, and to monitor its immediate impact and long-term effects on the population. The immediate objectives of the intervention were to contain the virus to the outbreak area, and to protect wolf packs in core territories. Wolves were caught in soft-catch leg-hold traps and vaccinated intramuscularly with a 1 or 2 ml dose of rabies vaccine (Nobivac Rabies, Intervet, batch nos. 79056A & 71032A). Vaccinated wolves were marked using coloured ear-tags. Trapping sessions were again conducted 1, 6 and 12 months post-vaccination to assess the humeral immune response to vaccination. A booster dose of 1ml was administered to all recaptured animals. In total, 84 animals were captured once, 25 twice and 2 a third time. All wolves captured at ~30 days post-vaccination seroconverted (n=19). There was no significant difference in titres between wolves which received 1 vs. 2 ml of vaccine (n=18, $F=2.49$, $p=0.14$). Three of seven unvaccinated wolves captured in the outbreak zone were seropositive for rabies virus neutralising antibodies. During the course of the epidemic, 74 of 95 animals died or disappeared from the outbreak zone, with 13 of 15 carcasses testing positive for rabies; by contrast, no rabies-confirmed deaths occurred in the vaccination zone. All but one vaccinated wolf were confirmed alive one week after vaccination, and all but three wolves were alive six months later. Although parenteral vaccination may have been successful in controlling this outbreak, its suitability in the long-term (as either a prophylactic or emergency control measure) is questionable, as capture rates decline significantly with time. There is thus still an urgent need to explore other potential disease management tools, such as the use of oral vaccination technology.

77) LYSSAVIRUSES FLOURISH AMONGST A BEVY OF BATS

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Rabies is an acute, progressive encephalitis caused by viruses in the Genus *Lyssavirus*, Family *Rhabdoviridae*. To date, at least 11 putative lyssavirus species have been defined. Except for Mokola virus, whose epidemiology is poorly understood, bats appear as major lyssavirus reservoirs on a global basis. Phylogenetic introspection, coupled with historical records, ecological observations, and epizootiological reports, imply group divergence millennia to centuries ago. Although all mammals are in theory susceptible to infection, particular viruses seem compartmentalised within certain species. Spillover infections take place to non-conspecifics, but often result in dead-end termini, via processes not well explained. In Africa, Lagos bat and Duvenhage viruses have been associated with several taxa, including *Eidolon*, *Epomophorus*, *Miniopterus*, and *Nycteris* species. Two major types of European bat lyssaviruses have been described widely from the insectivorous genera *Eptesicus* and *Myotis*. Recently, four new lyssaviruses, Aravan, Khujand, Irkut, and West Caucasian bat virus have been isolated from Eurasian Microchiroptera. Australian bat lyssaviruses exist among several species of *Pteropus*, as well as in non-pteropids. Throughout parts of Asia, from the Indian sub-continent to the Philippines, other lyssaviruses await characterisation, thus far identified only via serological surveys. The New World is the only region where the 'type species' for the entire Genus, rabies virus, has been documented among bats, existing as distinct variants in hosts as diverse as *Lasiurus*, *Tadarida*, *Pipistrellus*, and *Desmodus*. Despite previous suggestions that bats serve as 'carriers', typically excreting virus in saliva while remaining clinically normal, preliminary laboratory pathogenesis experiments do not support such contentions. Rather, bats appear to respond to lyssavirus infection much like other mammals, regardless of variant. Serum samples collected from hundreds of wild bats demonstrate the presence of virus neutralising antibodies in healthy individuals, supportive of the concept of abortive infection, sans illness or viral excretion. Unlike the historical control by oral vaccination of particular carnivore populations in Europe and North America, similar techniques have not been routinely engaged for bats. Bats respond as expected to parenteral or oral vaccination with rabies biologicals licensed for other species, and routine immunisation may be considered of wild caught bats when maintained under captive conditions during quarantine, for research, breeding or related conservation purposes. Considering the relatively poor cross-reactivity of extant commercial rabies vaccines against heterologous lyssaviruses, the opportunity for international translocation, and the potential establishment of exotic pathogens among indigenous fauna (including putative host shifts to native carnivores), a variety of new management strategies are desirable for bats. Notwithstanding selective population reduction by the application of topical anti-coagulants to vampire bats, actual prophylaxis and disease abatement in any of the Chiroptera under field conditions will require the development of more novel strategies than currently employed to approach any modicum for future success.

78) AUSTRALIAN BAT LYSSAVIRUS: OBSERVATIONS OF NATURAL AND EXPERIMENTAL INFECTION IN BATS

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Australian bat lyssavirus (ABLV) is a rabies-like virus that occurs as two virus variants (1) pteropid-ABLV, which affects the four common species of flying fox (*Pteropus alecto*, *P. poliocephalus*, *P. scapulatus* and *P. conspicillatus* and (2) ybst-ABLV that occurs in the insectivorous Yellow-bellied sheathtailed bat (YBST bat, *Saccolaimus flaviventris*). Both variants were first detected in 1996 and each variant has subsequently been diagnosed as the cause of single cases of fatal human encephalitis.

Between June 1996 and March 2002, surveillance of 1143 bats for ABLV by fluorescent antibody tests on fresh brain impression smears indicated that while ABLV is rare in whole bat populations (95% confidence <1%), it is common amongst sick, injured or orphaned *P. alecto*, *P. poliocephalus*, *P. scapulatus* and YBST bats. Multivariate analysis of data relating to wild megachiroptera (n=893) indicated that the prevalence of ABLV was associated with three factors: species, age and health status (clinical characterisation). No regional, temporal or seasonal association was found by analyses of epidemiological or phylogenetic (viral nucleoprotein sequence) data. ABLV was most prevalent in YBST bats (5 of 7, 71%) and P. scapulatus (21 of 124, 17%), common in *P. alecto* and *P. poliocephalus* (37 of 474, 8% and 8 of 175, 5% respectively) and least common in *P. conspiculatus* (1 of 95, 1%). One of 17 flying foxes not identified to species and none of 8 other fruit bats were ABLV-positive. It was significantly more common in sexually mature adults (>3 years) rather than juvenile flying foxes. The prevalence of ABLV was significantly higher (21%) amongst bats submitted with clinical signs suggesting central nervous system (CNS) disease (e.g. paralysis, paresis, overt aggression, seizures, cranial nerve deficits) than those with non-CNS signs (2%) and absent amongst clinically normal bats. Statistical analysis indicated that certain combinations of the three risk factors of species, health status and age gave rise to very high predicted prevalences for ABLV, e.g. the predicated prevalence of ABLV amongst adult *P. alecto* and *P. scapulatus* showing signs of CNS disease (of which 72 were submitted) were 35% and 59% respectively.

The range of clinical signs associated with naturally occurring ABLV were similar to those of rabies and included a most common 'paretic' form (unable to fly, progressive weakness) and a less common 'furious' form characterised by overt aggression towards other animals, humans and objects, including flying from trees to 'attack' humans and dogs. The incubation periods of only four natural cases of ABLV are known. Two bats developed clinical signs in captivity 36 to 57 days or 30 days after exposure to ABLV, and the two human patients became unwell 4 to 6 weeks and 27 months after being bitten by bats. The clinical duration of naturally occurring ABLV in bats is rarely observed in full. While most bats died or were humanely killed within 36 hours of being found, ten bats survived at least 3 (n=5), 5, 6 (n=2), 8 or 9 days while ill, during which time wildlife carers were at prolonged and repeated risk of exposure to ABLV.

It is clear that ABLV-infected bats commonly come into contact with humans and other animals. Consequently there is an ongoing risk of ABLV transmission to humans, their pets and wildlife. It should be noted that while YBST bats, flying foxes and humans are the only species in which ABLV infection has been diagnosed, the numbers of bats of other species, other wildlife and domestic animals that have been tested is too small to indicate an absence of ABLV in other animals. The use of animal rabies vaccines in Australia is restricted, limiting the management options for domestic animals. In the absence of any available or proposed mode of vaccine delivery for wild bats, there is no prospect of applying the effective wildlife vaccine strategies that have reduced the prevalence of terrestrial rabies in Europe and America. Consequently Australian ABLV management protocols rely on avoiding contact with bats, testing bats that bite or scratch people and pre- and post-exposure rabies virus vaccination of humans exposed to bats.

Experimental infection of 10 wild-caught *P. poliocephalus* with inocula prepared from the submandibular and sublingual salivary glands of a naturally infected *P. alecto* containing 105.2 to 105.5 MICDED50 of pteropid-ABLV, resulted in clinical disease in 7 of 10 bats. Infection with ABLV was confirmed by FAT, immunoperoxidase staining of formalin fixed tissue, virus isolation in mice, and TaqMan PCR assay. The incubation periods were comparatively short (10 to 19 days), with clinical signs lasting 1 to 4 days prior to euthanasia or death. Five of the 7 cases were overtly aggressive towards humans, other bats, and objects. One died during a seizure, while otherwise apparently well, approximately 1 hour after an uneventful recovery from anaesthesia, and the last became ataxic and moved incessantly and purposelessly about the cage. None developed the 'classic' calm progressive paresis described in most naturally occurring cases. The reasons for this are unclear.

Of the three that remained well until the completion of the trial on days 80 and 82, two showed no evidence of seroconversion in either rabies virus- or ABLV-based rapid fluorescent antibody tests (RFFITs). One developed a strong, apparently protective, neutralising response between days 7 and 35 suggesting subclinical resolution of experimental ABLV infection. This response was most evident in pteropid- and ybst-ABLV-RFFITs rather than rabies-RFFITs and subsequent pteropid-ABLV-based testing of pre-inoculation serum (which had appeared seronegative in rabies-RFFITs) indicated a low but apparently biologically significant pre-existing (naturally acquired) ABLV titre. The apparent failure of cross-reactive rabies-RFFITs to detect low but functionally significant ABLV titres has implications for the interpretation of all serological studies for ABLV that use cross-reactive rabies virus rather than ABLV-based assays. All three 'survivors' were negative for pteropid-ABLV by FAT, immunoperoxidase staining of formalin fixed tissue, and virus isolation in mice.

In summary, ABLV has never been detected in a clinically well bat, is common in sick injured or orphaned bats (5-10%) and very common (20-30%) in bats showing signs of CNS disease. Other causes of CNS disease in wild flying foxes include spinal and head injuries, neuro-angiostrongylosis (involving *Angiostrongylus cantonensis*, a nematode) congenital hydrocephalus, bacterial meningoencephalitis and tick paralysis (*Ixodes holocyclus*). Mass spectrometry of formalin fixed livers showed no evidence of plumbism (lead poisoning) in bats with undiagnosed CNS signs or clinically well urban bats caught in Brisbane (n=50). Veterinarians and carers need to consider the possibility of ABLV infection for bats and potentially other animals in their care. In particular those with CNS signs should be submitted for ABLV testing.

79) THE EMERGENCE OF AUSTRALIAN BAT LYSSAVIRUS – A REVIEW OF CURRENT HYPOTHESES

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Australian bat lyssavirus (ABLV) was first described in May 1996 in a black flying fox (*Pteropus alecto*). Six months later, a fatal rabies-like disease in a Queensland bat carer was attributed to ABLV - the first known human case. A second fatal human case occurred in December 1998.

Epidemiological studies indicate that ABLV is widespread in Australian bats. Evidence of current infection (virus detection) and/or previous infection (antibody detection) has been found in all four mainland *Pteropus* species (sub-order *Megachiroptera*) and in five of the six families of *Microchiroptera* represented in Australia. Importantly, the finding of antibody-positive individuals in non-nomadic microchiropteran populations beyond the known range of flying fox species strongly suggests that ABLV is cycling independently in microchiroptera.

Molecular studies provide some insight into the history of ABLV in Australia. While limited sequence variation has been reported between isolates from flying foxes (*Pteropus* spp.) collected at different locations and at different times, the isolation and subsequent sequencing of ABLV in the microchiropteran *Saccolaimus flaviventris* has revealed substantial difference to the pteropid variant. Combined, the molecular and the epidemiological findings indicate that ABLV has been present in Australian bat populations long enough for strain differentiation to occur and for infection to become geographically and taxonomically widespread in both micro- and megachiroptera.

Two hypotheses on the origins and emergence of ABLV are discussed based on phylogenetic data. The first proposes that the pteropid virus lineage is ancient and stable, that the molecular differences identified in pteropid viruses evolved in pteropids, and that the molecular differences between viruses of pteropids and *S. flaviventris* are maintained by purifying selection. The alternative interpretation of the phylogenetic data is that the pteropid lineage has only recently become adapted to pteropids, and that the narrow genetic diversity is the result of a recent radiation following the adaptation to and colonisation of pteropid species. Importantly, this hypothesis implies that ABLV is dynamic and potentially able to adapt to establish cycles in other mammal species. Recent changes in pteropid ecology may have caused the observed changes in ABLV prevalence, allowing ABLV either to become apparent to passive surveillance systems (first hypothesis) or to establish in the new pteropid hosts (second hypothesis).

SESSION 7:
DISEASES OF UNGULATES

80) HEMORRHAGIC DISEASE IN WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*) IN POPULATONS DERIVED FROM NATIVE AND INTRODUCED ANIMALS

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White tailed deer (WTD; *Odocoileus virginianus*) populations in the United States represent a mixture of populations that originated either from remnant animals or through reintroduction of animals from sometimes distant sources. Hemorrhagic disease (HD) is caused by related orbiviruses in the epizootic hemorrhagic disease and bluetongue virus serogroups, and is recognised as the most important viral disease affecting this species. Based on over 20 years of surveillance in the USA, it is apparent that reported WTD mortality and morbidity associated with HD exhibit distinct spatial patterns both regionally and within individual states. The objective of this work was to compare the distribution of reported HD mortality and morbidity within two states in relation to historic data on the WTD source populations used for restoration. Kansas represents a state where current deer populations are derived from remnant native populations. Georgia represents a state where current deer populations are derived from a combination of remnant native stock and introduced deer from varied sources. Local variation in reported HD was more apparent within Georgia, and this may be attributable to the source of deer that were used for WTD restoration.

81) BOVINE TUBERCULOSIS IN MICHIGAN WILDLIFE

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In 1994, a white-tail deer with bovine tuberculosis (TB) was shot by a hunter in southwestern Alpena County, Michigan. Since the fall of 1995 through 2004, wildlife surveys have been conducted in the surrounding area by Michigan Department of Natural Resources (MDNR) and over 138,290 white-tail deer, 1,390 elk, 21 moose, and 1,514 carnivores have been examined for bovine TB in the laboratory. During these surveys, 509 deer, 4 elk, 18 coyotes, 8 raccoons, 7 black bear, 3 red fox, 2 opossum and 4 bobcats have been confirmed to be infected with *Mycobacterium bovis* by the National Veterinary Services Laboratories (NVSL), Ames, IA, or the Michigan Department of Community Health *(*MDCH*),* Lansing MI or Michigan State University (MSU). All of the infected deer, elk, coyotes, raccoons, black bear, opossums, red foxes and bobcats have come from 13 northeastern Lower Peninsula counties in Michigan (Alpena, Montmorency, Alcona, Oscoda, Otesgo, Presque Isle, Osceola, Mecosta, Antrim, Crawford, Roscommon, Emmet, and Iosco).

One human case of *M. bovis* has been traced to a TB-infected hunter killed deer in Alcona County in October of 2004.While field dressing a deer with physical signs of bovine TB, the hunter cut himself on the hand. Upon seeking medical attention, physicians ran tests for bovine TB, which were confirmed by the MDCH. Another case of bovine TB occurred in a U.S. born resident of Alpena County who died of unrelated causes in 2002. This person had a family that hunted and may have consumed unpasteurised milk as a child. The other 8 TB positive individuals were either elderly and grew up on farms in Michigan or were foreign born.

Since testing began in 1996, the MDA, the USDA and private veterinary practitioners have tested nearly all of the state's 1 million livestock animals on 17,000 farms. As of January 28, 2005, bovine TB has been confirmed in 208 cows from 33 herds in 7 counties. Thus far there have been 7 dairy herds and 26 beef herds diagnosed with bovine TB. Positive herds have been found in Alpena County (13), Alcona County (11), Presque Isle County (28), Oscoda County (2), Emmet County (2), Antrim County (1) and in Montmorency County (2). Restriction fragment length polymorphism analysis (RFLP) was performed by the MDCH, the NVSL and the National Animal Disease Center (NADC). They have concluded that the index deer and subsequent deer, carnivore, and bovine isolates have identical IS6110 and TBN12 patterns*,* indicating that the same strain of *M. bovis* is involved in the outbreak in cattle and wildlife.

The most likely source of the infection in the carnivore and omnivore population was through the consumption of tuberculous white-tailed deer. The white-tailed deer in Michigan are recognised as a reservoir host of bovine TB. Once the disease is eliminated from the deer, the disease is expected to die out in the carnivorous and omnivorous species. As long as bovine TB exists in the wild, free-ranging deer population, there will be some risk to local wildlife species that feed on bovine TB infected deer carcasses or gut piles; therefore continued surveillance will be necessary.

Several strategies are being used to eradicate bovine TB from Northeastern Michigan. Strategy 1 involves keeping deer from concentrating by eliminating supplemental feeding and baiting. Strategy 2 involves reducing deer numbers through hunting to a level supported by the natural vegetation. MDA and USDA have implemented an electronic identification and movement permit and tracking system for all cattle in the Modified Accredited area of northeastern Michigan. In addition, annual herd surveillance testing and individual animal movement testing requirements have been instituted for all cattle in this area as of June 1, 2004.

USDA-Wildlife Services has also contributed to the eradication of bovine TB in Michigan by (1) assisting producers in removing deer under disease control permits issued by MDNR, (2) providing fencing to producers to prevent deer from feeding at storage areas as a means to prevent transmission, (3) making observations of wildlife patterns on TB positive farms, (4) assisting with trapping and removing deer that have tested positive and (5) providing assistance to researchers involved in studies dealing with psychological, physical and biological barriers to minimise interactions between white-tailed deer and livestock to reduce interspecies transmission of TB.

82) EXPERIMENTAL INFECTION OF ROCKY MOUNTAIN BIGHORN SHEEP (*OVIS CANADENSIS CANADENSIS*) WITH *BRUCELLA OVIS*

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The Rocky Mountain bighorn sheep (BHS*)* is a species that has been described as "…possibly the most exquisitely sensitive North American wild ungulate to common livestock diseases and parasites." (Jessup, 1985) Pneumonia (both viral and bacterial), bluetongue, scabies, contagious ecthyma, and lungworm are just a few of the diseases that are known to affect BHS. Brucellosis is a disease of concern. However, little is known about brucellosis in BHS and even less is known about *Brucella ovis* in BHS. During herd health assessments and relocations of BHS, it is not uncommon for *B. ovis* antibodies to be detected. However, culture of *B. ovis* from blood or tissue samples has not been reported. This raises the question as to whether positive serology results are indicative of *B. ovis* infection or perhaps a cross reaction with another organism. The purpose of this study was to determine if *B. ovis* causes clinical disease in BHS, if it could be detected by standard serologic and culture techniques, and to characterise any pathogenic changes associated with the infection. Male and female (both pregnant and non-pregnant) BHS and domestic sheep were inoculated intraconjunctivally with 10^8 colony forming units of *B. ovis* recently isolated from a domestic ram. The sheep were monitored and specimens periodically collected for serologic and microbiologic examination. Sheep were euthanased and tissues were collected for culture and histopathology. Preliminary results show that BHS challenged with *B. ovis* became positive on serology and culture. Abortions and still births occurred in inoculated BHS ewes, while epididymitis and testicular lesions occurred in inoculated BHS and domestic rams. Results of this study show that bighorn sheep are susceptible to *B. ovis* infection.

83) COULD WOLVES CONTROL CHRONIC WASTING DISEASE?

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The effect of chronic wasting disease (CWD) on deer and elk populations can be significant, both from the infection and from collateral impacts of management actions. Efforts to control CWD are limited to actions that reduce the number of infected animals in the population or otherwise alter the transmission rate. Current approaches to managing CWD are intensive, costly (often both economically and environmentally), and will require a long-term commitment to reduce prevalence or eliminate the disease in free-ranging populations. We suggest that restoring functional ecosystems, including top predators, is a promising management alternative that should be investigated. Wolves could influence CWD prevalence through several mechanisms including: increasing mortality rates, particularly selective removal of CWD positive deer and elk, redistributing deer and elk from areas of high concentration, and removing infected carcasses from the environment. Increasing mortality rates in diseased populations can retard disease transmission and reduce prevalence. Increasing mortality slows transmission by reducing the average lifetime of infected individuals. Reduced lifespan, in turn, can compress the time interval when animals are infectious, thereby reducing the number of infections produced per infected individual. The effect of reduced intervals of infectivity is amplified by reductions in population density that occur as mortality increases, reductions that cause declines in the number of contacts between infected and susceptible individuals. Further, if predators prey selectively on diseased individuals, it is reasonable to expect that they would reduce disease prevalence much more rapidly than would occur if mortality were non-selective. We used a simple mathematical model to forecast that predation by wolves could have potent effects on CWD prevalence. Moreover, in addition to impacts through mortalities, the presence of wolves would likely alter prey distribution and result in fracturing of large groups of sedentary prey species. Moving large concentrations of deer and elk off highly contaminated environments would reduce transmission rates further. Similarly, consumption and dispersal of carcasses of CWD positive deer and elk would dilute the concentration of PrP^{res} in the environment and would likely reduce transmission rates. Because we can currently only speculate and make informed predictions regarding the impact of wolves on CWD prevalence, we endorse well-designed field research to investigate this unique approach to CWD management.

84) PESTIVIRUS OF CHAMOIS (*RUPICAPRA* SPP.) IN EUROPE: AN EMERGING INFECTIOUS DISEASE?

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In 2001 and 2002, an outbreak of a new disease was observed in chamois of the Central Pyrenees, in Spain, Andorra and France. The population decreased between 40 and 45 % in some areas. Affected animals were cachectic and showed alopecia and neurological symptoms. By antigen ELISA test and RT-PCR, a pestivirus was detected. Virus isolation and sequence analysis revealed a new virus strain that forms a separate branch within the Border Disease virus cluster. Serologic analyses were performed on sera of healthy chamois from the affected area by a blocking ELISA test, and resulted in 79/99 positive (79.8 %). Serological studies in other areas from the Eastern Pyrenees showed different results. In the Orlu Reserve (France), a prevalence of more that 85% has been described since 1996, while a decrease of the population has been observed during this period. No apparently disease has been identified in this area until 2004, when one chamois was found with the pestivirus-associated disease. In the National Hunting Reserves of Cadí and Freser-Setcases (Spain), which have not been affected by the disease, a serological study revealed negative results.

At the same time, in the French Alps, serological studies showed a high prevalence of antibodies against a pestivirus in some areas, while negative results have been obtained in others. Virological studies have identified a pestivirus in one female chamois with keratitis and neurological symptoms in the Vanoise National Park and in apparently healthy seronegative chamois from the Hautes Alpes region.

To prove whether this new pestivirus strain isolated in the Pyrenees plays an aetiological role within this new disease, an experimental infection is needed and will be performed at the Veterinary Faculty of the Autonomous University of Barcelona.

85) CAPTURE AND ANAESTHESIA PROTOCOL OF ARABIAN ORYX AND ITS EFFECT ON BLOOD PARAMETERS IN CLIMATE CONDITION OF MIDDLE EAST

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Experience with 90 cases of a chemical immobilisation of Arabian oryx (*Oryx leucoryx*) during hot climate condition and during mild climate condition is presented by authors. Animals were free-ranging, forming different size family groups (5-25 individuals). Large area of sand dunes and extremely hot daily temperatures made animal capture very difficult in many aspects. In 18 cases of chemical immobilisation combination of etorphine (Immobilon, 1.8-2.2 ml/animal) and azaperone (Stresnil, 30 mg/animal) was used. This combination of anaesthetic was reversed by equal dose of diprenorphine (Revivon). The combination resulted in a good anaesthesia and analgesia in all darted animals. Darted animals shortly after darting usually tended to run until they become exhausted. Capture myopathy, hyperthermia and aspiration pneumonia was observed in 30% of darted animals. Through authors experience of animal capture in desert conditions, when summer temperature reaches 48°C, the preferred drug of choice is a mixture of ketamine (Ketavet, 0.8-1.5 mg/kg)-xylazine (Anased, 0.5-1.2 mg/kg), reversed with atipamezole (Antisedan, 0.5-0.9 mg/kg). Atipamezole was administered minimum 40 min. postdarting, half dose i.v., half dose i.m. Good results with atipamezole reversing xylazine in Arabian oryx was reported by other author as well (Ancrenaz, 1994). Ketamine-medetomidine combination was successfully used in gemsbok (*Oryx gazella*) (Grobler et al., 2001).

In 20 cases of anaesthesia and translocation with ketamine-xylazine in summer month, blood samples were collected to run acid-base, haematology and biochemistry and were compared with results obtained during anaesthesia and translocation in autumn and with blood results after inducing anaesthesia with medetomidine-ketamine-butorphanol mixture (Domitor-Ketavet-Torbugesic, 1.5-2 ml + 0.8-1 ml + 0.2 ml/animal i.m.). Heat stress can significantly increase body temperature, induce acid-base disturbances and serum electrolyte and enzyme responses. Also different anaesthetics and their combinations can alter blood gases and acid-base composition (Simeonova, 2004). After induction of anaesthesia by ketamine-xylazine in summer, acid-base status showed respiratory acidosis (average pH=7.205) caused by hypoventilation due to bradypnoe. After translocation blood samples revealed changes in some parameters. pH, blood bicarbonate ion (HCO_3^-), base excess (BE), partial oxygen pressure (pO_2) and oxygen saturation (O_2 Sat) increased. Blood carbon dioxide (pCO_2) decreased. These changes suggest development of respiratory alkalosis as a result of compensation of acidosis and effect of heat and capture stress (Odom et al., 1986). Haematology parameters showed decrease of haematocrit, white blood cell count and red blood cell count. These decreases were the effects of anaesthetics (Van der Linden et al., 2004). CK after translocation was elevated due to heat stress, reflecting heat stress-induced myopathy (Sandercock et al., 2001). Total protein was decreased and blood glucose concentration was increased, caused by release of corticoids due to capture stress. Concentrations of Na^+, Ca^{2+} decreased and K^+ increased due to stress and acidosis (Odom et al., 1986). Results obtained in November revealed similar changes. Shortly after inducing anaesthesia, acid-base status showed respiratory acidosis, which was later compensated towards higher values of pH, HCO_3^-, BE, pO_2, and O_2Sat.

These results suggest that the main changes in blood parameters are induced by anaesthesia and different anaesthetics. In all 7 cases of oryx anaesthetised by using medetomidine-ketamine-butorphanol mixture we did not see signs of acidosis (average pH=7.35). All three drugs combinations etorphine-azaperon, ketamine-xylazine and medetomidine-ketamine-butorphanol can be used for chemical capture and minor surgical procedures in Arabian oryx. Using etorphine in the extremely hot climate conditions and in animals in a free-ranging environment brings a much higher risk of occurrence of the negative effects and fatalities. Ketamine-xylazine is a relatively safe and cheap alternative to the ultrapotent narcotics with slight effect on blood parameters. Laboratory results of blood samples collected after inducing anaesthesia with medetomidine-ketamine-butorphanol are promising and showing possible safe anaesthesia protocol even during extremely hot climate conditions with no, or very little negative effect on acid-base status and other blood parameters.

Ancranez, M 1994. Use of atipamezole to reverse xylazine tranquilization in captive Arabian oryx (*Oryx leucoryx*). J. Wildl. Dis. 30, 592-595.

Grobler, D., Bush, M., Jessup, D., Lance, W. (2001). Anaesthesia of gemsbok (*Oryx gazella*) with a combination of A3080, medetomidine and ketamine. J. S. Afr. Vet. Assoc. 72, 81-83.

Odom, T.W., Harrison, P.C., Bottje, W.G. 1986. Effect of thermal-induced respiratory alkalosis on blood ionized calcium level in the domestic hen. Poultry Science 65(3):570-573.

Sandercock, D. A., Hunter, R.R., Nute, G.R., Mitchell, M.A. and Hocking, P.M. 2001. Acute heat stress-induced alterations in blood acid-base status and skeletal membrane integrity in broiler chickens at towages: implications for meat quality. Poultry Science 80(4):418-425.

Simeonova, G. 2004.Acid-base status and blood gas analysis in three different anaesthesia schemes in dogs. Turk J Vet Anim Sci 28: 769-774.

Van der Linden, P., De Hert, S., Mathieu, N., Degroote, F., Schmatrz, D., Zhan, H., Vincent, J.L. 2004. Tolerance to acute isovolemic hemodilution. Effect of anaesthetic depth. Anesthesiology 100(4):1034-1035.

SESSION 8:
MISCELLANEOUS TOOLS AND TECHNIQUES

86) ESTIMATING DISEASE INCIDENCE FROM AGE-SPECIFIC PREVALENCE DATA IN THE PRESENCE OF SIGNIFICANT DISEASE-INDUCED MORTALITY AND SPATIAL AGGREGATION

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Traditional methods for estimating transmission rates of human diseases from age-prevalence data assume that disease-induced mortality is negligible and/or disease is not spatially clustered. Breaching these assumptions causes the observed prevalence of disease in a random sample of individuals to start decreasing in older individuals, rendering standard models to analysing age-prevalence data unsuitable. This phenomenon is observed in a range of wildlife disease systems. In this paper we present a simple method for estimating disease transmission parameters within spatial clusters of disease using age-specific prevalence data collected from populations where disease is spatially aggregated and disease-induced mortality is high. The approach is applicable when confronted only with cross-sectional survey age-prevalence data and little other supporting information on the spatial distribution of disease. Monte Carlo Markov Chain empirical Bayesian methods are used to incorporate data on disease-induced mortality rates and levels of spatial aggregation where these data are available. The method is illustrated for *Mycobacterium bovis* infection in brushtail possums (*Trichosurus vulpecula*), and chronic wasting disease in mule deer (*Odocoileus hemionus*). Our results demonstrate how wildlife diseases that are seemingly rare have higher rates of disease transmission than are initially apparent based on their low prevalence.

87) A NOVEL MOLECULAR-ECOLOGY APPROACH TO ASCERTAINING EMIGRATION / IMMIGRATION AND POTENTIAL DISEASE SPREAD IN FERAL PIGS

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Our study used two consecutive years of aerial culling then molecular ecology techniques (micro-satellite analysis) to obtain parentage data from a widely dispersed and low density feral pig population during drought conditions in the semi-arid rangelands of Australia. This data was analysed geo-spatially to provide estimates on the actual minimum movements of feral pigs. The aim of this exercise was to obtain data that could be used to improve models that investigate rates of disease spread. The derived data revealed that some individuals will move much greater distances than previously recorded. The maximum recorded movement was 143km between a boar and pregnant sow in less than 1 year, with the mean boar to successfully mated sow distance being 43km. Thus, home ranges in this situation could be assumed to be much larger than previously calculated. Movement models were developed from this data and were based upon three assumptions of home range affinity; fixed home range, moving home range and no home range. Mean daily movements generated were 3km linear distance away from a previous mating for boars. Models revealed that currently planned disease eradication zones for Australia may be inadequate if an exotic disease outbreak remains undetected for only a short period of time (1 week). Using previously generated deterministic models for foot–and-mouth disease spread, and this data to alter home range size, the threshold population density below which disease will not transmit declines markedly.

88) USE OF INFRARED THERMOGRAPHY TO DETECT RABIES INFECTION IN RACCOONS (*PROCYON LOTOR)*

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We evaluated infrared thermography as a technique to determine if raccoons (*Procyon lotor*) experimentally infected with a virulent raccoon strain of rabies virus could be differentiated from non-infected and those infected with canine distemper virus. Six raccoons were used in the experiment using rabies virus. Each animal had a body temperature sensor (Advanced Telemetry Systems, Inc. (ATS), Isanti, Minnesota USA, Model 1310, weight 14 grams) surgically implanted subcutaneously in the dorsum of the neck. Each implant transmitted temperature data to a remote receiver (ATS, Isanti, Minnesota USA, Model R4500S Dsp Receiver w/Datalogger). This temperature was used as an index to body temperature and compared with temperature data derived from thermal imagery. Animals were housed in BSL3 facilities eight days prior to inoculation and thereafter until termination of the experiment. Animals were euthanased upon initial observance of severe clinical signs of rabies. The same six raccoons were used as controls (non-infected) during the first eight days prior to infection. At the end of eight days, the six raccoons were infected with a virulent raccoon strain of rabies virus via injection of the masseter muscle with 2 X 10^5 $TCID_{50}$, $n = 4$, and 1 X 10^6 $TCID_{50}$, $n = 2$, of virus at day 0. Twice daily, during the pre-and post-inoculation periods, infrared images of each raccoon were recorded using Forward Looking Infrared (FLIR Systems, Boston, North Bellerica, Massachusetts USA, Model Thermacam E65). Visual infrared images of the face, especially the nose and rostrum, clearly showed differences between infected animals showing clinical signs of rabies beginning at ≥ day 19 post-inoculation, compared with themselves while non-infected, and after infection but not yet presenting clinical signs. Data from the thermal images indicated a significant increase in surface temperature of the nose and rostrum (13%, 10^0C) but not in other areas of the body (e.g., eye, ear canal, average body surface) between raccoons showing clinical signs of rabies and those not presenting signs. We also compared the use of this technique using several raccoons naturally infected with canine distemper virus and found that the infrared images and surface temperatures of the nose and rostrum were distinct from those infected with rabies virus. This experiment provides data which indicates that infrared thermography can be used in an experimental setting to detect raccoons showing mild to severe clinical signs of rabies. This technique may represent a useful tool for detecting early/minimal to severe clinical signs of rabies, which may be overlooked by inexperienced observers using casual observation, or in situations where observance of clinical signs may be difficult.

89) USE OF SATELLITE TELEMETRY TO STUDY THE MOVEMENT OF THE MALAYAN FLYING FOX (*PTEROPUS VAMPYRUS*): IMPLICATIONS FOR CONSERVATION AND PUBLIC HEALTH

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Fruit bats of the genus *Pteropus* perform critical ecological functions and are also natural reservoirs for henipaviruses, zoonotic paramyxoviruses that cause significant disease in humans. This species is a natural reservoir for Nipah virus in Malaysia, and little is known about its distribution or long-range movements. The movements of three mature male Malayan flying foxes (*Pteropus vampyrus*) were monitored using satellite telemetry. Bat A traveled 780 km during the 3-month study period (December 2003 - March 2004), flying from Peninsular Malaysia to Sumatra, Indonesia and returning to Peninsular Malaysia through the Singapore Strait. The return journey from the Singapore Strait to Peninsular Malaysia, 270 km, was traversed within 10 days. Bats B and C were released near Lenggong, a village within 50km of the index farm where Nipah virus emerged) on the 9th and 12th of July 2004. Bat B left Lenggong after the 10th of August and flew 160 km north into Thailand. Bat C left Lenggong after the 12th of July and flew east 200 km. Between the 5th and the 24th of October both bats were 'reunited' and monitored roosting and foraging in the same area, west of Kota Baharu, 150 km north-east of Lenggong. During the 3 - 4 month study period, Bats A, B and C occupied 7, 4 and 9 roosting colonies and were monitored foraging up to 56 km from the roosting colony. This study demonstrates the mobility of this species and suggests that Nipah virus is endemic to a region broader than just Peninsular Malaysia.

90) DEVELOPMENT OF NEW IMMUNOLOGICAL DETECTION METHODS FOR USE IN THE DUCK

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Background

In order to improve efficacy of antiviral prevention and treatment regimes knowledge of the animal's immune response is critical. The immune response consists of the non-specific innate immune response and includes interferon (IFN) production, and activation of natural killer cells and macrophages. This is followed by the specific immune response consisting of the humoral and the cell mediated immune responses. The humoral response involves the production of antibody by B cells, that is generally neutralising and prevents viral attachment and infection of naïve cells. Once a virus has entered a cell the cell mediated immune response (CMI) becomes responsible for viral elimination. The T lymphocytes recognise processed viral antigens in association with MHC. The T helper cells (CD4+) produce cytokines which help regulate B cells and CD8+ T cytotoxic cells. The CD8+ cells interact with the infected cell and either cure (by cytokine production) or kill it. The investigation of the duck immune response has been hampered by a lack of duck-specific reagents.

Aims

1. Develop immuno-histochemical (IHC) assay for staining duck T lymphocytes
2. Develop Real time PCR for the detection duck IFN

Methods

1. Two different IHC methods were tried 1) the Avidin-Biotin (ABC) method, and, 2) the polymer detection system (DAKO EnVision™ + System, HRP). Using ABC, two different enzyme detection systems were used: a) Alkaline Phosphatase (VECTASTAIN ABC-AP Kit, Standard), and, b) Peroxidase (DAKO LSAB®+System, HRP) +/- a biotin-blocking step. Two anti-human CD3 antibodies were tested.
2. RNA extraction was performed on normal duck livers using the MasterPure™ Complete DNA and RNA Purification Kit (Epicentre®, Cat. No. MC85200; Madison, Wisconsin). The purified nucleic acid (6µl) was subsequently used in reverse-transcriptase PCR (RT-PCR) 4µl RT mix ($MgCl_2$ 3mM, dNTPs 500µM, DTT 0.1mM, RNasin 10U, SS III 20U and DEPC H_2O). Several combinations of primers for the Duck IFN-γ gene AF 100929 (Huang *et al.*, 1998) were designed for PCR using the PCRprimer primer program in ANGIS (www.angis.org.au) and experimentally tested.

Results

1. Th EnVision labelled polymers were more sensitive in detecting T cells than both the ABC methods. In addition little diffuse or non-specific staining was present.
2. Both Monoclonal Mouse, Anti-Human CD3, Clone F7.2.38, Code M 7254(diluted 1:100) and Polyclonal Rabbit, Anti-Human CD3, Code A 0452 (diluted 1:1000) were suitable primary antibodies (DakoCytomation, Sydney).
3. The optimal PCR mix was found to be ($MgCl_2$ 3mM, dNTPs 500µM, 200nM of each primer, 1x running buffer, 0.5U Tth). The most sensitive primers were:
   - Forward (PrF): 5'ACTGAACTGACGTGATACCC 3' (length: 20 bp)
   - Reverse (PrR): 5'CTCCTGAGTACAGAAACTTTCC 3' (length: 22 bp)

Conclusion

We have optimised two methods for evaluating the duck's immune response to infection.

91) EVALUATION OF CHEMICAL IMMOBILISATION AGENTS FOR ANAESTHETISING AMERICAN BEAVER (*CASTOR CANADENSIS)*

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Although, the literature contains some reports on immobilising agents used on beaver, critical evaluations of available compounds have not been conducted. We evaluated the efficacy of 12 anaesthetic protocols and 6 reversal agent protocols (i.e., 18 treatments) in American beaver using various doses of ketamine/xylazine, ketamine/medetomidine, ketamine/ acepromazine, tiletamine/zolazapam, yohimbine, and atipamazole. Eighteen beaver were randomly assigned to treatments. Each beaver received six anaesthetic protocols providing (n= 6 animals/treatment). Individuals were administered an intramuscular injection of an anaesthetic protocol at two week intervals, providing time between applications to metabolise residual compound and minimise the potential of refractory effects (e.g., sensitisation, desensitisation). Time from injection to sedation and anaesthesia were recorded. After becoming anaesthetised, heart rate, respiratory rate, sPO_2, respired CO_2, and blood pressure were monitored throughout anaesthesia. Beavers were allowed to recover from anaesthesia on their own or through the aid of a reversal agent. Time of post-anaesthetic sedation and full recovery were recorded. Results from this research will enhance wildlife professionals' ability to select the most appropriate anaesthetic protocol for immobilising beaver.

SESSION 9:
ENDANGERED AND CAPTIVE ANIMALS

92) DISEASE IN THE ENDANGERED MAURITIAN PINK PIGEON (*COLUMBA MAYERI*)

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The Pink Pigeon *Columba mayeri*, endemic to Mauritius, has been the subject of an intense species recovery programme, which resulted in a total population increase from less than 20 to over 300 free-living birds in five distinct subpopulations over 15 years. Parasitic diseases, in particular trichomoniasis and leucocytozoonosis, are thought to have contributed to the species' original decline and have been highlighted as a potential threat to its continued recovery. To investigate seasonal and site prevalence of *Leucocytozoon marchouxi* and *Trichomonas gallinae*, we collected bi-monthly epidemiological data from all five Pink Pigeon sub-populations on health status and presence/ absence of two pathogens, T. gallinae and L. marchouxi. 90% of the total population was sampled in each 2 month period. Several introduced species of dove were also screened for both parasites to determine potential reservoir species for these parasites. Where accurate squab mortality could be quantified, trichomoniasis was responsible for 65-76% of squab deaths at one lowland site in 2004. Both L. *marchouxi* and *T. gallinae* varied in prevalence between sites, and prevalence of T. gallinae differed across seasons. Prevalence of both parasites also varied with age. Overall prevalence of infection with T. gallinae across sites and seasons was approximately 50%. Results of this study will be used to formulate guidelines for controlling disease in Pink Pigeons in the long-term and thus improve the conservation management of the species in the wild. The research also has implications for other endemic island Columbids in which pathogens have been introduced with exotic dove species.

93) HEMATOLOGIC AND SERUM BIOCHEMISTRY VALUES FOR FREE-RANGING NORTHERN HAIRY-NOSED WOMBATS (*LASIORHINUS KREFFTII*)

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The northern hairy-nosed wombat (*Lasiorhinus krefftii*) is one of the world's rarest mammals, with a population limited to just 90 individuals. The entire population lives free-ranging in the tropical woodland savannah of Epping Forest National Park, central Queensland. There are no northern hairy-nosed wombats in captivity. Threats to the northern hairy-nosed wombat include predation by dingoes, wildfire, drought, loss of genetic diversity, demographic stochasticity, disease and loss of habitat. Northern hairy-nosed wombats are nocturnal, shy and spend a majority of time underground in complex burrow systems. Due to the geographical isolation of their habitat and the small size of the population, there have been limited opportunities to collect baseline health data, or investigate disease in this species.

Between May and September 1999, the Queensland Parks and Wildlife Service conducted a survey of northern hairy-nosed wombats within Epping Forest National Park. All identified burrow systems were trapped for up to 10 nights. Forty one adult and immature wombats were trapped and anaesthetised using injectable Zoletil and Isofluorane in oxygen. Individuals were closely examined and genetic, morphometric and baseline health data was collected. Blood was collected from thirty five animals into EDTA, fluoride oxalate and serum separator tubes. Serum was separated within 6 hours of collection. Serum chemistry values and complete blood counts were determined at a commercial veterinary laboratory within 96 hours of collection, using standard techniques.

The results were used to establish base line hematologic and serum chemistry parameters for this critically endangered species. Reference values from the present study were compared to published ranges for southern hairy-nosed and common wombats. Differences between immature and adult animals are recorded and comments are made on individuals whose parameters fall outside the suggested reference range.

94) COMPARISON OF ANTI-PHOSPHOLIPID ANTIBODIES BETWEEN WILD AND CAPTIVE BLACK RHINOCEROS (*DICEROS BICORNIS*): IMPLICATIONS FOR HEALTH AND REPATRIATION

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The antiphospholipid syndrome (APS) is defined as the occurrence of venous and arterial thrombosis, recurrent foetal losses, in the presence of the phospholipid antibodies (aPhL) This is a broad definition in a syndrome that can affect virtually any body system. Deep venous thromboses (DVT) and pulmonary embolism (PE) are among the most common clinical presentations of APS. Major-vessel occlusion has also been described in virtually every vessel, including the aorta, branches of the aorta, inferior vena cava, hepatic vein, portal vein, intra-abdominal and intracranial vessels, and the peripheral vasculature of the extremities. The aPhL antibody is associated with many cutaneous conditions, including livedo reticularis, superficial thrombophlebitis, cutaneous necrosis, digital ischemia, gangrene, stasis ulcers of the ankles, epidermal atrophy, splinter haemorrhages of the nailbeds, non-necrotising purpura, and blue-toe syndrome. Recurrent foetal loss is another major component of APS. Cardiac valvular disease is also common in patients with APS. The aPhL proteins result in anti-coagulant activity but actually cause a hypercoaguable state in vivo. The pathogenesis of APS is quite simply thrombosis regardless of the organ system involved. Black rhinos in captivity have been plagued by a host of clinical entities. These include superficial necrolytic dermatitis (SND), hemosiderosis, haemolytic non-haemolytic anaemia and recently the idiopathic hemorrhagic vasculopathy syndrome (IHVS) has been described in a group of black rhinos. Recent thoughts into IVHS suggest that this may indeed be manifestation of a microcoagulation state (D. Paglia pers.comm.). Other conditions affecting black rhinos include encephalomalacia and necrotic laminar disease. Secondary infectious conditions ranging from salmonella, aspergillus pneumonia, and leptospirosis have all been documented. Comparisons between APS and black rhino syndromes may not be obvious at first but there may be some parallels. Again the underlying pathogenesis for all the conditions may be thromboembolic events. Treatment of APS consists of anti-coagulation therapy. One of the most common forms, especially in women with recurrent foetal loss, is low dose aspirin and even warfarin. Warfarin has been utilised in one black rhino with resolution of clinical lameness, normalised fibrinogens, lowering of APS antibodies, and lowered serum ferritin levels. Appetite returned to normal. Nasal haemorrhage from an aspergillosis plaque was uncontrollable one month into sustained therapy before the rhinoceros was euthanased. In an effort to follow this lead, a black rhino specific IgG-aPL ELISA has been developed and validated under the direction of Dr. Sylvia Pierangeli at the Moorehouse School of Medicine in Atlanta. A standard human assay (APhL® ELISA Kit, Louisville APL Diagnostics, Inc., 3988 Flowers Rd. Ste. 620, Doraville, GA 30360, USA) was modified by substituting purified polyclonal black rhino Ig-G for the human Ig-G conjugate. To date 17/28 captive animals have tested positive. All 17 animals have some of the clinical signs associated with medical conditions in black rhinos. Of the 10 negative animals, 8 did not have any clinical signs. Several animals had increased titres with length in captivity. Twenty-one wild black rhinos tested at the Veterinary Science Services facility in the Kruger National Park all had negative titres.

Antiphospholipid antibodies are also elevated in generalised inflammatory conditions. Comparing the two populations of black rhinoceros, it is apparent that there is some inflammatory process that triggers an exaggerated response to APS antibodies in captive

black rhinoceros. The wild rhinoceros all exhibited some clinical manifestations of inflammation (tick loads, wounds, keratitis) but still had negative APS titres. An obvious difference between the two populations is the diet. Future work at BGT will focus on evaluating diet hypersensitivity and the physical form of the diet as the inciting causes. Planned evaluations include food allergy profiles and gastric biopsies during feeding trials with a browser diet consisting of a low starch and high physically effective fibre. Efforts to repatriate indigenous animals to home ranges should not only evaluate current health status and asses risk of infectious disease but also evaluate underlying or predetermining risk that may lead to secondary infectious diseases. Improved nutrition in captivity may lead to healthier animals with lowered risk of introducing infectious diseases to native populations.

95) VETERINARY DIAGNOSTIC EVALUATIONS OF GIANT PANDAS WITH CLINICAL AND SUBCLINICAL DISEASE AT THE CHENGDU RESEARCH BASE FOR GIANT PANDA BREEDING, CHINA

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The biomedical survey of captive giant pandas in China conducted in 1998-2000 under the direction of the IUCN Conservation Breeding Specialist Group revealed the presence numerous health issues in China's captive giant panda population that warrant further investigation. In June 2004 a team of veterinarians from the United States and the Chengdu Research Base of Giant Panda Breeding (Sichuan, China) performed extensive veterinary medical diagnostic examinations on 13 giant pandas at the latter institution. The selected individuals were matched for age (cub (<1yr), subadult (2-4 yr), adult (5-17 yr), old (17+ yr), sex and clinically normal and abnormal health status. The examinations were performed under general (inhalation) anesthesia and included gastroduodenoscopy, colonoscopy, abdominal ultrasonography, serum toxicology, fecal culture and parasitology, complete blood count and serum chemistry, urinalysis and histopathology of gastrointestinal and liver biopsies.

Results indicate the following points salient to the care of giant pandas at the Chengdu Panda Research Base: (1) The finding of an eosinophilic microabscess in the liver of one subadult giant panda and possible eosinophilic infiltrates in the duodenum of each animal from whom a duodenal biopsy was taken suggests that disease associated with parasites requires further investigation and an improved parasite control program; (2) The inability to detect *Helicobacter* sp. by light microscopy and by PCR in gastric biopsies suggests that clinical signs of gastroenteric disease in pandas at the Chengdu Panda Research Base is probably not associated with *Helicobacter* sp.; (3) Frequent findings of dental abnormalities that include broken teeth, exposed root canals, enamel hypoplasia or dysplasia and tooth rotations indicate an urgent need to evaluate the diet of giant pandas and to initiate training of Chinese veterinarians in dental care; (4) Elevated blood fluoride levels (3-4 times higher than normal values in other mammals) suggest that fluoride exposure may be a factor in the observed dental (particularly enamel) abnormalities and requires further investigation; (5) Fecal cultures for *Campylobacter* and *Salmonella* were negative on each panda, suggesting that an alternative explanation for the recurrent and/or chronic diarrhea in the giant pandas at the Chengdu Panda Research Base must be explored; (6) Intestinal and/or hepatic hemosiderosis was found in 6 of the 13 pandas and warrants investigation; (7) The finding of neutrophilia without left shift in 5 of the 13 pandas, together with the historical and physical evidence for chronic infectious and non-infectious disease in the examined individuals, suggests the need for a comprehensive preventive health program. Additional abnormal findings in specific cases include abnormal ultrasonographic renal shapes and cystic lesions in a 12 year old breeding male, a gastric ulcer in a 6 year old breeding male, an eosinophilic hepatic abscess in a 3.5 year old female and lymphoplasmacytic enteritis in a 19 year old breeding female.

Findings from these diagnostic exams have revealed the need for comprehensive preventive health programs and for advanced veterinary medical care and research for giant pandas in China.

96) AN OUTBREAK OF *BORDETELLA BRONCHISEPTICA* INFECTION IN A CAPTIVE COLONY OF TAMMAR WALLABIES (*MACROPUS EUGENII*)

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Bordetella bronchiseptica is an emerging infectious disease of marsupials. It has previously been implicated for mortality events in koalas (*Phascolarctos cinereus*) and bridled nail-tail wallabies (*Onychogalea fraenata*). In January 2003, several tammar wallabies from the Macquarie University Fauna Park were found to be exhibiting upper respiratory distress with serous to mucoid to purulent nasal discharges, inspiratory wheezing and intermittent sneezing. This was associated with a gradual decline in animal weight over the preceding weeks before clinical signs were detected. Several of the animals had more severe dyspnoea and radiographs in these animals revealed tracheal stenosis caused by peritracheal inflammation. *B. bronchiseptica* was isolated in the nasal passages of several affected animals, and was thought to be the cause of the outbreak. A total of 28 animals presented with clinical signs and of these four died and two were euthanased. *B. bronchiseptica* was cultured from the nasal passages of four out of five animals from which swabs were collected at the time of post-mortem.

All animals exhibiting clinical signs were treated with antibiotics (1ml/10kg s.c., enrofloxacin, Baytril®, Bayer Animal Health, Kansas) and bromhexine (0.5ml oral, Bisolvon® Chesty, Boehringer Ingelheim) daily. All animals in the colony, including those presenting clinical signs, were vaccinated with a *B. bronchiseptica* vaccine (Canvac®-BB, CSL Ltd., Parkville, Victoria, Australia). Treatment duration varied from 19 days to over 4 months (mean = 43 days) until all clinical signs had abated. Survival appeared to be dependent on the early detection of the disease and subsequent antibiotic treatment.

97) *MYCOBACTERIUM TUBERCULOSIS* INFECTION IN ELEPHANTS (*ELEPHAS MAXIMUS* and *LOXODONTA AFRICANA*) IN SWEDEN

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Human tuberculosis, caused by *Mycobacterium tuberculosis*, is an important zoonotic disease of elephants kept in captivity or in close association to humans. Tuberculosis in elephants was described in ancient Hindu literature with the name of "slow wasting fever". It was described and confirmed to be of the human type in several zoos in Germany at the beginning of the 19th century. Today tuberculosis in elephants probably occurs more frequently than recognised. It is estimated that 3% of the captive elephant population in North America is infected (Mikota el al, 2000). The information about the current occurrence of this important zoonotic disease in elephants in Europe is limited.

Since November 2001, *M. tuberculosis* infection has been diagnosed in Sweden in five Asian elephants (*Elephas maximus*) in one zoo, and in one African elephant (*Loxodonta africana*) in another zoo. The first case was detected during a routine control based on mycobacterial culture of trunk lavage. Earlier cultures of trunk lavages, and other *in vivo* tests including skin test and various types of serological ELISA tests had shown negative or uncertain results. Only the index case in the Asian group and the African elephant showed clinical signs (coughing, weight loss, polydipsia, polyuria and stiff gait). All the other elephants were in good condition.

All six elephants were euthanased. The post mortem examination revealed tuberculous lesions of various size, extension and activity in the different elephants, ranging from advanced disease with extensive granulomatous pneumonia to discrete calcified nodules in the lungs and mediastinal lymph nodes. The lungs, upper airways and respiratory lymph nodes were the tissues most frequently involved.

Histopathology showed diffuse granulomatous consolidation, formed by whirls of epithelioid cells surrounded by macrophages, eosinophils and lymphocytes. In the larger lesions there were multiple foci of central caseous necrosis. Calcification was mild. Variable numbers of acid-fast bacilli were observed in the Ziehl-Neelsen stained sections, but in many of the sections they were not detected.

M. tuberculosis was isolated from the lung lesions from the six elephants. The strains were subtyped by RFLP and spoligotyping. Four different subtypes of *M. tuberculosis* were identified in the Asian elephants, indicating various sources of initial infection. Some animals had mixed infections (more than one subtype).

The diagnosis of preclinical tuberculosis in elephants is difficult. *In vivo* tests (tuberculin skin test, serological ELISAs) were shown to have poor performance. Positive culture from trunk lavage provides a definite diagnosis. Since shedding is intermittent some cases remained undetected until advanced disease.

Tuberculosis acquired by humans from elephants is considered to result most often from close and repeated contact, such as that with elephant handlers. No evidence of human infection acquired from the elephants has been shown in the affected zoos. It is important that

monitoring and control programs for the detection of tuberculous elephants in captivity is implemented in order to prevent spreading of infection to humans and to other animals. There is an urgent need for sensitive and specific *in vivo* tests that can detect the presence of *M. tuberculosis* infection at early stages.

Mikota S.K., Larsen R.S. and Montali R.J. (2000). Tuberculosis in elephants in North America. Zoobiology 19 (5), 393-403

SESSION 10:
HEALTH OF MARINE ECOSYSTEMS

98) RECENT DISCOVERY OF BRUCELLA INFECTION IN NEW ZEALAND HECTOR'S DOLPHINS (*CEPHALORHYNCHUS HECTORI HECTORI*) – A POTENTIAL CAUSE OF LOW FECUNDITY AND HIGH PERINATAL MORTALITY

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Hector's dolphin (*Cephalorhynchus hectori hectori*) is found only around the South Island of New Zealand and there are as few as 6000 to 7000 animals in three separate populations, off the east, west and south coasts. Maui's dolphin (*C. hectori maui*) is critically endangered with probably less than 100 individuals off the west coast of the North Island. Low fecundity and high neonatal mortality are thought to contribute to the conservation status of both subspecies. In March 2004 a trial was conducted to evaluate satellite telemetry on Hector's dolphins in the waters surrounding the Banks Peninsula, South Island. This provided the opportunity to assess the health of three captured animals. One adult female was sero-positive for *Brucella abortus* in a cELISA test. This was the first evidence that *Brucella*, a potentially significant pathogen of dolphins, was present in New Zealand marine mammals. An investigation of all beached and bycaught Hector's and Maui's dolphins was initiated in collaboration with the Department of Conservation and the National Centre for Disease Investigation. Since June 2004 post mortem examinations have been conducted on 24 specimens from around the South Island. When the carcasses are suitable, the following tissues are collected for culture on *Brucella* selective media: mammary gland, spleen, lung, lymph nodes, liver, uterus, foetus if present, and testis. The same tissues are also used for nested *Brucella* PCR for 442bp and 272bp sequences of OMP25. PCR product of the expected size was amplified from all tissues of one subadult female dolphin. The genetic\ sequence of the amplified product is being compared to sequences for *Brucella* sp. from terrestrial and marine mammals. Further research is needed to determine the prevalence of this infection in Hector's and Maui's dolphins and to determine whether it is causing reproductive failure. This could have significant consequences for both subspecies but for the critically endangered Maui's dolphin in particular.

99) NEW VARIANT OF *BRUCELLA* FROM WHALES INHABITING WESTERN NORTH PACIFIC

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Brucellosis is known to cause reproductive disorders or abortion in terrestrial mammals, especially domesticated animals. The causative agent, *Brucella* has been isolated from a variety of wildlife species such as bison, elk, African buffalo, reindeer, caribou, feral swine, wild boars, foxes, and hares. The broad spectrum of *Brucella* infection has recently expanded to include marine mammals. Microbiological studies indicated that *Brucella* strains isolated from marine mammals were distinct from terrestrial isolates. Molecular analysis of the marine strains from the eastern North Atlantic also showed that they represent a separate group from terrestrial *Brucella* species. In the western North Pacific, a substantial number of common minke whales (*Balaenopera acutorostrata*) were shown to have marked granular lesions with caseation and calcification in their gonads, and serological investigations suggested *Brucella* infection. In contrast, neither pathological nor serological evidence of *Brucella* infection was detected in Antarctic minke whales (*Balaenoptera bonarensis*). Using polymerase chain reaction (PCR), *Brucella* was detected in granular testes of the minke whales in the western North Pacific. Insertion of the IS*711* element downstream of *bp26*, a molecular marker of marine strains, and specific DNA fragment for Atlantic seal strain was detected. Characterisation of outer membrane protein 2 (Omp2) genes (*omp2a* and *omp2b*) showed that the genes occupied a unique position among *Brucella* strains. Detailed analysis of the two genes revealed that they are chimeric, possessing the sequences shared with terrestrial and marine strains. As a whole, Pacific *Brucella* DNA showed the most similarity to Atlantic seal strain.

100) THE ST. LAWRENCE ESTUARY BELUGA: A TALE OF A WHALE

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The St. Lawrence beluga (*Delphinapterus leucas*) population is estimated to number approximately 1000 animals down from an estimated 5,000 in the 19th Century primarily due to hunting. Hunting was stopped in 1979 and the population has been protected since 1983. Recent surveys indicate that the population is stable but it is unknown why it has failed to rebuild to historic levels. Its failure to recover may be related to its habitat, the St. Lawrence estuary which, as part of the St. Lawrence Seaway, is subject to maritime traffic and associated noise as well as industrial, agricultural, atmospheric and municipal wastes. Since 1982 mortalities of St. Lawrence beluga have been documented and causes of mortality investigated through dissection of beach-cast carcasses on the shore or necropsy of transported carcasses at the University of Montreal, St-Hyacinthe. Since 1983, the first full year of the program, until 2004 over 330 mortalities have been documented with a mean of 14.7 (10 - 21) per year. The mean age of stranded beluga carcasses is 17 years. St. Lawrence beluga can live over 40 years, the oldest lifespan documented for this species in the world. There are few if any natural predators in the St. Lawrence estuary, although killer whales are occasionally observed. If we examine cause of death using carcasses coded 2 to 4 and stratify age classes we find that 50% of examined calves less than one year old die at parturition or shortly thereafter probably due to abandonment or separation. Seventy-three percent of juveniles (more than 1 year but less than 5 or 7 years old) die from parasitic infections, particularly lungworms (*Halocercus monocerus, Stenurus arctomarinus*), thus eliminating these animals from the population before they can reproduce. Thirty-three percent of adults (more than 5 or 7 years old) die from bacterial, viral or parasitic infections, 23% have terminal cancer, 2% of females die giving birth and 7% die from trauma (some due to propellers or ship collision). Terminal cancer is observed only in adults with a mean minimum age of 21 (11 – 29.5) years. Various degenerative diseases associated with age have been observed in this population as well. Cause of death could not be determined in 34% of adults. St. Lawrence beluga, like many marine mammals worldwide, have various chemical contaminants in their tissues but a cause and effect link between contaminants and cancer is elusive. The role of infectious diseases and possible interaction with immunotoxic contaminants is poorly understood and this population is considered at risk from a catastrophic epizootic. With an absence of predators and no hunting, clearly disease is playing a large role in this population. Since the early 1990's enforcement of current laws and new legislation on pollution, municipal and industrial discharge has helped improve water and air quality in the river and estuary but more remains to be done. Due to their long lifespan it will be many years before an improvement in the health of St. Lawrence beluga is observed as some chemical contaminants are passed from generation to generation. As a precautionary management measure a moratorium on rehabilitation of marine mammals is in place to protect this population from an epizootic that may arise due to the introduction of exotic or novel pathogens.

101) IMPACT OF GLOBAL WARMING ON THE NESTING OF MARINE TURTLES IN THE UNITED ARAB EMIRATES

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The Arabian Gulf contains numerous offshore islands which are used by turtles for nesting. From a data set of Sea Surface Temperatures (SST) and air temperatures it was determined that there has been a considerable rise in temperature in the study area over the last 20 years (Sheppard & Loughland 2002).

In the UAE marine turtles nest during spring (March - June), and a study on the effect of rising temperatures during the nesting period on incubation was undertaken. In 2001 temperature and humidity dataloggers were implanted into turtle nesting sites on three islands in the southern Arabian Gulf to determine nest temperature and humidity parameters. In 2005, the dataloggers were retrieved.

Data retrieved from the data loggers provided information on the average nest temperature and humidy during the spring nesting periods, and this was compared to average air temperatures during these times. The study presented for the first time micro climate data for marine turtle nest sites, on a background of data confirming global warming from the most temperature stressed, and fastest warming marine area in the world.

With a global warming scenario, it is proposed that criticle nest incubation temperatures will shift towards the winter season, therefore narrowing the incubation success of the current nesting period. As temperature also influences the sex ratio of turtle hatchlings, it is suggested that rising temperatures within the current nesting period may also favour females. Possibilities on whether turtles may alter their nesting behaviour in response to warming SST's are also discussed.

As the study examines the impact of elevated temperatures on incubation, sex ratio and nesting behaviour of marine turtles, it may be useful for predicting what will happen within a scenario of global warming in other tropical environments around the world.

Marine turtles are already threatened by a host of anthropological impacts, especially at nesting sites and additional impacts resulting from global warming could be fatal, and need to be considered when planning for the long-term survival of these important species.

102) SUB-LETHAL AND LONG-TERM EFFECTS OF EXPOSURE TO DOMOIC ACID IN STRANDED CALIFORNIA SEA LIONS

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Domoic acid is an excitatory water-soluble neurotoxin produced by a number of algae, including *Pseudo-nitzschia australis*. Acute domoic acid toxicosis resulting from the glutamate agonist action of domoic acid is well documented in California sea lions (*Zalophus californianus*) and is manifested as neurologic signs, including ataxia, disorientation, seizures, and death. However, the long-term and sub-lethal effects of domoic acid toxicity have not been fully investigated. Reproductive failure as a result of abortion and premature parturition was observed in 149 of 442 intoxicated adult females admitted to rehabilitation centres in California between 1998 and 2002 that survived acute toxicosis. Domoic acid was detected by liquid chromatography with tandem mass spectrometry in amniotic fluid, foetal urine and gastric fluid samples tested up to 2 weeks after initial stranding. This suggests the foetus acts as a sink for domoic acid that is typically rapidly cleared from model mammalian species (half life in primates is 4 hours). Of a further 179 California sea lions that stranded since 2002 showing neurological signs typical of domoic acid exposure, 46% exhibited neurological effects longer than 2 weeks after initial stranding. Magnetic resonance imaging on live animals and histopathology from animals that either died or were euthanased revealed varying degrees of unilateral and bilateral hippocampal atrophy, neuronal necrosis and gliosis in the limbic system. These data suggest that exposure to domoic acid can have effects on sea lion reproduction and survival beyond acute mortality documented to date.

103) SEROLOGICAL EVIDENCE OF INFLUENZA A VIRUS INFECTION IN SEALS INHABITING RUSSIA AND IN CETACEANS IN THE NORTH PACIFIC AND ANTARCTIC OCEAN

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Influenza A virus infects a variety of birds and mammals including marine mammals such as seals and cetaceans. Four subtypes of influenza A virus (H7N7, H4N5, H4N6, H3N3) and three subtypes of the virus (H1N3, H13N2, H13N9) were isolated from seals and from cetaceans. These viruses were shown to be related to avian influenza A viruses based on the results of antigenic and/or genetic analyses.

Serological surveillance was conducted in Caspian seals (*Phoca caspica*), Baikal seals (*Phoca sibirica*), and ringed seals (*Phoca hispida*) in Russia. Serum antibodies to influenza A virus were detected in the serum samples from 36% (28/77) of Caspian seals, from 29% (2/7) of Baikal seals, and 83% (5/6) of ringed seals using enzyme-linked immunosorbent assay (ELISA). In a hemagglutination-inhibition tests using H1-H15 reference influenza A viruses, these sera reacted to A/Aichi/2/68 (H3N2) and A/Bangkok/1/79 (H3N2) strains. One of the sera from ringed seals reacted with A/seal/Masschusetts/1/80 (H7N7). These results suggest that infection with H3 viruses of human origin were prevalent in the seals inhabiting those waters. Estimation of age in those three species of seals indicates that the viruses were prevalent in the seals after the counterpart viruses disappeared in humans.

Antibodies against influenza A virus were examined in two species of baleen whales and seven species of toothed whales inhabiting the western North Pacific, and in one species of baleen whales inhabiting the Antarctic ocean. Among 398 serum samples examined by ELISA, antibodies to influenza A virus were detected in seven among 103 samples from common minke whales (*Balaenoptera acutorostrata*) and in two among of 34 samples from Dall's porpoises (*Phocoenoides dalli*). The hemagglutinin subtype of the virus was not determined by the hemagglutination-inhibition test. The present serological evidence suggests that sporadic influenza A virus infection occurred in these whales in the western North Pacific.

104) AN IMPROVED CELL CULTURE METHOD FOR THE ISOLATION AND STUDY OF MARINE MAMMAL DISTEMPER VIRUSES

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The genus *Morbillivirus* is a member of the family *Paramyxoviridae* comprising six members, Measles virus (MV), Dolphin morbillivirus (DMV), Canine distemper virus (CDV), Peste-des-petits ruminants virus (PPRV), Phocine distemper virus, (PDV), and Rinderpest virus (RV). All are highly contagious and are considered dangerous pathogens to the host animals that they infect. Distemper viruses in marine mammals, the extension of the host range of CDV to include felids and endangered African wild dogs (*Lycaon pictus*) are recent examples of their ability to cause significant mortality in wild animal populations. Diagnosis of distemper virus infections has relied on results obtained from a number of testing methodologies including serology, histopathology with fluorescent antibody staining, reverse transcription polymerase chain reaction (RT-PCR) and less frequently, viral isolation. The study of distemper viruses to date has been limited in large part due to the lack of a good cell line system for their primary isolation and propagation. Isolation in mitogen-stimulated lymphocytes or co-cultivation of infected tissue with African green monkey kidney cells (Vero) is possible but is at best inefficient and time consuming requiring weeks of incubation and repeated "blind passaging". Recently, the cell receptor sites for MV was identified as the human signalling lymphocyte activation molecule (SLAM); also known as CD150. Subsequently, it has been determined that SLAM also acts as the cell receptor for other morbillivirus species including CDV and RV allowing successful virus isolation in as little as 24 hrs. The present study describes the use of a stable transfected Vero cell line expressing canine SLAM, obtained from Dr. Yusuke Yanagi, Kyushu University, Japan, and its usefulness in isolating and studying PDV from experimentally infected ferrets (*Mustela putorius*). RT-PCR was used to independently confirm the identity of the recovered virus.

105) EFFECTIVE, FIELD-BASED INHALATION ANAESTHESIA FOR CRABEATER SEALS (*LOBODON CARCINOPHAGUS*)

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Thirty-five adult crabeater seals (*Lobodon carcinophagus*) (17 females, 18 males) were anaesthetised with combinations of the sedative midazolam (n= 34) and the gaseous anaesthetic isoflurane (n= 35) during three research cruises to the Marguerite Bay region of the Antarctic Peninsula (~67°S, 67°W) in the austral winters of 2001 and 2002. Seals were captured and anaesthetised to enable instrument attachment (satellite relayed data loggers; SRDL) and experimental routines to be conducted as part of the U.S. Southern Ocean Global Ocean Ecosystems Dynamics Program. Modifications were required to gas anaesthetic equipment to achieve field portability and sufficient heating to allow operations in ambient temperatures of –20oC to 0oC. The thermal insulation and active temperature control of the equipment was effective in very low ambient temperatures, maintaining internal temperatures of the equipment within a 15oC-25oC range for periods of up to 4 hours in the field without the need for re-heating. While the anaesthetic equipment was heavy (approximately 45 kg) and reasonably cumbersome, a team of four people were able to handle it, as well as all the other equipment required to capture, weigh and restrain such a large mammal. The mean mass of the crabeater seals was 250 ± 53.4 kg (range 188-385 kg). Seals were sedated with an average intramuscular dose of midazolam of 0.55 ± 0.14 mg/kg (range 0.26 - 0.85 mg/kg) delivered via a pole syringe (n=32). One seal was not given midazolam and two seals were injected intravenously. The level of sedation provided by midazolam varied with each seal, but generally provided moderate sedation, making capture and masking for induction practical and safe. Mean induction time with isoflurane was 8 ± 4.8 minutes (range 2 - 27 min). Mean maintenance concentration over the anaesthetic period was 2.3 ± 0.9 % of isoflurane (range 1 - 5 %). Average recovery time was 18.2 ± 8.8 min (range 4 - 42 min). The duration of the anaesthesia was determined by the procedures to be performed on each seal and ranged from 15min through to 3hr 10min. For procedures estimated to take 60 min or longer, seals were intubated post induction for maintenance of gaseous anaesthesia (n=10); all others were maintained by mask. Two seals were anaesthetised for protracted periods (3 hr and 3 hr 10 min) to facilitate isotope equilibration. Both recovered uneventfully but showed evidence of hypothermia during the later part of the procedure and had among the longest recovery times. During the anaesthetic the mean respiratory rate was 5.2 ± 2.2 breaths per minute (range 1 - 10.1 breaths/min). No substantial difficulties were experienced and anaesthetics were easily managed. This drug combination and the use of modified, heated equipment provide an effective anaesthetic procedure for crabeater seals. Since this work was conducted a smaller, more field-portable heated anaesthetic machine has been developed which only heats the vaporiser. The use of heated inhalation systems represents a positive development that improves the quality and safety of chemically restraining ice seals in the field compared to the use of injectable anaesthetic agents.

106) DETECTION OF PATHOGENIC PROTOZOA IN MARINE ECOSYSTEMS USING MUSSELS (*MYTILUS* SPP.) AS BIOINDICATORS

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Pathogen pollution into the nearshore marine environment may be a significant threat to animal and human health. Bivalve shellfish (e.g. mussels, clams, oysters) have been shown to filter and retain pathogenic protozoa, viruses, and bacteria, so they may be useful bioindicators of pathogen pollution and water quality. Bivalves filter large amounts of water, often 2 litres/bivalve/hour, allowing them to concentrate pathogens from their environment. The nearshore marine environment along the California coast provides critical habitat for many wildlife species, including the threatened southern sea otter (*Enhydra lutris nereis*). Sea otters consume approximately 25% of their body weight in prey items each day, and a major part of their diet is shellfish. The link between pathogen pollution and sea otter mortality has yet to be proven; however, epidemiologic studies in California support this hypothesis.

In 2001 a three-year study was initiated to evaluate mussels as bioindicators of faecal contamination in coastal ecosystems of California. Hemolymph samples from 4680 mussels (*Mytilus* spp.) were tested for *Cryptosporidium* genotypes using PCR amplification and DNA sequence analysis. Our hypotheses were that mussels collected from sites near livestock runoff or human sewage outflow would be more likely to contain the faecal pathogen *Cryptosporidium* than mussels collected distant to these sites, and that the prevalence would be greatest during the wet season when runoff into the nearshore marine environment was highest. To test these hypotheses, 156 batches of sentinel mussels were collected quarterly at nearshore marine sites considered at higher risk for exposure to livestock runoff, higher risk for exposure to human sewage, or lower risk for exposure to both faecal sources. *Cryptosporidium* genotypes detected in hemolymph samples from individual mussels included *C. parvum*, *C. felis, C. andersoni*, and two novel *Cryptosporidium* spp. Factors significantly associated with detection of *Cryptosporidium* spp. in mussel batches were exposure to freshwater outflow and mussel collection within a week following a precipitation event. Detection of *Cryptosporidium* spp. was not associated with higher or lower risk status for exposure to livestock faeces or human sewage sources. This study showed that mussels can be used to monitor water quality in California and suggests that humans and animals ingesting faecal- contaminated water and shellfish may be exposed to both host-specific and anthropozoonotic *Cryptosporidium* genotypes of public health significance.

SESSION 11:
WILDLIFE HEALTH IN THE TROPICS

107) THE CONSEQUENCES OF HAEMOGREGARINE INFECTION IN A TROPICAL SNAKE

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Keelback snakes (*Tropidonophis marii*) are harmless, oviparous and semi-aquatic. They occur throughout coastal areas of northern Australia and their diet consists mainly of frogs. As part of an ongoing ecological study, a population of keelbacks inhabiting a coastal river floodplain near Darwin in the Northern Territory was surveyed for blood parasites. This revealed a high prevalence of infection by haemogregarines but that the intensity of infections varied widely among snakes. Because the ecology of the study population of snakes is well documented, it was possible to look for relationships between intensity of parasitaemia and measures of individual fitness. Specifically, the goal was to determine whether the blood parasites were detrimental to their host.

"Haemogregarines" are coccidial blood parasites in the Family Haemogregarinidae; most species in snakes belong to the *Hepatozoon* genus. The life cycle in a frog-eating snake would include sporogony in a blood-feeding arthropod vector (definitive host), ingestion of the arthropod and production of cystozoites by a frog (1st intermediate host) and merogony and gametogony in the snake (2nd intermediate host) following ingestion of the frog.

During April-May 2002, snakes were captured by hand and returned to the laboratory to be marked and measured and to have a blood smear taken. Several fitness-related variables were measured or assessed before the snakes were released back at their capture location. Fitness-related variables included antipredator behaviour, locomotor ability, reproductive status, feeding rate, growth rate, recapture rate, body condition and female reproductive output. Intensity of parasitaemia was determined on Giemsa-stained blood smears by counting the number of infected red blood cells in a total of 1000 cells.

There were no significant relationships between intensity of parasitaemia and any of the fitness-related variables ($N=92$, all $P>0.16$). Four explanations were considered for why snake fitness is independent of intensity of parasitaemia:

1. The apparent lack of effect on the host may be due to dissociation between blood stages and tissue stages of the parasite. This would be relevant if the tissue stages are more detrimental to the host than the blood stages. To test this, blood smears and histological slides of tissues were examined from 13 road-killed keelbacks from the study area during 2004. Tissue stages of the parasite were primarily in the lung and did not elicit an obvious inflammatory reaction. The number of parasite stages observed in the tissue was significantly correlated with the number of parasites detected in the blood ($r=0.83$, $P<0.0005$). Thus, the level of infection observed in blood smears also reflects the concurrent level of tissue infection.

2. Levels of infection may be too transitory to effect processes occurring over several months (e.g. growth, reproductive allocation). To assess changes in infection over time, blood smears of 27 snakes that were recaptured 12-568 days after their initial blood smear were examined. There was no relationship between intensity of parasitaemia at initial and subsequent captures ($r=0.25$, $P=0.21$). Therefore, levels of infection change over time.

3. Parasite infection may not elicit erythropoiesis. The detrimental effects of blood parasites in other reptiles have been associated with increased numbers of immature erythrocytes released into circulation and the decreased oxygen carrying capacity of the blood. The proportion of immature red blood cells in smears was assessed for a subsample of snakes showing wide variation in intensity of parasitaemia. We found no relationship between the proportion of immature red blood cells in circulation and intensity of parasitaemia ($N=42$, $r=0.24$, $P=0.19$). This suggests that the haemoparasites are not eliciting erythropoiesis and that aerobic capacity of the snakes is not affected.

4. Other detrimental factors may mask effects related to haemogregarine infection. For example, during post-mortem and histological examinations of several keelbacks, numerous metazoan parasites were encountered, sometimes in large numbers and causing tissue distortion or inflammation.

108) THE IMPACT OF DISEASE ON FREE LIVING CASSOWARY POPULATIONS IN FAR NORTH QUEENSLAND

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The Southern Cassowary (*Casuarius casuarius johnsonii*) occurs in the wet tropics region of far north Queensland from Ingham north to the tip of Cape York, a distance of approximately 2000 km. Within this area only 976,030 hectares remains as cassowary habitat, most of which is subject to pressure from one or more causes related to human activity.

The cassowary is a keystone species without which the continued diversity of rainforest plants is not possible. Only an estimated 1500 birds remain in the wild, a large number of which occur in and around human habitation. A number of significant threats exist in these areas for continued cassowary survival. They include vehicles, dogs, feral pigs, habitat clearing, fragmentation and degradation, hunting (both indigenous and recreational), feeding by tourists and locals, and natural disasters.

Diseases known to afflict free living cassowaries include internal parasites (particularly ascarids), aspergillosis (*Aspergillus fumigatus*) and avian tuberculosis (*Mycobacterium avium*). Immature birds (8-12 months of age) are most commonly affected, particularly in areas where habitat fragmentation and degradation are significant. The long term implications of these losses on recruitment into the population are not known.

Research into disease is currently limited to autopsy of birds which have died as a result of illness or injury, and opportunistic examinations and sample collection of free-living birds brought in for treatment or relocation. From this limited data source, a preliminary reference range for haematological and plasma biochemical parameters has already been established and a catalogue of diseases initiated. A research project is being developed to determine the prevalence of avian tuberculosis and other diseases in local free-living populations, and to correlate rate of infection with areas of significant habitat degradation and high human impact.

109) CAUSES OF DEATH FOR MOUNTAIN GORILLAS (*GORILLA BERINGEI BERINGEI* AND *G. B.* UNDECIDED) FROM 1968-2004: AN AID TO CONSERVATION PROGRAMS

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The Mountain Gorilla Veterinary Project's (MGVP) mission is to improve the sustainability of the extant mountain gorilla populations in the range countries of Rwanda, Uganda and the Democratic Republic of Congo. To achieve that mission, we provide clinical care in cases of human-induced or life-threatening illness or injury, and perform research to help clarify and ameliorate health threats. Post-mortem examination is important in both contexts, as it helps define causes of morbidity and mortality as well as providing insight into the most important health threats and high-risk categories within the populations.

Ideally, thorough gross and histopathologic examinations would be conducted on all dead gorillas. In reality, work is limited by the ability to recover dead gorillas, the field conditions under which gross post mortem exams are often conducted, and the quality of tissues available for histopathologic exam and ancillary testing. Recurrent episodes of war and instability have also caused the loss of valuable records and samples. The relatively recent developments of digital photography and internet communications have aided the field veterinarians' ability to communicate with specialists elsewhere. The development of molecular techniques coupled with preservation methods that don't require refrigeration or freezing has also improved diagnostic capabilities.

For this review, causes of death for 100 gorillas were analysed. Available records dated back to 1968, and some exams were performed by people with limited medical training. Post-mortem examination protocols were standardised in 1988, but have been implemented with varying rigor since then. The majority of cases (66/100) had at least some tissues available for histopathologic review, though some diagnoses (12/100) were made from gross examination only, and for the remainder the information on type of examination was unavailable.

Causes of death by age and sex are presented in Table 1. The leading cause of death for all age classes is trauma. For infants, the primary type of trauma is infanticide (13/15), while for juveniles (7/9) and adults (15/16) direct or indirect poaching is the main type of trauma. Respiratory disease is the second most common cause of death and, in this dataset, affects all age classes equally. For cases in which the cause of death is undetermined, infants are usually suffering from decomposition that hampers diagnosis, and adults, especially aged individuals, frequently have multiple subclinical and/or presumably chronic processes the impacts of which are difficult to assess in the absence of clinical laboratory data. Incomplete histology and autolysis also contributed to this category.

Understanding the most common causes of death for an endangered species like the mountain gorilla helps target activities and direct the use of resources for protecting populations. Infanticide, though the most common cause of death for infants, is a natural occurrence. Poaching, however, is clearly not a natural occurrence and the national parks' and legal authorities in all three countries devote significant resources to combating it.

More than 25% of mountain gorilla deaths are related to infectious disease. Due to the high level of exposure to humans that the gorillas experience and the ease of transmission of many pathogens, significant efforts are made to reduce the accompanying risk. Gorilla health monitoring and response efforts are also sensitive to clinical signs of potentially infectious diseases. Work with humans, associated livestock, and other sympatric wildlife also helps identify potential health threats so that programs can be developed to mitigate risks.

The pathology database will continue to grow and thus help inform the multidisciplinary effort to sustain mountain gorilla populations for the future. We believe that *in situ* veterinary care and attention to ecosystem health has, along with law enforcement and habitat protection, contributed to the growth of this population.

Table 1. Causes of death by age class

Cause	Infant (birth to < 3 years)	Juvenile (3 to <10♀ and	Adult (≥10♀ and 13♂)	% of Total
Trauma	15	9	16	40%
Respiratory	8	6	10	24%
Undetermined	9	1	7	17%
Multifactorial	1		4	5%
Gastrointestinal	1	1	2	4%
Metabolic	1	1	1	3%
Cardiac			3	3%
Infectious - other			1	1%
Developmental	1			1%
Neurologic	1			1%
Parasitic	1			1%
Total	38	18	44	100%

110) AN OUTBREAK OF APPARENT POXVIRUS INFECTION IN TWO GROUPS OF MOUNTAIN GORILLAS (*GORILLA BERINGEI BERINGEI*)

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Poxviruses are responsible for a variety of clinical diseases in nonhuman primates, some of which can be fatal. Symptoms and lesions are most commonly related to the integumentary system but can also involve the respiratory system as is often the case with monkeypox.

During January and February of 2005, a number of mountain gorillas (*Gorilla beringei beringei*) were found suffering with various lesions and ailments consistent with poxvirus infections. At least 10 gorillas (infants, juveniles, and adults) in two different groups had single facial ulcers near their nostrils. Two infant gorillas in two different groups suffered from upper respiratory tract infection evidenced by severe nasal discharge, one of which also had a facial ulcer. Three young infants in the same group suffered from multiple, diffuse, papules which seemed to progress to vesicles and eventually umbilicated erosions.

Two veterinary interventions were performed: one before the outbreak was fully evident to treat the infant (#1) that had severe nasal discharge but no facial lesions; and a second two weeks later to treat the most severely affected young infant (#2) with multiple papules. Blood was collected opportunistically from both anaesthetised mothers but only the second infant because of difficulties during the first intervention. Two full-thickness skin biopsies were also collected from infant #2 and nasal and throat swabs were collected from the first mother and infant.

Bacterial cultures of infant #1's nasal swab as well as the mother's nasal and throat swab all grew *Neisseria meningitides*. In mountain gorillas, this bacterium is suspected of being a secondary invader and has previously been associated with the pneumonia-related death of an infant gorilla after a respiratory disease outbreak in a different group.

Both mothers were seropositive for Epstein-Barr virus, adenovirus, and parainfluenza type 3, while the second mother also had titers for influenza A, hepatitis A and hepatitis B. Infant #2 was seropositive for Epstein-Barr, parainfluenza type 3 and hepatitis A. None of these gorillas were seropositive for monkeypox. Histopathologic analysis of the skin biopsies showed severe, multifocal, vesicular, ulcerative and pustular dermatitis with marked acute to subacute interface and periadnexal deep dermatitis. There were only equivocal intracytoplasmic inclusion bodies. Direct electron microscopy did not aid diagnosis and transmission electron microscopy and molecular diagnostics are currently being pursued. All affected gorillas appear to have recovered from this outbreak with no severe sequelae.

111) IMMOBILISATION AND ANESTHESIA OF AMERICAN ALLIGATORS (*ALLIGATOR MISSISSIPPIENSIS*): A BRIEF REVIEW AND UPDATE

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The American alligator (*Alligator mississippiensis*) is one of two living alligator species. It is distributed throughout freshwater to brackish swamp and riverine environments in the south-eastern United States. The Chinese alligator (*A. sinensis*) is highly endangered and confined to a limited geographical range primarily associated with the Yangtze River in China. Although once listed as an endangered species due to over hunting and habitat loss, American alligator populations have responded dramatically to protection. They are now a common and important part of wetland ecosystems throughout their range. Alligators are potentially very large and long-lived crocodilians. The largest animals may exceed 14 feet and may live to over 100 years. Procedures in free-living alligators that may require general anaesthesia include radiotelemetry implantation and visceral tissue biopsies. Local anaesthesia is used for osteoderm, dental and mandibular bone biopsies. Mandibular nerve blockade has recently been shown (Wellehan and Gunkle, unpublished) to be effective in providing analgesia for tooth and gingival biopsies in alligators and other crocodilians. General anaesthesia is indicated in captive alligators for diagnostic procedures (e.g., radiography, computed tomography, gastroscopy, tracheoscopy/ bronchoscopy), and surgery (e.g. laparoscopy, laparotomy, orthopaedics). Most, if not all aesthetic procedures, involve some form of physical restraint or containment of the alligator. Although hypothermia has been used for "anaesthesia", the adequacy of analgesia is unknown and is a major physiological stress on the immune system. Drugs used in the past for chemical immobilisation have included barbiturates, muscle relaxants (both depolarising and non-depolarising) and dissociative anaesthetics (ketamine and tiletamine). Recent advances include the use of medetomidine alone or in combination with ketamine, and propofol. Medetomidine offers the advantage of markedly decreasing ketamine dose, and it is able to be reversed with atipamezole. The benzodiazepine midazolam enhances the reliability of restraint with medetomidine/ketamine, while reducing overall dose. Midazolam can be reversed with flumazenil. Propofol is a short acting aesthetic that can be given to effect. Its major disadvantage is that it must be given intravenously. The authors have found the ventral coccygeal vein is readily accessible in crocodilians. It can be catheterised for continuous or intermittent drug administration. The supravertebral sinus (external jugular vein) is not recommended for aesthetic drug administration because of its close proximity to the subarachnoid space, as well as the head. Inhalation anaesthesia, using either isoflurane or sevoflurane in oxygen, is problematic because of unpredictable pulmonary shunting. Monitoring equipment for cardiovascular and respiratory function include a Doppler flow detector and capnograph, respectively. The Doppler flow probe is placed either directly over the heart or over the eye. The later is used to detect optic arterial blood flow. A probe placed in the cloaca may also detect blood flow. Further research is indicated to evaluate the physiological effects of currently available aesthetic drugs, and the influence of pulmonary shunting on inhalant aesthetic uptake and removal.

112) UNRECOGNISED AMPHIBIAN DISEASE PROBLEMS IN FAR NORTH QUEENSLAND, AUSTRALIA

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The Frog Decline Reversal Project Inc. is a non-profit, amphibian conservation organisation based in Cairns and servicing the region from Townsville to Torres Strait. One of the activities of the FDR Project is to run a small operation known as the Cairns Frog Hospital. Functioning as a rescue activity, its original goals of receiving sick and injured frogs were to assist more individuals to survive and reproduce and to gather information about the causes of mortality in local frogs. While the former goal has meant that more frogs have survived to reproduce than would have in our absence, the latter goal of identifying new threats has overwhelmed our community group in the numbers of animals affected and the range of conditions present.

Chytrid fungus is the only emerging pathogen officially recognised in Australia as having a dramatic impact on this country's amphibian populations. However, the Cairns Frog Hospital has seen only a few live chytrid cases which have been transported from cooler locations in the state. The diseased frogs we receive are affected by a variety of known diseases such as Mucor amphibiorium and Aeromonas, but more importantly, the overwhelming majority of cases involve pathogens which have not been documented or isolated. These include an immuno-deficiency complex in two species; a probable soil fungus which attacks the nervous system, deformities associated with a virus, and a disturbing number of neoplasia cases for which oncogenic viruses appear to be responsible.

An overview of these conditions will be presented.

SESSION 12:
MARSUPIALS

113) *CRYPTOSPORIDIUM* IN MARSUPIALS: OCCURRENCE, INFECTION PATTERNS, GENETIC CHARACTERISATION AND PUBLIC HEATH IMPACTS

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Cryptosporidium, an apicomplexan protozoan parasite, is a causative agent of enteric disease in a broad range of hosts. *Cryptosporidium* has been identified in greater than 170 vertebrates including 13 marsupials. In recent years much has been learnt about the epidemiology of *Cryptosporidium* in Australian native animals. A two year study in which 3,557 faecal eastern grey kangaroo samples were screened for *Cryptosporidium* oocysts demonstrated that prevalence varies with season and can be as high as 30%, and that infections appear to be asymptomatic and more common in juveniles. Similar patterns were observed in a smaller study of brushtail possums. Our understanding of the molecular epidemiology of *Cryptosporidium* in Australia has also been expanded. Novel *Cryptosporidium* genotypes have been identified in kangaroos and possums, and information on non-marsupial derived genotypes in marsupial species has begun to emerge. However, it remains apparent that further investigation into *Cryptosporidium* epidemiology in Australia is required. To enable the naming of *Cryptosporidium* marsupial - derived genotypes as species, data is required in areas of pathology and parasite host specificity. The transmission between marsupial and placental hosts is not clearly understood, nor are the public health impacts (if any) of marsupial-derived *Cryptosporidium* genotypes. Our current understanding of *Cryptosporidium* in Australian native animals and future directions will be presented.

114) EPIDEMIOLOGY AND HOST SPECIFICITY OF *CRYPTOSPORIDIUM* IN EASTERN GREY KANGAROOS

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Cryptosporidium is an apicomplexan protozoan that causes acute enteric disease in humans and animals. The current study investigates the epidemiology of *Cryptosporidium* in eastern grey kangaroos (*Macropus giganteus*) inhabiting two sites within the Sydney Hydrological Catchment. High prevalence of *Cryptosporidium* (20%) was associated with the autumn months, with a 10-fold increase in April compared to February or September. The highest *Cryptosporidium* incidence overlapped with the peak of young emerging from the pouch, at which time, greater than 50% of the young animals were infected. Oocyst shedding intensity varied between 10 and $3x10^5$ oocyst/g faeces.

Genetic analysis at the 18S rDNA locus has revealed the presence of two genotypes, EGK1 and EGK3, dominating at different periods of the year. *Cryptosporidium* strain-specific variation is being investigated at three genetic loci encoding for the sporozoite surface proteins P23, GP900 and S60, believed to be involved in parasite-host recognition, attachment and invasion.

115) INVESTIGATION OF CUTANEOUS PAPILLOMATOSIS AND OCULAR CHLAMYDIALES INFECTION AFFECTING ENDANGERED WESTERN BARRED BANDICOOTS (*PERAMELES BOUGAINVILLE*) IN THE WILD

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The western barred bandicoot (Perameles bougainville) is an endangered marsupial, occurring in the wild on only two islands off the coast of Western Australia (Dorre and Bernier Islands). Initial conservation efforts focussed on captive breeding programmes, with the ultimate goal being reintroduction of captive-bred individuals into predator-proof enclosures and habitat in historical distribution ranges on the mainland. Whilst the captive breeding programmes have generally been successful, continued progress is being hampered by a progressively debilitating, wart-like syndrome and ocular Chlamydiales infection. Cutaneous papillomatosis has been detected in both captive and wild populations of western barred bandicoots. The small skin lesions resemble papillomas, however, as the lesions increase in size there is histological evidence of malignant transformation into carcinomas. The results from preliminary examination of skin lesions using light microscopy, transmission electron microscopy and indirect immunohistochemistry is suggestive of a viral aetiology. Ocular chlamydiales infection was detected in wild western barred bandicoots and was associated with conjunctivitis, ocular discharge, blepharitis and corneal opacity. Four Chlamydiales types have been identified by gene sequencing, including a strain of C. pecorum (which was different from strains previously found in koalas) and several new Chlamydiales genotypes. Further research aimed at investigating the significance of both of these diseases to the captive breeding programme for western barred bandicoots will be undertaken over the next three years in collaboration with the Western Australian Department of Conservation and Land Management. This research will be funded by an ARC Linkage Grant.

SESSION 13:
REPTILES AND BIRDS

116) INVESTIGATIONS INTO SNAKE VIRAL DISEASES IN AUSTRALIA

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A series of epizootics in captive collections of snakes, and the discovery of inclusion bodies within the tissues of free-ranging snakes has triggered investigations into snake viral diseases in Australia.

Inclusion body disease of boids has previously been reported in captive Australian snakes (*Morelia spilota variegata* and *Morelia spilota spilota*), and although a viral aetiology was suspected in these animals, an aetiologic agent was not identified[1]. Iridovirus infections have been described in green tree pythons (*Chondropython viridis*) that were illegally imported into Australia[2]. An erythrocytic virus has also been identified in a diamond python (*Morelia spilota spilota*)[2]. Although Australia enjoys a great diversity of endogenous serpents, our understanding of their viral diseases is poor.

Diagnostic material submitted to the Australian Registry of Wildlife Health (the Registry) and NSW Department of Primary Industries have been reviewed and classified into the following entities:

- Snakes with progressive ascending paralysis, and other neurological signs, that have eosinophilic cytoplasmic inclusion bodies within cells in the brain, and possibly other parenchymatous organs, with or without mild non-suppurative meningitis. This entity has been listed as classical inclusion body disease.
- Captive and free ranging native pythons with no clinical signs, or progressive neurological dysfunction, and histological evidence of eosinophilic to basophilic intranuclear inclusion bodies within cells in the brain
- Captive king brown snakes (*Pseudechis australis*) with severe progressive neurological dysfunction and multifocal non-suppurative encephalitis
- Outbreaks of acute respiratory disease in captive snake populations, where histological findings include a proliferation of mesobronchial and infundibular epithelium. A single snake examined in this category had small numbers of eosinophilic cytoplasmic inclusion bodies within the respiratory epithelium
- Captive snakes with chronic neurological dysfunction and some evidence of demyelination of white matter tracts in the caudal brainstem and spinal cord
- Clinically normal snakes with small numbers of renal or pancreatic epithelial cells bearing intranuclear inclusions. Affected nuclei are large, with peripheralised chromatin and an amphophilic centre.

Clinical assessment, histopathology, electron microscopy, haemagglutination inhibition serology for ophidian paramyxovirus, degenerate polymerase chain reaction testing, and DNA *in-situ* hybridisation have been conducted on a variety of tissues from selected affected snakes. PCR testing has identified DNA fragments consistent with the presence of an inactive endogenous retrovirus. Electron microscopy has identified the presence of C type retroviral particles, reovirus particles and herpesvirus particles within the brains of snakes with neurological dysfunction. The presence of herpes-like and glassy inclusions have also been reported to the Registry, but these were thought to be artifacts associated with autolysis of the

brain samples. Serology has identified captive snakes positive to two paramyxovirus antigens (Paramyxovirus - 1 and Paramyxovirus - 7).

These serological, morphologic, and molecular data support the presence of a variety of viral agents in Australian snakes. A lack of specific diagnostic tests available locally, and the difficulty in obtaining appropriate permits to export tissues have hampered these studies. There is an immediate need for structured investigations to determine the aetiology, host range, epidemiology, and potential impact of these diseases in native Australian snakes.

1. Carlisle-Nowak MS, Sullivan N, Carrigan M, Knight C, Ryan C, Jacobsen ER. Inclusion body disease in two captive Australian pythons (*Morelia spilota variegata* and *Morelia spilota spilota*). *Aust Vet J* 1998, 76:98-100

2. Hyatt AD, Williamson M, Coupar BEH, Middleton D, Hengstberger SG, Gould AR, Selleck P, Wise TG, Kattenbelt J, Cunningham AA, Lee J. First identification of ranavirus from green pythons (*Chondropython viridis*). *J Wildl Dis* 2002, 38(2):239-252

Acknowledgements: The authors thank Drs George Reppas, Julie Barnes, Neil Sullivan, Peter Kirkland and Ruth Manvell for their contributions, and Jane Hall and Kaye Humphreys for their technical support.

117) OPHIDIAN PARAMYXOVIRUS AND INCLUSION BODY DISEASE OF BOID SNAKES: EXPERIENCES FROM THE UNITED STATES

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Ophidian paramyxovirus (OPMV) was first reported in the mid-1970s in a venom laboratory in Switzerland (Clark et al, 1979). This was followed by reports in the United States (Jacobson et al, 1980; Jacobson et al, 1981; Jacobson et al, 1992) and Europe (Essbauer and Ahne, 2001). While viperids were the first snakes diagnosed with OPMV, it is now known that members of multiple families of snakes are susceptible. A paramyxovirus also was isolated from caiman lizards (*Draecena guianensis*) that died with pneumonia following importation into the United States (Jacobson et al, 2001). Recently, carpet pythons (*Morelia spilotes*) with a neurological disease have been seen in the United States and Australia, and a paramyxovirus is suspected of being the causative agent. In the United States, identification of OPMV was originally based upon viral isolation in cell culture, identification of the virus using positive or negative staining electron microscopy, and immunohistochemistry (Richter et al, 1996). More recently, polymerase chain reaction has been used to amplify the partial sequence for the large (L) and hemagglutinin-neuraminidase (HN) genes of several snake isolates (Franke et al, 2001). Findings supported the splitting of these paramyxoviruses into two main groups, with bridging intermediate isolates. Polymerase chain reaction and an *in situ* hybridisation technique with a generic paramyxovirus cDNA probe are now being used to identify ophidian paramyxovirus nucleic acid in tissue section (Sand et al, 2004). The major serologic test used to screen snakes for exposure to OPMV is hemagglutination inhibition (Jacobson et al, 1992). In this assay an isolate of OMPV should be used as the antigen. In experimental challenge studies, snakes seroconvert approximately six to eight weeks following exposure. Thus in the early stages of an outbreak, snakes typically will not manifest an antibody response to the virus. For screening snakes in quarantine, samples should be taken at the time of arrival and then 8 to 12 weeks later. Many questions remain concerning OPMV including whether infected snakes can remain latent carriers and whether *in utero* transmission to neonates can occur in both viviparous and oviparous species. The presence (prevalence) of infection in wild populations of snakes is unknown.

A second important infectious disease of snakes has been named inclusion body disease (IBD). This disease is named for the characteristic intracytoplasmic inclusions seen in epidermal cells, visceral epithelial cells, and neurons and glial cells in the central nervous system (Schumacher et al, 1994). While originally thought to be a disease of boid snakes (boas and pythons), similar appearing inclusions have been reported in non-boid snakes (Raymond et al, 2001). The first reports of IBD were in the United States, followed by reports elsewhere in the world, including Australia in 1998 (Carlisle-Nowak et al, 1998). In the United States in the 1970s and 1980s, pythons were the most common snakes diagnosed with IBD. Starting in the 1990s there was a shift toward boa constrictors with relatively few pythons diagnosed over the last 10 years. The reason for this host change is unclear. As with OPMV, there is no information on the presence of this disease in wild populations. While the causative agent of IBD is thought to be a retrovirus (Schumacher et al, 1994), the original isolate used in a transmission study was lost and transmission studies with more recent isolates (Jacobson et al, 2002) have not been performed. While inclusions in IBD do not consist of viral particles, a 68 kd protein has been identified in the inclusions (Wozniak et al, 2002). Two-dimensional gel electrophoresis is being used to identify this protein so that its amino acid sequence can be determined. Inclusions may represent either an abnormal storage

product or viral-associated protein in an infected cell. Since the causative agent has not been firmly established, diagnosis involves identification of inclusions in antemortem biopsies or postmortem tissue samples. Occasionally, inclusions are present in lymphocytes in a peripheral blood film. Since some snakes have relatively few inclusions, false negatives are problematic. A test that can identify a marker for this disease is needed.

The movement of snakes in the pet trade and between zoological collections has given certain pathogens the opportunity to infect new species. Poor quarantine practices and mixing of species from different parts of the world has contributed to the problem. Release of captive snakes to the wild may result in the introduction of new unwanted pathogens into native populations of reptiles. Since serologic, molecular, and other specialised diagnostics are limited to a few laboratories working with these pathogens, rapid identification is not always possible. Identifying the ecology of these pathogens in wild populations will remain a challenge.

Franke J, Essbauer S, Ahne W, Blahak S. 2001. Identification and molecular characterisation of 18 paramyxoviruses isolated from snakes. Virus Res 80:67-74

Jacobson ER, Gaskin JM, Simpson CF, Terrell TG. 1980. Paramyxo-like infection in a rock rattlesnake. J Amer Vet Med Assoc 177:796-799

Jacobson ER, Gaskin JM, Page D, Iverson WO, Johnson JW. 1981. Illness associated with paramyxo-like virus infection in a zoologic collection of snakes. J Amer Vet Med Assoc 179:1227-1230

Jacobson ER, Gaskin JM, Wells S, Bowler K Schumacher J.1992. Epizootic of ophidian paramyxovirus in a zoological collection: pathological, microbiological, and serological findings. J Zoo Wildlf Med 23:318-327

Jacobson ER, Origgi F, Pessier AP, Lamirande EW, Walker I, Whitaker B, Stalis IH, Nordhausen R, Owens JW, Nichols DK, Heard D, Homer B. 2001. Paramyxovirus infection in caïman lizards, *Draecena guianensis.* J Vet Diag Investig 13:143-151

Raymond, J.T., Garner, M.M., Nordhausen, R.W., Jacobson, E.R. A Disease Resembling Inclusion Body Disease of Boid Snakes in Captive Palm Vipers (*Bothriechis marchi*). J. Vet. Diagn. Investig. 13:82-86

Richter GA, Homer BL, Moyer SA, Williams DS, Scherba G, Tucker SJ, Hall BJ, Pedersen JC, Jacobson ER. 1996. Characterisation of paramyxoviruses isolated from three snakes. Virus Res 43:77-83

Sand MA, Latimer KS, Gregory CR, Rakich PM, Jacobson E, Pennick KE. 2004. Molecular diagnosis of paramyxovirus infection in snakes using reverse transcriptase-polymerase chain reaction and complimentary deoxyribonucleic acid: ribonucleic acid in situ hybridisation. J Vet Diagn Invest 16:442-448

Schumacher, J., Jacobson, E.R., Homer, B. and Gaskin, J.M. 1994. Inclusion body disease of boid snakes. J Zoo Wildl Med 25:511-524

Wozniak E, McBride J, DeNardo D, Tarara R, Wong V, Osburn B. 2000. Isolation and characterisation of an antigenically distinct 68-kd protein from nonviral intracytoplasmic inclusions in boa constrictors chronically infected with the inclusion body disease virus (IBDV: Retroviridae). Vet Pathol 37:449-459

118) PSITTACID HERPESVIRUS (PsHV-1) IN AUSTRALIA

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There are 2 genetically distinct Psittacid herpesviruses (PsHV); PsHV-1 and PsHV-2. There are 4 genotypes of PsHV-1[4]. PsHV-1 is the causative agent of Pacheco's disease (PD) and internal papillomatous disease (IPD)[4,6,7]. The genetic variants of PsHV-1 are most likely host adapted to different species of New World parrots[4]. They do not typically cause disease in their host. Disease occurs when latently infected host species are co-mingled with other susceptible species. Not all susceptible species however develop disease and they themselves can become latently infected[4]. Old World parrots especially Pacific species, are relatively resistant. PD has been reported in Moluccan cockatoos, sulfur-crested cockatoos, gang-gang cockatoos, galahs, rosellas[1], fig parrots, eclectus parrots and cockatiels in captivity outside Australia (Phalen pers comm.). PsHV-1 can infect passerines and may cause disease in them. PsHV-1 infection has been suspected in a zebra finch and canaries[4].

PD is an acute and usually fatal disease and can result in massive die offs of parrots especially Amazons. PD has been reported in New Zealand (imported birds in quarantine) but has not been reported in Australia. IPD primarily affects macaws, amazons and less commonly conures. Most lesions occur in the cloacal and oral mucosa. Green-winged macaws are most commonly and severely affected. Blue and gold macaws are also commonly affected[4,6]. IPD has been reported in an Australian cockatiel in the USA[5]. IPD has not been reported in captive or wild native psittacine birds in Australia or in wild bird's elsewhere[4,6].

There are many different species on New World parrots that are well established in aviculture in Australia. These include those species that are known to be potential natural hosts for the different PsHV-1 genotypes.

In early 1997 two green-winged macaws in Australia were diagnosed with IPD[2,5]. These birds had been imported as part of a large consignment of macaws from the United Kingdom in 1993. There are anecdotal reports of other cases of IPD in green-winged macaws in Australia.

In February 2004 an imported, male green-winged macaw was diagnosed with IPD. The bird had been paired with an imported hen for 9 years. Both birds were part of the same consignment of birds that had been imported in 1993. Their current owner acquired the birds in 1995. The pair had bred and raised numerous chicks. All eggs laid for the previous two years however were infertile. The birds were presented for investigation of their infertility. The male had extensive cloacal papillomatosis and a single oral papilloma. The histologic appearance of a biopsy taken from the mass confirmed the diagnosis. The female bird was normal. The findings were reported to Biosecurity Australia and no action was required.

All New World parrots (30 birds of 5 species) in the collection from which these two birds came were tested for PsHV-1. Cloacal and oral mucosal scrapings were taken and sent to Texas A&M University. A sample of the papilloma from the affected male was also submitted. All samples were tested for the presence of PsHV-1 DNA using a nested primer set from the UL16 open reading frame and PCR. These primers have been shown to detect all four genotypes of PsHV-1. Additionally all samples were tested for the presence of the cytochrome oxidase gene by PCR to verify that adequate cellular DNA was in fact present in the sample. Twenty-nine of the 30 samples contained adequate DNA for testing. It was

confirmed that the male green-winged macaw with IPD was positive for PsHV-1 genotype 2 and the female with whom he had been paired was positive for PsHV-1 genotype 3. A third green-winged macaw, an offspring of the above pair was also positive. Attempts at sequencing the virus in him were unsuccessful because there were 2 sequences present, most probably genotypes 2 and 3. All other birds tested were negative.

This is the first report of PSHV-1 in Australia. The 3 affected birds have been isolated. It is highly likely that the 2 imported birds came into Australia already infected. State and Federal agriculture agencies have been informed. Owners of offspring of this pair have been informed of the findings.

Confirmation of the presence of PsHV-1 in green-winged macaws in Australia is significant. While it is rare for these genotypes to cause PD in macaws, cases of PD caused by genotype 3 have been reported in macaws and conures (Phalen pers comm.). IPD can spread widely in collections of captive macaws and genotype 3 is associated with the development of bile duct and pancreatic carcinomas. Of greater significance is the fact that the virus that causes IPD is now known to be the same virus that causes PD. Birds with IPD must be considered a potential source for PD outbreaks. Australian native psittacines are susceptible and PsHV-1 may pose a threat to both captive and wild Australian native birds2.

1. Arnold ID. An outbreak of psittacine herpesvirus in rosellas. *Proceedings of the Association of Avian Veterinarians* 1990. pp283-291

2. Gallagher A and Sullivan ND. Internal papilloma disease in green-winged macaws (*Ara chloropter*). *Australian Veterinary Journal* 1997. 75 (9): 675

3. Macwhirter P and Perry R. Introduction of exotic avian diseases through legal importation. *Australian Veterinary Journal* 1997. 75 (9): 677-678

4. Phalen DN, Tomaszewski EK and Styles DK. Epizootiology, diversity and pathogenicity of Psittacid Herpesviruses. *Proceedings of the Association of Avian Veterinarians* 2004. pp3-6

5. Roe RT. Internal papillomatous disease. *Australian Veterinary Journal* 1997. 75(9):676-677

6. Styles DK, Tomaszewski EK and Phalen DN. Psittacid herpesviruses associated with mucosal papillomas of neotropical parrots. *Virology* 2004. 235:24-35

7. Styles DK, Tomaszewski EK and DN Phalen. A novel psittacid herpesvirus in African gray parrots (*Psittacus erithacus erithacus*). *Avian Pathology* 2005. (In press).

Acknowledgements:
The authors would like to thank Frances Hulst, Andrea Reiss, Julie Barnes, Bob Donnelly, Kaye Humphries and Elizabeth Tomaszewski for their assistance with this work and the bird keepers at Taronga Zoo for their professionalism and dedication to the care of these birds.

119) PERSISTENCE OF ANTIBODIES TO WEST NILE VIRUS IN NATURALLY INFECTED ROCK PIGEONS (*COLUMBA LIVIA*)

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In order to use feral pigeons as WNV indicators, WNV antibody persistence in this species is vital information. The objectives of this study were to 1.) determine the persistence of WNV antibodies in feral pigeons over a one year period, 2.) identify trends in antibody titer, 3.) compare serologic assays commonly used in WNV antibody detection [Plaque reduction neutralisation test (PRNT), hemagglutination inhibition test (HAI), and enzyme linked immunosorbent assay (ELISA)], and 4.) evaluate squabs for maternal antibody formation and persistence against WNV. Feral pigeons were captured during April 2003 in Atlanta, Georgia, housed in a mosquito-free environment, and blood samples drawn every three weeks for 60 weeks. All birds with antibodies to WNV on entry retained an antibody response until the end of the study. ELISA results were consistent with PRNT results. HAI results were inconsistent with PRNT results. Neutralising antibody titers to WNV present in the squab serum persisted at reciprocal titers $\geq$ 10 for a range of 19 to 33 days. To our knowledge, this is the first report detailing the persistence of avian maternal antibodies to a North American strain of WNV.

SESSION 14:
LAGOMORPHS

120) THE IMPACT OF OVINE NEMATODES ON THE EUROPEAN HARE (*LEPUS EUROPAEUS*)

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Noting that the European hare appears to be in decline in Europe, that the decline is believed to be associated with agricultural intensification, that the decline appears to be more marked in pastural areas, that adult stages of ovine nematodes have been recorded in wild lagomorphs, that larval stages are pathogenic in several species, and that hares are often sympatric on a fine scale with livestock, we hypothesised that hares are adversely affected by exposure to the strongyle parasites of livestock.

In a preliminary trial, we dosed naïve captive hares with a mixed population of 10,000 infective larvae derived from sheep. We sacrificed those hares at 15 or 21 days post infection and examined the impact of the parasites. We recovered a small proportion of the *Ostertagia circumcincta* as both larvae and adults from the stomach of the hares, and individual immature *Cooperia sp.*and *Nematodirus sp.* from the small intestine. Most of the *Trichostrongylus colubriformis* were recovered, and nematode eggs were found in the faeces of the test animals, indicating that the infections were patent. Duodenal villous atrophy with lymphoplasmacytic infiltration was observed at 15 days. No nematode of any species was recovered from a sympatric sentinel hare, and no nematode was recovered from the large intestine of any hare. Although plasma pepsinogen levels suggested that histotrophic *Ostertagia* larvae had caused some damage to the gastric mucosa, no evidence of damage was seen histologically.

Although the preliminary trial is not yet complete, these early results suggest that further investigation is warranted.

121) EVOLUTIONARY ASPECTS OF EUROPEAN BROWN HARE SYNDROME VIRUS (EBHSV)

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European brown hare syndrome (EBHS) is a highly contagious, acute disease of the European brown hares (*Lepus europeaus*) and mountain hares (*Lepus timidus*), first described in the early 1980s in Northern Europe. EBHS is caused by a non-enveloped positive-strand RNA virus (EBHSV) with a diameter of about 32-35 nm, showing morphological characteristics indistinguishable from those of the rabbit haemorrhagic disease virus (RHDV) and biochemical features typical of the Caliciviridae family. EBHSV is transmitted directly or indirectly, mainly by oro-faecal and respiratory routes. Humans, insects, and birds can act as vectors but no reservoir hosts have been identified yet. Infection via ingested vegetation is also likely, with virus being secreted and excreted, and also being spread in the droppings of predators that have consumed infected hares. EBHSV is highly robust, resisting acid of pH3, and may remain infectious for 3-4 months in the field.

EBHS in hares lasts slightly longer and causes a lower mortality rate (around 50%) than RHD in rabbits (peak of mortality 72-90 hours post-infection). Death can be sudden, lacking any clinical signs but more often aberrant behaviour (such as lack of fear, dullness, jumping into the air, circling, staggering incoordination and convulsion) may be observed before death. During an outbreak, around 30-50% of hares may show a chronic or subclinical course of the disease, which is characterised by generalised jaundice clearly visible at a mucosal and subcutaneous level. Such affected hares can die after several days or may finally recover. At necropsy, the principal findings are oedema and congestion of tracheal mucosa with foamy haemorrhagic contents, liver enlargement, degeneration and decolouration with sharply demarcated and friable lobes, enlargement of the spleen and generalised jaundice. The disease has not been observed in hares younger than approximately 40-50 days, and although those of 2-3 months of age may contract infection they do not usually develop clinical disease. Mortality is highest in the fall, when the population is most dense and the young of the year become susceptible. The impact on local hare populations can vary from 7% to 90% mortality. Under certain circumstances local European brown hare populations can be reduced dramatically. EBHS has been reported in many European countries: Germany, Italy, Belgium, United Kingdom, Croatia, Sweden, Finland, Austria, Spain, Poland, Switzerland, Greece and Slowenia. However, it was not known outside Europe until 2003. EBHS occurred in Europe many years before the appearance of RHD. The earliest confirmed case of an apparently "new disease" called European brown hare syndrome (of which hunters had been aware since the 1970s), was from Sweden in 1981.

All known EBHSV isolates appear to belong to one serotype. The phylogenetic analysis of geographically different EBHSV strains revealed close overall homology in terms of genome sequence (maximum nucleotide divergence of 11.7%) indicating a high level of conservation between isolates. In contrast, antigenic characterisation using a panel of 13 different specific MAbs, which recognise at least 9 epitopes exposed on the surface of the virus, can indicate the existence of different viral strains. The origins of these viruses are difficult to trace. They may have mutated from an avirulent calicivirus of Eurasian lagomorphs or by the introduction of a new virus avirulent in its native host, possibly South American rabbits (*Sylvilagus* spp.) or hares (*Lepus* spp.) imported into Europe in large numbers for recreational hunting in the

1970s and 1980s. Positive identification of EBHSV in European brown hares from Argentina and Uruguay has been shown either directly by virus identification or indirectly by serological evidence of specific antibodies. It is assumed that a less pathogenic or even non-pathogenic form (strain) of EBHSV may have been carried from Europe to South America in the 19th century by the importation of European brown hares, and remained apathogenic until now. This hypothesis is supported (1) detection of EBHSV-antigen by polymerase chain reaction in paraffin embedded specimens collected in the 1970s in Sweden, (2) Lesions consistent with EBHS have been described from England since 1976, and (3) specific antibodies were found in sera archived since 1962, although confirmed clinical cases of EBHS were first diagnosed by pathological and EM investigations in the UK in 1990. This demonstrates that EBHSV might have occurred in European hare populations years before clinical signs of EBHS were described. In conclusion, it is conceivable that a less pathogenic variant of EBHSV may exist among European brown hares in Argentina and that an ancestor of the present European EBHSV strain might have been apathogenic.

122) EPIDEMIOLOGICAL AND MOLECULAR EVIDENCE FOR THE FIRST BIOLOGICAL VECTOR AND RESERVOIR HOST OF MYXOMA VIRUS IN THE UK

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The myxoma viruses naturally occur in the Americas in cottontail rabbit species, *Sylvilagus brasiliensis* and *S. bachmani.* In their natural *Sylvilagus* hosts the myxoma viruses cause benign lesions but in the European rabbit, *Oryctolagus cuniculus*, they cause the more severe, oedematous lesions and systemic disease, known as myxomatosis. Brazilian myxoma virus was introduced into European rabbit populations in Australia, France and the UK in the early 1950's as a biological control agent. The main vectors have always been believed to be mosquitoes and the European rabbit flea, *Spilopsyllus cuniculi*, both of which transmit the virus mechanically.

Research has been carried out on a free-living rabbit population on the University of East Anglia campus (UK) for 25 years, into behaviour, population dynamics, social organisation, genetics, disease and reproduction. Myxomatosis occurs in annual late summer/early autumn epizootics, within the same 14 week period each year. All young of the year become infected and those that recover remain immune to subsequent infection. Mortality rates vary between 59 and 100% among susceptible young of the year. This long-term research highlights gaps in our knowledge concerning the timing, transmission of myxomatosis and the location of the virus between epizootics.

A range of potential arthropod vector species were collected from the UEA study site and screened for the presence of myxoma virus using a PCR assay. This analysis revealed that myxoma infected rabbit fleas, *Spilopsyllus cuniculi*, were present in every month of the year and that mosquitoes play a relatively minor role in myxoma transmission in this study population. Myxoma virus was also detected in a new arthropod species. Compelling circumstantial, epidemiological and molecular evidence is presented for the first biological vector and reservoir host of myxoma virus.

SESSION 15:
GENERAL PAPERS

123) *SALMONELLA* SPP. IN CALIFORNIA WILDLIFE: A COMPARISON WITH INVERTEBRATE, DOMESTIC ANIMAL, AND HUMAN ISOLATES

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Salmonella spp. are common enteric pathogens that may cause diarrheal disease in animals and humans under certain combinations of microbe, host, and environmental conditions. A variety of *Salmonella* spp. are known to infect domestic animals and humans but reports on *Salmonella* spp. in wildlife are more limited. We hypothesised that if fecal pollution is flowing from terrestrial to aquatic ecosystems, then the same *Salmonella* fingerprints may be detected in *Salmonella* from aquatic wildlife and invertebrates as is seen in terrestrial wildlife, domestic animals, and humans. A variety of free ranging California wildlife species were tested for *Salmonella* spp. using selenite enrichment broth and XLT4 agar, with isolates further characterised using antimicrobial testing and chromosomal fingerprints. Barn owls (*Tyto alba*) and northern harriers (*Circus cyaneus*) were chosen to represent raptor species; common murres (*Uria aalge*), western grebes (*Aechmorphorus occidentalis*), common loons (*Gavia immer*), western gulls (*Larus occidentalis*), and surf scoters (*Melanitta perspicillata*) were chosen to represent marine birds; and California sea lions (*Zalophus californianus*), harbor seals (*Phoca vitulina*), northern elephant seals (*Mirounga angustirostris*), and southern sea otters (*Enhydra lutris nereis*) were chosen to represent marine mammals. *Salmonella* spp. were detected in raptor, marine bird, and marine mammal species in 1999-2000, with a mean prevalence of 4% (9/212). *Salmonella* serotypes detected in wildlife species included Johannesberg, Montevideo, Newport, Ohio, Saint Paul, Enteritidis Group D, and 4,5,12:1 Monophasic. One western gull had two serotypes. Antimicrobial resistance was observed to gentamicin, amoxicillin/clavulanic acid, and ampicillin in some *Salmonella* isolates. Chromosomal fingerprints differed among *Salmonella* serotypes but not within serotypes. *Salmonella* isolates obtained between 1999-2005 from wildlife species were compared with isolates from filter-feeding invertebrates, domestic animals and humans in California. A variety of serotypes and antimicrobial resistance patterns were observed among *Salmonella* isolates from aquatic animals, terrestrial animals, and humans. These findings suggest that while *Salmonella* characterisation can be used as a tool to investigate the epidemiology of fecal pathogen pollution, in this study there was not a predominant unique strain of *Salmonella* isolated from aquatic wildlife that was also isolated from aquatic invertebrates, terrestrial animals and humans in California.

124) AVIAN INFLUENZA VIRUS: A NEW PATHOGEN FOR WILD AND DOMESTIC FELIDS

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During the ongoing epidemic of highly pathogenic avian influenza virus (subtype H5N1) infection in poultry in South East Asia, unusual mortality of domestic cats, tigers, and leopards was reported in the affected area. This mortality was linked to feeding on poultry carcasses, implicating H5N1 virus infection as the cause. This is unusual because influenza virus is usually not considered as a mortality factor in domestic or wild felids. In order to determine the pathogenicity and possible routes of infection of H5N1 virus in domestic cats, we experimentally inoculated cats with an H5N1 virus isolate from a fatal human case in Vietnam in 2004. Cats were inoculated intratracheally (n = 3), orally by feeding on infected chicks (n = 3), or by contact with intratracheally inoculated cats (n = 2). Cats infected with another subtype of influenza virus (H3N2) (n = 2) or fed on sham-infected chicks (n = 2) were used as controls. Cats were examined daily for clinical signs and pharyngeal swabs were collected at 1, 3, 5, and 7 days post infection (dpi) for virological examination. Cats were euthanased at 7 dpi and pathological, immunohistochemical and virological studies were performed on respiratory tract samples. The results were similar for all H5N1 virus-inoculated cats, irrespective of route of inoculation. The cats developed clinical signs including lethargy and dyspnoea from 2 dpi, and started excreting virus from 3 dpi. The primary lesion was multifocal pulmonary consolidation, characterised histologically by diffuse alveolar damage. H5N1 virus was confirmed as the cause of these lesions by virus isolation and immunohistochemistry. The control cats showed no evidence of virus infection or disease. Our results show that this H5N1 virus can productively infect domestic cats, cause pulmonary lesions, and result in disease or death. They also show that cats can be infected both by horizontal transmission and by feeding on virus-infected birds. This implies that domestic cats are at risk of potentially fatal disease during H5N1 virus outbreaks in poultry, can play a role in the spread of the virus, and may form an opportunity for the virus to adapt to efficient replication in mammals, including in humans.

125) USE OF ANTICOAGULANT RODENTICIDES AND WILDLIFE POISONING: FROM THE BEST TO THE WORST

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Anticoagulant rodenticides are being used worldwide against rodents but also in many situations against invading mammalian or bird species. The products used include the older first generation compounds (warfarin, chlorophacinone) and the newer second-generation products, active after a single feeding. Many reports of wildlife poisoning exist (e.g. in New Zealand with the use of brodifacoum).

The objective of this presentation is to synthesise field and experimental data obtained in France and to discuss situations in which anticoagulants provided a satisfactory means of controlling invading species, and situations in which they resulted in severe effects on non-target populations.

In the sub-antarctic Kerguelen archipelago, chlorophacinone has been used on wheat bait to eradicate rabbits. Baiting was conducted on small islands as a test, during winter. Baiting was highly effective, with almost 100% lethality, and wild life effects were only observed in some birds (Dominican gull, Eaton's duck). In continental France, bromadiolone (second generation) is currently approved for use against coypu (*Myocastor coypu*, introduced in the 19th century in France) and field voles (*Arvicola terrestris*). In the country, a sanitary network for the surveillance of wildlife diseases (on animals found dead) exists and deals with 2000 to 3000 cases per year.

Until recently, there were only few reports of unexpected poisoning after coypu control operations. A survey on small mustelids (*Mustela lutreola*, European mink, endangered species in France) was conducted to investigate all causes of death. Over the 10 years of body collection, anticoagulant exposure was detected in 9% of the animals found dead. However, only three cases of anticoagulant poisoning could be confirmed (presence of hemorrhages and anticoagulants in the liver). This survey shows that the use of bromadiolone against coypu has some effects on unexpected predators such as the European otter (*Lutra lutra*) and that caution should be exercised when applying the bait.

In several areas, field voles undergo cycles of high population expansion (every 4-6 years), especially in small mountain areas. In the eastern part of France, several years of survey resulted in the analysis of hundreds of animals for bromadiolone, with over 90% confirmed poisoning cases. Although baiting was conducted properly, the wide area covered by bromadiolone use, together with population dynamics of rodents, resulted in massive outbreaks of bromadiolone poisoning in predators and scavengers, including protected and endangered species such as the common buzzard (*Buteo buteo*), red kites (*Milvus milvus*) and black kites (*Milvus migrans*).

The analysis of habitat modification, field use and compound of choice is discussed in this presentation. Several risk factors can be pointed out: second generation products are more at risk, the surface area covered is involved, severe habitat modification and agricultural use of the land have an impact and biological characteristics of the invading species are important in the understanding of undesired wildlife poisoning. Should anticoagulant rodenticides be used against invading species, care should be taken in order to avoid

NON-STUDENT POSTERS

126) PENTASTOMIASIS AND SPARGANOSIS IN A BANDED MONGOOSE (*MUNGOS MUNGO*)

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Reports of diseases in mongoose are few, hence this report of a double infection of parasites in a previously free-ranging animal. Five Banded Mongoose (*Mungos mungo*) were put down at the end of a winter season in a zoo in central Sweden due to frostbites leading to loss of toes and tips of tails. The animals had lived several years in the zoo, but it was the first time this kind of lesion was noted. At necropsy of an adult 1.5 kg male, originally wild caught in Africa, a massive parasite infestation was seen, with numerous white tapeworms in the subcutaneous tissue, and large number of arthropod pentastomids both in the abdominal wall, free in the abdomen and in the spleen. Some abdominal lesions were calcified, other were intact larval cysts. The mongoose was in good body condition, and no further diseases were seen at necropsy or at histopathology. The other four euthanased mongoose were captive bred, and parasites were not found in these animals. The pentastomids were identified as *Armillifer armillatus* and the subcutaneous parasites as spargana larvae of the genus Spirometra.

Armillifer pentastomids are most commonly found in the respiratory tract of snakes, especially in the African python (*Python sebae*). The female parasite discharges eggs containing a fully developed embryo. Mammals typically ingest the eggs, the larvae hatch in the intestines and actively penetrate the gut wall, migrating into internal organs where they are encysted and stay through several moults. When a snake eats the intermediate mammal host containing the nymphs, the parasite develops into the adult stage in the reptile. Infection with A. armillatus is most common in West and Central Africa, and has been seen in antelopes, monkeys, rodents, hedgehogs, warthog, lion, and man. Mongoose is mentioned as a host for Armillifer sp. in only one reference, without any more detailed information. Humans often contract the disease by eating snakes. Usually there is little or no clinical disease due to the parasites, but space-occupying lesions may cause symptoms, and granulomatous reactions have been seen in the liver of humans.

Sparganosis has been reported in different animal species, e.g. crocodiles, feral pigs, dogs, cats, echidnas, and platypus, as well as humans. Various mammal and bird species become infected by feeding on parasitised frogs or snakes, or from natural water sources containing infected copepods, a planktonic crustacean, being the first of the two required intermediate hosts. The copepods ingest embryos that develop from the tapeworms eggs when they reach the water with the faeces of the worms normal host, a dog or a cat. The infection rate in man is low, and sparganosis is in humans acquired mainly by ingesting larvae contained in raw or undercooked meat of infected animals. After ingestion the larvae undergo visceral migration and end up in various tissues where they in humans can grow to up 14 inches in length. The ‘spargana’, or cysts, often develop in subcutaneous connective tissue and superficial muscles. The nodular lesion develops slowly, and may be itchy, inflamed, and painful. The subcutaneous lesion may resemble a lipoma, fibroma, or a sebaceous cyst. The only treatment is surgery.

The wild caught mongoose male was presumably infected when feeding in Africa. Subsequently the alien parasites were brought undetected to Sweden, which stresses the point that live animal shipments seldom involves only the obvious and intended organisms.

127) ANTIBODY FREQUENCY, PREVALENCE AND POTENTIAL IMPACT OF *COXIELLA*, *SALMONELLA*, AND *CHLAMYDOPHILA* INFECTIONS ON CHAMOIS (*RUPICAPRA RUPICAPRA)*

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In order to get a first approach of abortive bacterial disease on chamois population (*Rupicapra rupicapra*) in the massif of Bauges (North Prealps, France), we carried out a retrospective survey of Q fever caused by *Coxiella burnetii*, chlamydial infections caused by *Chlamydophila abortus* and salmonellosis caused by *Salmonella abortus* ovis.

The targeted chamois population has been surveyed for 25 years by demographical studies (thanks to 657 tagged individuals) and sanitary investigations. Serological methods used to test the three abortive infections are similar to those employed for domestic ungulates for Q fever, Chlamydia and Salmonella infection. Lactation status at capture time and offspring/mother longitudinal observation has been used to monitor reproductivity rate.

The impact of these infectious diseases on reproductive failure at the in individual level has been assessed. A logistic regression of reproductive success on 252 females caught from 1980 up to 2003 has been used to test for the influence of antibody prevalence. In a preliminary analysis stage, factors influencing fecundity which were not infectious has been tested: individual age, season and year at capture time can all influence productivity. In a second stage serological status has been added in the explanatory model of reproductive failure.

Appropriate statistical tests were used to evaluate the likelihood of individual antibody titre and reproduction failure correlation. Whereas Q fever and chlamydial infections cannot be significantly correlated with individual reproduction, anti-salmonella antibody titre significantly does reduce the probability of reproductive success: females with high antibody titre (> 320) have 2.8 more chances to abort of fail to breed than females with lower antibody titre. This risk increases together with the antibody titre.

Ongoing studies are carried out to test for the influence of these infections on productivity at the population scale; parallel examination has been attempted to assess the link between infection in domestic stock and possible transmission toward free ranging chamois, or *vice et versa*.

128) REVIEW: RE-EMERGING PARASITES AND INVASIVE SPECIES ISSUES IN JAPAN

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Since the Japanese Government launched a new act to prevent the destruction of natural ecosystems by alien species in June 2004, such species have become an increasingly important issue in Japan. Even veterinarians should actively become involved in tackling the problem of alien species, for example, by conducting educational campaigns, implementing management policies in line with animal ethics, developing painless methods of euthanasia for captured pest animals, and by conducting epidemiological surveys of zoonotic pathogens carried by such animals. Above all, we have investigated and educated parasitological aspects related to zoogeography and ecology of the alien terrestrial vertebrates including reptiles, birds and mammals since 1995. Up to now, alien helminths do not parasitise endemic Japanese vertebrate species. On the other hand the Japanese endemic parasites (i.e., *Ixodes* spp., *Calodium hepatica*, *Trichinella* spp., *Fasciola* spp., *Taenia taeniaformis* etc) parasitised to the alien vertebrates. These parasites are endemic species which are consisted of natural ecosystem of our islands. However, because these alien vertebrates use a vacant niche for the Japanese endemic (host) vertebrates and transport these endemic parasites to the new niches, there seems to be a possibility that re-emerging parasites in not only man but also wildlife will be brought. Namely, even the endemic parasites could be pathogens with using alien hosts as new "vehicles". In this presentation, the review of the results obtained will be given, and implications for the potential strategies for the invasive species issues are considered.

129) RESEARCH AND EDUCATIONAL ACTIVITIES OF THE WILD ANIMAL MEDICAL CENTER IN RAKUNO GAKUEN UNIVERSITY, JAPAN

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Although Rakuno Gakuen University (RGU) was established as an institute for studies on agricultural and dairy science, the Wild Animal Medical Centre (WAMC) was established in April 2004. It is funded by the High Technological Project by the Ministry of Education and Culture, Japan. We presents the aims of the WAMC and the trends in research and educational activities based on these aims, namely, 1) to provide facilities for sampling infectious pathogens and toxic agents from dead wildlife under the P2 level, 2) to tentative provide facilities for maintaining injured wildlife for veterinary training and/or sampling 3) to provide facilities for research and diagnosis of diseases among wildlife, zoo and aquatic captive animals and exotic pets by zoo vets, university staffs, NGOs and so on, registered with the High Technological Project, and 4) to provide educational facilities for studies of zoo and wildlife medicine and natural history. Undergraduate education staff in the school of veterinary medicine of RGU is involved in the management of WAMC, that is, in coordinating university researchers, zoo veterinarians, national and local government officers, and students, obtaining budgets, making documents, writing and selecting research papers, and so on. To perform the field research and educational activities of zoo and wildlife medicine, it is ideal that such facilities are located in rural areas. To show the present status and future trends of WAMC, the following provides an outline of our resent research activities. The research fields presented here are divided into3 types, namely, those involving alien, endemic and captive wild animal species. Consequently, these results are connected to host ecology, and such educational trends could provide information from micro to macro levels.

130) TYPE E BOTULISM IN WILD BIRDS ON THE LOWER GREAT LAKES: A CONSEQUENCE OF INVADING ALIEN SPECIES?

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Spores of *Clostridium botulinum* Type E are widely distributed in aquatic ecosystems, but Type E intoxication has only rarely been associated with large-scale wildlife mortality. Since 1998, annual outbreaks of Type E botulism have occurred in southern Lake Huron and in Lakes Erie and Ontario, involving many thousands of shorebirds, gulls, terns, diving ducks, mergansers, grebes and loons. Unusual in terms of the number of avian species involved, their geographic scope, their size and their repetitive nature, these outbreaks may reflect fundamental shifts in the ecology of the lower Great Lakes, possibly associated with invading alien species. The harbinger of this series of annual outbreaks occurred on the southeastern shore of Lake Huron in autumn 1998, when Type E botulism killed hundreds of common loons (*Gavia immer*). In subsequent years (1999, 2002) outbreaks in this area involved not only loons but gulls (*Larus* spp.) and grebes (*Podiceps* spp.).

Type E botulism on Lake Erie was first confirmed in gulls dying during summer 1999 at Presque Isle, Pennsylvania on the south shore. In autumn 1999, Type E botulism killed about 6,000 red-breasted mergansers (*Mergus serrator*), loons and grebes along the north shore of the west basin of Lake Erie. Over the next four years, Type E botulism on Lake Erie adopted a general annual pattern. In summer, local, usually relatively small-scale outbreaks involved scores to hundreds, rarely thousands, of resident gulls, terns (*Sterna* spp), double-crested cormorants (*Phalacrocorax auritus*) and shorebirds (Scolopacidae). In autumn larger outbreaks killed many hundreds to many thousands of southbound migrant fish-eating birds (mainly red-breasted mergansers, common loons, grebes) and diving ducks (mainly long-tailed ducks *Clangula hyemalis*). Fish-eating birds and diving ducks generally died off-shore; where the carcasses drifted in was determined by the prevailing winds.

The location of major outbreaks on Lake Erie shifted from the west basin (1999) to involve both the central basin and east basin (2000-2004), latterly concentrating in the east basin (2002-2004). Major mortalities were those of 1999 referred to above; 2000, when about 6,000 fish eating birds washed onto the New York shore at the east end of the lake; 2001, when 3,000 gulls, fish-eating birds and long-tailed ducks died along the New York shore; 2002, when over 3,000 ring-billed gulls (*Larus delawarensis*) died near Buffalo NY, and 12,600 long-tailed ducks and over 3,000 fish-eating birds came ashore on the New York coast; 2003 when 2,000 loons and hundreds of gulls and long-tailed ducks died on both sides of the east basin; 2004, when about 2,800 loons, 2,700 long tailed ducks, and hundreds of birds of other species were found on the New York shore.

Type E botulism was first confirmed on Lake Ontario in 2002, when it occurred in gulls and affected about 675 long-tailed ducks along the New York shore. About 1,500 deaths attributed to botulism occurred in gulls, diving ducks, cormorants and loons on the New York side of Lake Ontario in 2003, and botulism occurred in great black-backed gulls at the east end of the lake on the Canadian side. In 2004, over 1,750 carcasses were counted on breeding

colonies and beaches at the east and west ends of Lake Ontario in late summer/fall: mainly double-crested cormorants, great black-backed gulls, long-tailed ducks and white-winged scoters (*Melanitta fusca*). On the New York shore about 1700 birds died, including long-tailed ducks, ring-billed gulls, cormorants, and common loons.

Since 1998 Type E botulism has been confirmed by mouse protection test in the following avian species from the lower Great Lakes: common loon, red-throated loon (*Gavia stellata*), horned grebe (*Podiceps auritus*), red-necked grebe (*Podiceps grisegena*), eared grebe (*Podiceps nigricollis*), great blue heron (*Ardea herodias*), ring-billed gull, herring gull (*Larus argentatus*), great black-backed gull (*Larus marinus*), Bonaparte's gull (*Larus philadelphia*), double-crested cormorant, red-breasted merganser, long-tailed duck, white-winged scoter, greater scaup (*Aythya marila*), common goldeneye (*Bucephala clangula*), American golden plover (*Pluvialis dominica*), sanderling (*Calidris alba*), semipalmated sandpiper (*Calidris pusilla*), American coot (*Fulica americana*), bald eagle (*Haliaeetus leucocephalus*), American crow (*Corvus americanus*). The population implications of recurrent botulism mortality on less common avian species are unknown.

Fish-eating birds dying during botulism outbreaks often had remains of fish in the ventriculus, the most common species identified being the round goby *(Neogobius melanostomus)*. A few abnormally behaving live fish, especially sheepshead (*Aplodinotus grunniens*), collected in New York waters, have contained detectable botulinus Type E toxin, supporting the contention that live intoxicated fish are available for consumption by fish-eating birds. Die-offs of mudpuppies (*Necturus maculosus*) sometimes were associated with mortalities, especially among gulls, while zebra mussels (*Dreissena polymorpha*) and quagga mussels (*Dreissena bugensis*) were present in the ventriculi of many long-tailed ducks and a few fish-eating birds. The presence of incompletely digested food items in the ventriculus of some birds suggests that they died rapidly after ingestion of toxin-laden prey.

Outbreaks were first observed on the southeast shore of Lake Huron and in the west basin of Lake Erie, moving eastward down Lake Erie, and ultimately into Lake Ontario, over the seven-year period. Predisposing factors may include ecological perturbations associated with eruptions of invasive alien zebra and quagga mussels, natives of the Black and Caspian Seas, first observed in the St. Clair river between Lakes Huron and Erie in 1988, and round gobies, also native to the Black and Caspian Seas, first detected in the same area in 1990. *Dreissena* spp. and gobies have rapidly expanded their range throughout the Great Lakes, and produce enormous biomass. In eastern Lake Erie the round goby first emerged as a prominent component of the forage fish community beginning in 2000, coincident with the first observations of widespread bird and fish mortalities in this area. Type E botulism toxin may be produced by clostridia proliferating in a suitable redox environment in the extensive mussel beds on the lake bottom, and may be concentrated in mussels. Fish such as gobies, which are specialist mussel predators, may acquire toxin through feeding in mussel beds, and may then act as a source of toxin for predatory fish or for fish-eating birds higher in the food web. Mussel-feeding diving ducks may acquire toxin directly, rather than via a fish 'vector'. Scavengers such as gulls may acquire toxin through consumption of toxin-containing carcasses, and shorebirds through consumption of toxic invertebrates.

131) EFFECTS OF *CLOSTRIDIUM BOTULINUM* TYPE E NEUROTOXIN IN GREAT LAKES FISH: IMPLICATIONS FOR TRANSMISSION OF AVIAN BOTULISM TO FISH-EATING BIRDS

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Since 1999, large-scale mortalities of fish-eating birds (common loons *Gavia immer*; red-breasted mergansers *Mergus serrator*; grebes *Podiceps* spp.) attributable to Type E botulism have been observed on the lower Great Lakes, especially Lake Erie. The mechanism of Type E botulism exposure in fish-eating birds is unknown. Given that they exclusively eat live fish, it seems likely that their prey play a key part in toxin transmission, but the role of live fish as potential transport vectors of botulinum neurotoxin Type E to birds has not been adequately investigated.

We report here use of a fish botulism exposure model to compare the sensitivity of rainbow trout (*Oncorhynchus mykiss*), round goby (*Neogobius melanostomas*), walleye (*Stizostedion vitreum*) and yellow perch (*Perca flavescens*) to *Clostridium botulinum* neurotoxin Type E (BoNT/E) at four doses: 0, 800, 1,500 and 4,000 Mouse Lethal Doses (MLD). Forty-eight fish of each species (n = 12 per treatment X 4 treatments) were given a gelatine capsule by gastric intubation containing one of the four doses of toxin. Fish were housed individually in tanks and observed continuously by video recorder for 10 days or until death. Behaviour and appearance during this period, time to onset of observed abnormalities, and time to death were recorded.

Each species expressed a unique combination of clinical signs, consisting of changes in behaviour and/or skin pigmentation, prior to death. Behavioural changes varied among species, but included increased opercular movements; opercular abduction; intermittent bursts of agitated swimming followed by periods of immobility on the bottom; loss of coordinated fin control; transient periods of inversion; head-up orientation in the water column; terminal loss of voluntary motor function; immobility and inversion on the tank bottom. Intoxicated round gobies developed a hyperpigmented (black) band around their girth behind the pectoral fins, which, over time, extended anteriorly and posteriorly until the entire fish was almost black. Intoxicated yellow perch also became somewhat to markedly hyperpigmented, which progressed in some cases to darken the entire fish. Intoxicated walleye became somewhat darker than normal. Hyperpigmentation was not observed in rainbow trout.

Mortality rate and time to maximum mortality was species dependent. Round gobies had the highest mortality rates (92-100% depending on BoNT/E dose) and shortest time to maximum mortality (1-1.25 days). Yellow perch were the least susceptible to BoNT/E, having mortality rates of 25-67% depending on dose, and times to maximum mortality of 5.75- 8.5 days, depending on dose. Yellow perch survived significantly longer ($p < 0.05$) than the three other species at all treatments. Since there was variation in the weight of experimental fish between species, times to death for all fish was expressed as a function of BoNT/E dosage (BoNT/E MLD per gram body weight). Although there was a negative linear trend, the slope of the regression line did not differ significantly from 0.

Free toxin was sought by mouse bioassay in muscle tissue and the remainder of the carcass of

each fish collected at the end of the experiment (death, or 10 days in survivors). Free toxin was present in muscle tissue of only two of 192 fish, both round gobies dosed with 4000 MLD BoNT/E. However, many non-muscle carcass samples contained detectable free toxin. The proportion of intoxicated fish in which free toxin was detected was a function of species and dose, varying from 0-92%. Overall, 83% of goby samples tested positive over the three toxin-treated groups.

Results of this study support the contention that live intoxicated fish can represent a vector for transfer of BoNT/E to birds, since intoxicated fish, containing free BoNT/E, can survive for up to several days, depending on species and toxin dose. Furthermore, intoxicated fish might be preferentially selected as prey by fish-eating birds, due to changes in behaviour and/or appearance that make them more conspicuous, and probably easier prey, in comparison with non-intoxicated conspecifics.

132) BASELINE PHYSIOLOGICAL PARAMETERS FOR TASMANIAN DEVILS

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Devil Facial Tumour Disease (DFTD) is a neoplastic disease of Tasmanian Devils with no known aetiology at this stage. At present, diagnosis is based on histology of a suspected lesion. One of the initial components of the diagnostic investigation into DFTD, has been to determine baseline physiologic parameters for Tasmanian Devils. Before this project was undertaken, little was documented regarding haematological and biochemical reference values for Devils. Fieldwork undertaken in 2003-2004 has generated a bank of over 300 blood samples which has allowed a comprehensive study of haematological, biochemical and some endocrine parameters. The analysis of these parameters has allowed comparisons to be made with blood samples from devils diagnosed as suffering from Devil Facial Tumour Disease. The tumour has also been hypothesised to be producing secretory hormones and as such, the biochemical parameters have been beneficial in determining the direction of further endocrinology testing. This study should also be of use in monitoring the health of Tasmanian Devils in captive populations.

133) FIELD LABORATORY DIAGNOSIS OF *BACILLUS ANTHRACIS* INFECTION FROM SWAB AND INSECT SAMPLES COLLECTED FROM DEAD BISON

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The Wood Buffalo National Park (WBNP) and adjacent land managed by the Resources Wildlife and Economics District (RWED) in the Northwest Territory, Canada, have a recurrent history of bison deaths due to *Bacillus anthracis* infection. Collaborative studies between the United States Army Medical Research Institute of Infectious Diseases (USAMRIID), the Defence Research Department Canada (DRDC), the WBNP, and the RWED were conducted in 2001 for animal diagnostic and environmental assessments for *B. anthracis*. Because there is a concern of spreading *B. anthracis* spores through necropsy procedures, it is desirable to evaluate carcasses by non-invasive procedures whenever possible. This paper focuses on the diagnosis of *B. anthracis* infection from swab and insect samples collected from six dead bison with a clinical history and/or gross pathology consistent with cases of anthrax observed in the WBNP- and RWED-managed land. Swab samples (n=17) were collected from the anus, anal discharge fluids, nostrils, mouth, eye, muscle, and unidentifiable decomposed tissue from five different bison. Composite swab samples (n=4) were collected from the mouth, skin, nostrils, and anus from a single dead bison. Maggots (n=2) and an insect (n=1) were collected from two bison carcasses. A standard buffer solution was used to generate samples by washing swabs and the insect and in producing maggot homogenates. Buffer solutions from all sample preparations were analysed in a field laboratory by polymerase chain reaction (PCR) for the presence of DNA associated with the px01 *B. anthracis* plasmid, and by electrochemiluminescence (ECL) for the presence of *B. anthracis* protective antigen. Samples were also germinated in HIBAAAUS-HS before ECL analyses, to determine the presence of viable spores. Two different systems were used to conduct PCR analyses; the Cepheid Smartcycler XC and Idaho Technology's RAPID. Eighteen of 24 samples were positive by at least one of the two PCR systems. Eleven of 24 samples tested positive by ECL without pre-analysis germination. After germination, only 4 of 23 swab samples tested positive by ECL. The four composite swab samples of the mouth, skin, nostrils, and anus all tested positive by both PCR systems and by ECL without pre-analysis germination. Follow-on analyses of samples at USAMRIID included PCR testing for the *B. anthracis* px02 plasmid and the presence of PCR inhibitors, and culture on sheep blood agar.

134) COMPARISON OF STANDARD SEROLOGICAL TESTS TO THE FLUORESCENCE POLARISATION ASSAY FOR THE DETECTION OF SERUM ANTIBODIES TO *BRUCELLA ABORTUS* IN MONTANA ELK (*CERVUS ELAPUS*)

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The Greater Yellowstone Area (GYA) encompassing portions of the states of Idaho, Montana, and Wyoming is the last reservoir of Brucella abortus infected wildlife in the United States. The elk resident in south central Montana, as an element of the 120,000 elk in the ecosystem, have significant exposure to B. abortus. Annual surveys show the average percentage of seropositive animals in this population to be between 2 and 4 percent.

Brucellosis is a nationally regulated disease with only four validated and approved serological tests for detection of the disease in elk (Rivanol, Card, Standard Plate, Complement Fixation). The fluorescence polarisation assay (FPA) is approved for the detection of B. abortus antibodies in cattle, bison, and swine but not cervids. In these species the FPA has the ability to differentiate between field strain and other cross reactants, including strain 19 vaccine antibody. This attribute coupled with the high degree of sensitivity and specificity, the minimum of equipment needed to perform the assay, and the availability of units manufactured for field use, make the fluorescence polarisation assay an attractive candidate for the detection of B. abortus antibodies in wild elk.

One step in the validation of the FPA in elk is assessing the degree of agreement of this test with a battery of standard serologic brucellosis tests. Using data collected from three different populations (n= 783) of wild elk within Montana, we independently measured the association between the FPA and the BAPA, Card, Standard tube, Standard plate, Rivanol, and CF tests for brucellosis. The BAPA and Card test results are dichotomous, but the FPA is a continuous assay. Therefore, FP results are determined to be positive or negative using pre-established FP cut-off value (i.e. >15 mp units above the negative control). A Kappa statistic is calculated for the dichotomised FP results versus BAPA and Card test results. The remaining standard tests for brucellosis are more or less continuous. Standard correlation analysis was conducted to measure the degree of association between these tests and the FP assay.

The distribution of FP results among uninfected elk can be used to evaluate the effect of changing FP cut-off values on the specificity of this test. To assess this distribution, data were assessed from a population of 96 elk that, through repeated testing, was epidemiologically determined to be uninfected. The FP results from this population were used to construct an empirical frequency distribution.

135) SUSCEPTIBILITY OF GREATER SAGE GROUSE TO EXPERIMENTAL INFECTION WITH WEST NILE VIRUS

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Populations of greater sage-grouse (*Centrocercus urophasianus*) have declined 45 – 80 % in North America since 1950. While much of this decline has been attributed to habitat loss, recent field studies have indicated that West Nile virus (WNV) has had significant negative impact on local populations of grouse. We confirm the susceptibility of greater sage grouse to WNV infection in laboratory experimental studies. Grouse were challenged by subcutaneous injection of WNV (1770 pfu/0.1ml). All grouse died within 7 days. The mean estimate for 50% survival was 4.5 days, with peak viremia at time of death at 5.9 log10 pfu /ml (+ 0.2 SEM). Virus was shed cloacally (2.9 log10pfu/ml +0.3 SEM) and orally (3.1 log10 pfu/ml +0.3 SEM). A small pilot study evaluating the effectiveness of a propriety DNA vaccine was also conducted. Four of the five grouse died, but survival time was increased (50% survival = 9.5 days), with one grouse surviving to the end-point of the experiment (14 days) with no signs of illness. Peak viremia for the vaccinated birds was 0.9 log10 pfu/ml (+1.2 SEM). Two birds cleared the virus from their blood. These data emphasise the high susceptibility of greater sage-grouse to infection with WNV. Because greater sage-grouse are a lekking species, it may be possible to capture large numbers of local populations. Hence there is a possibility of using vaccination to protect strategically valuable populations. More work is needed to develop a successful vaccine, but the preliminary results are encouraging for the development of a vaccine as a conservation tool for threatened populations.

136) CAPTIVE VERTEBRATE MANAGEMENT BY DISTANCE EDUCATION THROUGH CHARLES STURT UNIVERSITY

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About the Captive Vertebrate Management Program:
Three articulated, distance education courses in Captive Vertebrate Management (GradCertCaptVertMgt, GradDipCaptVertMgt and MasterCaptVertMgt) have been developed by Charles Sturt University in co-operation with Western Plains Zoo, Dubbo. Other people involved in the courses include professionals from the University of Queensland, Taronga Zoo, Melbourne Zoo and Sea World as well as academics from Charles Sturt University. The ongoing support of the industry for the courses enables us to remain abreast of current issues and provide education of exceptional quality that is relevant to industry personnel. These courses are intended to provide a means for people working in zoos or wildlife parks to extend their knowledge of the successful management of vertebrate species and upgrade their qualifications. The course is also open to interested persons outside these industries (see admission criteria below). The Graduate Certificate was the first tertiary course in Australia to specialise in captive vertebrate management and is recognised by the Australian Regional Association of Zoological Parks and Aquaria (ARAZPA) and Australasian Society of Zoo Keeping (ASZK) as meeting the advanced training needs of their members.

Captive Vertebrate Management Courses:
The courses are structured to be flexible. This allows people with busy lifestyles and heavy work commitments to complete either 1 or 2 subjects of study each semester in each year. That is a total of 2 or 4 subjects per year (completion in either 1 or 2 years). Graduation from the Graduate Certificate allows students to continue to the Graduate Diploma and finally the Masters Degree if so desired.

Admission Criteria:
Applicants for the GradCertCaptVertMgt will be expected to have had a minimum of five years appropriate industry experience; or completed a TAFE zookeeping certificate or equivalent; or completed relevant previous tertiary study. Applicants for the GradDipCaptVertMgt must have completed the Graduate Certificate and at a Credit grade average or better. Applicants for the MCaptVertMgt must have completed the Graduate Diploma.

Course Structure:
Session (semester) 1 (Graduate Certificate)
BIO440 Reproduction Biology
BIO443 Captive Animal Management

Session 2 (Graduate Certificate)
BIO327 Wildlife Ecology and Management (double subject)

Session 3 (Graduate Diploma)
BIO442 Captive Population Management
BIO445 Captive Animal Behaviour

Session 4 (Graduate Diploma)
BIO447 Captive Animal Health
BIO448 Master Planning and Exhibit Design

Session 5 (Master)
SCI501 Special Topics 1
STA201 Scientific Statistics or BIO437 Captive Avian Management

Session 6 (Master)
BIO446 Captive Reptilian Management
SCI502 Special Topics 2

Residential Schools:
Students are required to attend compulsory residential schools of up to 6 days at Western Plains Zoo or Albury campus in sessions 1, 2 and 3.

Enquiries:
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137) SALMONELLA SPECIES IN PINNIPEDS IN NEW ZEALAND

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A survey was carried out to determine the prevalence of *Salmonella* species in pinnipeds in New Zealand waters. Over a three and a half-year period from January 1998 to August 2001, intestinal and faecal samples from pinniped species were examined for the presence of *Salmonella* species. Samples were collected from the intestine of stranded, by-caught or captive pinnipeds at routine necropsy, or from the faeces of healthy free-living or rehabilitating pinnipeds. Subtyping of *Salmonella* species was performed by serotyping, phage-typing and pulsed-field gel electrophoresis (PFGE).

Salmonella Typhimurium phage type (PT) 101 was isolated from apparently healthy and from diseased New Zealand fur seals (*Arctocephalus forsteri*) from the mainland and *S.* Typhimurium PT1 from by-caught seals. *Salmonella* Cerro, *S.* Derby, *S.* Newport, and *S.* Enteritidis PT4 and PT8 were isolated from New Zealand sea lions (*Phocarctos hookeri*) in the Auckland Islands during a mass mortality event in 1998. Isolates of *S.* Cerro and *S.* Newport from sea lions and feral pigs from the Auckland Islands were indistinguishable by PFGE, suggesting an epidemiological link. In June 2001, an animal carer in Otago was believed to have contracted *S.* Enteritidis PT1 from a sick New Zealand fur seal pup that later died from septicaemia.

It is useful to monitor *Salmonella* serotypes in the New Zealand environment to increase our knowledge and understanding of the epidemiology of *Salmonella* in this country. Understanding the epidemiological links between wild animals, domestic animals, and people will assist in formulating recommendations for mitigating transmission risk to people at risk such as agricultural workers, fishers, biologists, animal carers, and Department of Conservation staff. It will also assist in preventing the introduction of infection to relatively pristine Sub-Antarctic and Antarctic environments through human waste, introduced animals or contaminated food products.

138) *LIBYOSTRONGYLUS* SP. IN OSTRICHES: ASSESSING DISEASE RISKS TO KIWI

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Libyostrongylus douglassii (wireworm) causes verminous gastritis in young ostriches and is thought to be predominantly restricted to this species. *Libyostrongylus* spp. has recently been detected in ostriches in New Zealand.

Due to the fact that New Zealand's icon, the Kiwi, is phylogenetically linked to the ostrich there is a concern that the kiwi may be at risk especially where kiwi habitat overlaps with land contaminated by larvae from parasites carried by farmed ratites. Biosecurity New Zealand has been working with staff from the Department of Conservation to establish what the real risks are to kiwi.

The parasite has never been reported in kiwi despite recent evidence to suggest that ostriches in New Zealand may have had the parasite for several years before it was detected. Due to the fact that kiwi have different feeding habits & behaviour to ostrich, and because kiwi are not held captive in high stocking rates, the real risk to kiwi is currently considered to be very small. It is possible that kiwi may be exposed to the parasite but it is hard to predict what the outcome would be. Increased passive surveillance of kiwi has been initiated where kiwi habitat overlaps contaminated farm land.

Farms where *L. douglassii* has been identified have been targeted for surveillance work. In addition, an education programme for kiwi conservancy staff, ostrich farmers and veterinarians was implemented to encourage monitoring of wild kiwi and the treatment of infected ostriches along with good management to reduce parasite loading of pasture.

139) ORAL DELIVERY MICROBIAL VACCINES FOR WILDLIFE

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Several microbial and viral vaccines rely on the delivery of live agents to confer protection. Oral-delivery live vaccines are particularly attractive for use in wildlife, yet the protective agents (bacterial or viral) are usually degraded by gastric hydrolysis, rendering them ineffective. We have developed a lipid-encapsulation matrix that facilitates the oral delivery of live, microbial vaccination agents while retaining immunogenicity and protective efficacy. The matrix has been developed primarily for the delivery of *Mycobacterium bovis* BCG to wildlife tuberculosis (TB) vectors in New Zealand (principally the brushtail possum, *Trichosurus vulpecula*). Oral delivery of a TB vaccine to wildlife in bait form is an attractive method to use as an adjunct to current control strategies.

Present frontline strategies for the control of wildlife TB vectors in New Zealand involve lethal control (poisoning). Alternative control strategies are required where wildlife reservoirs of *M. bovis* persist, including other countries where wildlife vectors are protected (e.g. badgers, *Meles meles*, in the British Isles). Control of TB in wildlife by vaccination is being considered as a means of reducing the spread of disease to domestic animals. The lipid-encapsulated oral BCG vaccine has been tested in mice and possums, and shown to provide similar levels of protection against experimental challenge with *M. bovis* to those seen with needle vaccination. In New Zealand, the oral BCG vaccine is currently being tested in two field trials with free-living possums. These trials, and further testing of the oral BCG vaccine in a range of wildlife species (Europe, North America), will assist in determining whether vaccination can be used as a strategy to reduce the spread of TB from wildlife to farmed animals. Additionally, the lipid encapsulation matrix is being considered as a vaccine delivery vehicle for other wildlife diseases that pose a significant risk to farmed livestock.

140) THE UNITED STATES DEPARTMENT OF AGRICULTURE, ANIMAL AND PLANT HEALTH INSPECTION SERVICE, WILDLIFE SERVICES' NATIONAL WILDLIFE DISEASE SURVEILLANCE AND EMERGENCY RESPONSE SYSTEM

Thomas J. DeLiberto

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Countries conducting disease surveillance in wildlife populations are more widely recognised as being able to understand the epidemiology of specific infectious diseases and zoonotic infections. Therefore, these countries are better prepared to protect wildlife, domestic animals, and humans from the detrimental effects of diseases. Active surveillance for known diseases of economic or public health importance among wildlife is particularly beneficial to the national interest, and the World Organisation for Animal Health (OIE) encourages all countries to develop and maintain wildlife disease surveillance systems. The goal of the United States Department of Agriculture/Animal and Plant Health Inspection Service/Wildlife Services' (WS) involvement in disease monitoring, surveillance, and emergency response is to develop and implement a National Wildlife Disease Surveillance and Emergency Response System (SERS) for the purpose of providing assistance to safeguard American agriculture, human health and safety, and natural resources. This SERS is designed to provide an infrastructure capable of assisting State, Federal, and Tribal agencies and private co-operators with wildlife disease issues. Supplementing these pre-existing programs with a nationally coordinated wildlife surveillance system provides support in sample collection, facilitate information exchange among programs, and ensure diseases of national biosecurity concern are adequately sampled. In addition, the SERS can provide additional laboratory infrastructure that would be available for assisting other agencies with disease diagnosis in emergency outbreaks.

141) FIELD LABORATORY DIAGNOSIS OF *BACILLUS ANTHRACIS* INFECTION FROM TISSUE AND BODY FLUID SAMPLES FROM DEAD BISON AND A MOOSE IN THE WOOD BUFFALO NATIONAL PARK

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The Wood Buffalo National Park (WBNP) in the Northwest Territory and Alberta, Canada, has a recurrent history of bison deaths due to *Bacillus anthracis* infection. A research permit was granted by the WBNP headquarters in 2001 for animal diagnostic and environmental assessment collaborative studies between the United States Army Medical Research Institute of Infectious Diseases (USAMRIID), the Defence Research Department Canada (DRDC), and the Wood Buffalo National Park. This paper focuses on the diagnosis of *B. anthracis* infection from tissue and body fluid samples harvested from the relatively fresh carcasses, 6-36 hours postmortem, of two dead bison and a moose with a clinical history and gross pathology consistent with cases of anthrax observed in the WBNP. Data are also presented from diagnostics conducted on a bison that had been dead for 18 days before necropsy. Animal carcasses were located by helicopter surveillance and a necropsy team was transported to the carcass sites as soon as possible for tissue and body fluid sample collection. Samples were analysed in a field laboratory by two different polymerase chain reaction (PCR) systems for the presence of DNA associated with the px01 *B. anthracis* plasmid, and by electrochemiluminescence (ECL) for the presence of *B. anthracis* protective antigen. A total of 28 samples were analysed from the two fresh bison and the moose carcasses. All samples tested positive by at least one of the two different PCR systems; the Cepheid Smartcycler XC and the Idaho Technology RAPID. Positive test results were observed in 24 of 28 samples by the Igen Origen 1.5 ECL system. All negative ECL test results were observed in samples taken from the carcass with the longest estimated interval between death and necropsy, 24-36 hr. After storage of sample aliquots for 6 days at 4°C, all samples, n=24, tested positive by at least one of the two PCR systems, but only 13 of 24 samples tested positive by ECL. A total of 12 tissue and bowel content samples were analysed from the bison that had been dead for 18 days before necropsy. Positive test results were observed in 8 of 12 samples by both PCR systems. Only of 2 of 12 samples from this bison tested positive by ECL. Those samples were collected from contents in the rumen and bowel. Follow-on analyses of samples at USAMRIID included PCR testing for the *B. anthracis* px02 plasmid and the presence of PCR inhibitors, ECL testing following germination in HIBAAAUS-HS germination medium, culture on sheep blood agar, and immunohistochemical staining of formalin-fixed tissues.

142) EXPRESSION OF IMMUNOLOGICAL MOLECULES IN THE DEVELOPING IMMUNE TISSUES OF THE TAMMAR WALLABY (*MACROPUS EUGENII*)

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Marsupials make ideal models for studying the development of the immune system as their immune tissues develop after birth in the non-sterile pouch environment unlike their "placental" mammal counterparts at the same stage of development that develop their immune tissues in the sterile uterine environment. It is also well recognised that marsupials are unable to mount specific immune responses shortly after birth. This study investigates the expression of several key markers of lymphocyte maturity in a range of developing immune tissues in the tammar wallaby using molecular techniques. The key molecules are limited to the T-cell markers, Rag-1, TdT, TCR-α and TCR-δ, and the B-cell markers, IgG, IgM, IgE and IgA that have been isolated to date from a range of marsupials. The results from this study will be discussed and a time suggesting when the lymphocytes become mature and hence, the neonate may be able to mount an immune response of its own will be discussed.

143) IMPACT OF *KLEBSIELLA PNEUMONIAE* EPIDEMICS ON NEW ZEALAND SEA LION RECRUITMENT

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Epidemics among New Zealand sea lion pups in the 2001/02 and 2002/03 breeding seasons highlight the importance of examining the role of disease in the population dynamics of this threatened species. The pathogen implicated in both events was the gram-negative bacterium *Klebsiella pneumoniae.* Isolates from both seasons were genetically indistinguishable suggesting that the events were caused by a single introduction of an epidemic strain of the pathogen. The events were characterised by a sharp rise in the pup mortality rate approximately three weeks after the start of the pupping season. On post mortem examination, affected pups had one or more of the following lesions: acute suppurative arthritis or polyarthritis, cellulitis, peritonitis, pleuritis, or meningitis. Adults were not apparently affected. Prior to the appearance of *Klebsiella,* mortality to mid-January (~six weeks of age) among pups was 6.2%, with mortality by mid-February (~10 weeks) 16.7%. Mean values for the years in which *Klebsiella* affected pup mortality were estimated at 18.5% (6 weeks) and 26.8% (10 weeks). The marginal increase in mortality attributable to *Klebsiella* was 12.3% at 6 weeks and 10.1% at 10 weeks and the latter value was used in survivorship calculations. There is no evidence of increased mortality in the 2003/04 season given the comparability of mortality data, and temporal pattern with pre-*Klebsiella* seasons. Using survival data from tagged fully mature cohorts, the reduction in the number of females recruiting to the adult population from epidemic years (2001/02 & 2002/03) was estimated. Between 42 and 72 fewer females from the 2002 cohort would reach age five, while between 47 and 80 fewer females from the 2003 cohort would reach age five. In 2008 when both the 2002 and 2003 cohorts will contribute significantly to the mature female population between 93 and 144 fewer females will be present in the population. These missing females potentially represent between 2.3% and 4.6% of the adult female population at a marginal increase in mortality attributable to *Klebsiella* of 10.1%.

144) PATTERNS OF GENETIC RELATEDNESS AMONG WHITE-TAILED DEER FROM A CHRONIC WASTING DISEASE ENDEMIC AREA

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Chronic wasting disease (CWD), a fatal transmissible spongiform encephalopathy of captive and free-ranging cervid ruminants (Williams and Young 1980), was first detected in northern Colorado and south-east Wyoming, and has increasing distribution in mid-western regions of North America (Belay et al. 2004). The aim of our project was to provide individual and population genetic information to help clarify the ecology and epidemiology of CWD through examination of relationships of parentage, kinship, population structure and genetic diversity among CWD positive white-tailed deer in an endemic area in Nebraska (USA). We tested the hypothesis that deer testing positive for CWD were more likely to share higher levels of pairwise relatedness than deer testing negative.

Deer DNA samples (n=150) for which there was accompanying sex, age, CWD pathology, prion protein precursor gene (PRNP) and unexpressed processed pseudogene genetic sequence information were analysed for parentage and kinship using nuclear microsatellite loci. Results of CWD pathology and PRNP analyses have been reported by O'Rourke et al. 2004. Multiplex PCR of 35 microsatellite DNA loci was conducted using standard conditions, followed by capillary electrophoresis using an ABI 3730 DNA analyser. Multilocus genotypes were determined using the analysis software STRand (Toonen and Hughes 2001). Data analyses were conducted using the following methods: parentage relationships (Marshall et al. 1998; Slate et al. 2000); pairwise genetic relatedness indices (Hardy & Vekemans 2002; Goodnight and Qweller), and population structure (Raymond and Rousset 1995; Pritchard et al. 2000; Schneider et al. 2000). Heterozygosity levels did not indicate that extensive inbreeding had occurred among the deer tested. Results of kinship and population structure analyses, along with implications for CWD epidemiology and ecology will be discussed.

Belay E. D., R.A. Maddox, E. S. Williams, M. W. Miller, P. Gambetti, and L. B. Schonberger. 2004. Chronic Wasting Disease and Potential Transmission to Humans. Emerging Infectious Diseases (serial on the Internet). 2004 June (date cited)

Goodnight, K.F and D. C. Queller. 1999. Computer software for performing likelihood tests of pedigree relationship using genetic markers. Molecular Ecology 8:1231-1234

Hardy, O. J. and X. Vekemans (2002). SPAGeDi : a versatile computer program to analyse spatial genetic structure at the individual or population levels. Molecular Ecology Notes 2: 618-620

Marshall, T. C., J. Slate, L. E. B. Kruuk, and J. M. Pemberton. 1998. Statistical confidence for likelihood-based paternity inference in natural populations. Molecular Ecology 7(5): 639-655

O'Rourke, K. I., T. R. Spraker, L. K. Hamburg, T. E. Besser, K. A. Brayton, and D. P. Knowles. 2004. Polymorphisms in the prion precursor functional gene but not the pseudogene are associated with susceptibility to chronic wasting disease in white-tailed deer. Journal of General Virology, 85: 1339–1346

Pritchard, J. K., M. Stephens and P. Donnelly. 2000. Inference of Population Structure Using Multilocus Genotype Data. Genetics 155: 945–959

Queller D. C., K. F. Goodnight. 1989. Estimating relatedness using genetic markers. Evolution 43,258-275

Raymond M. and F. Rousset. 1995. GENEPOP (version 3.3): population genetics software for exact tests and ecumenicism. J. Heredity, 86:248-249

Schneider, S., D. Roessli and L. Excoffier. 2000. Arlequin: A Software for Population Genetics Data Analysis, Version 2.000. Genetics and Biometry Laboratory, Dept. of Anthropology, University of Geneva, Switzerland

Slate J., T. C. Marshall, and J. M. Pemberton. 2000. A retrospective assessment of the accuracy of the paternity inference program CERVUS. Molecular Ecology 9(6): 801-808

Toonen, R. J. and S. Hughes. 2001. Increased throughput for fragment analysis on an ABI 377 automated sequencer using a 100-lane RapidLoad membrane comb and STRand software. BioTechniques 31:1320-1324

Williams, E. S. and S. Young. 1980. Chronic wasting disease of captive mule deer: a spongiform encephalopathy. J Wildl Dis 16, 89–98

145) PATCH: A SPATIALLY EXPLICIT WILDLIFE POPULATION MODEL FOR ASSESSING RISKS OF PESTICIDES TO SONGBIRDS

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Wildlife populations are exposed to varying habitat structure and quality, as well as an array of human-induced environmental stressors. Predicting the consequences to a real population of one perturbation (e.g. a pesticide application) without considering other human activities and naturally changing environmental conditions is unrealistic and frequently results in inaccurate predictions. The U.S. EPA has been challenged to develop risk assessment tools that predict with reasonable accuracy long-term effects of pesticides on songbird populations. Current methods address only the fate of individual animals exposed to a single stress, viz. pesticides. The PATCH wildlife simulation model (Program to Assist in Tracking Critical Habitat; Schumaker 1998) is a spatially-explicit, individual based life history simulator that incorporates GIS representations of landscapes and provides a tool for scaling up to populations in a manner that addresses the complexities of real landscapes, and evaluates the cumulative effects of pesticide use and other environmental stressors. A 3-year study of the western bluebird (*Sialia mexicana*) is being conducted in the Willamette Valley, OR encompassing multiple habitat types (e.g., tree farms, grass seed farms, residential and natural areas) to develop information about the importance of habitat selection and movement patterns of songbirds on the realism of the PATCH model simulations. Reproductive data, such as double-brooding, and juvenile and adult survival rates also will be used in the model. Information collected about interactive effects of stressors (e.g., pesticides and nest parasites) as well as indirect effects of pesticides (e.g., reduced food supply) will aid in understanding long-term impacts of pesticide use on wildlife.

146) NON-NATIVE ANIMAL PATHOGENS AS INDICATORS OF ECOSYSTEM CONDITION

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The distribution of disease organisms is of increasing concern not only for public health, but for livestock industries as well as our natural habitats. Non-native animal pathogens can be especially problematic since naïve host populations are often more susceptible to infection, and novel surroundings may alter disease dynamics in ways that favour the spread of introduced pathogenic organisms. Here we present an ecological indicator based on the distribution of non-native animal pathogens in the United States. Using the presence of disease organisms, we demonstrate how a relatively simple measure (the percent area with zero, one or more animal pathogens) may provide a useful indication of ecosystem condition in terms of the extent and number of introduced animal pathogens. Implementation of this indicator at a national level, however, will require a coordinated effort of disease surveillance and monitoring.

147) HEAVY METAL POISONING AS A REASON FOR PRESENTATION IN WILD AUSTRALIAN PARROTS

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Heavy Metal Poisoning appears to be under-diagnosed as a cause of weakness in wild parrots presenting to wildlife carer networks throughout Australia. The birds do not always present with 'typical' signs seen in pet parrots. The presenting complaints vary from being weak and able to be caught, with predation wounds secondary to poor mobility, and fractures from vehicle trauma. It is well understood that heavy metal poisoning is common in parrots in captivity with the consumption of dags from zinc aluminium wire cages. Heavy metal Poisoning is also recognised as a cause of weakness and mortality in waterbirds that consume gun pellets from the bottom of waterways and in birds of prey that consume shot carcasses (rabbits, birds etc). It appears to be not well-recognised that free-ranging birds have access to a range of sources of heavy metal particles where consumption of a number of particles may result in clinical signs. This condition does not appear to be restricted to parrots feeding in manufacturing areas, but is also seen in rural and semi-rural settings. Sources of heavy metal are varied, and a location or source is not often found in each case. Diagnosis is made by radiography that reveals the presence of metal particles in the gizzard. Treatment of heavy metal poisoning is intensive. Recent overseas work that recommends the use of CaEDTA at 100mg/kg bid for 5 days instead of at 40mg/kg has been tried with birds in care with the view of minimising the time spent in care. Supportive care also involves the use of fluids; antibiotics, such as enrofloxacin (Baytril) at 15mg/kg bid for 5 days; B complex injections, and use of Psyllium (Metamucil) given with hand-rearing mix to promote the movement of particles from the gizzard.

148) LACK OF PASSIVE TRANSFER IN A NEONATAL RIGHT WHALE CALF (*EUBALENA GLACIALIS*)

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Only about 350 Right Whales remain in the critically endangered North Atlantic population, which spends most of the year breeding and feeding off the New England and southern Canadian coasts. Between December and March, predominantly pregnant females head south to the shallow waters off the Georgia and Florida east coast. This area is the only known calving ground for this species.

On February 3, 2004, a male, approximately 1,800kg, 478 cm, neonatal Right Whale calf was found alive on Fernandina Beach, St. Augustine, St. John's County, Florida at 7 am by a lay observer. Initial examination revealed a thin, neonatal animal that appeared to be tachypneic and hypothermic in water contaminated with feces. Treatment included removal of fecal contaminated water and attempts were made to warm the animal. Emergency medications including intramuscular injection of 100cc Dexamethasone, 40cc Vitamin E/Selenium, and 100cc calphosan calcium were administered at 2pm. Blood samples were originally taken from the dorsal fluke veins though it was noted that venipuncture quickly became more difficult. On-site analysis revealed a severe hypoglycemia. An intramuscular injection of Duopen was administered at 3:30pm and 10cc of Demerol were administered at 5:15pm. Large equipment was organised by NOAA coordinators and attempts were made to move the calf for temporary housing at Marineland, FL. The calf expired naturally during transport at 5:15pm.

No reference intervals for Right Whales exist and this is believed to be the first complete blood analysis in a Right Whale. Using other reference intervals for domestic and cetacean species some inferences about significant changes may be attempted. Complete blood count revealed a low, normal white blood cell count with a predominance of neutrophils. Upon slide review, platelets were clumped but assessed as adequate. The most significant finding was a low plasma and serum protein both on refractometry and using the biuret method of analysis. Albumin was within normal range to increased while the globulin component was markedly decreased. Biochemical analysis also revealed a markedly increased alkaline phosphatase and aspartamine aminotransferase. Markedly decreased blood glucose was documented. It is possible that creatinine, blood urea nitrogen, and creatine kinase may have been mildly to moderately increased but this cannot be proven with existing data. A plasma gel electrophoresis performed based on the decreased globulin concentration revealed an agammaglobulinemia.

Necropsy revealed a patent urachus and a constricted but patent ductus arteriosus and foramen ovale. These findings are consistent with an animal <48 hours old and likely did not contribute to the animal's death. Extramedullary hematopoiesis was noted in the sublumbar and mesenteric lymph nodes.

Mild lymphocytic and histiocytic infiltrate was noted in the lungs along with the presence of squamous cells. No infectious agents were identified with special stains (B and B, GMS). The presence of squamous cells without infectious agent and significant inflammation is considered to be a normal finding in a neonate. Marked atelectasis was noted, consistent with

a large animal stranded on the beach. It is difficult to ascertain if the atalectasis was pre or postmortem.

The stomach contained approximately one liter of dark brown fluid and no ingesta. Meconium was present in the colon. Histopathology revealed a mild to moderate eosinophilic and lymphoplasmacytic enterocolitis. Colonies of bacteria, present in the submucosa, extended as deeply as the lamina propria in the colon. Culture revealed a 95% growth of Vibrio sp.

Acknowledgements: The authors would like to acknowledge all of the National Oceanographic and Atmospheric Administration, SeaWorld, the Florida Fish and Wildlife Conservation Commission employees and volunteers and others who worked very hard to respond to and coordinate the response to this stranding event. Teri Rowles, of National Oceanographic and Atmospheric Administration, who was the onsite coordinator was instrumental in the successful scientific collection of this specimen. We would also like to acknowledge the responders and employees from the University of Florida, including Ruth Francis-Floyd, Martha Keller, and Hendrik Nollens, who helped facilitate the necropsy and sample collection.

149) EXPERIMENTAL CROSS-SPECIES TRANSMISSION OF TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES (TSEs) AT THE NATIONAL ANIMAL DISEASE CENTER (NADC), AMES, IOWA, USA: AN UPDATE

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Experimental transmission of TSE agents provides valuable information for identification of potential host ranges, and generates much needed prion-infected tissues for research. Such investigations have been conducted at NADC since 1990 and have been confined to scrapie, chronic wasting disease (CWD), and transmissible mink encephalopathy (TME). Most TSE investigations require long incubation periods and need BL-2 conditions for conducting the experiments. Initially our studies were restricted to farm livestock (cattle and sheep). However, as a result of increased demand from our stakeholders, we now also conduct research on wildlife (herbivores and carnivores).

Some of the significant findings of past studies at NADC are as follows:

- Intracerebral inoculation of sheep scrapie to cattle resulted in a neurologic disease that was distinct from bovine spongiform encephalopathy (BSE).
- Oral inoculation of cattle did not result in clinical disease during eight years of observation.
- Sheep scrapie was transmitted to elk by intracerebral route and the resulting disease could not be distinguished from CWD in elk.
- Intracerebral inoculation of CWD from mule deer resulted in transmission of prion disease to small numbers of cattle and one sheep.
- TME and scrapie agents were transmissible to raccoons via intracerebral route, whereas CWD was not.
- Intracerebral inoculation of TME to cattle resulted in a neurologic disease that could not be differentiated from BSE by the currently available diagnostic tests.

150) EFFECTS OF TEMPERATURE AND DURATION OF SAMPLE STORAGE ON HAEMATOLOGICAL CHARACTERISTICS OF WESTERN GREY KANGAROOS (*MACROPUS FULIGINOSUS*)

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Blood samples are commonly obtained during studies on wild animals to aid in the evaluation of health status. Any delay in analysis or differing storage conditions may affect sample quality and consequently the results of haematological tests. The artefactual changes that occur with storage of human blood have been well documented but effects on the blood of other animal species are less well known. The present study was undertaken to investigate the effect of storage on blood from a representative species of macropodid. Blood samples were collected from western grey kangaroos and evaluated over a 5day period using an Advia 120 haematology analyser and manual differential leukocyte counts. In agreement with the outcomes of human studies, the blood samples maintained optimal stability at 4°C. Samples stored at 36°C showed rapid degeneration of haematological values within 12 hours whereas samples stored at room temperature and 4°C remained stable for 48 hours and 72 hours respectively. Apart from an increase in lysed cells on blood smears from 72 hours, refrigerated samples remained essentially unchanged for the duration of the study. Storage at 4°C is therefore recommended for optimum maintenance of sample quality.

151) ADVERSE EFFECT OF SELECTED XENOBIOTIC ON CHOLINESTERASE ACTIVITY IN *RANA CYANOPHLYCTIS* BRAIN, LIVER AND KIDNEY

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Frogs and toads are very important to the overall ecosystem balance. The large biomass of these amphibians makes them significant prey for other animals. Currently amphibian populations are declining in a number of geographical locations throughout the world including Pakistan. In most cases, the cause or causes are unknown, but are assumed to result from man-made alterations in the environment. In this study, *Rana cyanophlyctis* was exposed to two selected xenobiotics; cypermethrin, and endosulfan in the laboratory experiments. Two different concentrations i.e. 1 % and 0.5 % of both pesticides were used, and cholinesterase activity was observed in the brain, liver and kidney of *Rana cyanophlyctis*. Under the treatment of cypermethrin it was decreased up to 45.32 and 26.44 % in the brain and 38.00 and 22.60 % in the liver and 40.25 and 38.87 % in the kidney, respectively. While under the effect of endosulfan, it was decreased up to 20.06 and 2.24 % in the brain and 9.03 and 5.56 in the liver and 17.72 and 16.77 % in the kidney, respectively. This study has shown that xenobiotics chemicals cypermethrin and endosulfan decreased the cholinesterase activity in the amphibians

152) DEVIL FACIAL TUMOUR DISEASE (DFTD) IN TASMANIAN DEVILS *(SARCOPHILUS HARRISII)* - THE GROSS, HISTOLOGICAL, ULTRASTRUCTURAL AND IMMUNOHISTOCHEMICAL CHARACTERISTICS OF THE NEOPLASM

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Tasmanian Devils (*Sarcophilus harrisii*) have been diagnosed with Devil Facial Tumour Disease (DFTD) across their island state of Tasmania, Australia. Eighty one cases of DFTD neoplasms examined showed unusual consistency in their gross, histological, ultrastructural and immunohistochemical properties across individuals from geographically different populations. They are typically multi-centric, involving the oral, head or neck regions. Histologically, the neoplasms occur in the subepithelium, forming expansile masses of round to spindloid cells encased within a pseudocapsule. They are locally aggressive and metastasise in 22% of cases. Ultrastructural characteristics were non-specific and lack features of well differentiated cells. Immunohistochemically, the DFTD neoplasms were negative for cytokeratin (48/48), smooth muscle actin (15/16), CD-3 (10/18), CD-16 (13/13), CD-57 (43/43), epithelial membrane antigen (42/42), vWF (11/11), desmin (42/47), glial fibrillary acid protein (13/13). DFT was positive for melan A (34/44), vimentin (50/50) and S-100 (39/47). The histiogenic origin of the neoplasms will be discussed.

153) RELATIONSHIP BETWEEN INTER-TIDAL VEGETATION AND RAINFALL IN ABU DHABI, THE UNITED ARAB EMIRATES

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A study of changes in inter-tidal vegetation (mangrove and saltmarsh) was undertaken in an area in eastern Abu Dhabi emirate using satellite images covering a 30 year period (1972-2002). The changes in vegetation cover were examined in relation to total and maximum rainfall in the study area prior to the capture of each image to determine if rainfall effected intertidal vegetation growth and cover (NDVI).

The study area is arid, averaging less than 120mm of rain per year, and often experiences extended drought. Dust deposition is high, and this may impede the ability of standard remote sensing techniques to detect all vegetation cover.

This study indicated that rainfall effects inter-tidal vegetation growth and cover, and that the intensity of rainfall also affects the amount of vegetation detected by the satellites. The results indicate that rare heavy rainfall events wash the vegetation of accumulated dust, and this is then interpreted by satellites as a sudden increase in total vegetation cover. It is suggested that these sudden increases in vegetation cover detected after heavy rain events are mostly attributed to the cleansing of inter-tidal foliage from dust.

The accuracy of remote sensing for determining vegetation cover in arid regions is therefore discussed in light of these results.

154) TICK PARALYSIS AND CLEFT PALATE SYNDROME IN SPECTACLED FLYING FOXES

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There is a high incidence of tick paralysis in Spectacled flying foxes (*Pteropus conspicillatus*) on the Atherton Tablelands, near Cairns in northern Australia. Since its discovery in 1990, a number of bat hospitals have treated hundreds of adults each year, and reared hundreds of resulting orphans. It is believed the bats are being infested with ticks largely while feeding on wild tobacco *(Solanum mauritianum)*. Tick paralysis (*Ixodes holocyclus)* is lethal for the bats unless treated with tick anti-toxin and good nursing care. Tick season is October to December each year, the same months the bats give birth. A daily presence in the colonies at this time has resulted some years in the discovery of a high number of abandoned young with a cleft palate syndrome. This is characterised by a severe midline defect often extending into the throat, missing and rudimentary claws, other craniofacial anomalies and wiry facial hair. Functionally many of them have trouble hanging.

155) FIELD SURVEY: SOUTH AFRICAN CLAWED FROGS (*XENOPUS LAEVIS*) IN GOLDEN GATE PARK, SAN FRANCISCO, CA

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South African Clawed Frogs, native to the Ivory Coast of South Africa, are an invasive, highly adaptable, long-living (15-25 years), carnivorous, fully aquatic amphibian species that has a high affinity for disturbed or artificial habitats. A feral population of *Xenopus laevis* is currently established in a small (< 0.4 hectares), easily wadeable (< 1.2m deep), man-made pond in Golden Gate Park, San Francisco, CA. The source of the *Xenopus* introduced into the pond is unknown. However, the Park is an area of refuge for California red-legged frogs and other endangered amphibians within the San Francisco urban expanse. The objectives of this investigation were to determine the relative abundance, reproductive effort, and reproductive efficiency of *Xenopus*, to determine their diet by examining stomach contents and thus identify other species that may be affected by their presence, and to determine the parasite load and diseases they carry. Between June 2004 and February 2005, 360 *Xenopus* from this population were examined. Relative abundance was estimated to be ~10,000 frogs. The population appeared to be free of common aquatic amphibian bacterial and fungal diseases. Neither *Mycobacterium sp.* nor chytrid fungus, *Batrachochytrium dendrobatidis*, was detected in any of the specimens collected. The stomach contents of *Xenopus* collected in the summer months included predominantly mosquito fish and juvenile *Xenopus*. January and February, when the pond was essentially devoid of all other fish or small aquatic species, *Xenopus laevis* consumed plant matter, zooplankton (>0.1cm), and insect larvae (~0.5cm-1.5cm). *Xenopus* in the pond were also relatively free of parasites, the only identified to date being the intestinally encysted *Balantinum spp*. and unspeciated nematodes, encysted in serosal surfaces. Water quality tests on pond water showed levels of copper at 0.20 mg/L during the summer months, a level that exceeds what is reported to be compatible with successful *Xenopus* egg development. The pH and water temperature ranged from 5.8-7.6 and 40 C to 65 C, in the winter and summer months, respectively. The biology and dynamics of this feral population of invasive *Xenopus laevis* are currently under study with the aim of using the data to develop an effective control/eradication plan.

156) SEROSURVEY FOR ANTIBODIES TO WEST NILE VIRUS IN WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*) FROM IOWA (1999-2003)

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White-tailed deer sera were collected in Iowa during the winter months of 1999-2003; two years before and after West Nile Virus (WNV) was first reported in Iowa (2001), and were analysed for antibodies to WNV. Samples from 1999-2001 were antibody negative by blocking enzyme-linked immunosorbent assay (ELISA) and plaque reduction neutralisation test (PRNT90). There were no differences between the prevalence estimates derived from blocking ELISA (2002, 12.7%; 2003, 11.3%) and the WNV PRNT90 (2002, 13.8%; 2003, 15.6%) assays. All samples were negative for antibodies against Saint Louis encephalitis virus as determined by PRNT. Antibodies to flaviviruses were detected by direct ELISA prior to the first WNV cases reported in Iowa (1999-2001) with prevalence ranging from 2.2-3.0%, suggesting the circulation of an additional undescribed flavivirus prior to the introduction of WNV into the area. Flavivirus prevalence as determined by direct ELISA increased in 2002 and 2003 (22.9% and 32.3%, respectively). The increase in prevalence exceeded estimates of WNV prevalence, suggesting that conditions favouring general flavivirus transmission (including WNV) existed during the 2002-2003 epizootic. These data indicate that serologic analysis of deer sera collected from hunter harvests may prove useful for surveillance and evidence of local transmission of WNV and other pathogens.

157) INFECTIVITY OF *CRYPTOSPORIDIUM* OOCYSTS FROM *MACROPUS GIGANTEUS* (EASTERN GREY KANGAROO) IN HUMAN GUT EPITHELIAL CELLS

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Cryptosporidium is a globally important cause of water-borne diarrhoea in humans. In immunocompetent individuals *Cryptosporidium* causes severe, self-limiting infection, but in immuncompromised humans it can be life-threatening. *Cryptosporidium* belongs to the protozoan phylum Apicomplexa. It is an obligate intracellular parasite that forms oocysts that are shed in the faeces of infected hosts and are highly resistant to normal water treatment processes including chlorine treatment.

Cryptosporidium have been identified in more than 170 vertebrate species. Zoonotic sources (eg *C. parvum* from cattle) have been identified as being infectious to humans. *Macropus giganteus* (eastern grey kangaroo) is the largest biomass of herbivores in the Sydney water catchment area. *Cryptosporidium* have been identified in the faeces of eastern grey kangaroo, although at low levels. However, it is not known if this poses any health-threat to humans. We are using a cell culture assay to determine if *Cryptosporidium* genotypes from *Macropus giganteus* can infect human gut epithelial cells as a first step in determining if they could be infectious to humans.

158) PRESENCE OF ENDOVIRAL (EV) GENES OF AVIAN LEUKOSIS AND SARCOMA VIRUSES IN SOME WILD SPECIES OF BIRDS

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The various species included in this study were Silver Pheasant *(Lophura nycthemera*), Green Pheasant (*Phasianus versico*lor), PeaFowl (*Pavo cristatus*), Emu (*Dromaius novae*), Hawk (*Accipiter badius*), Horn Bill (*Tockus birostris*), Sparrows (*Passer domesticus*), and from migratory Siberian Crane (*Grus leucogeramus*). The whole DNA of these species was extracted (Sambrook, 1989) and was amplified with the following primer set designed from the variable region of eV genes of sub group E viruses.

Forward Primer: GGTATCCCGTCCCCTATT-18 mer
Reverse Primer: CTATCCGCTGTCACCACCGTAAAC-24 mer

The primers were able to amplify a part of eV genes giving an expected product of size 398 bp. Restriction endonuclease (RE) analysis of the product was attempted with four different enzymes viz . StyI, Dde I, PvuII, and SspI. RE products were similar in all the amplified eV sequences of different species showing that there is no or little variation in these.

159) DETECTION OF ANTI- *TOXOPLASMA GONDII* ANTIBODIES IN CAPTIVE AND WILD TOOTHED WHALES BY SEROLOGICAL METHODS FOR DIAGNOSIS OF *TOXOPLASMA GONDII* INFECTION

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Antibodies against *T. gondii* were examined by both latex agglutination test (LAT) and indirect hemagglutination test (IHAT) for 77 serum or plasma samples obtained from 59 individuals of 6 species, including 2 hybrids belonging to Odontceti. Antibody titres greater than 1:64 in LAT and greater than 1:640 in IHAT, were indicative of the presence of *T. gondii.* In 7 samples that showed a positive reaction by IHAT, *T. gondii* titres were examined for each immunoglobulin fraction separated by sucrose gradient centrifugation. The antibody peaks in each fraction were divided into 3 types, thought to be a reaction of IgM (Type 1), IgG (Type 2), and IgM with IgG (Type 3). Serum samples from 58 bottle-nosed dolphins (*Tursiops truncatus*) were also tested by LAT, enzyme-linked immunosorbent assay (ELISA) and immunoblotting. In 25 out of the 58 samples, no antibody activity was detected by LAT and ELISA. In 8 samples, the anti-*T. gondii* IgG antibody was detected by both LAT and ELISA with titres of greater than 1:64 and 1:160. In 11 samples, the antibody was detected by LAT and ELISA with titres of 1:16~64 and 1:20~160. In 14 samples, the antibody was detected by either LAT or ELISA with titres of 1: 8~16 or 1:10~20. In immunoblotting, the 8 serum samples producing higher titres showed specific antibody IgG reactivity to several antigens on the *T. gondii* lane, but not on the *Neospora caninum* lane. No specific bands were noted on the lanes for either parasite in the 25 serum samples for which no antibody activity was detected. In 9 of the 11, and 3 of the 14 serum samples producing lower titres, reactivity to antigens having molecular masses of 92, 70, 50, 39 and 24 kDa was observed on both *T. gondii* and *N. caninum* lanes. These results indicate that the IgG antibody in serum samples from dolphins producing higher titres show specific reactivity to *T. gondii* antigens.

160) SEROLOGIC EVIDENCE OF DISTEMPER, BRUCELLOSIS, AND TOXOPLASMOSIS CIRCULATING IN STELLER SEAL LIONS IN THE NORTH WESTERN PACIFIC OCEAN

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The present study reports preliminary serologic evidence of some significant marine mammal pathogens circulating in a population of Steller sea lions (*Eumetopias jubatus*) in the North Western Pacific Ocean. Morbilliviruses are known to cause severe epizootics in a number of phocid species while brucellosis and toxoplasmosis are known to have a more subtle effect by affecting reproductive function in infected animals. We tested 169 serum samples from sea lion pups collected in 2001-2002 from three pupping sites in the Sea of Okhotsk (Kuril Strait n= 96, Commander Island n=20, and Okhotsk Sea Islands n= 53). The samples were tested for the presence of binding antibodies to all three pathogens using enzyme linked immunoassay (ELISA) technology. Overall, 13% of the pups had evidence of exposure to toxoplasmosis and more positive animals (15.4%) were detected from the Kuril Strait while fewer positive animals (5%) were detected from Commander Island. Antibodies to brucellosis were detected in 11.2% of the pups and more positive reactors (25%) were detected from the Commander Island but the prevalence dropped to 1.9% in samples obtained from the Okhotsk Sea Islands. Morbillivirus antibodies were detected in 23.7% of the samples and more reactors (30.2%) were detected in animals from the Kuril Strait while fewer positives (15%) were detected in sea lions from Commander Island. Both toxoplasmosis and brucellosis are capable of transmission from mothers to their offspring *in utero* and maternal antibody transmission from a previous infection may also be responsible for some of the positive results in all three tests. Results from all three analyses support the hypothesis that distemper, brucellosis and toxoplasmosis are enzootic in the Steller sea lions of the North Western Pacific.

161) CLEFT PALATE IN A NEONATAL VIRUNGA MOUNTAIN GORILLA (*GORILLA BERINGEI BERINGEI*)

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Infant mortality in mountain gorillas is approximately 11% by 1 year of age (Gerald 1995). While infanticide accounts for more than 25% of deaths, other causes include infectious diseases (primarily respiratory) and aspiration. Since infant survival impacts population dynamics of this slowly-reproducing species, and since infectious diseases are recognised as a significant threat to mountain gorilla survival, elucidating non-traumatic causes of death is important. Historically, gross and histopathologic post-mortem examinations have been hampered by autolysis, as mothers frequently carry their dead infants for one to many days before abandoning them. In an effort to help clarify causes of infant mortality, the Mountain Gorilla Veterinary Project has begun intervening to recover deceased infants for more informative examination. The full-term infant of a clinically normal primiparous mother died at 6 days of age with no premonitory signs. The mother was immobilised and the infant's body was recovered for examination. Gross examination showed a large midline cleft involving both soft and hard palate to the level of the dental ridge, and evidence of inanition and possible aspiration pneumonia. The defect was compatible with a diagnosis of bilateral complete cleft palate. Oral-facial clefts have been reported in lemurs, New and Old World monkeys, and one captive western lowland gorilla (Seibert et al 1998). Causes of cleft palates include a variety of genetic, dietary, and teratogenic factors, however the aetiology is often undetermined. A possible case of cleft palate was found in a neonatal Virunga mountain gorilla in 1994, but severe autolysis precluded confirmation. Interestingly, the mother of the 1994 case is the full sister of the mother of the 2004 case. Also, the 2004 infant was likely the result of a consanguineous mating, as the co-dominant silverbacks of the group are the female's father and brother. Additional radiographic studies of the 2004 infant, as well as genetic analysis, are planned to help further characterise this case.

Gerald CN. 1995. Demography of the Virunga mountain gorilla (*Gorilla gorilla beringei*), MS thesis, Princeton University, 81p

Seibert JR, Williams B. Collins D, Winkler LA, Swindler DR. 1998. Spontaneous cleft palate in a newborn gorilla (*Gorilla gorilla gorilla*). *Cleft Palate-Craniofacial Journal* 35(5):436-441

162) MANGE CAUSED BY *PANGORILLALGES GORILLAE* (FAIN 1962) IN THREE VIRUNGA MOUNTAIN GORILLAS (*GORILLA BERINGEI BERINGEI*)

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Clinically significant sarcoptic mange has been previously reported in the mountain gorillas (*Gorilla beringei* undecided) of Bwindi Impenetrable National Park (BINP), Uganda (Graczyk et al 2001, Kalema-Zikusoka et al 2002). Numerous gorillas were affected, and one infant died from the disease, while the remainder recovered after treatment with ivermectin or were lost to follow up. Due to the observed morbidity and mortality, and the difficulty experienced in treating all affected animals at BINP, the index of suspicion remains high whenever clinical signs consistent with sarcoptic mange are observed, such as hair loss, dry, crusty skin, and pruritus. In December 2002, a young adult male (blackback) Virunga mountain gorilla (*Gorilla beringei beringei*) from a tourist-habituated group in Parc National des Volcans (PNV), Rwanda, was observed with hair loss on his flanks, shoulders and thighs, mild thickening of skin, and pruritus. He was immobilised for examination and treatment (ivermectin 200µg/kg), during which mites were visible on his flanks and shoulders. Specimens collected were identified as *Pangorillalges gorillae* (Fain 1962), and a biopsy showed moderate lymphohistiocytic dermatitis. No other significant abnormalities were found and he responded well to treatment, but has since suffered from waxing and waning hairloss and pruritus.

In January 2004, a 3-month-old infant from a habituated group in Parc National des Virunga (PNVi), Democratic Republic of Congo was reported with hair loss. Upon observation, the infant was depressed, appeared small for her age, and had significant hair loss and dry, flaky skin. Mother and infant were immobilised for examination and treatment, and skin lesions were evident on both. They consisted of round, dry, flaky areas, approximately 0.5 cm diameter; some areas were raised and some were flat. The lesions on the mother were confined to the left side of her chest, where she was observed to hold the infant most of the time. The lesions on the infant covered her entire body, and resulted in significant hair loss. The remaining hair coat was rough and dry, skin was thickened, the infant was thin for her age and was listless and depressed. Skin scrapings and biopsies from both mother and infant showed *Pangorillalges gorillae*, and moderate acanthosis and basketweave orthokeratosis, respectively. Both were treated with ivermectin as above, and responded well to treatment.

Pangorillalges gorillae was first described from western lowland gorillas in 1957 (previously called *Psoroptoides gorillae*), and was later mentioned as an incidental finding in an 8-month old Virunga mountain gorilla that was the victim of infanticide. There are no previous reports of clinical disease associated with this mite in mountain gorillas. Anecdotally, all 3 gorillas clinically affected to date may have suffered or continue to suffer underlying abnormalities. The blackback from the 2002 case is considered to be small for his age and late in developing and is missing a hand from a snare injury. The mother from the 2004 case is of unknown age but is thought to be at least 30 years old. Both her previous infants have died before weaning, at least 1 from infectious disease, which in conjunction with some suspicious laboratory results from the mother suggest the possibility of underlying maternal disease. No other gorillas in either the PNV or PNVi group have shown similar clinical signs, which also suggests the virulence of *Pangorillalges gorillae* (Psoroptidae) is significantly less than that

of *Sarcoptes scabiei* (Sarcoptidae). We hypothesise that *P. gorillae* is an incidental finding, and that special circumstances may be required for this mite to cause clinical disease.

Fain A. 1962. *Pangorillalges pani* g.n., sp.n. Acarien psorique du Chimpanzé (Psoralgidae: Sarcoptiformes). *Revue de zoologie et de botanique africaines.* 66:283-290

Graczyk TK, Mudakikwa AB, Cranfield MR, Eilenberger U. 2001. Hyperkeratotic mange caused by Sarcotes scabei (Acariformes: Sarcoptidae) in juvenile human-habituated mountain gorillas (*Gorilla gorilla beringei*). *Parasitology Research* 87:1024-1028

Kalema-Zikusoka G, Kock RA, Macfie EJ. 2002. Scabies in free-ranging mountain gorillas (Gorilla beringei beringei) in Bwindi Impenetrable National Park, Uganda. *Veterinary Record* 150(1):12-15

163) THE USE OF A TADPOLE SURVEY METHOD COMBINED WITH A CHYTRID-SPECIFIC POLYMERASE CHAIN REACTION TEST TO DECTECT CHYTRIDIOMYCOSIS INFECTION IN FREE-RANGING TASMANIAN AMPHIBIAN POPULATIONS

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Tasmania is one region in Australia that had not reported chytridiomycosis in its local frog populations despite declines in the range and abundance of species of frog considered likely to be susceptible to this serious fungal infection. The Central North Field Naturalists received funding from the *Natural Heritage Trust - Invasive Species Program* with particular focus on developing a field survey method and trialling a Polymerase Chain Reaction test for the presence of the chytrid fungus in tadpoles at frog habitats.

The survey confirmed the presence of the chytrid fungus, *Batrachochytrium dendrobatidis* in a number of frog habitats close to major cities and towns. It is believed that chytrid infection established in Tasmania through the release of infected frogs recovered from imported fresh produce (like bananas) at these peri-urban wetlands. The detection of the chytrid infection in remote wetlands at high altitude on the Tasmanian Central Plateau is of particular concern. Declines in the range and abundance of the endemic Tasmanian Tree Frog *(Litoria burrowsae)* and the Green & Gold Frog *(L. raniformis)* could be directly linked to the establishment and spread of chytridiomycosis in Tasmania. The fungal pathogen may have been in Tasmania for up to two decades with the local transfer of frogs & tadpoles by frog seekers and recreational fishers extending its range.

The presence of putative chytrid infections can be assessed by looking for depigmentation and asymmetry in jaw sheaths and tooth rows of tadpoles using a hand lens. Field surveys of up to 60 tadpoles in combination with the Taqman chytrid PCR test were useful diagnostic tools for the detection of chytridiomycosis at targeted amphibian habitats. Combined with appropriate hygiene protocols to prevent anthropogenic spread of chytrid infection, this field survey method with the PCR test as back-up has application in baseline and follow up monitoring surveys for chytrid infection in frog populations.

164) CLINICAL COCCIDIOSIS DUE TO *CARYOSPORA* (APICOMPLEXA) IN TAWNY FROGMOUTHS, *PODARGUS STRIGOIDES* (CAPRIMULGIFORMES) IN AUSTRALIA

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This is a preliminary report of a new coccidian parasite species causing clinical disease in two subadult wild tawny frogmouths from two suburbs in Sydney, Australia. Both birds appeared thin and stunted and were presented to the Taronga Zoo Wildlife Clinic with a history of weakness and regurgitation. The birds were treated supportively but died several days after arrival. At necropsy, both birds had depleted fat deposits and markedly distended caecae containing brown fluid contents. A direct smear of the gut contents revealed numerous coccidial oocysts which were sporulated in 2% potassium dichromate solution and identified as belonging to the genus *Caryospora.* The parasite species has not previously been described and is unique in morphology. Subspherical oocysts (28-34 x 28-32 μm) lacked a micropyle, residuum and polar granules but contained a single ovoid sporocyst (19-24 x 18-23 μm) lacking a Stieda body but containing 8 sporozoites and a granular sporocyst residuum.

Numerous endogenous developmental stages (schizonts and gamonts) were evident in histological sections of the small (and to a lesser extent large) intestines together with extensive mucosal degeneration and necrosis. The clinical course and mortality was attributed to the extensive intestinal coccidiosis (caryosporosis) in these birds. Similar clinical findings were noted in another three tawny frogmouths that died from one of the suburbs, but no necropsy material was available.

Caryospora spp. are coccidian parasites infecting reptiles, birds and rodents[1]. They have monoxenous and heteroxenous life-cycles, with some species exhibiting cyclic transmission between predator and prey host species. A total of 19 *Caryospora* spp. have been described in birds[2]; 13 species in Falconiformes, 2 in Strigiformes, 2 in Charadriiformes and 2 in Passeriformes. This is the first report of infections in birds belonging to the order Caprimulgiformes, and only the second report in Australian avifauna, an infection having previously been found in a cuckoo-shrike (Passeriformes)[3].

Most infections appear to be asymptomatic but several *Caryospora* spp. have been associated with acute/subacute disease in raptors in Europe and the Americas, with clinical signs including regurgitation, anorexia, weight loss, weakness, lethargy, depression, haemorrhagic faeces, diarrhoea and death[4,5,6]. Clinical infections have also been reported widely in squamata and chelonids[7], including within Australia. The implications of caryosporosis in wild tawny frogmouths in Australia are currently uncertain and will require further study and surveillance.

1. Upton SJ, Current WL, Barnard SM. A review of the genus *Caryospora* Leger, 1904 (Apicomplexa: Eimeriidae). *Syst Parasitol* 1986, 8:3-21

2. Upton SJ, Campbell TW, Weigel M, McKown RD. The Eimeriidae (Apicomplexa) of raptors: review of the literature and description of new species of the genera *Caryospora* and *Eimeria. Can J Zool* 1990, 68:1256-65

3. O'Donogue PJ. Protozoan parasites of wildlife in south-east Queensland. Tribe A (ed) Proc Aust Assoc Vet Conserv Biol, Brisbane 1997, pp119-136

4. Volf J, Koudela B, Modry D. Two new species of *Caryospora* Leger, 1904 (Apicomplexa, Eimeriidae) from accipitrid raptors. *Syst Parasitol* 2000, 46:23-7

5. Papazahariadou MG, Georgiades GK, Komnenou AT, Ganoti M. *Caryospora* species in a snowy owl (*Nyctea scandiaca*). *Vet Rec* 2001, 148:54-5

6. Forbes NA, Fox MT. Field trial of a *Caryospora* species vaccine for controlling clinical coccidiosis in falcons. *Vet Rec* 2005, 156:134-8

7. Gordon AN, Kelly WR, Lester RJ. Epizootic mortality of free-living green turtles, *Chelonia mydas*, due to coccidiosis. *J Wildl Dis* 1993, 29:490-4

Acknowledgements: The authors thank Jane Hall and Kaye Humphreys for their technical support, Dr. Larry Vogelnest and the Taronga Zoo veterinary clinic and nursing staff for clinical assistance, and Libby Hall for her care of wildlife.

165) HAEMATOLOGY AND SERUM BIOCHEMISTRY OF THREE NATIVE AUSTRALIAN DESERT MURIDS

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Approximately 25% of Australian mammals are murids (rats and mice) with much research to date having been conducted on our unique marsupials. However, like the marsupials, Australia's murid population has also been affected by changes in the environment such as the introduction of predators and domestic stock, changes in fire regimes and human development.

This study was aimed to measure haematological and serum biochemistry parameters from three species of Australian native murids from three different genera that live in the central arid region of Australia. The Spinifex Hopping-mouse (*Notomys alexis*) is a common desert inhabitant of inland Australia. The Plains Rat (*Pseudomys australis*) is listed as vulnerable nationally whilst the Central Rock-rat (*Zyzomys pedunculatus*) is listed as endangered nationally. The study aims to determine the blood parameters for captive murids and will allow a comparison to be made to those in the wild population and is part of a larger project looking at the health of the wild murid populations in the central desert region.

Captive animals currently held in the Alice Springs Desert Park were sampled and blood parameters determined. Most blood parameters were similar to those of other murids located worldwide, however some parameters differed. The parameters that differed may be due to the harsh desert conditions that these animals live in or may be unique to Australian native murids and will be discussed.

166) INCIDENCE OF CRYPTOCOCCAL DISEASE AT PERTH ZOO – AN EPIDEMIOLOGICAL INVESTIGATION

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Cryptococcosis is an important disease of humans and animals worldwide. The disease is most commonly caused by two species of the genus *Cryptococcus*, C. *neoformans* (consisting of two varieties C. *neoformans* var. *grubii* and C. *neoformans* var. *neoformans*) and *C. gattii (*formerly *C. neoformans* var. *gattii* or *C. bacillisporus),* although other species have been known to cause disease on rare occasions.

In Australia, C. *neoformans* var. *grubii* (serotype A) is the most common isolate from humans and animals and is strongly associated with disease in immunocompromised patients. C. n. var. *grubii* is commonly associated with bird (usually pigeon) guano, which provides a nitrogen rich environment that favours cryptococcal growth. C. *gattii* (serotype B) is closely associated with several species of *Eucalyptus* trees. In contrast to C. n. var *grubii*, disease caused by C. *gattii* occurs almost exclusively in immunocompetent hosts. Koalas (*Phascolarctos cinereus*) have been shown to amplify the number of cryptococci in certain environments, and may or may not succumb to clinical disease in these situations. Both C. n. var. *grubii* and *C. gattii* have been isolated from the nasal mucosa of koalas, however all clinical infections recorded in koalas have been attributable to C. gattii.

Perth Zoo has housed koalas of both the Victorian and Queensland subspecies for over 20 years, and currently houses three Queensland koalas that have been in the collection since October 2001. The koalas are fed freshly cut eucalyptus branches on a daily basis, from trees grown on off-site plantations. These branches are subsequently used in enclosures throughout the zoo to provide enrichment and shelter for other animals. A number of the species fed extensively to the Perth Zoo koalas include species with a well-documented association with C. *gattii*, including River Redgum *(Eucalyptus camaldulensis)*, Forest redgum (E. *tereticornis)*, and Flooded Gum (E. *rudis* and E. *grandis)*.

Since October 2004, cryptococcal disease caused by C. *gattii* has been diagnosed in three individuals at Perth Zoo: an Australian King-parrot (*Alisterus scapulatus*), a Long-nosed Potoroo (*Potorus tridactylus*) and a Greater Stick-nest Rat (*Leporillus conditor*).

This paper describes the investigation into the role of the koala browse as a potential reservoir for C.*gattii*. The potential link between recycling of koala browse within the zoo and the incidence of cryptococcosis in other species is also examined.

167) SEQUENCING OF THE HEPATITIS B VIRUS IN A SILVERY GIBBON (*HYLOBATES MOLOCH*) AT PERTH ZOO

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Hepatitis B virus (HBV) infections involving strains that are distinct from human HBV genotypes, have been identified in gibbons (GiHBV), orangutans (OuHV), chimpanzees (ChHBV), gorillas (GoHBV) and woolly monkeys (WMHBV). Recent studies have demonstrated widespread infection of wild primate populations with these species-specific HBV infections.

The silvery gibbon (*Hylobates moloch*) is a critically endangered primate, with less than 2,500 animals remaining in the wild in the rainforests of Java, Indonesia. Populations are under threat due to habitat destruction and fragmentation, in addition to poaching and disease. The recent tsunami disaster may further jeopardise these fragile populations as the thousands of displaced Indonesians move further inland and utilise the valuable forest resources which are home to the gibbons.

There are currently no published sequences of the entire HBV viral genome from a silvery gibbon. Analysis of partial viral sequences obtained from silvery gibbons suggests that they carry a gibbon-specific variant, most likely GiHBV. Sequence analysis involving the entire genome is required to determine the HBV genotype, and therefore is necessary to confirm that the virus found in silvery gibbons is a nonhuman primate-specific strain and not of human origin.

This paper describes the successful sequencing of the entire HBV genome obtained from Perth Zoo silvery gibbon *Hecla*. Sequencing confirms that *Hecla* is infected with GiHV, an indigenous strain of HBV previously identified in a number of gibbon species, but not previously confirmed in the silvery gibbon. *Hecla*'s strain of HBV was shown to be distinct from all previously sequenced viruses, and more closely related to other nonhuman primate strains of HBV than to any of the human strains of HBV.

This finding confirms that silvery gibbons can be infected with a nonhuman primate-specific strain of hepatitis B. These findings may have implications for the preventative veterinary management of silvery gibbons in captivity, including those for release into the wild.

168) CYTOGENETIC SUPPORT OF THE ALLOGRAFT THEORY OF TRANSMISSION OF DEVIL FACIAL TUMOUR DISEASE (DFTD) IN TASMANIAN DEVILS *(SARCOPHILUS HARRISII)*

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Like other dasyurids the constitutional karyotype of the Tasmanian devil is 14XX in the female and 14XY in the male, the Y chromosome is minute. Tasmanian devils differ from the other dasyurids in having an apparent peri-centric inversion of chromosome 5. The G banded chromosomes of 51 animals were studied. Of these, 11 animals (7 males, 4 females) had neoplasms and the G banded chromosomes of these animals were studied. They were complex and apparently identical in 9 of the animals. In a further two animals the chromosomal aberrations appeared to have transformed from the complex rearrangement seen in the other 9 animals. Both of the sex chromosomes were missing in the cancer cells of all animals. It was found that the cancers generally had a mixture of normal cells, an aneusomic diploid line plus an aneusomic tetraploid line that was the diploid line doubled.

Because of the complex nature of the chromosome rearrangements found and the consistency of these rearrangements it is hypothesised that the means of transmission of the cancer between animals is implantation of the cells from the neoplasm itself. This hypothesis is supported by the finding of one animal with a constitutional anomaly that was not present in his cancer cell tine.

169) SEROLOGICAL EVIDENCE OF EXPOSURE OF WILD MAMMALS TO FLAVIVIRUSES IN THE CENTRAL AND EASTERN UNITED STATES

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Serosurveys were conducted to obtain flavivirus and West Nile virus (WNV) seroprevalence data from mammals. Sera from 513 small and medium-sized mammals collected during late summer and fall 2003 from Colorado, Louisiana, New York, Ohio, and Pennsylvania were screened for flavivirus-specific antibodies. Sera samples containing antibody to flaviviruses were screened for WNV-specific antibodies by epitope-blocking enzyme-linked immunosorbent assays and confirmed with plaque reduction neutralisation tests. Prevalence of WNV antibodies among study sites ranged from 0 – 42.8% among the mammal communities sampled. High prevalence rates for WNV were noted among raccoons (100%, {with a very small sample size, n=2}), Virginia opossums (50.0%), fox squirrels (49.1%), and eastern grey squirrels (48.3%). The high WNV antibody prevalence noted for tree squirrels, the peri-domestic tendencies of several of these species, and their ease of observation could make these species useful sentinels for monitoring WNV activity within urban communities. Future work should assess the duration and levels of viremia in select mammals and their reservoir potential for WNV.

170) MANAGEMENT OF EPISTAXIS IN A TIGER

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Epistaxis means bleeding of fresh blood from the nasal septum. A rare case of Epitaxis in a male tiger aged approx. 16 years is described here, which was encountered during early summer due to sudden rise in ambient temperature at Gwalior Zoo, MP (India). Approximately 10-15 ml of bleeding was observed from right nostril, and the probable site was the anterior portion of nasal passage.

The animal was symptomatically treated with Injection Chromostat intramuscularly along with supportive treatment, complete rest in the cool housing.

On 12th day post treatment epistaxis was again seen from the nostrils and again controlled by giving Inj. Chromostat i/m. Though the bleeding stopped but few drops were noticed 2-3 times in a day. For detailed investigation of nasal septum and to know the cause of epistaxis, the animal was sedated by giving xylazine and ketamine. With the help of ENT specialist, thorough examination of the anterior nasal passage was conducted using rhinoscope. The septal mucosa and both sides of turbinates were found normal and there was no pathology involved.

Oral medication consisted of Vit. B complex, Vit. C, Vit. D, Vit. E, and calcium was given along with meal for fifteen days.

Looking to the circumstance, especially the age and the season, it was believed that epistaxis was due mucosal injury by dry climate and high ambient temperature. Temperature inside the tiger housing was cool down with air coolers along with circulation of cold fresh air. The tiger showed marked improvement and no signs of epistaxis were recorded thereafter.

171) THE IMPACT OF PHYTOESTROGENIC INFERTILITY ON THE EUROPEAN HARE (*LEPUS EUROPAEUS*)

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European populations of the European hare appear to be in decline, and Australian populations are remaining at low densities. Populations can be sensitive to maintenance of the breeding stock, which is influenced by infertility in the females. In our study, 245 adult female hares from three Australian populations were dissected between 1996 and 1999, and their reproductive systems examined for abnormalities.

Abnormalities were found in 51 hares. Cystic endometrial hyperplasia was relatively common, as was hydrosalpinx. Extra-uterine foetuses, neoplasms, pseudopregnancies and resorptions were also found. Multiple abnormalities not including resorption were found in 16 individuals. However, although pseudopregnancies and resorptions were found in young adults (< 12 months) as well as older hares, conditions inconsistent with fertility were almost always in older hares, with a prevalence at one site of 46.2%. Only hares with access to known sources of oestrogens exhibited pathological conditions, but sympatric European rabbits (*Oryctolagus cuniculus*) did not, which is consistent with the known difference in responses between the corpora lutea of the two species to exposure to exogenous oestrogen.

Cystic endometrial hyperplasia was first reported in southern Australian sheep exposed to phyto-oestrogens, and each of the other abnormalities has been associated with hyper-oestrogenism in another species. We suggest, therefore, that phyto-oestrogenic infertility occurs in Australian hare populations exposed to leguminous crops and pastures. Infertility at such a high prevalence would compound and extend the impact of years of low juvenile survival on recruitment. We also suggest that the condition is widespread, because seven studies of hares (in Europe, North America, New Zealand and Australia) have recorded extra-uterine foetuses, similar uterine or tubal abnormalities have been recorded in hares in Austria, France and Germany, and a high incidence of ovarian tumours has been recorded in New Zealand.

172) AVIAN MALARIA IN NEW ZEALAND: HAWAII ALL OVER AGAIN?

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Avian malaria has been responsible for the most dramatic loss of avifauna seen anywhere in the world. After its introduction, this protozoan pathogen vectored by mosquitoes played a key role in the extinction of much of Hawaii's endemic bird species. It is thought, however, that it was the subsequent introduction of the exotic mosquito *Culex quinquefasciatus* that actually enabled the introduced pathogen to reach epizootic proportions. Over the past three decades this same invasive mosquito has been extending its distribution south through New Zealand from its original sites of introduction in Northland and Auckland. Since avian malaria has been found infecting birds in New Zealand, this raises the alarming possibility that, as climate change continues to alter vector distributions, much of the native avifauna may suffer the same fate as that seen in Hawaii. Recent outbreaks of avian malaria among captive collections, causing over 60% mortality, attest to this possibility. Here I present results from the first survey for malarial parasites in wild bird populations in New Zealand for more than fifty years, relating patterns observed to the spreading distributions of native and introduced mosquitoes.

173) USE OF TRICLABENDAZOLE/LEVAMISOLE MEDICATED FEED PELLETS IN THE CONTROL OF NATURALLY INDUCED LARGE AMERICAN LIVER FLUKE (*FASCIOLOIDES MAGNA*) INFECTION IN FREE-RANGING RED DEER (*CERVUS ELAPHUS ELAPHUS*) IN CROATIA

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In the early years of 1990's, the Red deer (*Cervus elaphus elaphus*) population occupying a relatively small area of the north-eastern region of Croatia, Europe, enclosed within the Danube basin and sequestered by the Drava and Danube river valleys and bordering Hungary and Serbia, counted more than 10,000 animals. During that time there were no records of liver lesions suggestive of fascioloidosis. Nevertheless, when the counting of the animals was resumed during 1997, the population was estimated to be not more than 4,000. In the following years it was observed that deer herds were in generally poor condition. Many animals were emaciated; there was an increased mortality rate and poor fawn progression, along with a lowering antler trophy value. Peculiar liver lesions were observed and specimens of unusually large liver flukes were collected in harvested animals. The incriminated trematode was soon determined to be the Large American Liver Fluke (*Fascioloides magna*). An extremely high percentage (88%) of the examined deer livers exhibited pathomorphological lesions compatible with F. magna infection and the diagnosis was confirmed by observing different number of adult parasites as well as their positive identification by means of parasitological methods.

In grading the severity of the liver involvement among positive animals it was observed that more than half the animals exhibited very seriously compromised livers, while for the roughly 1/3 of the involved animals recuperation was thought to be questionable even after the anthelmintic treatment.

The primary aim of the conducted program was not meant to evaluate the already well recognised efficacy of triclabendazole in treating F. magna infection in deer nor was it to try to determine the optimal dose of triclabendazole but to start a treatment and control project of fascioloidosis of the free ranging deer population inhabiting the Croatian part of the Danube river basin which by its hydro-terrestrial conditions offers an ideal environmental niche.

Although the imidazothiazole component of levamisole is known not to have fascioloidicidal effect, the two-drug composition was used intentionally in order to be able to use levamisole as a specific nematode anthelmintic drug in the treatment and control of the nematode parasite population. Especially lung worms which are often to be found. An ideal combination was presented in the form of an oral broad-spectrum anthelmintic containing 12% w/v triclabendazole and 7.5% w/v levamisole hydrochloride (Combinex ® Cattle, Novartis). No specific group of animals was chosen but the population involved, counted around 3,500 individuals. Since the animals were free-ranging the duration of infection could not have been precisely established but it varied from acute, larval infections to chronic as well as superinfections.

Triclabendazole/levamisole oral broadspectrum anthelmintic solution was offered via medicated pellets (Ø 8mm). The solution was administered by way of cold-spray-on technique in order to preserve the parasiticidic effect of the thermally sensitive drug. Special blackberry flavored deer feed was formulated in order to promote the organoleptic qualities of

the feed and subsequently to boost and encourage the feed intake. Animals of all age categories including calves, young adults and mature adults and animals of both sexes had an undisturbed access to the total of 43 feeding sites on approximately 23,000ha where medicated feed was made available.

Recommended triclabendazole doses of 10-12mg/kg body weight/day were used.
The assumption was that approximately 5,000 animals would have gravitated the study area, with an average body mass of 150kg, each animal on average consuming 0.5kg of feed/day.
In the season of 2002/03 and the years preceding, 88% of 368 cadavers examined upon the harvest were presented with F. magna infection (40% severely or very severely affected). After a single treatment with triclabendazole alone (Fasinex 10% ®, Novartis) in July 2003 (7 days, 10mg/kg b.w./day) and after examining 285 cadavers still all adult animals were found to be infected, but gradation of changes was more moderate compared to 2002/03. There was a lower number of severely affected animals and evidence of healing with some dead parasites were observed in 55%. However, adult intact parasites and egg shedding were still largely present. No egg shedding and dead or no parasite with evidence of liver reparation was present in less than 5% of examined cases.

After the second and the third triclabendazole/levamisole (Combinex Cattle ®, Novartis) treatment in March 2004 and June 2004 (7 days, 12mg/kg b.w./day) 263 cadavers were examined during past November, December and January.
A significantly lower number of animals were infected, thus as much as 32% were presented with no pathognomonic changes, even often lacking typical black hematin pigmentation throughout the hepatic parenchyma. The hematin pigment is produced in the intestine of immature and adult flukes as a byproduct of feeding on blood and so presents an extremely good diagnostic tool. Also, less than 7% of the population was presented with severe changes and no very severe infections with total destruction of the liver parenchyma, which used to predominate before the treatment, were observed. Evidence of healing with dead parasites or no parasites at all was presented in approx. 33%, regardless of sex and age. Nevertheless, intact mature flukes and egg shedding were still present in at least 35% of the population and superinfections were still to be found.

In conclusion it is fair to say that medicated feed pellets were outstandingly well accepted by the deer and that triclabendazole has attained the expected positive effect well within the results of the past studies in treating F. magna infection in the free-ranging deer.

174) SEROPREVALENCE OF INFECTIOUS AGENTS IN FREE LIVING MOUNTAIN GORILLAS (*GORILLA BERINGEI* SSP)

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The remaining populations of mountain gorillas (*Gorilla beringei beringei* and *G. b. undecided*) in Central Africa are subjected to relatively intense human contact because they are habituated to human presence for tourism and research programs. Their habituation also allows them to benefit from emergency veterinary care in cases of human-induced or life threatening injury or illness. Serum samples (N= 41) collected opportunistically during these interventions were analysed for the presence of various infectious agents. The majority of samples analysed were collected after 2001. Mountain gorillas had antibodies for or antigens from 14 different viruses including influenza A and B, parainfluenza types 1, 2, and 3, measles, human herpes simplex 1, simian agent 8/African monkey herpesvirus, Epstein-Barr virus, chimpanzee cytomegalovirus, adenovirus, hepatitis A and B, and simian agent 11/rotavirus. Positive titres were also detected for *Mycoplasma pneumoniae*. No gorillas had titres for respiratory syncytial virus, human herpes simplex 2, human varicella zoster, simian retrovirus, simian immunodeficiency virus, simian T-lymphotrophic virus, foamyvirus, encephalomyocarditis virus, lymphocytic choriomeningitis virus, filovirus, mumps, human immunodeficiency virus type 1, Q fever, eastern equine encephalitis, western equine encephalitis, St. Louis encephalitis, California encephalitis, hepatitis C, simian hemorrhagic fever virus, reovirus, or monkeypox The highest seroprevalences were for Epstein-Barr and adenovirus for which nearly all gorillas were positive. There were no obvious correlations between seroprevalence and active or recent disease. Anecdotal evidence suggests that severe respiratory disease outbreaks in mountain gorillas may be caused at least partly by parainfluenza type 3 as nearly all gorillas seropositive for this virus were from groups which had recently experienced such outbreaks.

175) EQUINE HERPESVIRUS 9 (EHV-9)-INDUCED ENCEPHALITIS IN ANIMALS PATHOLOGY AND EXPERIMENTAL INFECTIONS

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An outbreak of acute encephalitis occurred in a herd of Thomson's gazelles (*Gazella Thomsoni*) in a Japanese zoo. Seven of 9 gazelles died with or without neurological symptoms in a 3-week period. A herpesvirus was isolated from brain of the dead gazelles. The virus was neutralised by anti-EHV-1 serum, but its DNA fingerprint differed from those of EHV-1 and other equine herpesviruses. The isolated virus was named equine herpesvirus 9 (EHV-9) based on DNA analysis. Pathologically, all animals examined had nonsuppurative encephalitis characterised by necrosis and degeneration of neurons, gliosis and perivascular cuffing in the cerebrum. Five cases had intranuclear inclusion bodies in neurons compatible with herpesvirus. The neuropathology of EHV-9 infection clearly differed from EHV-1-induced encephalitis in the horse, which is characterised by vasculitis, thrombosis, ischemia, and lack of intranuclear inclusions in neurons.

We are now conducting experimental studies to clarify the infectivity of EHV-9 in rodents and domestic animals. After inoculation of EHV-9, mice, rats, goats, cats and dogs showed fatal acute encephalitis, while horses and cattle had only moderate nonsuppurative encephalitis and survived.

Suckling mice and rats inoculated intracerebrally showed growth deterioration and neurological symptoms, including depression and seizures, and dies within 8 days of inoculation. The brain of dead animal had severe neuronal degeneration and necrosis accompanied by numerous intranuclear inclusion bodies characteristic of herpesviruses.

Goats, pigs, cats and dogs: These animals inoculated intranasally showed sudden neurological symptoms consisting of marked convulsion, tremor and ataxia and died around a week after inoculation. Dead animals had fulminant encephalitis characterised by neuronal degeneration and necrosis with intranuclear inclusion bodies.

Horses and cattle inoculated intranasally showed no clinical symptom except a moderate fever. The brains showed a moderate degree of nonsuppurative encephalitis characterised by perivascular cuffings and gliosis. Neither neuronal necrosis nor intranuclear inclusions were observed.

EHV-9 showed infectivity in a wide variety of animals including gazelles. EHV-9 caused fulminant acute encephalitis in all animals examined except horses and cattle. It is likely that the gazelle was a foreign host for EHV-9 and, as a result, fatal infection occurred. Because the gazelles shared the same field with zebras, the latter must be considered a possible reservoir of EHV-9.

Fukushi,H., Tomita,T., Taniguchi,A., Ochiai,Y., Kirisawa,R., Masumura,T., Yanai,T., Yamaguchi,T. and Hirai,K.: Gazelle herpesvirus 1: A new neurotropic herpesvirus immunologically related to equine herpesvirus 1. Virology, 227, 34-44, 1997

Yanai,T., Sakai,T., Fukushi,H., Hirai,K., Narita,M., Sakai,H.and Masegi,T.: Neuro-pathological study of Gazelle Herpesvirus 1 (Equine Herpesvirus 9) infection in Thomson Gazelles (*Gazella thomsoni*). J. Comp.Pathol.119: 158-168, 1998

Taniguchi, A., Fukushi, H., Matsumura, T., Yanai, T., Masegi, T. and Hirai, K.: Pathogenicity of a new neutrotropic equine herpesvirus 9 (Gazelle Herpesvirus 1) in horses. J. Vet. Med. Sci. 62: 215-218, 2000

Taniguchi, A., Fukushi, H., Yanai, T., Masegi, T. and Hirai, K.: Equine herpesvirus 9 induced lethal encephalitis in experimentally infected goats. Arch. Virol. 145: 2619-2627, 2000

Fukushi, H., Taniguchi, A., Yasuda, K., Yanai, T., Masegi, T., Yamaguchi, T. and Hirai, K: A hamster model of equine herpesvirus 9 induced encephalitis. J. NeuroVirol. 6:314-319, 2000

Narita, M., Uchimura, A., Kimura, K., Tanimura, N., Yanai, T., Masegi, T., Fukushi, H. and Hirai, K.: Brain lesions and transmission of experimental equine herpesvirus type 9 in pigs. Vet. Pathol. 37: 476-479, 2000

Yanai, T., Tsujioka, S., Sakai, H., Fukushi, H., Hirai, K., Masegi, T.: Experimental infection with equine herpesvirus 9 (EHV-9) in cats. J. Comp. Pathol. 128:113-118, 2003

Yanai, T., Fujishima, N., Fukushi, H., Hirata, A., Sakai, H., Masegi, T.: Experimental infection of equine herpesvirus 9 in dogs. Vet. Pathol. 40: 263-267, 2003

STUDENT POSTERS

176) ISOLATION AND IDENTIFICATION OF *TRYPANOSOMA* SPECIES IN GILBERT'S POTOROO (*POTOROUS GILBERTII*)

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Little is known of the prevalence and life cycle of trypanosomes in Australia. The first record of Australian trypanosomes in mammals was made by T.L. Bancroft in 1888 with the discovery of *T. lewisi* in rats. Since then *T. pteropi* from the flying fox, *T. hipposideri* from the dusky horseshoe-bat (*Hipposideros bicolor albanensis*), *T. binneyi* from the platypus (*Ornithorhynchus anatinus*), *T. thylacis* from the northern brown bandicoot (*Thylacis obesulus*) and more recently novel *Trypanosoma* sp. from the Kangaroo (*Macropus giganteus)* and a common wombat (*Vombatus ursinus)*, have been detected (Mackerras1959; Noyes *et al*., 1998). Recently, trypanosomes were identified in blood smears from the critically endangered Gilbert's potoroo (*Potorous gilbertii*). This is the first record of trypanosomes in Western Australia and within the family Potoroidae. This novel trypanosome was characterised using conventional light microscopy and molecular tools to determine morphological and molecular characteristics. From stained blood smears various stages of the trypanosome life cycle were observed and the trypanosomes were also successfully established *in vitro* culture systems. Phylogenetic analyses of partial *Trypanosoma* sequences of the 18S rRNA gene indicated that Gilbert's potoroo trypanosome was closely related to an isolate from the wombat discovered in Victoria. Morphological characterisation confirmed that the isolate belongs to the Stercorarian group of trypanosomes in the subgenus *Herpetosoma*, an observation consistent with the phylogenetic tree analyses.

177) CONSERVING OUR NATIVE CARNIVORES: THE APPLICATION OF MOLECULAR GENETICS TO THE CONSERVATION MANAGEMENT OF QUOLLS

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Quolls (*Dasyurus* spp.) are amongst the largest of the carnivorous marsupials in the world, only exceeded in size by the Tasmanian devil. All six known species are endemic to Australasia and function as top-level native marsupial predators in all environments in which they occur. All four of the Australian quolls are under severe threats due to a variety of interacting factors, including habitat loss, direct and indirect effects of introduced predators, indirect mortality from non-target 1080 poisoning, the spread of cane toads, and purported disease epidemics. With the recent introduction of foxes in Tasmania and the alarming disease outbreak in devils, Tasmania's eastern and spotted-tailed quolls could come under serious threat, while cane toad invasions into the Northern Territory are having a significant impact on one of the few remaining strongholds for northern quolls. More and more frequently, molecular techniques are being used to address significant questions in ecology, evolution, behaviour and conservation. The overall aim of this project is to use genetic markers to gather information on, and better understand Australia's large carnivorous marsupials. Microsatellites and the mitochondrial DNA (mtDNA) control region (CR) are highly variable markers that have a broad range of applications in conservation and evolutionary genetics and have been widely used in population, parentage, and phylogeographical studies. These markers, previously developed for Dasyurids, will be used to identify and characterise genetic partitions within quoll species for biodiversity assessment, measure population parameters of concern for management (genetic diversity, effective population size, and degree of gene flow between populations) and to understand the evolutionary forces acting on translocated populations. The results obtained from these studies will help guide management practices for both short- and long-term conservation of these species and provide a model system for genetic management of other Australian species.

178) INVESTIGATIONS INTO THE DEVELOPING GUT MICROFLORA OF THE TAMMAR WALLABY

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Macropus eugenii, commonly known as the tammar wallaby only has a short gestation period of 28-32 days. The neonate weighs approximately 0.4 g, climbs through the urogenital tract and up into the pouch where it continues the rest of its development. When the neonate is born, it only has a rudimentary gastro-intestinal tract, forelimbs to climb from the urogenital tract into the pouch and a tongue to suck milk from the teat. The rest of the features are very much undeveloped and the neonate is totally dependent on its mother. Since the neonatal does not have a functional immune system at this stage, it must, we presume, rely on maternal defences to prevent pathogenic microbial infection.

In the first three weeks, there are no mature lymphoid tissues and the neonate lacks immune competence. It is not until tissues mature at 120 days that the animal is believed to be immunocompetent. Since it is well known in humans that the microflora of the gut changes dramatically from facultative anaerobes (e.g. Streptococci) to obligate anaerobes (e.g. *Bacteroides*) when the baby is weaned off milk to solid foods, it is hypothesised that the microflora of the neonate tammar wallaby will change and that these changes can be correlated with changes in the neonate immune system. Identifying the dominant bacteria in the neonate gut at these various stages of immune development is the first step in allowing us to evaluate which bacteria may be responsible for protecting the neonate from pathogens. Evaluation of the nature of this microflora will direct our research towards understanding how these probiotic bacteria and their products protect the mucosal surfaces and assist in immune system development.

The initial focus of this research is therefore to investigate the developing gut microflora of the neonate at different stages of its immune development. To achieve this, the bacterial microflora from maternal samples taken from the faeces, cloaca, pouch, skin, oral cavity and milk will be analysed along with those of the neonate, since, the neonate is inoculated through the birth process. This information will then be used to elucidate the relation between bacterial microflora and mucosal immune system development. The planned approach for identifying the bacterial microflora will be discussed as it involves molecular/microbial methods to detect uncultivable bacteria.

179) URBAN INDUCED INFLUENCES ON THE HEALTH OF FRAGMENTED POPULATIONS OF *ISOODON OBESULUS*

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The Southern Brown Bandicoot (*Isoodon obesulus*) was once one of the commonest of Australian mammals and widespread in NSW. Since European settlement and the associated urbanisation, its habitat range has been restricted to a few non-contiguous locations along the coast fringe of NSW. The continuing influence of urbanisation threatens the species. Garigal National Park is a small patch of land surrounded by urban developments and a major Sydney road. This fragmented habitat is home to a reportedly unhealthy and declining population of *I. obesulus*. In contrast, the nearby Ku-ring-gai National Park harbours a relatively successful and healthy population. The residual effects of the urban developments and housing around Garigal National Park on *I. obesulus* is yet to be determined. Research is proposed to assess the health, disease and genetic variation of both populations to provide managers with a tool for conservation. Live trapped *I. obesulus* will undergo individual health assessments to evaluate their immune functions and presence of parasites. Molecular techniques based on analysing micro-satellite variation will be undertaken to examine the variability/relatedness of the two populations *I. obesulus*. This study will enable us to determine specific harmful diseases and determine genetic variation within and between the two populations. Without specific conservation measures to counteract these threatening processes and knowledge of potential diseases, the continuation of these Northern Sydney populations is in jeopardy.

180) SEROPREVALENCE OF PATHOGENS IN AN ISLAND POPULATION OF WILD OCELOTS (*LEOPARDUS PARDALIS*)

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Spillover from domestic species has been frequently implicated as a source of disease in wildlife populations. Previous study of wild ocelots (*Leopardus pardalis*) has demonstrated that populations in close proximity to human settlements can become infected with multiple pathogens of domestic dogs and cats. In contrast, prior serosurveys have determined that ocelots have an extremely low level of feline immunodeficiency virus (FIV)-like infection (0-5%). This low seroprevalence is remarkable because other wild felids have species-specific FIV infection rates approaching 100%. This study was designed to determine the seroprevalence of domestic animal pathogens and feline lentivirus (FIV) in an isolated island population of ocelots that has practically no contact with domesticated species. We collected serum samples from 12 ocelots native to Barro Colordao Island (BCI) and screened for antibodies to: (1) feline calicivirus, canine distemper virus, and feline herpesvirus using serum neutralisation; (2) feline corona virus using immunofluorescence assay; (3) feline parvovirus using hemagglutination inhibition and (4) feline lentivirus using immunoblots with virus from three antigenically distinct FIV strains (puma lentivirus, lion lentivirus, and feline immunodeficiency virus). Sera from naïve or exposed animals were used as controls. All twelve individuals were seronegative for exposure to all pathogens with the exception of lentivirus infection. Five of seven females were positive for lentiviral infection, and one female's sera was inconclusive. Interestingly, only one of five males was positive for lentiviral-reactive antibodies. These results are suggestive of female ocelot predilection for lentiviral infection but were only marginally significant when analysed using non-parametric methods ($p=0.08$). Gender bias for lentiviral infection has not been reported previously in FIV infection, and should such a relationship be validated by analysis of additional samples, this population would provide a rare opportunity to determine behavioural or ecological factors underlying this observation. Overall, these results suggest that isolation from domestic reservoirs will prevent the introduction of disease in small, high density populations of wild felids. We speculate that lentiviral seropositivity in this population represents infection with a species-specific virus that was enzootic prior to isolation of the BCI ocelot population. We hypothesise that the high density of ocelots on BCI leads to greater contact rates among individuals and may explain the higher seroprevalence of lentivirus exposure in this population as compared to previously studied populations.

181) INTERACTION OF MULTIPLE PATHOGENS AND HABITAT ALTERATION ON CALIFORNIA RED-LEGGED FROGS (*RANA AURORA DRAYTONII) ALONG* THE CENTRAL CALIFORNIA COAST

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The California red-legged frog (*Rana aurora draytonii*) is listed on The World Conservation Unions' Red List of Threatened Species and faces growing threats across its range. Our work has centred on populations in and around the Elkhorn Slough National Estuarine Research Reserve (ESNERR), located along the central coast of California. Data collected through ESNERR's amphibian monitoring project from 1997 through 2004 revealed directional declines in numbers of adults and juveniles. To investigate the factors driving these declines, we surveyed at total of 40 ponds within 5 km of ESNERR. These surveys revealed several habitat and disease factors likely to account for regional declines in California red-legged frog populations. We found a significant positive correlation between occurrence of California red-legged frogs and densities of the snail intermediate host for *Riberoia ondatrae*, as well as negative correlations between observations of California red-legged frogs and increasing turbidity and ammonia levels. Surprisingly, we did not find that their presence was affected by proximity to agriculture. We documented malformations caused *Riberoia ondatrae,* and infection with *Batrachochytrium dendrobatidis* and *Serratia fonticola* in California red-legged frogs and/or co-occurring anuran species. Individuals at two ESNERR sites and two regional sites tested positive for infection with *Batrachochytrium dendrobatidis,* while malformed amphibians were detected at one ESNERR site and at a regional site. Based on these preliminary findings we have designed an ongoing monitoring and experimental program to investigate population viability and disease effects.

182) PLASMA BIOCHEMISTRY AND HEMATOLOGICAL VALUES FOR WILD-CAUGHT FLYING FOXES (*PTEROPUS GIGANTEUS*) IN INDIA

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Bats of the genus *Pteropus* are ecologically important and also are natural hosts for zoonotic pathogens, yet little is known about their basic biology. We collected blood samples from 41 wild-caught flying foxes (*Pteropus giganteus*) in northern India to evaluate baseline haematology and plasma biochemistry values. There were no differences between plasma biochemistry values of male and female bats, between juveniles and adults, or between lactating and non-lactating females. Variation in aspartate aminotransferase was seen between bats grouped by body condition score. The mean lymphocyte count was higher for juveniles than adults. There were no differences in haematological values between male and female *P. giganteus* or between groups based on body condition. No haemoparasites were observed. Photomicrographs from a captive *Pteropus giganteus* are presented to demonstrate blood cell morphology. Blood urea nitrogen and cholesterol values were lower than reported values for other mammals. Alanine aminotransferase and aspartate aminotransferase values were higher than those reported for *Pteropus vampyrus,* a closely related pteropid species. This study provides basic physiological information which can be used in future health and disease studies of wild or captive Indian flying foxes.

183) PORPHYRIA OBSERVATIONS ON THE CANEFIELD RAT (*RATTUS SORDIDUS*)

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Congenital erythropoietic porphyria (CEP) is a rare autosomal recessive disease caused by defects in heme synthesis. Uroporphyrin 1 is deposited in tissues, particularly bones and teeth, resulting in pink coloration and fluorescence of bones, tissues and urine under long wave ultraviolet (UV) light. CEP is detrimental and has been detected as a rare pathological condition in cattle, swine, cats, dogs, rodents and humans. But fox squirrels (*Sciurus niger*) have a physiological porphyria that is characteristic to the species and causes no dermal lesions and no detrimental effects to the animal.

As a result from a study focusing on animal species in banana fields and their prevalence of leptospirosis in Far North Queensland, 2 canefield rats (*Rattus sordidus*) were removed exhibiting CEP characteristics. All the bones and teeth were bright red and the cartilaginous tissue was pink. Under the UV light, the skeleton had a bright red fluorescence. Among mammals, it was previously believed that only in the *Sciuridae* did really red bones appear to occur. The animals were mature and appeared to be in good health with the clinical manifestations associated with CEP seemingly absent.

The implications of this observation suggest *R. sordidus* as an alternative animal model for porphyria research. Until now, the fox squirrel represented the only animal model suitable to determine the genetic basis and physiological conditions of the disease, and an easy and inexpensive laboratory animal for the studies. The capture of affected rats and the establishment of a breeding colony are warranted to determine the condition of CEP in the animals and to determine their suitability as laboratory research animals.

184) PRESENCE OF *ANGIOSTRONGYLUS* SPECIES IN POPULATIONS OF *RATTUS RATTUS* AND *RATTUS FUSCIPES* IN COASTAL FORESTS OF NSW, AUSTRALIA

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Angiostrongylus is a genus of nematode comprising at least 20 species that infect mammals throughout the world. In Australia two species of *Angiostrongylus* have been described from rodents. Both species inhabit the pulmonary arteries of their definitive rat hosts, and complete their life cycle in intermediate gastropod hosts. *Angiostrongylus cantonensis* has been recorded from introduced *Rattus rattus* and *Rattus norvegicus* in south-eastern Queensland and the Sydney metropolitan area. It has not been found in native rodents and is presumed to have been introduced to Australia from Asia with invasive rodent species. Its continued presence in rodent populations and potential spread has been enabled by using a wide range of invertebrate intermediate and invertebrate and vertebrate paratenic hosts. Many native and introduced snails and slugs are suitable intermediate hosts. *Angiostrongylus mackerrasae* is an endemic species reported from native rodents in south-eastern Queensland (*Rattus fuscipes*) and Tasmania (*Rattus lutreolus*). It has been found to co-infect with *A. cantonensis* in *R. norvegicus*, but not in *R. rattus*.

The distribution of *Angiostrongylus* species in introduced and native rodents in Australia is unknown. This research aims to determine the presence of *Angiostrongylus* spp. in *R. rattus* and *R. fuscipes* populations in southern NSW, and whether crossover of lungworm species occurs in the two rodent species.

Presence of *Angiostrongylus* spp. across 14 study sites was measured seasonally for 12 months from faecal samples (which detect presence of larvae) and autopsies. Sites occur in coastal forests surrounding the north and south of Jervis Bay. In the south, *A. mackerrasae* was sampled from *R. fuscipes* populations on 7 of 8 sites. Recurrent presence of larvae in faeces and 17% prevalence of adult lungworms in autopsies, suggests that *A. mackerrasae* may be well established in native rodents in the area. Lungworm was also sampled from *R. rattus* in the south. While the species in *R. rattus* has not been confirmed, it is possibly the native *A. mackerrasae* based on very low larval loads in faeces and degraded adult worms in an autopsied rat. These findings suggest that the black rat may not be a suitable host for the native lungworm.

In the north of Jervis Bay *R. rattus* are infected with *A. cantonensis*. The incidence appears low with a prevalence of 2% among autopsied animals. This is the most southern recording of *A. cantonensis* on the east coast of Australia and indicates likely distribution from Sydney. Infections of lungworm have only recently been found in *R. fuscipes* in the north. Low larval loads have been detected in faeces from animals on 3 of 6 sites. The lungworm species has not been confirmed by autopsy to date.

The presence of *A. cantonensis* in bushland close to campgrounds in the north of Jervis Bay, has possible human health implications. The parasite may infect humans (via ingestion of infected snails or larvae released in slime trails on improperly washed vegetables like lettuce) and cause neurological symptoms from severe headaches to disease (eosinophilic meningitis) and death. Risk of infection to humans around the campgrounds is low, with potential sources of infection being small children ingesting infected molluscs.

The presence of *A. cantonensis* in *R. rattus* populations also has potential negative implications for native wildlife that may include snails and slugs as part of their diet, including native rats, birds, possums, bandicoots and bats. If the lungworm becomes established, it may limit populations of native species by causing neurological disease and death of individuals.

185) SPATIAL PATTERNS OF THE AMPHIBIAN PATHOGEN, *BATRACHOCHYTRIUM DENDROBATIDIS* IN EUROPE

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In 1998 mass mortalities of *Alytes obstetricans* in Peñalara natural Park, central Spain, led to the first documented case of chytridiomycosis in a wild European amphibian assemblage. The causal agent, the chytrid fungus, *Batrachochytrium dendrobatidis* has been associated with amphibian declines globally. However until recently it was unknown whether or not this apparently isolated outbreak represented the actual distribution of *B. dendrobatidis* in Europe. We report here on recent surveillance using molecular diagnostics at several geographical scales: Peñalara, Spain and Europe. Within Spain we have identified further outbreaks of chytridiomycosis in three geographically distinct, high altitudinal populations of *A. obstetricans*. While chytrid related mortalities in Spain have only been associated with *A. obstetricans, Salamandra salamandra* and *Bufo bufo*, we have shown that the fungus has a broader host range, infecting at least 60% of the amphibian species known from the country. Across Europe, we report *B. dendrobatidis* to be present in at least five countries and from over 20 European species and one invasive species. We have been investigating how environmental variables might be driving these distributions using spatial statistical models.

186) HAEMATOLOGY OF HEALTHY AND MORBID SOUTHERN BROWN BANDICOOTS (*ISOODON OBESULUS*)

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Investigations of the health of wild animals may be aided by the assessment of haematological values. However, the haematological characteristics in health and in response to disease are poorly understood for many Australian marsupials. This study investigated the haematology of a population of wild southern brown bandicoots (*Isoodon obesulus*) in the Perth area and bandicoots suffering from injury or disease that were presented to a wildlife rehabilitation centre.

Blood samples were collected from animals from a wild population (n=65), and from individuals suffering from injury or disease (n=8). The blood of these animals was assessed using an automated haematology analyser (Advia 120, Bayer, Tarrytown, NY), manual methods (total solids, fibrinogen) and microscopy. The samples from clinically healthy individuals from the wild population were used to develop reference intervals to investigate the changes in the unwell animals. This included animals suffering from injuries from an attack by a domestic dog, abscess, toxoplasmosis, periodontal disease, aural and ocular infections, and general malaise.

The morphology of erythrocytes, leukocytes and platelets in healthy animals were similar to previous descriptions for other bandicoot species. The haematological characteristics of the healthy animals are presented in Table 1. Of the unwell animals, 6/8 had increased leukocyte concentrations, and 2/6 had increased fibrinogen concentration. The neutrophils of one animal exhibited morphological changes. Four of eight animals were anaemic, and of these three had an increased concentration of polychromatophilic erythrocytes. A haemoparasite, morphologically consistent with a *Hepatozoon* sp., was present in approximately half the wild population, and in some unwell animals, but did not appear to cause disease.

The values for the haematology analytes from the wild population may be used in the investigation of health and disease in southern brown bandicoots. Of the morbid animals, most had evidence of an inflammatory response, in the form of a leukocytosis. However, fibrinogen concentration was an inconsistent indicator of acute inflammation in bandicoots. Haematological changes consistent with glucocorticoid release were seen in one animal, and this may make interpretation of values difficult in animals that are “stressed” prior to blood collection. Anaemia was usually associated with systemic illness, and a regenerative response was not seen in all cases of anaemia.

Table 1: Haematological characteristics of healthy southern brown bandicoots

Erythrocyte concentration ($x10^{12}$/L)	n=65	5.42-8.4
Haematocrit (L/L)	n=65	0.32-0.51
Haemoglobin concentration (g/L)	n=63	120-167
Polychromatophils (%)	n=52	<4.4
Leukocyte concentration ($x10^9$/L)	n=65	1.25-7.68
Neutrophil concentration ($x10^9$/L)	n=65	0.32-3.97
Lymphocyte concentration ($x10^9$/L)	n=65	0.34-5.68
Monocyte concentration ($x10^9$/L)	n=65	<0.38
Eosinophil concentration ($x10^9$/L)	n=65	<0.91
Basophil concentration ($x10^9$/L)	n=65	<0.05
Annular leukocyte concentration ($x10^9$/L)	n=65	<0.33
Fibrinogen concentration (g/L)	n=47	1-6

187) REAL-TIME PCR DETECTION OF *CAMPYLOBACTER* IN WILD MOUNTAIN GORILLAS (*GORILLA BERINGEI BERINGEI*)

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Health monitoring of wildlife populations could greatly benefit from rapid, local, non-invasive molecular assays for important pathogens. Fecal samples (N=157) from free-living mountain gorillas were tested for *Campylobacter* DNA using a portable, real-time polymerase chain reaction instrument. An 87% prevalence was detected with no statistically significant differences between different gorilla groups, sexes, or age classes. A statistically significant higher prevalence was detected in samples collected from identified individuals during behavioral observations (90%) compared to samples collected from night nests (73%). A modification of the standard protocol improved assay consistency, and increased detection sensitivity. The modified procedure was more sensitive than bacterial culture with *Campylobacter* specific media, and commercially available EIA tests. A subset of fifty samples was negative for *Salmonella sp., Listeria monocytogenes,* and *E. coli* O157 by the same instrument. We speculate that the high prevalence of *Campylobacter* detected by real-time PCR in non-invasively collected fecal samples from wild gorillas represents normal intestinal flora.

MARK TWAIN
IN
AUSTRALIA AND NEW ZEALAND

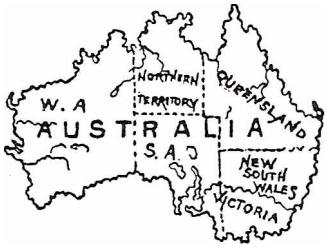
Mark Twain
(Samuel Clemens)
Penguin Books Ltd, Harmondsworth, Niiddlesex England
Penguin Books Australia Ltd Ringwood, Victoria, Australia

The material in this book first appeared
in *Following the Equator*, published by
The American Publishing Company in 1897
This edition published by Penguin Books 1973

"But Nature is always stingy of perfect climates; stingier in the case of Australia than usual. Apparently this vast continent has a really good climate nowhere but around the edges.

If we look at a map of the world we are surprised to see how big Australia is. It is about two-thirds as large as the United States was before we added Alaska.

But where as one finds a sufficiently good climate and fertile land almost everywhere in the United States, it seems settled that inside of the Australian border-belt one finds many deserts and in spots a climate which nothing can stand except a few of the hardier kinds of rocks. In effect, Australia is as yet unoccupied. If you take a map of the United ·States and leaye the Atlantic Sea-9Qard States in their places; also the fringe of Southern States from Florida west to the Mouth of the Mississippi; also a narrow, inhabited streak up the Mississippi half-way to its head waters; also a narrow, inhabited border along the Pacific c'oast: then take a brushful of paint and obliterate the whole remaining mighty stretch of country that lies between t,he Atlantic States and the Pacific-coast strip, your map will look like the latest map of Australia.



"In the Zoological Gardens of Adelaide I saw the only laughing jackass that ever showed any disposition to be courteous to me. This one opened his head wide and laughed like a demon; or like a maniac who was consumed with humorous scorn over a cheap and degraded pun. It was a very human laugh. If he had been out of sight I could have believed that the laughter came from a man. It is an odd-looking bird, with a head and beak that are much too large for its body. In time man will exterminate the rest of the wild creatures of Australia, but this one will probably survive, for man is his friend and lets him alone. Man always has a good reason for his charities towards wild things, human or animal - when he has any. In this case the bird is spared because he kills snakes. If L J. will take my advice he will not kill all of them."

LAUGHING JACKASS.

“I already knew a good deal about the rabbits in Australasia and their marvelous fecundity, but in my talks with him I found that my estimate of the great hindrance and obstruction inflicted by the rabbit pest upon traffic and travel was far short of the facts. He told me that the first pair of rabbits imported into Australasia bred so wonderfully that within six months -rabbits were so thick in the land that people had to dig trenches through them to get from town to town.

He told me a great deal about worms, and the kangaroo, and other coleoptera, and said he knew the history and ways of all such pachydermata. He said the kangaroo had pockets, and carried its young in them when it couldn't get apples. And he said that the emu was as big as an ostrich, and looked like one, and had an amorphous appetite and would eat bricks. Also, that the dingo was not a dingo at all, but just a wild dog; and that the only difference between a dingo and a dodo was that neither of them barked; otherwise they were just the same.”

“The magpie was out in great force, in the fields and on the fences. He is a handsome large creature, with snowy white decorations, and is a singer; he has a murmurous rich note that is lovely. He was once modest, even diffident; but he lost all that when he found out that he was Australia's sole musical bird. He has talent, and cuteness, and impudence; and in his tame state he is a most satisfactory pet - never coming when he is called, always coming when he isn't, and studying disobedience as an accomplishment. He is not confined, but loafs all over the house and grounds, like the laughing jackass. I think he learns to talk, I know he learns to sing tunes, and his friends say that he knows how to steal without learning. I was acquainted with a tame magpie in Melbourne. He had lived in a lady's house several years, and believed he owned it. The lady had tamed him, and in return be had tamed the lady. He was always on deck when not wanted, always having his own way, always tyrannizing over the dog, and always making the cat's life a slow sorrow and a martyrdom. He knew a number of tunes and could sing them in perfect time and tune; and would do it, too, at any time that silence was wanted; and then encore himself and do it again; but if he was asked to sing he would go out and take a walk.”

“The Moa stood thirteen feet high, and could step over an ordinary man's head or kick his hat off; and his head, too, for that matter. He said it was wingless, but a swift runner. The natives used to ride it. It could make forty miles an hour, and keep it up for four hundred miles and come out reasonably fresh. It was still in existence when the railway was introduced into New Zealand; still in existence, and carrying the mails. The railroad began with the same schedule it has now: two expresses a week - time, twenty miles an hour. The company exterminated the moa to get the mails.”

OFF GOES HIS HEAD

“Speaking of the indigenous coneys and bactrian camels, the naturalist said that the coniferous and bacteriological output of Australasia was remarkable for its many and curious departures from the accepted laws governing these species of tubercles, but that in his opinion Nature's fondness for dabbling in the erratic was most notably exhibited in that curious combination of bird, fish, amphibian, burrower, crawler, quadruped, and Christian called the Ornithorhyncus - grotesquest of animals, king of the animalculæ of the world for versatility of character and make-up. Said he-

> "You can call it anything you want to, and be right. It is a fish, for it lives in the river half the time; it is a land animal, for it resides on the land half the time; it is an amphibian, since it likes both and does not know which it prefers; it is a hybernian, for when times are dull and nothing much going on it buries itself under the mud at the bottom of a puddle and hybernates there a couple of weeks at a time; it is a kind of duck, for it has a duck-bill and four webbed paddles; it is a fish and quadruped together, for in the water it swims with the paddles and on shore it paws itself across country with them; it is a kind of seal, for it has a seal's fur; it is carnivorous, herbivorous, insectivorous, and vermifuginous, for it eats fish and grass and butterflies, and in the season digs worms out of the mud and devours them; it is clearly a bird, for it lays

eggs, and hatches them; it is clearly a mammal, for it nurses its young; and it is manifestly a kind of Christian, for it keeps the Sabbath when there is anybody around, and when there isn't, doesn't. It has all the tastes there are except refined ones, it has all the habits there are except good ones.

"It is a survival - a survival of the fittest. Mr. Darwin invented the theory that goes by that name, but the Ornithorhyncus was the first to put it to actual experiment and prove that it could be done. Hence it should have as much of the credit as Mr. Darwin. It was never in the Ark; you will find no mention of it there; it nobly stayed out and worked the theory. Of all creatures in the world it was the only one properly equipped for the test. The Ark was thirteen months afloat. and all the globe submerged; no land visible above the flood, no vegetation, no food for a mammal to eat, nor water 'for a mammal to drink; for all mammal food was destroyed, and when the pure floods from heaven and the salt oceans of the earth mingled their waters and rose above the mountain tops, the result was a drink which no bird or beast of ordinary construction could use and live. But this combination was nuts for the Ornithorhyncus, if I may use a term like that without offense. Its river home had always been salted by the flood-tides of the sea. On the face of the Noachian deluge innumerable forest trees were floating. Upon these the Ornithorhyncus voyaged in peace; voyaged from clime to clime, from hemisphere to hemisphere, in contentment and comfort, in virile interest in the constant change of scene, in humble thankfulness for its privileges, ill ever-increasing enthusiam in the development of the great theory upon whose validity it had staked its life, its fortunes, and its sacred honor, if I may use such expressions without impropriety in connection with an episode of this nature.

WAS NEVER IN THE ARK

"It lived the tranquil and luxurious life of a creature of independent means. Of things actually necessary to its existence and its happiness not a detail was wanting. When it wished to walk, it scrambled along the tree trunk; it mused in the shade of the leaves by day, it slept in their shelter by night; when it wanted the refreshment of a swim, it had it; it ate leaves when it wanted a vegetable diet, it dug under the bark for worms and grubs; when it wanted fish it caught them, when it wanted eggs it laid them. If the grubs gave out in one tree it swam to another; and as for fish, the very opulence of the supply was an embarrassment. And finally, when it was thirsty it smacked its chops in gratitude over a blend that would have slain a crocodile.

"When at last, after thirteen months of travel and research in all the Zones it went aground on a mountain-summit, it strode ashore, saying in its heart, 'Let them that come after me invent theories and dream dreams about the Survival of the Fittest if they like, but I am the first that has *done* it!

"This wonderful creature dates back like the kangaroo and many other Australian hydrocephalous invertebrates, to an age long anterior to the advent of man upon the earth; they date back, indeed, to a time when a causeway hundreds of miles wide, and thousands of miles long, joined Australia to Africa, and the animals of the two countries were alike, and all belonged to that remote geological epoch known to science as the Old Red Grindstone Post-Pleosaurian. Later the causeway sank under the sea; subterranean convulsions lifted the African continent a thousand feet higher than it was before, but Australia kept her old level. In Africa's new climate the animals necessarily began to develop and shade off into new forms and families and species, but the animals of Australia as necessarily remained stationary, and have so remained until this day. In the course of some millions of years the African Ornithorhyncus developed and developed and developed, and sluffed off detail after detail of its make-up until at last the creature became wholly disintegrated and scattered. Whenever you see a bird or a beast or a seal or an otter in Africa you know that he is merely a sorry surviving fragment of that sublime original of whom I have been speaking - that creature which was everything in general and nothing in particular - the opulently endowed *e pluribus unum* of the animal world.

"Such is the history of the most hoary, the most ancient, the most venerable creature that exists in the earth to day – *Ornithorhyncus Platypus Extraordinariensis* – whom God preserve!"

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ADDENDUM 1

EUROPEAN BROWN HARE SYNDROME (EBHS): A LARGE OUTBREAK IN LAST AUTUMN AND WINTER (2004-2005) IN FRANCE

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Set up in 1986 by the "Office National de la Chasse et de la Faune Sauvage" (ONCFS), the French Game and Wildlife Agency, SAGIR is the national surveillance network for wildlife in France, the main goal of which is to detect the principal causes of wildlife mortality.

SAGIR has collected reliable data on wildlife diseases through some 3,001 laboratory-tested animals in 2002 and 2,686 in 2003, i.e. more than 49,000 between 1986 and the end of January 2005. These data can already be used to make in-depth studies of one important game species: the European hare (*Lepus europeanus*) (1,156 animals in 2002, 816 in 2003 and 1,625 in 2004, most of them in autumn and the beginning of winter).

SAGIR network has obtained 470 EBHS (29%) positive results last autumn and winter 2004, compared to 109 (12%) in the hunting season 2003.

The cases have occurred all over France, including in the mountain areas where hares are at low densities.

Those results have led to hunting interdictions in several regions all over France but no proven explanation has been found.

Does the EBHSV (European Brown Hare Syndrome Virus) go through a mutation to a higher virulence strain? Or has a new way of dissemination of the virus has arisen?

As the "Departemental" (N.B.: France is divided into 95 administrative regions called "départements") Federations of Hunters are much involved in SAGIR network operations, it is important to give them answers to the following questions:

- Will the disease be as brutal next autumn?
- Is it interesting to try to find a vaccine against it (We have in France many demands for such a vaccine against VHD (Viral Hemorrhagic Disease) in rabbits)?
- What are the best management measures to take to limit the impact of the disease?

Last hunting season we proposed to limit the quantity of huntable hares in all the territories where the losses were important and most of the hunter associations accepted this slowdown.

As a matter of fact, since it has shown the effect of certain agricultural practices on wildlife, the SAGIR network has become the key point for all accidents involving wildlife. To find an answer to EBHS epizootics is its next challenge.

ADDENDUM 2

COLONY SPATIAL DYNAMICS INFLUENCE THE TRANSMISSION OF SYLVATIC PLAGUE IN BLACK-TAILED PRAIRIE DOGS

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Sylvatic plague (*Yersinia pestis*) is highly virulent in black-tailed prairie dogs (*Cynomys ludovicianus*) and causes mortality rates approaching 100%. Little is known of how plague is transmitted among prairie dog colonies. Currently, the major hypotheses include: (1) an intraspecific plague cycle among prairie dogs and (2) an interspecific cycle within small mammals, such as mice. If transmission is interspecific, species such as northern grasshopper mice (*Onychomys leucogaster*) or deer mice (*Peromyscus maniculatus*), which are often found in association with prairie dogs, may maintain plague and occasionally transmit plague-infected fleas to prairie dogs. Conversely, intraspecific transmission is likely the result of dispersing prairie dogs with infected fleas or perhaps the transfer of plague-infected fleas to neighbouring colonies by coyotes or other predators. We identified colony spatial characteristics that may impact intercolony transmission and distinguish between intra- and interspecific modes of transmission. In the case of intraspecific transmission, plague transmission may be influenced by colony size, distance to the nearest neighbouring colony, and distance to the nearest dry creek drainage, which may serve as prairie dog dispersal corridors. If interspecific transmission is dominant, colony infection may be independent of nearest neighbour or distance to drainage, but colony size may still be important. Multistrata mark-recapture modelling was used to investigate which of these spatial characteristics were most important to the transmission of plague in prairie dog colonies at Thunder Basin and Rita Blanca National Grasslands. Both grasslands recently experienced plague epizootics. At both sites, as the distance to the nearest neighbouring colony and distance to the nearest drainage increased, the probability of plague declined, and as colony area increased, the probability increased. The distance to the nearest neighbouring colony was most important at Thunder Basin, but colony area was also significant (ΔAICc<2.00). The distance to the nearest drainage was the most important factor at Rita Blanca National Grassland. The significance of the colony spatial characteristics outlined here provides support for intraspecific transmission of plague in these grassland systems.