52nd Annual

Wildlife Disease Program &

August 11 – 14, 2003
Saskatoon, Saskatchewan

52nd Annual Wildlife Disease Association Conference

Program and Abstracts of Papers and Posters Presented August 11 – 14, 2003

Hosted by:

The Canadian Cooperative Wildlife Health Centre

Canadian Cooperative Wildlife Health Centre

Centre canadien coopératif de la santé de la faune

UNIVERSITY OF SASKATCHEWAN

Western College of Veterinary Medicine, University of Saskatchewan

Held at:
The Delta Bessborough Hotel
Saskatoon, Saskatchewan

CONFERENCE AGENDA

DATE AND TIME	EVENT	LOCATION
Saturday, August 9th		
8:00 am – 5:00 pm	WDA Strategic Planning	Kelsey Room
1:00 pm – 6:00 pm	IUCN – VSG Meeting	Carlton Room
Sunday, August 10th		
8:00 am – 5:00 pm	ACZM Preparatory Course	Rm. 1655, WCVM*
8:00 am – 5:00 pm	WDA Editorial and Council Meeting	Salon Batoche
9:00 am – 12:00 noon	IUCN – VSG Meeting	Carlton Room
1:00 pm – 6:00 pm	WWHC Meeting	Kelsey Room
4:00 pm – 9:00 pm	Registration	William Pascoe Room
6:00 pm – 7:00 pm	Student Reception	William Pascoe Room
7:00 pm – 11:00 pm	Welcoming Reception Music by: The Lighthouse Penguins	William Pascoe Room
Monday, August 11th		
7:30 am – 12:00 noon	Registration	Convention Floor Foyer
8:30 am – 9:00 am	Welcome and General Introduction	Adam Ballroom
8:30 am – 5:00 pm	Poster Set-Up and Display	Convention Floor Foyer
9:00 am – 10:00 am	Diseases and Populations Session	Adam Ballroom
10:00 am – 10:30 am	Break	Convention Floor Foyer
10:30 am – 12:00 noon	Diseases and Populations Session cont.	Adam Ballroom
12:00 noon – 1:30 pm	Lunch	
1:30 pm – 3:00 pm	General Session A	Adam Ballroom
3:00 pm – 3:30 pm	Break	Convention Floor Foyer
3:30 pm – 4:30 pm	General Session A cont.	Adam Ballroom
5:00 pm – 8:00 pm	CAZWV Meeting	Kelsey Room
5:00 pm – 8:00 pm	AAWV Meeting	Salon Batoche
7:00 pm – 11:00 pm	Auction	Adam Ballroom
Tuesday, August 12th		
7:30 am – 12:00 noon	Registration	LaRonge Room
8:00 am – 5:00 pm	Poster Display	Convention Floor Foyer
8:00 am – 8:45 am	Conservation Biology & Disease Invited Speaker – Dr. Paul Paquet	Adam Ballroom
8:45 am – 10:00 am	Conservation Biology & Disease	Adam Ballroom

	Session	
10:00 am – 10:30 am	Break	Convention Floor Foyer
10:30 am – 12:00 noon	Conservation Biology & Disease Session cont.	Adam Ballroom
12:00 noon – 1:00 pm	Lunch	
1:00 pm – 3:00 pm	Student Research Presentations	Adam Ballroom
3:00 pm – 3:30 pm	Break	Convention Floor Foyer
3:30 pm – 5:00 pm	Student Research Presentations cont.	Adam Ballroom
5:30 pm – 6:30 pm	Bus Pick-up for Picnic (every 15 minutes)	Front of Delta Bessborough
5:30 pm – 9:00 pm	Picnic	Wanuskewin Heritage Park
Wednesday, August 13th		
6:30 am – 8:00 am	WDA Prayer Breakfast	Salon Batoche
8:00 am – 11:00 am	Registration	LaRonge Room
8:00 am – 5:00 pm	Poster Display	Convention Floor Foyer
8:00 am – 10:00 am	General Session B	Adam Ballroom
DATE AND TIME	**EVENT**	**LOCATION**
Wednesday, August 13th cont.		
10:00 am – 10:30 am	Break	Convention Floor Foyer
10:30 am – 12:00 noon	General Session B cont.	Adam Ballroom
12:00 noon – 1:30 pm	Lunch	
1:30 pm – 3:00 pm	General Session C	Adam Ballroom
3:00 pm – 3:30 pm	Break	Convention Floor Foyer
3:30 pm – 4:30 pm	General Session C cont.	Adam Ballroom
4:30 pm – 5:30 pm	WDA Business Meeting	Adam Ballroom
4:30 pm – 6:30 pm	CCM – CCWHC Meeting	Kelsey Room
4:30 pm – 6:00 pm	Poster Take-Down	Convention Floor Foyer
6:00 pm – 7:00 pm	Cash Bar	Convention Floor Foyer
7:00 pm – 11:00 pm	WDA Banquet AAWV Speaker: Dr. William Karesh Music by: Vesty and the Vexations	Adam Ballroom
Thursday, August 14th		
8:00 am – 10:30 am	Registration	LaRonge Room
8:00 am – 10:00 am	Large Ungulates Session A	Adam Ballroom

10:00 am – 10:30 am	Break	Convention Floor Foyer
10:30 am – 12:00 noon	Large Ungulates Session A cont.	Adam Ballroom
12:00 noon – 1:30 pm	Lunch	
1:30 pm – 3:00 pm	Large Ungulates Session B cont.	Battleford Room
3:00 pm – 3:30 pm	Break	Battleford Foyer
3:30 pm – 5:00 pm	Large Ungulates Session B cont.	Battleford Room
Friday, August 15th		
7:30 am – 12:00 noon	Registration for Chronic Wasting Disease Workshop	Rm. 2302, WCVM*
8:00 am – 5:00 pm	International Workshop on Chronic Wasting Disease	Rm. 2302, WCVM*

* Location of Western College of Veterinary Medicine (WCVM) relative to Delta Bessborough Hotel is displayed on Saskatoon City map.

PRESENTATION SCHEDULE

MONDAY, AUGUST 11

8:30 – 9:00	**WELCOME AND GENERAL INTRODUCTION** Conference Chair: Ted Leighton	
9:00 – 9:15	**DISEASES AND POPULATIONS SESSION** Moderator: Judit Smits	
	SPEAKER	***[ABSTRACT #] TITLE****
9:15 – 9:30	Gilbert, M.	[1] THE ASIAN VULTURE CRASH: INVESTIGATING MORTALITY OF ORIENTAL WHITE-BACKED VULTURE (*GYPS BENGALENSIS*) IN PUNJAB PROVINCE, PAKISTAN
9:30 – 9:45	Aguirre, A.A	[2] SEROEPIDEMIOLOGY OF HAWAIIAN MONK SEALS: ARE INFECTIOUS DISEASES LIMITING POPULATION RECOVERY?
9:45 – 10:00	Cattet, M.R.L.	[3] ASSESSING ENVIRONMENTAL HEALTH – THE FOOTHILLS MODEL FOREST GRIZZLY BEAR RESEARCH PROJECT
10:00 – 10:30	***BREAK***	
10:30 – 10:45	Dufour, K.W.	[4] POPULATION-LEVEL IMPACTS OF AVIAN BOTULISM IN PRAIRIE CANADA: INSIGHTS FROM HUNTER RECOVERIES OF BANDED MALLARDS (*ANAS PLATYRHYNCHOS*)
10:45 – 11:00	Joly, D.O.	[5] BOVINE TUBERCULOSIS AND BRUCELLOSIS AS FACTORS LIMITING POPULATION GROWTH OF BISON IN NORTHERN CANADA
11:00 – 11:15	Lankester, M.	[6] PARASITE BARRIERS AND CONSEQUENCES OF BREACHING THEM
11:15 – 11:30	Samuel, M.D.	[7] TRANSMISSION MODELS AND POPULATION EFFECTS OF VECTOR-BORNE DISEASES: AVIAN MALARIA IN HAWAIIAN FOREST BIRDS
11:30 – 11:45	Schock, D.	[8] POPULATION-LEVEL DIFFERENCES IN SUSCEPTIBILITY TO INFECTIOUS DISEASES: AN AMPHIBIAN – VIRUS CASE STUDY
11:45 – 12:00	Mörner, T.	PREVIEW OF EUROPEAN WILDLIFE DISEASE ASSOCIATION MEETING, SWEDEN 2004
12:00 – 1:30	***LUNCH***	

MONDAY, AUGUST 11 cont.

1:30 – 4:30	**GENERAL SESSION A** Moderator: Karen Machin	
	SPEAKER	***[ABSTRACT #] TITLE****
1:30 – 1:45	Hunter, B.	[9] WEST NILE VIRUS DIE-OFF IN CAPTIVE NORTH AMERICAN OWLS IN ONTARIO, CANADA
1:45 – 2:00	Hars, J.	[10] SURVEILLANCE OF WEST NILE VIRUS ON THE AVIFAUNA IN THE SOUTH OF FRANCE
2:00 – 2:15	Van Riper, C. III	[11] HABITAT PARTITIONING BY NEOTROPICAL MIGRANT WARBLERS ALONG THE LOWER COLORADO RIVER CORRIDOR AND POTENTIAL INFLUENCES ON WEST NILE VIRUS TRANSMISSION
2:15 – 2:30	Barker, I.K.	[12] ENHANCED PASSIVE SURVEILLANCE FOR WEST NILE VIRUS IN DEAD WILD BIRDS, CANADA – 2002-2003
2:30 – 2:45	Barker, I.K.	[13] WEST NILE VIRUS INFECTION IN FREE-RANGING AND CAPTIVE WILD BIRDS AND MAMMALS, ONTARIO, CANADA, 2002
2:45 – 3:00	Ezenwa, V.O.	[14] USING RS/GIS TO ASSESS WILDLIFE AND HUMAN WEST NILE VIRUS DISEASE RISK
3:00 – 3:30	***BREAK***	
3:30 – 3:45	Kuiken, T.	[15] DESCRIPTION OF THE HIGHLY PATHOGENIC AVIAN INFLUENZA A (H7N7) EPIDEMIC IN THE NETHERLANDS IN 2003
3:45 – 4:00	Ley, D.H.	[16] *MYCOPLASMA GALLISEPTICUM* CONJUNCTIVITIS IN HOUSE FINCHES (*CARPODACUS MEXICANUS*). CORRELATIONS AMONG CLINICAL SIGNS AND DETECTION BY POLYMERASE CHAIN REACTION FROM CONJUNCTIVAL AND CHOANAL SWABS
4:00 – 4:15	Campbell, G.D.	[17] TYPE E BOTULISM IN BIRDS ON LAKE HURON AND LAKE ERIE, 1998-2002
4:15 – 4:30	Moccia, R.D.	[18] PUTATIVE ROLE OF FISH IN THE EPIDEMIOLOGY OF AVIAN BOTULISM IN LAKE ERIE

TUESDAY, AUGUST 12

8:00 – 12:00	**CONSERVATION BIOLOGY AND DISEASE SESSION**	
	Moderator: Catherine Soos	
	SPEAKER	***[ABSTRACT #] TITLE****
8:00 – 8:45	Paquet, P.C.	CONSERVATION BIOLOGY AND DISEASE INVITED SPEAKER
8:45 – 9:00	Hilsberg, S.	[19] CONSERVATION MEDICINE IN ACTION: ONGOING INVESTIGATIONS INTO A SEVERE DISEASE OUTBREAK IN THE NGORONGORO CRATER, TANZANIA DURING 2000/2001?
9:00 – 9:15	Loeffler, I.K.	[20] BIOMEDICAL SURVEY OF GIANT PANDAS IN CHINA *EX SITU*: OBSERVATIONS OF A STUNTED GROWTH SYNDROME, AND THE NEED FOR SEROEPIDEMIOLOGICAL STUDIES
9:15 – 9:30	Mansfield, K.	[21] DISEASE AS A FACTOR LIMITING COLUMBIA BASIN PYGMY RABBIT RECOVERY
9:30 – 9:45	Nishi, J.S.	[22] RISK ASSESSMENT AS A TOOL TO EVALUATE HEALTH STATUS OF A SALVAGED HERD OF CAPTIVE WOOD BISON
9:45 – 10:00	Dein, F.J.	[23] THE NBII WILDLIFE DISEASE INFORMATION NODE
10:00 – 10:30		***BREAK***
10:30 – 10:45	Raphael, B.L.	[24] VETERINARY INVOLVEMENT IN CHELONIAN CONSERVATION PROGRAMS THROUGH THE TURTLE SURVIVAL ALLIANCE
10:45 – 11:00	Sleeman, J.M.	[25] THE ROLE OF WILDLIFE CENTERS AND REHABILITATORS IN WILDLIFE DISEASE MONITORING
11:00 – 11:15	Work, T.M.	[26] PATHOLOGY OF NATIVE AND INTRODUCED REEF FISH IN HAWAII
11:15 – 11:30	Raphael, B.L.	[27] HEALTH MONITORING PROGRAMS ASSOCIATED WITH CONSERVATION OF THE GRAND CAYMAN IGUANA (*CYCLURA NUBILA LEWISI*)
11:30 – 11:45	Rose, K.	[28] AN EPIZOOTIC OF SYSTEMIC COCCIDIOSIS (*CARYOSPORA CHELONIAE*) IN GREEN TURTLES (*CHELONIA MYDAS*) ALONG COASTAL NSW – A MARINE INDICATOR OF DROUGHT

TUESDAY, AUGUST 12 cont.

	SPEAKER	***[ABSTRACT #] TITLE****
11:45 – 12:00	Dubay, S.	[29] BIGHORN SHEEP (*OVIS CANADENSIS*) DISEASES: A BRIEF LITERATURE REVIEW AND RISK ASSESSMENT FOR TRANSLOCATION
12:00 – 1:00	***LUNCH***	
1:00 – 5:00	**STUDENT RESEARCH PRESENTATIONS** Moderator: Bill Samuel	
	SPEAKER	***[ABSTRACT #] TITLE****
1:00 – 1:15	Tate, C.M. (2003 Student Research Award Recipent)	[30] EXPERIMENTAL INFECTIONS OF WHITE-TAILED DEER WITH *ANAPLASMA (EHRLICHIA) PHAGOCYTOPHILUM*, THE ETIOLOGIC AGENT OF HUMAN GRANULOCYTIC EHRLICHIOSIS
1:15 – 1:30	Jolles, A.E.	[31] TUBERCULOSIS IN AFRICAN BUFFALO (*SYNCERUS CAFFER*): POPULATION EFFECTS OF A CHRONIC DISEASE
1:30 – 1:45	Appelbee, A.	[32] TRANSMISSION OF ZOONOTIC ISOLATES OF *GIARDIA* AND *CRYPTOSPORIDIUM* TO HARP SEALS
1:45 – 2:00	Yabsley, M.J.	[33] MOLECULAR VARIATION IN THE VARIABLE LENGTH PCR TARGET (VLPT) AND 120-KDA ANTIGEN GENES OF *EHRLICHIA CHAFFEENSIS* FROM WHITE-TAILED DEER
2:00 – 2:15	Ellis, A.E.	[34] PATHOLOGY OF NATURAL WEST NILE VIRAL INFECTION OF RAPTORS IN GEORGIA
2:15 – 2:30	Varela, A.S.	[35] EVIDENCE OF TICK-BORNE DISEASE AGENTS IN LONE STAR TICKS (*AMBLYOMA AMERICANUM*) FROM NORTHEASTERN GEORGIA
2:30 – 2:45	Gibbs, S.E.J.	[36] WEST NILE VIRUS IN AVIAN SPECIES OF GEORGIA
2:45 – 3:00	Pollock, E.	[37] EVALUATION OF TISSUE CONTAMINANT CONCENTRATIONS AND SELECTED HEALTH PARAMETERS IN CARIBOU (*RANGIFER TARANDUS*) IN LABRADOR, CANADA
3:00 – 3:30	***BREAK***	
3:30 – 3:45	Gillespie, T.R.	[38] LONG-TERM EFFECTS OF LOGGING ON PARASITE DYNAMICS IN AFRICAN PRIMATE POPULATIONS

TUESDAY, AUGUST 12 cont.

	SPEAKER	***[ABSTRACT #] TITLE****
3:45 – 4:00	Nemeth, N.	[39] NATURAL AND EXPERIMENTAL WEST NILE VIRUS INFECTION IN FOUR RAPTOR SPECIES
4:00 – 4:15	Hanni, K.D.	[40] EVALUATION OF SUCCESS OF A SEA OTTER REHABILITATION PROGRAM AND IMPLICATIONS FOR POPULATION MANAGEMENT
4:15 – 4:30	Hwang, Y.T.	[41] DYNAMICS OF RABIES AND STRIPED SKUNK (*MEPHITIS MEPHITIS*) POPULATIONS: AN INDICATION OF DISEASES AS POPULATION REGULATION MECHANISM
4:30 – 4:45	Holmes, B.E.	[42] FLEA COMMUNITIES AND SMALL MAMMALS IN PHILLIPS COUNTY, MONTANA: IMPLICATIONS FOR THE MAINTENANCE OF SYLVATIC PLAGUE
4:45 – 5:00	Farmer, K.	[43] PHENOTYPIC DIFFERENCES IN SEVERAL ISOLATES OF THE HOUSE FINCH STRAIN OF *MYCOPLASMA GALLISEPTICUM*

WEDNESDAY, AUGUST 13

8:00 – 12:00 **GENERAL SESSION B**

Moderator: Nigel Caulkett

	SPEAKER	***[ABSTRACT #] TITLE****
8:00 – 8:15	Fauquier, D.	[44] CAUSES OF MORTALITY IN BOTTLENOSE DOLPHINS (*TURSIOPS TRUNCATUS*) STRANDED ALONG CENTRAL SOUTHWEST FLORIDA FROM 1985-2002
8:15 – 8:30	Forbes, L.B.	[45] THE OCCURRENCE AND FOOD SAFETY IMPLICATIONS OF TRICHINELLOSIS IN MARINE MAMMALS
8:30 – 8:45	Gajadhar, A.A.	[46] SUSCEPTIBILITY OF GREY SEALS TO *TOXOPLASMA GONDII* OOCYSTS, AND FOOD SAFETY IMPLICATIONS
8:45 – 9:00	Rijks, J.M.	[47] DESCRIPTION OF THE PHOCINE DISTEMPER IN THE NETHERLANDS DURING 2002
9:00 – 9:15	Uhart, M.M.	[48] IMMOBILIZATION OF FREE-RANGING MALE SOUTHERN SEA LIONS (*OTARIA BYRONIA*) WITH TILETAMINE-ZOLAZEPAM AND ISOFLURANE

WEDNESDAY, AUGUST 13 cont.

	SPEAKER	***[ABSTRACT #] TITLE****
9:15 – 9:30	Measures, L.	[49] MARINE MAMMALS AND 'WILDLIFE REHABILITATION' PROGRAMS
9:30 – 9:45	Beckmen, K.B.	[50] THE EFFECTS OF ENVIRONMENTAL CONTAMINANTS ON IMMUNE FUNCTION AND HEALTH IN FREE-RANGING PINNIPEDS IN ALASKA
9:45 – 10:00	Richey, L.	[51] PATHOLOGY, MICROBIOLOGY, AND CLUTCH VIABILITY OF FLORIDA AMERICAN ALLIGATOR (*ALLIGATOR MISSISSIPPIENSIS)* EMBRYOS AND NEONATES NATURALLY EXPOSED TO ORGANOCHLORINES
10:00 – 10:30	***BREAK***	
10:30 – 10:45	Wheler, C.L.	[52] COMPARISON OF CHOLINESTERASE LEVELS BETWEEN CAPTIVE AND WILD JUVENILE BURROWING OWLS (*AHTENE CUNICULARIA*)
10:45 – 11:00	Smits, J.E.G.	[53] DIETARY, AND SPATIAL INFLUENCES ON CONTAMINANT LEVELS AND RELATED BIOLOGICAL EFFECTS IN TREE SWALLOWS (*TACHYCINETA BICOLOR*) IN POINT PELEE NATIONAL PARK, ONTARIO
11:00 – 11:15	Fischer, J.R.	[54] VACUOLAR MYELINOPATHY OUTBREAKS IN MULTIPLE SPECIES AT A SOUTHEASTERN RESERVOIR
11:15 – 11:30	Campbell, G.D.	[55] SALMONELLOSIS IN PASSERINE BIRDS IN ONTARIO, CANADA, 1996-2002
11:30 – 11:45	Driscoll, C.P.	[56] 2001 MARYLAND MORBIDITY EVENT INVOLVING MICROCYSTIN TOXICITY AND STEATITIS IN GREAT BLUE HERONS (*ARDEA HERODIAS*)
11:45 – 12:00	Black, S.	[57] SHORT-TERM TRANQUILIZATION OF SANDHILL CRANES (*GRUS CANADENSIS*) USING TRIAZOLAM
12:00 – 1:30	***LUNCH***	
1:30 – 4:30	**GENERAL SESSION C** Moderator: Margo Pybus	
	SPEAKER	***[ABSTRACT #] TITLE****
1:30 – 1:45	Cook, W.	[58] TESTING RECOMMENDATIONS FOR WILD TURKEYS

WEDNESDAY, AUGUST 13 cont.

	SPEAKER	***[ABSTRACT #] TITLE****
1:45 – 2:00	Andrews, S.	[59] THE CANADA DATABASE OF ANIMAL PARASITES (CDAP): MONITORING PARASITES IN WILDLIFE IN CANADA
2:00 – 2:15	Hegglin, D.	[60] THE RISE OF URBAN FOXES AND THE ZOONOTIC PARASITE *ECHINOCOCCUS MULTILOCULARIS* – ECOLOGICAL AND EPIDEMIOLOGICAL ASPECTS OF AN URBAN PARASITE LIFE CYCLE
2:15 – 2:30	Jessup, D.A.	[61] UPDATE ON HEALTH, RECOVERY AND MANAGEMENT ISSUES FOR THE SOUTHERN SEA OTTER (*ENHYDRA LUTRIS NEREIS*)
2:30 – 2:45	Gavier-Widen, D.	[62] NECROTIZING ENCEPHALITIS IN SWEDISH ARCTIC FOXES (*ALOPEX LAGOPUS*)
2:45 – 3:00	Brown, C.M.	[63] INVESTIGATION OF A SEASONAL SEPTIC ARTHRITIS IN STRIPED SKUNKS (*MEPHITIS MEPHITIS*) ON CAPE COD
3:00 – 3:30	***BREAK***	
3:30 – 3:45	Miera, V.	[64] SUSCEPTIBILITY OF SALAMANDERS AND FROGS TO CHYTRID INFECTIONS
3:45 – 4:00	Raffel, T.R.	[65] STRESS-INDUCED IMMUNOSUPPRESSION IN RED-SPOTTED NEWTS: EFFECTS OF PHYSIOLOGICAL AND CHEMICAL STRESSORS
4:00 – 4:15	Brunner, J.	[66] THE ECOLOGY OF A TIGER SALAMANDER RANAVIRUS
4:15 – 4:30	Howerth, E.W.	[67] *ICHTHYOPHONUS*-LIKE INFECTION IN A MARBLED SALAMANDER (*AMBYSTOMA OPACUM*) AND A SHORT REVIEW OF MICROORGANISMS IN THE CLASS MESOMYCETOZOEA

THURSDAY AUGUST 14

8:00 – 12:00	**LARGE UNGULATES SESSION A** Moderator: Helen Schwantje	
	SPEAKER	***[ABSTRACT #] TITLE****
8:00 – 8:15	Gibbs, P.	[68] FOOT-AND-MOUTH DISEASE AND DEER: REFLECTIONS ON THE 2001EPIDEMIC IN THE UK AND IMPLICATIONS FOR NORTH AMERICA
	SPEAKER	***[ABSTRACT #] TITLE****
8:15 – 8:30	Palmer, M.V.	[69] WEST NILE VIRUS INFECTION IN REINDEER (*RANGIFER TARANDUS*)
8:30 – 8:45	Kreeger, T.J	[70] BRUCELLOSIS IN CAPTIVE ROCKY MOUNTAIN BIGHORN SHEEP (*OVIS CANADENSIS*) CAUSED BY *BRUCELLA ABORTUS* BIOVAR 4
8:45 – 9:00	Haigh, J.C.	[71] BRUCELLOSIS IN UGANDAN WILDLIFE: A PILOT STUDY
9:00 – 9:15	Godfroid, J.	[72] RFLP POLYMORPHISM IN *BRUCELLA* SP. BASED ON 3 NEWLY IDENTIFIED INSERTION SEQUENCES IN *BRUCELLA MELITENSIS* 16M
9:15 – 9:30	Roffe, T.J.	[73] PROTECTION AGAINST CHALLENGE-INDUCED ABORTION AND INFECTION BY *BRUCELLA ABORTUS* STRAIN 19 BY SINGLE CALFHOOD VACCINATION OF ELK (*CERVUS ELAPHUS*)
9:30 – 9:45	Lees, V.W.	[74] EPIDEMIOLOGY OF BOVINE TUBERCULOSIS IN HARVESTED ELK (*CERVUS ELAPHUS MANITOBENSIS*) NEAR RIDING MOUNTAIN NATIONAL PARK, MANITOBA, CANADA
9:45 – 10:00	Bergeson, D.	[75] BOVINE TUBERCULOSIS IN THE RIDING MOUNTAIN NATIONAL PARK REGION
10:00 – 10:30	***BREAK***	
10:30 – 10:45	Waters, W.R.	[76] EXPERIMENTAL INFECTION OF REINDEER (*RANGIFER TARANDUS*) WITH *MYCOBACTERIUM BOVIS*: DIAGNOSTIC IMPLICATIONS
10:45 – 11:00	Godfroid, J.	[77] MORTALITIES DUE TO MYCOBACTERIAL INFECTIONS IN WILD RED DEER (*CERVUS ELAPHUS*) IN BELGIUM

THURSDAY AUGUST 14 cont.

	SPEAKER	***[ABSTRACT #] TITLE****
11:00 – 11:15	Hars, J.	[78] FIRST ISOLATION OF *MYCOBACTERIUM BOVIS* FROM FREE-LIVING WILD BOARS (*SUS SCROFA*) AND RED DEER (*CERVUS ELAPHUS*) IN FRANCE
11:15 – 11:30	Cattet, M.R.L.	[79] INTRANASAL ADMINISTRATION OF SEDATIVE-TYPE DRUGS TO REDUCE STRESS IN ELK CAPTURED BY NET GUN
11:30 – 11:45	Haigh, J.C.	[80] A PRELIMINARY REPORT ON THE USE OF THIOFENTANYL OXALATE FOR THE CAPTURE OF UGANDA KOB
11:45 – 12:00	Perera, B.V.P.	[81] WILD ELEPHANT (*ELEPHAS MAXIMUS MAXIMUS*) IMMOBILIZATION PROCEDURES FOR TREATMENTS – A REVIEW OF 27 CASES
12:00 – 1:30	***LUNCH***	
1:30 – 5:00	**LARGE UNGULATES SESSION B**	
	Moderator: Stacy Tessaro	
	SPEAKER	***[ABSTRACT #] TITLE****
1:30 – 1:45	Artois, M.	[82] INTERACTIONS BETWEEN CLASSICAL SWINE FEVER VIRUS AND WILD BOAR (*SUS SCROFA*) IN FRANCE; TEN YEARS OF SURVEY: 1992-2002
1:45 – 2:00	Gerhold, R.W.	[83] RETROSPECTIVE REVIEW OF DEMODECTIC MANGE AND DERMATOPHILOSIS IN SOUTHEASTERN WHITE-TAILED DEER *(ODOCOILEUS VIRGINAINUS*)
2:00 – 2:15	Rose, K.	[84] *LEISHMANIA* SPECIES ASSOCIATED WITH NON-SUPPURATIVE DERMATITIS IN RED KANGAROOS (*MACROPUS RUFUS*) IN AUSTRALIA'S NORTHERN TERRITORY
2:15 – 2:30	Cornish, T.	[85] HERD HEALTH SURVEY AND ISOLATION OF A NEW PESTIVIRUS OF PRONGHORN (*ANTILOCAPRA AMERICANA*) IN WYOMING
2:30 – 2:45	Wild, M.A.	[86] EFFECTS OF GNRH AGONIST (LEUPROLIDE) ON REPRODUCTION, BODY CONDITION, AND BEHAVIOR IN FREE-RANGING FEMALE WAPITI

THURSDAY AUGUST 14 cont.

	SPEAKER	***[ABSTRACT #] TITLE****
2:45 – 3:00	Ogunremi, O.	[87] SEROLOGICAL DIAGNOSIS OF *PARELAPHOSTRONGYLUS TENUIS* INFECTION IN MOOSE
3:00 – 3:30		***BREAK***
3:30 – 3:45	Powers, J.G.	[88] SURVEILLANCE AND MANAGEMENT OF CHRONIC WASTING DISEASE IN THE UNITS OF THE NATIONAL PARK SYSTEM
3:45 – 4:00	Grear, D.A.	[89] DEMOGRAPHIC PATTERNS OF CWD PREVALENCE IN A HIGH-DENSITY WHITE-TAILED DEER POPULATION
4:00 – 4:15	Jewell, J.E.	[90] AN APPARENT DUPLICATION OF THE PRION PROTEIN CODING SEQUENCE IN MULE DEER (*ODOCOILEUS HEMIONUS*)
4:15 – 4:30	Joly, D.O.	[91] SPATIAL DISTRIBUTION OF CHRONIC WASTING DISEASE IN FREE-RANGING WHITE-TAILED DEER IN WISCONSIN
4:30 – 4:45	Schettler, E.	[92] STUDIES ON TSE (CWD, BSE, AND SCRAPIE) IN CERVIDS FROM GERMANY – PRELIMINARY RESULTS
4:45 – 5:00	Hamir, A.	[93] EXPERIMENTAL INOCULATION OF TME, SCRAPIE, AND CWD TO RACCOONS (*PROCYON LOTOR*) AND THE UTILIZATION OF RACCOONS FOR STRAIN-TYPING OF UNKNOWN TSES IN THE UNITED STATES

CONFERENCE AGENDA

DATE AND TIME	EVENT	LOCATION
Saturday, August 9th		
8:00 am – 5:00 pm	WDA Strategic Planning	Kelsey Room
1:00 pm – 6:00 pm	IUCN – VSG Meeting	Carlton Room
Sunday, August 10th		
8:00 am – 5:00 pm	ACZM Preparatory Course	Rm. 1655, WCVM*
8:00 am – 5:00 pm	WDA Editorial and Council Meeting	Salon Batoche
9:00 am – 12:00 noon	IUCN – VSG Meeting	Carlton Room
1:00 pm – 6:00 pm	WWHC Meeting	Kelsey Room
4:00 pm – 9:00 pm	Registration	William Pascoe Room
6:00 pm – 7:00 pm	Student Reception	William Pascoe Room
7:00 pm – 11:00 pm	Welcoming Reception Music by: The Lighthouse Penguins	William Pascoe Room
Monday, August 11th		
7:30 am – 12:00 noon	Registration	Convention Floor Foyer
8:30 am – 9:00 am	Welcome and General Introduction	Adam Ballroom
8:30 am – 5:00 pm	Poster Set-Up and Display	Convention Floor Foyer
9:00 am – 10:00 am	Diseases and Populations Session	Adam Ballroom
10:00 am – 10:30 am	Break	Convention Floor Foyer
10:30 am – 12:00 noon	Diseases and Populations Session cont.	Adam Ballroom
12:00 noon – 1:30 pm	Lunch	
1:30 pm – 3:00 pm	General Session A	Adam Ballroom
3:00 pm – 3:30 pm	Break	Convention Floor Foyer
3:30 pm – 4:30 pm	General Session A cont.	Adam Ballroom
5:00 pm – 8:00 pm	CAZWV Meeting	Kelsey Room
5:00 pm – 8:00 pm	AAWV Meeting	Salon Batoche
7:00 pm – 11:00 pm	Auction	Adam Ballroom
Tuesday, August 12th		
7:30 am – 12:00 noon	Registration	LaRonge Room
8:00 am – 5:00 pm	Poster Display	Convention Floor Foyer
8:00 am – 8:45 am	Conservation Biology & Disease Invited Speaker – Dr. Paul Paquet	Adam Ballroom
8:45 am – 10:00 am	Conservation Biology & Disease Session	Adam Ballroom
10:00 am – 10:30 am	Break	Convention Floor Foyer
10:30 am – 12:00 noon	Conservation Biology & Disease Session cont.	Adam Ballroom
12:00 noon – 1:00 pm	Lunch	
1:00 pm – 3:00 pm	Student Research Presentations	Adam Ballroom
3:00 pm – 3:30 pm	Break	Convention Floor Foyer
3:30 pm – 5:00 pm	Student Research Presentations cont.	Adam Ballroom
5:30 pm – 6:30 pm	Bus Pick-up for Picnic (every 15 minutes)	Front of Delta Bessborough
5:30 pm – 9:00 pm	Picnic	Wanuskewin Heritage Park
Wednesday, August 13th		
6:30 am – 8:00 am	WDA Prayer Breakfast	Salon Batoche
8:00 am – 11:00 am	Registration	LaRonge Room
8:00 am – 5:00 pm	Poster Display	Convention Floor Foyer
8:00 am – 10:00 am	General Session B	Adam Ballroom
DATE AND TIME	**EVENT**	**LOCATION**

Wednesday, August 13th cont.

10:00 am – 10:30 am	Break	Convention Floor Foyer
10:30 am – 12:00 noon	General Session B cont.	Adam Ballroom
12:00 noon – 1:30 pm	Lunch	
1:30 pm – 3:00 pm	General Session C	Adam Ballroom
3:00 pm – 3:30 pm	Break	Convention Floor Foyer
3:30 pm – 4:30 pm	General Session C cont.	Adam Ballroom
4:30 pm – 5:30 pm	WDA Business Meeting	Adam Ballroom
4:30 pm – 6:30 pm	CCM – CCWHC Meeting	Kelsey Room
4:30 pm – 6:00 pm	Poster Take-Down	Convention Floor Foyer
6:00 pm – 7:00 pm	Cash Bar	Convention Floor Foyer
7:00 pm – 11:00 pm	WDA Banquet AAWV Speaker: Dr. William Karesh Music by: Vesty and the Vexations	Adam Ballroom

Thursday, August 14th

8:00 am – 10:30 am	Registration	LaRonge Room
8:00 am – 10:00 am	Large Ungulates Session A	Adam Ballroom
10:00 am – 10:30 am	Break	Convention Floor Foyer
10:30 am – 12:00 noon	Large Ungulates Session A cont.	Adam Ballroom
12:00 noon – 1:30 pm	Lunch	
1:30 pm – 3:00 pm	Large Ungulates Session B cont.	Battleford Room
3:00 pm – 3:30 pm	Break	Battleford Foyer
3:30 pm – 5:00 pm	Large Ungulates Session B cont.	Battleford Room

Friday, August 15th

7:30 am – 12:00 noon	Registration for Chronic Wasting Disease Workshop	Rm. 2302, WCVM*
8:00 am – 5:00 pm	International Workshop on Chronic Wasting Disease	Rm. 2302, WCVM*

* Location of Western College of Veterinary Medicine (WCVM) relative to Delta Bessborough Hotel is displayed on Saskatoon City map.

PRESENTATION SCHEDULE

MONDAY, AUGUST 11

8:30 – 9:00	**WELCOME AND GENERAL INTRODUCTION** Conference Chair: Ted Leighton	
9:00 – 9:15	**DISEASES AND POPULATIONS SESSION** Moderator: Judit Smits	
	SPEAKER	***[ABSTRACT #] TITLE****
9:15 – 9:30	Gilbert, M.	[1] THE ASIAN VULTURE CRASH: INVESTIGATING MORTALITY OF ORIENTAL WHITE-BACKED VULTURE (*GYPS BENGALENSIS*) IN PUNJAB PROVINCE, PAKISTAN
9:30 – 9:45	Aguirre, A.A	[2] SEROEPIDEMIOLOGY OF HAWAIIAN MONK SEALS: ARE INFECTIOUS DISEASES LIMITING POPULATION RECOVERY?
9:45 – 10:00	Cattet, M.R.L.	[3] ASSESSING ENVIRONMENTAL HEALTH – THE FOOTHILLS MODEL FOREST GRIZZLY BEAR RESEARCH PROJECT
10:00 – 10:30	***BREAK***	
10:30 – 10:45	Dufour, K.W.	[4] POPULATION-LEVEL IMPACTS OF AVIAN BOTULISM IN PRAIRIE CANADA: INSIGHTS FROM HUNTER RECOVERIES OF BANDED MALLARDS (*ANAS PLATYRHYNCHOS*)
10:45 – 11:00	Joly, D.O.	[5] BOVINE TUBERCULOSIS AND BRUCELLOSIS AS FACTORS LIMITING POPULATION GROWTH OF BISON IN NORTHERN CANADA
11:00 – 11:15	Lankester, M.	[6] PARASITE BARRIERS AND CONSEQUENCES OF BREACHING THEM
11:15 – 11:30	Samuel, M.D.	[7] TRANSMISSION MODELS AND POPULATION EFFECTS OF VECTOR-BORNE DISEASES: AVIAN MALARIA IN HAWAIIAN FOREST BIRDS
11:30 – 11:45	Schock, D.	[8] POPULATION-LEVEL DIFFERENCES IN SUSCEPTIBILITY TO INFECTIOUS DISEASES: AN AMPHIBIAN – VIRUS CASE STUDY
11:45 – 12:00	Mörner, T.	PREVIEW OF EUROPEAN WILDLIFE DISEASE ASSOCIATION MEETING, SWEDEN 2004
12:00 – 1:30	***LUNCH***	

MONDAY, AUGUST 11 cont.

1:30 – 4:30 **GENERAL SESSION A**

Moderator: Karen Machin

	SPEAKER	***[ABSTRACT #] TITLE****
1:30 – 1:45	Hunter, B.	[9] WEST NILE VIRUS DIE-OFF IN CAPTIVE NORTH AMERICAN OWLS IN ONTARIO, CANADA
1:45 – 2:00	Hars, J.	[10] SURVEILLANCE OF WEST NILE VIRUS ON THE AVIFAUNA IN THE SOUTH OF FRANCE
2:00 – 2:15	Van Riper, C. III	[11] HABITAT PARTITIONING BY NEOTROPICAL MIGRANT WARBLERS ALONG THE LOWER COLORADO RIVER CORRIDOR AND POTENTIAL INFLUENCES ON WEST NILE VIRUS TRANSMISSION
2:15 – 2:30	Barker, I.K.	[12] ENHANCED PASSIVE SURVEILLANCE FOR WEST NILE VIRUS IN DEAD WILD BIRDS, CANADA – 2002-2003
2:30 – 2:45	Barker, I.K.	[13] WEST NILE VIRUS INFECTION IN FREE-RANGING AND CAPTIVE WILD BIRDS AND MAMMALS, ONTARIO, CANADA, 2002
2:45 – 3:00	Ezenwa, V.O.	[14] USING RS/GIS TO ASSESS WILDLIFE AND HUMAN WEST NILE VIRUS DISEASE RISK
3:00 – 3:30		***BREAK***
3:30 – 3:45	Kuiken, T.	[15] DESCRIPTION OF THE HIGHLY PATHOGENIC AVIAN INFLUENZA A (H7N7) EPIDEMIC IN THE NETHERLANDS IN 2003
3:45 – 4:00	Ley, D.H.	[16] *MYCOPLASMA GALLISEPTICUM* CONJUNCTIVITIS IN HOUSE FINCHES (*CARPODACUS MEXICANUS*). CORRELATIONS AMONG CLINICAL SIGNS AND DETECTION BY POLYMERASE CHAIN REACTION FROM CONJUNCTIVAL AND CHOANAL SWABS
4:00 – 4:15	Campbell, G.D.	[17] TYPE E BOTULISM IN BIRDS ON LAKE HURON AND LAKE ERIE, 1998-2002
4:15 – 4:30	Moccia, R.D.	[18] PUTATIVE ROLE OF FISH IN THE EPIDEMIOLOGY OF AVIAN BOTULISM IN LAKE ERIE

TUESDAY, AUGUST 12

8:00 – 12:00	**CONSERVATION BIOLOGY AND DISEASE SESSION** Moderator: Catherine Soos	
	SPEAKER	***[ABSTRACT #] TITLE****
8:00 – 8:45	Paquet, P.C.	CONSERVATION BIOLOGY AND DISEASE INVITED SPEAKER
8:45 – 9:00	Hilsberg, S.	[19] CONSERVATION MEDICINE IN ACTION: ONGOING INVESTIGATIONS INTO A SEVERE DISEASE OUTBREAK IN THE NGORONGORO CRATER, TANZANIA DURING 2000/2001?
9:00 – 9:15	Loeffler, I.K.	[20] BIOMEDICAL SURVEY OF GIANT PANDAS IN CHINA *EX SITU*: OBSERVATIONS OF A STUNTED GROWTH SYNDROME, AND THE NEED FOR SEROEPIDEMIOLOGICAL STUDIES
9:15 – 9:30	Mansfield, K.	[21] DISEASE AS A FACTOR LIMITING COLUMBIA BASIN PYGMY RABBIT RECOVERY
9:30 – 9:45	Nishi, J.S.	[22] RISK ASSESSMENT AS A TOOL TO EVALUATE HEALTH STATUS OF A SALVAGED HERD OF CAPTIVE WOOD BISON
9:45 – 10:00	Dein, F.J.	[23] THE NBII WILDLIFE DISEASE INFORMATION NODE
10:00 – 10:30		***BREAK***
10:30 – 10:45	Raphael, B.L.	[24] VETERINARY INVOLVEMENT IN CHELONIAN CONSERVATION PROGRAMS THROUGH THE TURTLE SURVIVAL ALLIANCE
10:45 – 11:00	Sleeman, J.M.	[25] THE ROLE OF WILDLIFE CENTERS AND REHABILITATORS IN WILDLIFE DISEASE MONITORING
11:00 – 11:15	Work, T.M.	[26] PATHOLOGY OF NATIVE AND INTRODUCED REEF FISH IN HAWAII
11:15 – 11:30	Raphael, B.L.	[27] HEALTH MONITORING PROGRAMS ASSOCIATED WITH CONSERVATION OF THE GRAND CAYMAN IGUANA (*CYCLURA NUBILA LEWISI*)
11:30 – 11:45	Rose, K.	[28] AN EPIZOOTIC OF SYSTEMIC COCCIDIOSIS (*CARYOSPORA CHELONIAE*) IN GREEN TURTLES (*CHELONIA MYDAS*) ALONG COASTAL NSW – A MARINE INDICATOR OF DROUGHT

TUESDAY, AUGUST 12 cont.

	SPEAKER	***[ABSTRACT #] TITLE****
11:45 – 12:00	Dubay, S.	[29] BIGHORN SHEEP (*OVIS CANADENSIS*) DISEASES: A BRIEF LITERATURE REVIEW AND RISK ASSESSMENT FOR TRANSLOCATION
12:00 – 1:00	***LUNCH***	
1:00 – 5:00	**STUDENT RESEARCH PRESENTATIONS** Moderator: Bill Samuel	
	SPEAKER	***[ABSTRACT #] TITLE****
1:00 – 1:15	Tate, C.M. (2003 Student Research Award Recipent)	[30] EXPERIMENTAL INFECTIONS OF WHITE-TAILED DEER WITH *ANAPLASMA (EHRLICHIA) PHAGOCYTOPHILUM*, THE ETIOLOGIC AGENT OF HUMAN GRANULOCYTIC EHRLICHIOSIS
1:15 – 1:30	Jolles, A.E.	[31] TUBERCULOSIS IN AFRICAN BUFFALO (*SYNCERUS CAFFER*): POPULATION EFFECTS OF A CHRONIC DISEASE
1:30 – 1:45	Appelbee, A.	[32] TRANSMISSION OF ZOONOTIC ISOLATES OF *GIARDIA* AND *CRYPTOSPORIDIUM* TO HARP SEALS
1:45 – 2:00	Yabsley, M.J.	[33] MOLECULAR VARIATION IN THE VARIABLE LENGTH PCR TARGET (VLPT) AND 120-KDA ANTIGEN GENES OF *EHRLICHIA CHAFFEENSIS* FROM WHITE-TAILED DEER
2:00 – 2:15	Ellis, A.E.	[34] PATHOLOGY OF NATURAL WEST NILE VIRAL INFECTION OF RAPTORS IN GEORGIA
2:15 – 2:30	Varela, A.S.	[35] EVIDENCE OF TICK-BORNE DISEASE AGENTS IN LONE STAR TICKS (*AMBLYOMA AMERICANUM*) FROM NORTHEASTERN GEORGIA
2:30 – 2:45	Gibbs, S.E.J.	[36] WEST NILE VIRUS IN AVIAN SPECIES OF GEORGIA
2:45 – 3:00	Pollock, E.	[37] EVALUATION OF TISSUE CONTAMINANT CONCENTRATIONS AND SELECTED HEALTH PARAMETERS IN CARIBOU (*RANGIFER TARANDUS*) IN LABRADOR, CANADA
3:00 – 3:30	***BREAK***	
3:30 – 3:45	Gillespie, T.R.	[38] LONG-TERM EFFECTS OF LOGGING ON PARASITE DYNAMICS IN AFRICAN PRIMATE POPULATIONS

TUESDAY, AUGUST 12 cont.

	SPEAKER	*[ABSTRACT #] TITLE**
3:45 – 4:00	Nemeth, N.	[39] NATURAL AND EXPERIMENTAL WEST NILE VIRUS INFECTION IN FOUR RAPTOR SPECIES
4:00 – 4:15	Hanni, K.D.	[40] EVALUATION OF SUCCESS OF A SEA OTTER REHABILITATION PROGRAM AND IMPLICATIONS FOR POPULATION MANAGEMENT
4:15 – 4:30	Hwang, Y.T.	[41] DYNAMICS OF RABIES AND STRIPED SKUNK (*MEPHITIS MEPHITIS*) POPULATIONS: AN INDICATION OF DISEASES AS POPULATION REGULATION MECHANISM
4:30 – 4:45	Holmes, B.E.	[42] FLEA COMMUNITIES AND SMALL MAMMALS IN PHILLIPS COUNTY, MONTANA: IMPLICATIONS FOR THE MAINTENANCE OF SYLVATIC PLAGUE
4:45 – 5:00	Farmer, K.	[43] PHENOTYPIC DIFFERENCES IN SEVERAL ISOLATES OF THE HOUSE FINCH STRAIN OF *MYCOPLASMA GALLISEPTICUM*

WEDNESDAY, AUGUST 13

8:00 – 12:00 **GENERAL SESSION B**

Moderator: Nigel Caulkett

	SPEAKER	*[ABSTRACT #] TITLE**
8:00 – 8:15	Fauquier, D.	[44] CAUSES OF MORTALITY IN BOTTLENOSE DOLPHINS (*TURSIOPS TRUNCATUS*) STRANDED ALONG CENTRAL SOUTHWEST FLORIDA FROM 1985-2002
8:15 – 8:30	Forbes, L.B.	[45] THE OCCURRENCE AND FOOD SAFETY IMPLICATIONS OF TRICHINELLOSIS IN MARINE MAMMALS
8:30 – 8:45	Gajadhar, A.A.	[46] SUSCEPTIBILITY OF GREY SEALS TO *TOXOPLASMA GONDII* OOCYSTS, AND FOOD SAFETY IMPLICATIONS
8:45 – 9:00	Rijks, J.M.	[47] DESCRIPTION OF THE PHOCINE DISTEMPER IN THE NETHERLANDS DURING 2002
9:00 – 9:15	Uhart, M.M.	[48] IMMOBILIZATION OF FREE-RANGING MALE SOUTHERN SEA LIONS (*OTARIA BYRONIA*) WITH TILETAMINE-ZOLAZEPAM AND ISOFLURANE

WEDNESDAY, AUGUST 13 cont.

	SPEAKER	***[ABSTRACT #] TITLE****
9:15 – 9:30	Measures, L.	[49] MARINE MAMMALS AND 'WILDLIFE REHABILITATION' PROGRAMS
9:30 – 9:45	Beckmen, K.B.	[50] THE EFFECTS OF ENVIRONMENTAL CONTAMINANTS ON IMMUNE FUNCTION AND HEALTH IN FREE-RANGING PINNIPEDS IN ALASKA
9:45 – 10:00	Richey, L.	[51] PATHOLOGY, MICROBIOLOGY, AND CLUTCH VIABILITY OF FLORIDA AMERICAN ALLIGATOR (*ALLIGATOR MISSISSIPPIENSIS)* EMBRYOS AND NEONATES NATURALLY EXPOSED TO ORGANOCHLORINES
10:00 – 10:30		***BREAK***
10:30 – 10:45	Wheler, C.L.	[52] COMPARISON OF CHOLINESTERASE LEVELS BETWEEN CAPTIVE AND WILD JUVENILE BURROWING OWLS (*AHTENE CUNICULARIA*)
10:45 – 11:00	Smits, J.E.G.	[53] DIETARY, AND SPATIAL INFLUENCES ON CONTAMINANT LEVELS AND RELATED BIOLOGICAL EFFECTS IN TREE SWALLOWS (*TACHYCINETA BICOLOR*) IN POINT PELEE NATIONAL PARK, ONTARIO
11:00 – 11:15	Fischer, J.R.	[54] VACUOLAR MYELINOPATHY OUTBREAKS IN MULTIPLE SPECIES AT A SOUTHEASTERN RESERVOIR
11:15 – 11:30	Campbell, G.D.	[55] SALMONELLOSIS IN PASSERINE BIRDS IN ONTARIO, CANADA, 1996-2002
11:30 – 11:45	Driscoll, C.P.	[56] 2001 MARYLAND MORBIDITY EVENT INVOLVING MICROCYSTIN TOXICITY AND STEATITIS IN GREAT BLUE HERONS (*ARDEA HERODIAS*)
11:45 – 12:00	Black, S.	[57] SHORT-TERM TRANQUILIZATION OF SANDHILL CRANES (*GRUS CANADENSIS*) USING TRIAZOLAM
12:00 – 1:30		***LUNCH***
1:30 – 4:30		**GENERAL SESSION C** Moderator: Margo Pybus
	SPEAKER	***[ABSTRACT #] TITLE****
1:30 – 1:45	Cook, W.	[58] TESTING RECOMMENDATIONS FOR WILD TURKEYS

WEDNESDAY, AUGUST 13 cont.

	SPEAKER	***[ABSTRACT #] TITLE****
1:45 – 2:00	Andrews, S.	[59] THE CANADA DATABASE OF ANIMAL PARASITES (CDAP): MONITORING PARASITES IN WILDLIFE IN CANADA
2:00 – 2:15	Hegglin, D.	[60] THE RISE OF URBAN FOXES AND THE ZOONOTIC PARASITE *ECHINOCOCCUS MULTILOCULARIS* – ECOLOGICAL AND EPIDEMIOLOGICAL ASPECTS OF AN URBAN PARASITE LIFE CYCLE
2:15 – 2:30	Jessup, D.A.	[61] UPDATE ON HEALTH, RECOVERY AND MANAGEMENT ISSUES FOR THE SOUTHERN SEA OTTER (*ENHYDRA LUTRIS NEREIS*)
2:30 – 2:45	Gavier-Widen, D.	[62] NECROTIZING ENCEPHALITIS IN SWEDISH ARCTIC FOXES (*ALOPEX LAGOPUS*)
2:45 – 3:00	Brown, C.M.	[63] INVESTIGATION OF A SEASONAL SEPTIC ARTHRITIS IN STRIPED SKUNKS (*MEPHITIS MEPHITIS*) ON CAPE COD
3:00 – 3:30	***BREAK***	
3:30 – 3:45	Miera, V.	[64] SUSCEPTIBILITY OF SALAMANDERS AND FROGS TO CHYTRID INFECTIONS
3:45 – 4:00	Raffel, T.R.	[65] STRESS-INDUCED IMMUNOSUPPRESSION IN RED-SPOTTED NEWTS: EFFECTS OF PHYSIOLOGICAL AND CHEMICAL STRESSORS
4:00 – 4:15	Brunner, J.	[66] THE ECOLOGY OF A TIGER SALAMANDER RANAVIRUS
4:15 – 4:30	Howerth, E.W.	[67] *ICHTHYOPHONUS*-LIKE INFECTION IN A MARBLED SALAMANDER (*AMBYSTOMA OPACUM*) AND A SHORT REVIEW OF MICROORGANISMS IN THE CLASS MESOMYCETOZOEA

THURSDAY AUGUST 14

8:00 – 12:00 **LARGE UNGULATES SESSION A**

Moderator: Helen Schwantje

	SPEAKER	***[ABSTRACT #] TITLE****
8:00 – 8:15	Gibbs, P.	[68] FOOT-AND-MOUTH DISEASE AND DEER: REFLECTIONS ON THE 2001EPIDEMIC IN THE UK AND IMPLICATIONS FOR NORTH AMERICA

	SPEAKER	***[ABSTRACT #] TITLE****
8:15 – 8:30	Palmer, M.V.	[69] WEST NILE VIRUS INFECTION IN REINDEER (*RANGIFER TARANDUS*)
8:30 – 8:45	Kreeger, T.J	[70] BRUCELLOSIS IN CAPTIVE ROCKY MOUNTAIN BIGHORN SHEEP (*OVIS CANADENSIS*) CAUSED BY *BRUCELLA ABORTUS* BIOVAR 4
8:45 – 9:00	Haigh, J.C.	[71] BRUCELLOSIS IN UGANDAN WILDLIFE: A PILOT STUDY
9:00 – 9:15	Godfroid, J.	[72] RFLP POLYMORPHISM IN *BRUCELLA* SP. BASED ON 3 NEWLY IDENTIFIED INSERTION SEQUENCES IN *BRUCELLA MELITENSIS* 16M
9:15 – 9:30	Roffe, T.J.	[73] PROTECTION AGAINST CHALLENGE-INDUCED ABORTION AND INFECTION BY *BRUCELLA ABORTUS* STRAIN 19 BY SINGLE CALFHOOD VACCINATION OF ELK (*CERVUS ELAPHUS*)
9:30 – 9:45	Lees, V.W.	[74] EPIDEMIOLOGY OF BOVINE TUBERCULOSIS IN HARVESTED ELK (*CERVUS ELAPHUS MANITOBENSIS*) NEAR RIDING MOUNTAIN NATIONAL PARK, MANITOBA, CANADA
9:45 – 10:00	Bergeson, D.	[75] BOVINE TUBERCULOSIS IN THE RIDING MOUNTAIN NATIONAL PARK REGION
10:00 – 10:30		***BREAK***
10:30 – 10:45	Waters, W.R.	[76] EXPERIMENTAL INFECTION OF REINDEER (*RANGIFER TARANDUS*) WITH *MYCOBACTERIUM BOVIS*: DIAGNOSTIC IMPLICATIONS
10:45 – 11:00	Godfroid, J.	[77] MORTALITIES DUE TO MYCOBACTERIAL INFECTIONS IN WILD RED DEER (*CERVUS ELAPHUS*) IN BELGIUM

THURSDAY AUGUST 14 cont.

	SPEAKER	***[ABSTRACT #] TITLE****
11:00 – 11:15	Hars, J.	[78] FIRST ISOLATION OF *MYCOBACTERIUM BOVIS* FROM FREE-LIVING WILD BOARS (*SUS SCROFA*) AND RED DEER (*CERVUS ELAPHUS*) IN FRANCE
11:15 – 11:30	Cattet, M.R.L.	[79] INTRANASAL ADMINISTRATION OF SEDATIVE-TYPE DRUGS TO REDUCE STRESS IN ELK CAPTURED BY NET GUN
11:30 – 11:45	Haigh, J.C.	[80] A PRELIMINARY REPORT ON THE USE OF THIOFENTANYL OXALATE FOR THE CAPTURE OF UGANDA KOB
11:45 – 12:00	Perera, B.V.P.	[81] WILD ELEPHANT (*ELEPHAS MAXIMUS MAXIMUS*) IMMOBILIZATION PROCEDURES FOR TREATMENTS – A REVIEW OF 27 CASES
12:00 – 1:30	***LUNCH***	
1:30 – 5:00	**LARGE UNGULATES SESSION B** Moderator: Stacy Tessaro	
	SPEAKER	***[ABSTRACT #] TITLE****
1:30 – 1:45	Artois, M.	[82] INTERACTIONS BETWEEN CLASSICAL SWINE FEVER VIRUS AND WILD BOAR (*SUS SCROFA*) IN FRANCE; TEN YEARS OF SURVEY: 1992-2002
1:45 – 2:00	Gerhold, R.W.	[83] RETROSPECTIVE REVIEW OF DEMODECTIC MANGE AND DERMATOPHILOSIS IN SOUTHEASTERN WHITE-TAILED DEER *(ODOCOILEUS VIRGINAINUS*)
2:00 – 2:15	Rose, K.	[84] *LEISHMANIA* SPECIES ASSOCIATED WITH NON-SUPPURATIVE DERMATITIS IN RED KANGAROOS (*MACROPUS RUFUS*) IN AUSTRALIA'S NORTHERN TERRITORY
2:15 – 2:30	Cornish, T.	[85] HERD HEALTH SURVEY AND ISOLATION OF A NEW PESTIVIRUS OF PRONGHORN (*ANTILOCAPRA AMERICANA*) IN WYOMING
2:30 – 2:45	Wild, M.A.	[86] EFFECTS OF GNRH AGONIST (LEUPROLIDE) ON REPRODUCTION, BODY CONDITION, AND BEHAVIOR IN FREE-RANGING FEMALE WAPITI

THURSDAY AUGUST 14 cont.

	SPEAKER	***[ABSTRACT #] TITLE****
2:45 – 3:00	Ogunremi, O.	[87] SEROLOGICAL DIAGNOSIS OF *PARELAPHOSTRONGYLUS TENUIS* INFECTION IN MOOSE
3:00 – 3:30		***BREAK***
3:30 – 3:45	Powers, J.G.	[88] SURVEILLANCE AND MANAGEMENT OF CHRONIC WASTING DISEASE IN THE UNITS OF THE NATIONAL PARK SYSTEM
3:45 – 4:00	Grear, D.A.	[89] DEMOGRAPHIC PATTERNS OF CWD PREVALENCE IN A HIGH-DENSITY WHITE-TAILED DEER POPULATION
4:00 – 4:15	Jewell, J.E.	[90] AN APPARENT DUPLICATION OF THE PRION PROTEIN CODING SEQUENCE IN MULE DEER (*ODOCOILEUS HEMIONUS*)
4:15 – 4:30	Joly, D.O.	[91] SPATIAL DISTRIBUTION OF CHRONIC WASTING DISEASE IN FREE-RANGING WHITE-TAILED DEER IN WISCONSIN
4:30 – 4:45	Schettler, E.	[92] STUDIES ON TSE (CWD, BSE, AND SCRAPIE) IN CERVIDS FROM GERMANY – PRELIMINARY RESULTS
4:45 – 5:00	Hamir, A.	[93] EXPERIMENTAL INOCULATION OF TME, SCRAPIE, AND CWD TO RACCOONS (*PROCYON LOTOR*) AND THE UTILIZATION OF RACCOONS FOR STRAIN-TYPING OF UNKNOWN TSES IN THE UNITED STATES

* Abstracts from poster presentations ([94] to [111]) follow abstracts from stage presentations beginning at p. 126 in the proceedings.

ABSTRACTS FROM STAGE PRESENTATIONS

[1] THE ASIAN VULTURE CRASH: INVESTIGATING MORTALITY OF ORIENTAL WHITE-BACKED VULTURE, *GYPS BENGALENSIS* IN PUNJAB PROVINCE, PAKISTAN

MARTIN GILBERT, J. LINDSAY OAKS, MUNIR Z. VIRANI, RICHARD T. WATSON, ALEEM A. KHAN, SHAKEEL AHMED, JAMSHED CHAUDHRY, MUHAMMAD ARSHAD, SHAHID MAHMOOD and AHMAD ALI.

Until recently, the Oriental White-backed Vulture *Gyps bengalensis* was considered among the most abundant species of large raptor in the world. Over the last 10-15 years large scale declines in the populations of the *Gyps* vultures have been reported across the Indian subcontinent. This long-term study provides data on the mortality rates and patterns through the continuous monitoring of three large colonies of Oriental White-backed Vultures (n=758, 413, and 445 breeding pairs respectively in 2000) within the Punjab Province, Pakistan. The activity and productivity of nests within fixed study sites were determined over three consecutive breeding seasons. All dead vultures were collected and removed from transects during the breeding and non-breeding seasons. Mortality of adult vultures at each site has continued at a rate indicative of a population in decline. Numbers of active nests at the three study colonies have declined by 33%, 88% and 97% from the 2000/01 to the 2002/03 nesting season. The majority (~80%) of birds examined post mortem have been associated with a clinical syndrome of renal failure (visceral gout). Veterinary diagnostic investigation began in November 2000. Extensive testing including histopathology, toxicology, bacteriology, virology, and electron microscopy excluded all typical recognized causes of renal failure in wild birds. Consequently studies have focused on non-conventional aetiologies. Latest results will be discussed.

[2] SEROEPIDEMIOLOGY OF HAWAIIAN MONK SEALS: ARE INFECTIOUS DISEASES LIMITING POPULATION RECOVERY?

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The Hawaiian monk seal (*Monachus schauinslandi)* is considered the most endangered pinniped in U.S. waters. As a result of a severe population decline, the species was protected in 1972. Population trends most likely will continue to decline due to high juvenile mortality and low reproductive recruitment at French Frigate Shoals, North Western Hawaiian Islands, where the largest of the six main reproductive populations can be found. Captive care and release programs were an integral part of management efforts to conserve the species from 1981 to 1994. Three strategies have been used including on-site protection and release, direct translocation from one site to another, and transport to Oahu for rehabilitation followed by release into the wild population. Rehabilitation efforts have been halted by the development of an ocular condition of unknown etiology affecting 12 female pups captured during the summer of 1995. The seals have not been released because of the risk of spreading the disease to the wild population. Serum specimens were obtained from 332 Hawaiian monk seals (*Monachus schauinslandi*) captured at six sites in the northwestern Hawaiian Islands between 1997-2001. Specimens were screened for antibodies to some potential pathogenic viruses, bacteria and parasites known to cause morbidity and mortality in other marine mammal species. Antibody titers were found to phoocine herpes virus 1 (PH-1) when using the ELISA test. Positive titers were detected to *L. Bratislava, L. hardjo, L. icterohaemorrhagica* and *L. Pomona* at several sites. Positive titers to *Brucella abortus* ranged from one to 50% at all populations depending on the serologic test employed except Kure Atoll where no seropositives were identified. Prevalence of adult seals (84%) compared to weanlings (47%) and juveniles (51%) but was similar to most sites. Antibodies to *Toxoplasma gondii* and *D. immitus* were insignificant. All specimens were serologically negative to jCDV, PMV, DMV and PMV, PH-1 when using the virus neutralization test, seal influenza virus, canine adenovirus, caliciviruses (32 serotypes), vesicular exanthema of swine viruses (17 serotypes), fur seal adenovirus, California sea lion rotavirus, walrus enterovirus 7-19 and *Brucella canis*. Isolation attempts for herpesvirus, *Brucella* and *Chlamydophila* have been unsuccessful. This is the first serologic epidemiology study of Hawaiian monk seals in the wild. Serologic testing remains to be the strongest tool to avoid the introduction of emerging infectious diseases and endemic pathogens. These findings support the need to continuous monitoring and evaluation of health in future conservation efforts of Hawaiian monk seals. Minimizing the risk of introducing diseases to a population can be accomplished by using the best available diagnostics to test seals for evidence of infection or disease; conduct epidemiological surveys; and taking a proactive approach to disease management, i.e. treatment or immunization as technology becomes available. The current knowledge of biomedical impact on declining survival and the role of disease in the success or failure of translocation experiments in the monk seal population are unknown. Epidemiological research needs to continue to provide recommendations for future translocation efforts.

[3] ASSESSING ENVIRONMENTAL HEALTH – THE FOOTHILLS MODEL FOREST GRIZZLY BEAR RESEARCH PROJECT

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For many years the health of animal populations has typically been assessed with the tools of population dynamics: estimations of trends in abundance, mortality, and reproductive rates. However, for species like grizzly bears (*Ursus arctos*) that have long generation times, this approach is expensive and often too slow and insensitive to provide an early warning about the impact of environmental stressors such as pollution, human activities, and climatic warming. Further, although evident in some individuals, signs of compromised health (e.g., disease, loss of condition, failed reproduction) may be difficult to recognize and quantify at the population level. As a consequence, efforts to link environmental stress with the health of populations are often speculative, lacking in convincing supportive data. The Foothill Model Forest Grizzly Bear Research Project focuses on management issues and questions by assessing grizzly bear populations, bear response to human activities, and habitat conditions to provide land managers with tools to integrate grizzly bear "needs" into the land management decision-making framework. A major objective of the project is to detect and assess the effects of environmental (chronic) stress on the health of grizzly bears in the study area by seeking biochemical and molecular indicators to quantify the level of chronic stress in a bear and by applying multivariate analyses on existing data to seek significant associations among measures of the health and environment of grizzly bears.

[4] POPULATION-LEVEL IMPACTS OF AVIAN BOTULISM IN PRAIRIE CANADA: INSIGHTS FROM HUNTER RECOVERIES OF BANDED MALLARDS (*ANAS PLATYRHYNCHOS*)

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Catastrophic losses of waterfowl to avian botulism are well documented, but few studies have attempted to quantify impacts of botulism on population parameters. We used band-recovery data from adult mallards (*Anas platyrhynchos*) trapped at nine botulism outbreak sites in prairie Canada (late June - early August, 1998-2000) to determine the extent to which exposure to botulism during the post-breeding season results in reduced late-summer survival. Specifically, we tested the prediction that direct recovery rates (indicative of survival during the period between banding and harvest) would be lower among individuals banded at outbreak sites (n = 6594) than among conspecific individuals banded at non-outbreak control sites (n = 15241). Banding data from two of the three prairie provinces (Saskatchewan and Manitoba) were uniformly consistent in supporting the predicted association between exposure to botulism and direct recovery probability: recovery rates of mallards banded at outbreak sites in these areas were 14-44% lower than expected based on comparisons with control data. This pattern was evident among both males and females and occurred in all three years of study. In contrast, results from a third province (Alberta) were mixed and generally did not support the predicted association. Overall, our results support the suggestion that exposure to botulism during the post-breeding season can have a measurable impact on survival at the population level. However, our findings from Alberta suggest a need for caution when generalizing from these results. Future investigations should seek to (1) refine our ability to predict when and where botulism-related mortality is likely to be most severe, and (2) define population effects of botulism in terms of the size of the population at risk.

[5] BOVINE TUBERCULOSIS AND BRUCELLOSIS AS FACTORS LIMITING POPULATION GROWTH OF BISON IN NORTHERN CANADA

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Two diseases of domestic cattle, tuberculosis (*Mycobacterium bovis*) and brucellosis (*Brucella abortus*), were introduced to Wood Buffalo National Park, Canada in the late 1920s. Forty-nine percent of WBNP bison tested in the winters of 1997 -1999 tested positive on either the buffered-plate or complement fixation test for brucellosis. Forty-five percent of bison tested positive using the caudal fold test and fluorescent polarization assay for tuberculosis. Prevalence for both diseases increases with age and males are more likely to test positive for tuberculosis. These diseases are endemic and unlikely to disappear as the population declines. Each disease has the potential to alter the predator-prey relationship between wolves *(Canis lupus*) and bison. This effect can act in both direct and indirect fashions. Tuberculosis can reduce exertion tolerance in bison through the development of granulatomous pulmonary lesions, resulting in an impaired ability to flee during a wolf attack. Infection of joints by brucellosis and subsequent arthritis can impair the ability of a bison to avoid predation by reducing running ability. Disease can also impact the predator-prey dynamics in an indirect manner. Disease may reduce the overall productivity of the bison herd by reducing fecundity and increasing mortality rates from non-predation sources. As predation rate may vary with bison density in a nonlinear fashion as proposed for moose (*Alces* alces) and wolf systems, a small reduction in herd productivity could drastically alter the equilibrium density of bison. In particular, the bison population may be shifted from a high, food-regulated density where predation is density-independent or inversely density-dependent, to a low predator-regulated density where predation is strongly density-dependent. This permanent presence of two economically important diseases in northern Canada results in a conflict with respect to further recovery of bison as a species, and migration of diseased bison from WBNP presents a quantifiable, albeit low, risk of transmitting the disease to nearby, disease-free populations.

[6] PARASITE BARRIERS AND CONSEQUENCES OF BREACHING THEM

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Two foreign, metastrongyloid nematodes occur in Newfoundland where they were introduced from Europe. The French heartworm (*Angiostrongylus vasorum*) infects the pulmonary arteries of native foxes (*Vulpes vulpes*) with no measurable effects but in dogs causes severe respiratory disease and disseminated intravascular coagulation. It=s distribution is not restricted by geography as previously thought nor by competitive interference from the fox lungworm (*Crenosoma vulpis*). Instead, *A. vasorum* persists only in the extreme southeastern portion of the island province due to its mild winters. First-stage larvae were quickly killed by freezing and the parasite was absent where mean winter temperatures (December to February) were below - 4 C. Likely the French heartworm would similarly be restricted to mild climatic zones if it were accidentally translocated to mainland North America.

The reindeer muscleworm (*Elaphostrongylus rangiferi*) occurs in the central nervous system and among skeletal muscles of native caribou (*Rangifer tarandus caribou*) and is responsible for periodic epizootics of cerebrospinal elaphostrongylosis (CSE), a neuromuscular disease affecting young animals. It occurs in northern and central Newfoundland but not on the Avalon Peninsula, probably because a narrow isthmus of developed land discourages travel by large ungulates. However, in the early 1990s, the parasite inexplicably appeared in the previously unexposed Avalon caribou herd with dramatic results. Instead of only young animals succumbing to CSE, adults were affected as well. The herd, initially numbering over 7000, declined to less than 2500 within 3 years. The opportunity to observe this initial infection of a naive herd was a stark reminder of the important role of concomitant immunity in mitigating the impact of disease caused by certain helminths. This lesson also underscores an often over-looked aspect of the risk to naive wild animal populations presented by new and emerging diseases.

[7] TRANSMISSION MODELS AND POPULATION EFFECTS OF VECTOR-BORNE DISEASES: AVIAN MALARIA IN HAWAIIAN FOREST BIRDS

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Determining the rates of disease transmission is of primary importance in understanding the epidemiology and dynamics of most disease systems. In conjunction with studies on vector-borne malaria in Hawaiian forest birds we are currently developing generalized disease transmission models that can incorporate data from both concurrent infection (e.g., parasitemia, viremia) and cumulative infection (e.g., seroconversion). These models can be applied to known age animals, to marked animals studied over time, and to cross-sectional data collected from populations. Transmission rates can be corrected to account for bias that occurs when acute infection causes mortality that reduces the probability of detecting recently infected individuals. These models are illustrated by estimating seasonal transmission of avian malaria in Hawaiian forest birds. By combining information on disease transmission rates and case fatality rates, with the number of susceptible and immune animals, we can estimate the effects of disease of wild populations.

[8] POPULATION-LEVEL DIFFERENCES IN SUSCEPTIBILITY TO INFECTIOUS DISEASES: AN AMPHIBIAN – VIRUS CASE STUDY

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Heightened awareness and surveillance account for the apparent "emergence" of some diseases, but in most cases fundamental changes in disease dynamics have occurred including changes in geographic range, host range, and/or virulence of the pathogen. Factors responsible for changes in disease dynamics are not well understood but hypothesized mechanisms range from changes in climate patterns to introduction of exotic species. Identifying patterns of susceptibility and elucidating the factors responsible for those patterns are key to understanding what precipitates infectious disease emergence and how to manage the effects. Our research focuses on identifying and understanding factors that can generate population-level differences in host susceptibility to infectious diseases. In particular, we are investigating tiger salamanders (*Ambystoma tigrinum*) and a group of closely-related lethal salamander viruses, Ranaviruses, to explore susceptibility in the larger context of Host-Pathogen theory.

Tiger salamanders are found throughout much of North America in habitats ranging from wetlands in alpine meadows to artificial ponds on the Great Plains. Ranaviruses cause tiger salamander die-offs in all habitats across western North America. Preliminary genetic evidence of a long-term association between tiger salamanders (*Ambystoma tigrinum*) and Ranaviruses prompted a cross-infection laboratory study. Such long-term associations are expected to result in local adaptation: virus isolates should perform better (e.g., exhibit higher infection rates) in their natal tiger salamander populations than in tiger salamander populations from elsewhere. Tiger salamander eggs were collected from several ponds in each of two rural focal regions ~800 km apart in south-west Manitoba (MB) and south-central Saskatchewan (SK). Larvae were reared for 3 months and then larvae from each focal region were subjected to one of four treatments: i) a virus isolate from the SK focal region, ii) an isolate from the MB focal region, iii) an isolate from northern Arizona as an outgroup, or iv) no virus as a control treatment.

Rather than seeing evidence of local adaptation, the SK tiger salamanders were highly susceptible to all three virus isolates, while the MB tiger salamanders were resistant to all three isolates. The underlying cause(s) of the differences in susceptibility between regions is not obvious. Subsequent experiments suggest two likely explanations: 1) Some component of the SK environment is suppressing the salamanders' immune system, either acting on eggs directly or through the mother (i.e., mother is exposed and passes it to her eggs). 2) SK populations are suffering from detrimentally low genetic diversity (inbreeding depression). On-going research is testing which of these two hypotheses is likely driving the observed patterns in disease susceptibility.

[9] WEST NILE VIRUS DIE-OFF IN CAPTIVE NORTH AMERICAN OWLS IN ONTARIO, CANADA

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West Nile Virus (WNV) was identified as the agent responsible for a die-off of owls that occurred at a breeding and rehabilitation facility (The Owl Foundation) in Vineland, ON, Canada during July-September 2002. At the time of the outbreak this facility held 260 birds representing 17 species of owls and 2 species of falcons. By the middle of September 2002, 108 (43%) of the owls kept outdoors had died, making this the first and the largest outbreak of WNV in a captive collection of birds in Canada.

A serological survey of the outbreak survivors combined with the mortality results, demonstrated that over 87% of the birds in the facility were infected with the virus. Yet some species such as the Eastern Screech owl (*Otus asio*), were not clinically affected, while in others (mainly northern species such as Great Gray Owls (*Strix nebulosa*), Snowy Owls (*Nyctea scandiaca*), Northern Hawk Owls (*Surnia ulula*) and Boreal Owls (*Aegolius funereus*)) the mortality rate reached 100%. This supports the hypothesis that a marked difference in the susceptibility of different owl species to this virus exists.

The higher susceptibility of several, not closely related, northern species may be the result of their geographic isolation, perhaps leading to a less competent immune response. It is also possible that higher stress levels in these species (e.g. due to the high temperatures in the late summer) could have contributed to the overall outcome. Many of the birds were heavily infested with the Hippoboscid fly *Icosta americana*. The highest loads were seen once again in northern species. These exceeded 360 flies/bird in two Great Gray Owls, and must have been a major stress factor for these species. The possible role of the fly in the rapid spread of the disease and its capability of acting as either a mechanical and/or biological vector for WNV are still under investigation. Hippoboscid flies removed from dead owls were PCR positive for WNV. Microdissection together with PCR demonstrated that the virus was located in the fly's digestive tract but not in other organs such as the head, the salivary glands or the reproductive tract. In addition, 70 pools of fly pupae collected during the outbreak and 67 pools of unfed flies (collected as pupae and allowed to hatch) were tested by PCR for WNV. 3 pools of pupae and one pool of unfed flies were positive. These results may suggest that transovarian transmission of WNV exist in the Hippoboscid fly, however we cannot at this point rule out the possibility of sample contamination (e.g. by fecal material from the cage floors). During and after the outbreak tens of owls were vaccinated at TOF using the Fort Dodge© (equine) vaccine. Each bird was injected intramuscularly with 0.5cc of the vaccine (half the equine dose) 2-3 times, 3 weeks apart. Out of 14 owls that were initially negative and that were vaccinated at least twice, only 6 (43%) showed elevation in titer (as measured by ELISA) on rechecks 1-4 months after the last vaccination. Only 1 out of 11 owls that had positive titers before the first vaccination showed an elevated titer 3 month after the last of 3 vaccinations, while in the other 10 a marked decrease in antibody titer was noted.

[10] SURVEILLANCE OF WEST NILE VIRUS ON THE AVIFAUNA IN THE SOUTH OF FRANCE

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West Nile fever is a mosquito-borne viral disease, including wild birds as amplifying hosts. Human and horse, which sometimes may show lethal symptoms of encephalitis (dead-end infection), are accidental victims. The disease has recently been observed in Algeria (1994), Morocco (1996), Romania (1996), Italy (1998), Western Russia (1999), Israel (1997-2000) and the United States and Canada since 1999. The presence of the virus was also reported in mosquitoes in the Czech Republic (1997).

In France, it was described in 1962-65 in horses (500 clinical cases) and humans (19 clinical cases) living in the Camargue, a wetland situated in the South of France where *Culex modestus* mosquitoes were incriminated in the transmission of the virus. After an epidemiological silence of thirty-five years, the disease emerged in 2000 in the Camargue, not far from the town of Montpellier.

Between September 4th and November 15th 2000, the Pasteur Institute confirmed 76 cases in horses among 141 suspected. Twenty of them died. It seemed that horses were the only host sensitive to the disease, neither human clinical cases nor avian abnormal mortality have been reported in the area.

At the request of the Ministry of Agriculture, in September 2000, the French game and wildlife agency (Office national de la chasse et de la faune sauvage) started a preliminary survey to identify the wild bird species that were infected by the virus. Since no cases of mortality have been observed, it was rather difficult to select the bird species that should be investigated. Therefore, we selected mostly sedentary species with large populations: the house sparrow (*Passer domesticus*), the yellow-legged herring gull (*Larus cachinnans*), the black-headed gull (*Larus ridibundus*), the mallard (*Anas plathyrynchos*) and the black-billed magpie (*Pica pica*). 440 birds were captured in order to take a blood sample (and brain sample in the dead ones). RT-PCR was used for WN virus detection, ELISA and neutralisation tests for WN antibody detection. Neither virus nor antibodies was isolated in sparrows and gulls. Only low seroprevalences were detected in mallards (8%, n = 100) and in magpies (22%, n = 18).

In 2001 and 2002, the surveillance for West Nile virus was based on:

- Surveillance for encephalitis cases both in humans and horses particularly focussed in the Camargue area
- Viral detection in mosquitoes collected in the Camargue from July to October 2001,
- Surveillance for abnormal mortalities in wild birds
- Longitudinal serosurveys in sentinel birds theorically on a monthly basis from June to November, including 150 mallards and 150 chicken located in 30 sites distributed in all the Camargue area.

In 2001 and 2002, no clinical case was confirmed in humans (among 34 investigated suspicions) or in horse (among 50 suspicions); no virus was isolated among 997 mosquitoes pools tested. No abnormal mortality was observed in the avifauna. Only two seroconversions were detected in sentinel birds, one in October 2001 in a mallard and one in August 2002 in a chicken).

These results show that West Nile virus is still present in Camargue, circulating at a very low level within mosquitoes and birds. It is impossible to determine at that stage if the virus is endemic in the area or periodically introduced. Focus would be to isolate the virus to provide evidence of endemic maintenance.

[11] HABITAT PARTITIONING BY NEOTROPICAL MIGRANT WARBLERS ALONG THE LOWER COLORADO RIVER CORRIDOR AND POTENTIAL INFLUENCES ON WEST NILE VIRUS TRANSMISSION

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We examined foraging ecology of spring and fall migrant warblers in native and introduced vegetation habitat patches along the Lower Colorado River corridor, in an effort to predict future risks of West Nile Virus infections. Study areas were located on the Rio Hardy and Rio Colorado rivers in Sonora, Mexico, Cibola and Bill Williams National Wildlife Refuges in Arizona. From our census and mist-net capture data, we found that warbler species' arrival and departure dates differed among species, but were more predictable during the spring migration period. Plant species abundance and phenology patterns dramatically influenced location of warbler foraging. Preliminary analysis of invertebrate samples revealed significant differences, among tree species and particularly between native and introduced plant species. Hence, vector access to different bird species in a vegetation patch may be important factor in future West Nile Virus transmission to foraging migrants. We found that warbler species partitioned foraging habitat in similar manners during both migration periods, preferring native over introduced vegetation and thus potentially impacting WNV prevalences. Lucy's Warblers preferred the highest vegetation strata, while Yellow Warblers occurred primarily in the middle foliage regions. Orange-crowned Warblers were observed most often in the lower third of the vertical vegetation strata, while Black-throated Grey, Wilson's, Nashville and MacGillivray's Warblers all preferred the lowest vegetation strata. We found a threshold of native plant species composition that appears to influence migrating warbler abundance within differing vegetation patches. It thus appears that vegetation species, structure, phenology, abundance, and responses to insect prey base all appear to potentially play a role in determining migrating warbler susceptibility to WNV infection along the lower Colorado River corridor.

[12] ENHANCED PASSIVE SURVEILLANCE FOR WEST NILE VIRUS IN DEAD WILD BIRDS, CANADA – 2002-2003

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The Canadian Cooperative Wildlife Health Centre (CCWHC), under contract to and in partnership with Health Canada, acted as a clearing house to organize the collection, processing and submission to the National Microbiology Laboratory (NML), Health Canada, Winnipeg, or to the Veterinary Services Branch, Manitoba Dept. of Agriculture and Food, of tissues from wild birds as part of the West Nile virus (WNv) surveillance programme in Canada in 2002. Surveillance was focussed on members of the Corvidae (American crow, blue jay, common raven, gray jay, and northwestern crow and black-billed magpie in the west), since among wild birds they seem exceptionally susceptible to mortality caused by WNv. WNv was detected in tissue specimens using a real time RT-PCR system (TaqMan) or standard PCR that employed primer-probe sets specific for 5' and 3' portions of the WNv genome. Initial cases detected in a local health jurisdiction were confirmed by cell culture and viral isolation. The CCWHC also collected sightings of dead wild birds reported by the public, and maintained a data base for tracking specimens, recording and reporting results, and mapping and epidemiologic analysis of events.

In all provinces from Saskatchewan eastward, systems customized to the province or region were put in place to promote awareness of the surveillance programme, and to collect and transmit to the four CCWHC Regional Centres carcasses of dead birds and information on sightings. They utilized various combinations of local public health authorities, and provincial health ministries, agriculture ministries and wildlife agencies.

Between approximately 15 May and 31 October 2002, CCWHC accessioned 5,451 carcasses of wild birds (of which 3,660 specimens were suitable for submission for WNv detection and 3,478 were tested), and data on an additional 16,591 sightings of dead birds, for a total of 22,042 records in the data base. Ontario/Nunavut Region

CCWHC handled over 30% of the carcass submissions; Quebec Region ~over 27%; Manitoba Agriculture and Food ~16%; Atlantic Region ~15%, and Western Region the remainder. Carcasses of a total of 3,927 crows (488/2,589 tested +); 676 blue jays (26/403 tested +); 223 ravens (5/177 tested +); 187 black-billed magpies (20/139 tested +); 87 northwestern crows (0/81 tested +); 3 gray jays (0/3 tested +); and 348 other species (24/87 tested +) were accessioned.

West Nile Virus was detected first just west of Toronto in a bird picked up on 19 May 2002, in an area that had been epidemic with WNv in 2001. A total of 289 WNv-positive birds were detected in 36 of the 37 Ontario Health Units, encompassing Ontario north into the boreal forest. In Québec, WNv activity was detected in the vicinity of Montréal in a bird picked up on June 4, and 138 cases were distributed throughout most of the southern and western part of the province, again extending north into the boreal forest. In Manitoba, 88 WNv positive birds were detected, beginning in the Winnipeg area with a bird picked up on July 8, and activity was detected throughout the southern half of the province, up to about latitude 51EN. In Saskatchewan, the first positive bird was picked up in the Regina area on July 28, and 44 WNv cases were distributed west nearly to the Alberta border, and north to about latitude 52E. In Nova Scotia, 4 WNv cases were picked up, the first in the Halifax area on September 13. Surveillance was truncated or ceased in most areas when WNv was clearly established in a public health region, so the number of birds affected is a vast underestimate. The peak of WNv activity over the country as a whole was from the end of July to the end of August.

WNv activity in Canada in 2002 reflected the greatly expanded geographic range and intensity of activity of the virus across North America in 2002. In southern Ontario, where WNv was epidemic in 2001, activity may have been due to recrudescence of endemic WNv overwintering in diapausing *Culex* spp mosquitoes. Activity in the remainder of Canada seems to represent northward and westward movement of virus, perhaps in migratory birds in spring 2002. The date at which WNv activity was detected may integrate duration of time since introduction of the virus, and degree-days of heat, which affects the rate of mosquito development and of virus replication in infected mosquitoes, and hence opportunity for virus amplification in the ecosystem.

Dead bird surveillance in 2003 was extended from coast-to-coast. Testing was decentralized to CCWHC regional or provincial agricultural laboratories, and was based on an antigen capture wicking Elisa, the VecTestJ, using the eluate of an oropharyngeal swab taken from dead birds. Work done in 2002 indicated that in crows, VecTestJ had a sensitivity of ~85%, and a specificity of ~95-96%, in comparison with PCR. The advantages of the VecTestJ, in comparison with the centralized PCR-based system previously employed, include: simplicity of specimen collection and test methodology; no high cost equipment; local testing with rapid turnaround time (15 minutes to a result); competitive cost per test, and increased operator safety, since no dissection is required. The first Canadian bird detected with WNv in 2003 was noted dead on April 15 near Newmarket, north of Toronto, ON, but was not submitted for testing until 24 April. It was reported positive on April 25, 2003. Early WNv activity in this part of southern Ontario again may reflect overwintering of *Culex* spp. infected in summer 2002. Perhaps significantly, WNv had been detected in wild birds in this general area up until the end of the 2002 testing season.

[13] WEST NILE VIRUS INFECTION IN FREE-RANGING AND CAPTIVE WILD BIRDS AND MAMMALS, ONTARIO, CANADA, 2002

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During 2002, West Nile Virus (WNv) activity was monitored in the province of Ontario, Canada by enhanced passive surveillance for sightings of dead American crows and common ravens; by determination of the WNv infection status of selected dead crows and ravens; and by passive surveillance for mortality attributed to WNv in other avian species in wild populations, in a major zoo, and in wildlife rehabilitation facilities. Realtime polymerase chain reaction (RT-PCR) was used to determine infection status of crows submitted for surveillance. Other free-ranging and captive wildlife species were examined by one or more of the following means: histopathology, immunohistochemistry (IHC), RT-PCR and wicking antigen capture assay (VecTest™, Medical Analysis Systems, Camarillo, CA).

As part of the Canadian national program for enhanced passive surveillance for WNv, substantial mortality was detected in wild corvids, in all but one of the 37 health regions in Ontario. Over 11,500 sightings of dead birds were reported in Ontario in 2002. Of these almost 2700, as well as over 3000 reports of mortality in species other than crows, were mapped in Halton Region, southwest of Toronto, where numerous cases of WNv infection were documented in people, and WNv was isolated from mosquito pools at minimum infection rates of up to ~80/1000 *Culex* spp. Crow mortality in Halton Region over summer/fall 2002 exceeded 150/square mile in some areas.

In 2002, West Nile Virus activity was associated with a noticeable increase in the number of submissions of some species of raptors to the Canadian Cooperative Wildlife Health Centre, Ontario/Nunavut Region, for diagnosis of cause of death; WNv infection was detected by IHC and/or PCR in about 2/3rds of them. Among wild bird species diagnosed with WNv infection were: redtailed hawk, great horned owl, sharp-shinned hawk, Cooper's hawk, northern goshawk, osprey, American kestrel, great black-backed gull, American robin, Canada goose, and ring-billed gull.

We evaluated VecTest™ as a diagnostic tool, using oropharyngeal or swabs, and triturates of various tissues. VecTest™ was acceptable for detection of WNv in oropharyngeal swabs of American crows used for surveillance, but sensitivity was unsatisfactory for routine surveillance, diagnostic pre-screening, or etiologic diagnosis in non-corvid species examined.

WNv was detected in wild birds found sick or dead on the Toronto Zoo grounds (2 crows, 1 house sparrow, 1 northern goshawk), in birds from the collection (1 black-

billed magpie, 1 green-naped lorikeet, 5 loggerhead shrikes from a captive breeding program, 1 American flamingo), and it caused the death of a Barbary ape *Macaca sylvanus*. In addition, during the period of WNv activity, a bald eagle, a Demoiselle crane, a second green-naped lorikeet, and 2 American kestrels from the captive collection, and 5 wild Canada geese, were observed ill. Typically, sick birds were depressed and anorectic, and stayed on the ground if normally arboreal. The Demoiselle crane and the Canada geese were ataxic, and the geese had tremors. The Demoiselle crane recovered within a few days, but 3 Canada geese were euthanatized due to the severity of signs and failure to recover under hospitalization, while the other 2 were released. However, a released bird was recaptured and euthanatized in January 2003, due to inability to fly, and persistent fine tremors. One of the kestrels and several of the shrikes were found paralyzed or in convulsions; while the shrikes died, the kestrel survived, but did not improve under hospitalization, and was euthanatized a month later. A second kestrel that was moved indoors as a precaution, and never showed signs, died suddenly in late October. WNv was detected by RT-PCR and/or IHC in the wild corvids that died on site, in the loggerhead shrikes, and in the flamingo. Non-suppurative encephalitis and myocarditis were present in the 2 kestrels, but WNv was not detected, probably because of the chronicity of the disease. All Canada geese examined had non-suppurative encephalitis, but WNv was not detected. Only one surviving shrike had antibody to WNv, while other surviving birds suspected of infection with WNv were seropositive.

Major mortality also occurred in a large captive collection of owls, located in southern Ontario (reported elsewhere at this meeting – see abstract [9]).

Several pathologic syndromes are associated with WNv infection. These may, in part, be related to the relative susceptibility of the species to infection, and related mortality, as well as duration of infection. In some avian species, experiencing acute to peracute infection, WNv may be present in large amounts in the myocardium; the endothelium and media of vessel walls; in macrophages in the spleen and liver; and in glandular epithelium lining the proventriculus and intestine at all levels, the upper and lower respiratory tract, and kidney tubules. Necrosis of infected cells is evident, but usually there is no inflammatory response. In such species (eg. corvids, shrikes, some raptors), virus may or may not be found in neurons and other cells in the central nervous system, and encephalomyelitis is not evident.

In the second syndrome, most obvious in some raptors, virus may be found in cells within the nervous system and myocardium especially, associated with inflammation in both sites, but typically, virus infection in other sites is more spotty or unpredictable. In the third syndrome, WNv is found only in isolated or scattered cells or tissues, and death may be ascribed to a cause other than WNv infection (eg. Canada goose, American robin). In the fourth syndrome, mild to severe meningoencephalomyleitis and myocarditis are found in birds (American kestrels, Canada geese) euthanatized or dying weeks or months after the time of circumstantial exposure to WNv, which is not detected in their tissues. Such animals typically have circulating antibody to WNv.

Among wild mammals, WNv was detected as a cause of meningoencephalitis in gray squirrels, in which sporadic mortality or local epidemics of neurologic disease were reported.

The experience with WNv infection in wild birds and mammals in Ontario in 2002 is similar to that reported over much of the upper midwestern United States via various informal communications media. The opportunity to carry out controlled infection studies to determine the susceptibility of various species of wild birds to WNv is very limited in Canada, and relatively so in the United States. Systematic study of naturally-occurring WNv in free-ranging and captive populations of wild birds can provide data on species susceptibility basic to models predicting the impact of this agent on populations of wild birds, including endangered species, in the western hemisphere.

[14] USING RS/GIS TO ASSESS WILDLIFE AND HUMAN WEST NILE VIRUS DISEASE RISK

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West Nile Virus (WNV) was introduced into the United States in 1999 and has since spread to 44 States and the District of Columbia. Although the dynamics of the disease remain obscure, its effects on humans and naïve wildlife populations are readily apparent across the country. In Louisiana, WNV was first detected in dead birds in 2001, and in 2002 329 human cases and 16,570 dead birds were reported, suggesting that the state was a major focus of the epidemic. As of mid-May 2003, 6000+ dead bird reports have been logged in the state indicating that virus activity continues to be high in the region. The aim of our study is to use remote sensing (RS) and Geographical Information Systems (GIS) techniques to investigate the processes underlying disease spread in Louisiana and to understand the links between hosts, vectors and pathogens in the environment. To do this, we are studying the ecological and environmental variables associated with virus amplification and wildlife and human disease incidence. Preliminary results linking our studies of mosquito ecology with human infection data suggest that disease presence in humans corresponds most highly with the distribution of *Culex quinquefasciatus* mosquitoes than with the distribution of any other WNV-carrying mosquito species within our study area. Future work will look at associations between landscape characteristics, weather conditions, mosquito distributions, dead bird clusters and equine cases and the potential for using mosquito distribution maps to predict areas of high risk for both humans and wildlife.

[15] DESCRIPTION OF THE HIGHLY PATHOGENIC AVIAN INFLUENZA A (H7N7) EPIDEMIC IN THE NETHERLANDS IN 2003

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Starting in February 2003, an outbreak of highly pathogenic avian influenza occurred in commercial poultry farms in the Netherlands, and subsequently spread to humans. The objective of this study was to determine the origin of the epidemic and to describe its epidemiology. Tissue samples and swabs from birds and humans were tested by a combination of virus isolation, amplification of parts of the hemagglutinin and neuraminidase genes by RT-PCR, and nucleotide sequencing. The subtype of the causative influenza A virus in commercial poultry was identified as H7N7. It showed high homology to low pathogenic influenza viruses isolated from migratory waterfowl during surveillance studies in the Netherlands in 1999 and 2000, and to a low pathogenic influenza virus detected in free-range poultry in the Netherlands in 2002. The outbreak started in central Netherlands in February, and spread to southern Netherlands in April. Measures to control the spread of highly pathogenic avian influenza consisted mainly of culling poultry and other captive birds and banning transport of livestock in and around infected areas. As of 21 April, 18.3 million birds were culled on 1001 farms. A high proportion of people involved in culling poultry became infected with H7N7 virus. As of 5 May, H7 virus was detected in conjunctival or throat swabs, or both, of 85/309 (28 %) individuals suffering from conjunctivitis, influenza-like illness, or both. Six individuals had H3 positive throat swabs, but no simultaneous infection with H7 and H3 influenza virus was detected. One veterinarian who had visited an infected poultry farm died with an interstitial pneumonia associated with H7N7 virus infection. Of 85 wild birds of 24 species found dead or euthanized around infected poultry farms, all free-living birds were negative for influenza virus. Highly pathogenic H7 virus was detected in four mallards and three mute swans (*Cygnus olor*), all semi-captive birds kept in close proximity to infected poultry. A possible source of highly pathogenic H7N7 virus is transfer of low pathogenic H7N7 virus from migratory waterfowl to free-range poultry in 2002, and mutation to highly pathogenic H7N7 virus during amplification within poultry. The explosive growth of free-range poultry operations in recent years may have increased the risk of spill-over of influenza virus from wild birds to free-range poultry. The chance of transmission of this virus from poultry to humans was likely increased due to the large scale of the culling operation. Strategies for the poultry industry as a whole and for free-range operations in particular need to be reconsidered in the light of the above epidemic.

[16] *MYCOPLASMA GALLISEPTICUM* CONJUNCTIVITIS IN HOUSE FINCHES (*CARPODACUS MEXICANUS*). CORRELATIONS AMONG CLINICAL SIGNS AND DETECTION BY POLYMERASE CHAIN REACTION FROM CONJUNCTIVAL AND CHOANAL SWABS

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Mycoplasma gallisepticum conjunctivitis emerged in 1994 as a disease of free-ranging house finches (*Carpodacus mexicanus*) in the eastern United States, and has since spread to house finches throughout their entire eastern range including Canada. The disease is now endemic in the eastern range of this species and is clinically characterized by conjunctivitis, sinusitis, and debilitation. The resulting epidemic of mycoplasmal conjunctivitis produced an unprecedented decline of eastern house finch populations, and the endemic disease remains associated with repeating seasonal peaks of disease and limitation of host populations. A similar clinical disease and isolation of *M. gallisepticum* has been described in free-ranging purple finches (*Carpodacus purpureus*), American goldfinches (*Carduelis tristis*), evening grosbeaks (*Coccothraustes vespertinus*), pine grosbeaks (*Pinicola enucleator*), and from a captive blue jay (*Cyanocitta cristata*).

NSF has funded a 5-year multi-disciplinary multi-institutional (Cornell University, Princeton University, North Carolina State University, University of Wisconsin) project to study *M. gallisepticum* conjunctivitis of house finches, a unique opportunity to study the interactions between an emerging pathogen and its introduced host. An important objective of this project is continued monitoring of the disease and *M. gallisepticum* prevalence in house finches. The prevalence of disease may be monitored by observing, either remotely (via binoculars or spotting scopes) or physical exam of captured birds, typical clinical signs and/or gross lesions. The occurrence of disease may or may not coincide with evidence of *M. gallisepticum* infection. For example, house finches could be infected with *M. gallisepticum* with the absence of disease in the incubation period and/or as recovered and inapparent carriers. It may also be possible for house finches to have the disease (typical clinical signs and gross lesions) in the absence of detectable infection. For example, in a convalescent phase when previous infection may be cleared or undetectable yet house finches continue to show clinical signs and/or lesions typical of the disease. Therefore, an important component of this objective is to study and understand the dynamics and alternatives related to categorizing house finches under observation as either diseased and/or infected. We are attempting to study and understand these important dynamics using data from both controlled experimental infections in aviary trials, and that obtained from captured free-ranging house finches – the focus of this presentation.

A program to systematically capture, assess, band, and take samples from house finches within a 5 km radius of the Laboratory of Ornithology in Ithaca, NY was initiated in October 2000, and has been followed by a re-sighting program. From 30 July to 4 December 2002, house finches captured in the Ithaca study area were evaluated for *M. gallisepticum* disease by physical examination and observation of typical clinical signs and gross lesions, and sampled for detection of the organism by polymerase chain reaction (PCR). Specifically, determination of disease was based on examination of both eyes and adnexa of each captured house finch for signs of conjunctivitis, such as eyelid or conjunctival edema, erythema, and discharge. Each eye was given a score of 0 (no lesions, normal ocular health), 1 (mild), 2 (moderate), or 3 (severe). An eye lesion score of 1 or above was considered clinically positive for the disease. Samples for *M. gallisepticum*-specific PCR were collected from both eyes using swabs of the conjunctiva (calcium alginate fiber tipped ultrafine aluminum) placed in Frey's broth medium with 15% swine serum (pooled sample, 2 swabs per broth). The choanal cleft of each bird was also swabbed and the sample inoculated to a separate broth for the purpose of comparing these sites (conjunctiva vs. choana) for the presence of the organism. *M. gallisepticum*-specific PCR results were scored either negative or positive.

Preliminary examination of the data showed that among house finches with no clinical signs, the PCR positive rates were similar for both choanal and conjunctival swabs, approximately 23%. However, there was not a complete concordance between the two sample sites. Therefore, of house finches without disease, approximately 34% were PCR positive from either sample site, and only 7% were PCR positive from both sites. Among house finches that had clinical signs approximately 70% were PCR positive from both sample sites, while the combined PCR positive detection rate was approximately 93%. More extensive and detailed analyses of these data are in progress. Additionally, we are looking at individual recapture histories relative to clinical disease and PCR test results to evaluate temporal relationships of disease and organism detection from conjunctival and choanal sample sites.

Combining these types of analyses from natural infections occurring in free-ranging house finches with controlled exposures of captive birds will provide valuable information on the epidemiology *M. gallisepticum* conjunctivitis of house finches.

[17] TYPE E BOTULISM IN BIRDS ON LAKE HURON AND LAKE ERIE, 1998-2002

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Type E botulism has been the confirmed or suspected cause of numerous die-offs of birds during the period of June to November on Lake Huron and Lake Erie since 1998. The annual pattern has been one of sporadic mortality of gulls and shorebirds during the summer months, followed by more intense epizootics, involving common loons, red-breasted mergansers and more recently, long-tailed ducks during the fall migration. The disease was initially detected on Lake Huron where it caused epizootics in 1998 and 1999, and from which it was apparently absent until its reappearance in 2002. It has occurred annually on Lake Erie since 1999, with a gradual shift in activity from the western to central and eastern basins. The disease is suspected to have reached Lake Ontario in 2002, with positive cases in ring-billed gulls on the Niagara River and suspected cases in long-tailed ducks from Lake Ontario. Analysis of gizzard contents of birds dying in 2002 indicated that in common loons with identifiable material (48 of 65 birds), 40 of 48 contained fish. In contrast, 22 of 27 long-tailed duck gizzards contained zebra or quagga mussels. In previous years, the round goby was the species of fish most frequently identified in the gizzard content of affected birds. These findings indicate an expanding geographic range of this condition and confirm that multiple routes of exposure to toxin are possible, depending upon the feeding habits of the species of bird affected.

[18] PUTATIVE ROLE OF FISH IN THE EPIDEMIOLOGY OF AVIAN BOTULISM IN LAKE ERIE

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Since 1999, significant mortalities involving several species of fish-eating birds, ducks and gulls, have been reported along both the Canadian and US borders of Lake Erie. Type E botulism is considered to be the primary cause of death associated with these annually recurrent epidemics, involving species such as common loons, common and red-breasted mergansers, grebes, diving ducks, ring-billed and herring gulls, Bonaparte's gulls and others. Cumulative mortalities since 1999 now number in the tens of thousands of birds.

Serious animal as well as human health implications are associated with this situation. Most importantly, these recurring events may be an early sentinel of serious, underlying problems with the health and integrity of the Lake Erie ecosystem.

Many of the affected bird species are scavengers, and could easily consume organic material or animal carcasses (esp. fish) contaminated with Type E botulinum toxin. However, other bird species, such as the loons and mergansers, feed predominantly on live fish, which, until now, have been regarded as an unlikely source of the botulism toxin.

A temporal and geographical association has been made between Type E botulism outbreaks in Lake Erie, and the colonization of the lake by invading zebra and quagga mussels, as well as the fish which feed on them, namely, the round gobie. It has been hypothesized that there is a link involving mussels and gobies that transports botulinum toxin up through the water column in live, toxin-laden fish. It is these fish which are then consumed by birds such as loons and mergansers.

Little is known about the epidemiology or pathogenesis of botulism in fish, and there may be significant interspecies variation in the sensitivity of fish to Type E toxin. If common prey fish are highly sensitive to Type E toxin, then it is unlikely that many of them live long enough (while carrying a bird-lethal dose), to be eaten 'live' by the bird species in question. On the other hand, if prey fish are relatively tolerant to type E toxin, they could conceivably carry enough toxin to kill a bird without displaying clinical signs themselves. In the latter scenario, the fish could represent a living 'vector' which transports toxin from the substrate or other anaerobic environment where it is produced, to a bird which otherwise would not come into contact with it.

A Fish Botulism Exposure Model (FBEM) has been developed to compare the sensitivity of various fish species found in Lake Erie to known doses of Type E botulinum toxin, as well as to characterize the associated clinical signs and tissue distribution of the toxin. Based on this model, it is apparent that fish intoxicated with Type E botulinum toxin can survive for considerable periods of time, during which they exhibit various behavioural displays which may cause them to be selectively predated by

fish-eating birds. The application of this model to characterization of the relative sensitivity to Type E botulinum toxin in several species of fish will be reported. These studies will aid in a better understanding of the mechanisms of botulinum transport in the L. Erie ecosystem, and may help elucidate the exact cause(s) of the avian botulism epidemics observed.

[19] CONSERVATION MEDICINE IN ACTION: ONGOING INVESTIGATIONS INTO A SEVERE DISEASE OUTBREAK IN THE NGORONGORO CRATER, TANZANIA DURING 2000/2001?

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Between May 2000 and April 2001, there was a major die-off of wildlife in the Ngorongoro Crater, Tanzania. A team of experts was brought into the Crater to investigate the causes of this severe die-off. All experts agreed on the fact that several interacting factors probably contributed to this abnormally high mortality. Tick-related diseases were identified in rhinos, lions and buffalo, but the impact of these diseases was exacerbated by other factors, such as malnutrition caused by prolonged drought, which almost certainly increased susceptibility to disease. It is unlikely that these diseases alone would have resulted in high mortality in the absence of other contributory factors. On the other hand, it had to be investigated, where this disease came from, as two black rhinos had been translocated into the Ngorongoro Crater in December 1997 from Addo Elephant National Park.

Between May 2000 and January 2001, five black rhinos (out of 18), 800 buffalo (out of 5000), 30 lions (out of 60), one of the three cheetahs, 200 Gnus and a few zebras died. The death of the South African female black rhino was attributed to an accident, probably a fight with an elephant. Her calf was killed by lions a few weeks earlier. Mortality of the other three black rhino individuals was linked to a tick-borne disease caused by two protozoan parasites just recently identified as *Babesia bicornis* spec. nov. and *Theileria bicornis* spec. nov.. This disease was probably fatal in rhinos this year only because of nutritional- and other stress. The cause of death in the 30 lions has not been identified definitively, but probably resulted from an interaction of several factors, including heavy ectoparasite burdens, high levels of blood parasites and stress. Canine distemper virus, which has infected a large proportion of the crater lion population, is not thought to have been a primary cause of death in this outbreak but may have modified susceptibility to other diseases. The death of 800 Buffalo were associated with tick-borne diseases concomittant with massive tick infestations and severe malnutrition and heavy tooth-ware from old grass.

In all death cases, the question arose, how this tick-infestation, and hence tick-born disease, could have spread in the Crater with such high mortality. For this complex investigation experts from many different disciplines, such as ecologists, water-, tick-, vegetation and fire-experts, were brought to the Crater for short or serial investigations. The outcomes of these investigations as they relate to the cause of death are presented.

The introduction of more rhinos into the Serengeti Ecosystem is believed necessary for the long-term survival of black rhinos in Tanzania. Currently it is not clear if the Babesia/Theileria parasite that finally killed three rhinos was introduced to the Crater via the translocated animals from South Africa. Currently it is believed that this parasite - isolated from the rhinos in the Crater - is a new parasite. A similar parasite has been identified in the South African population, but further investigation is needed to

determine whether the parasite also occurs naturally in East Africa and was just not studied before. Whatever the outcome, animals for possible future translocations must be screened more thoroughly for diseases in the population of origin. Further information is also required on the prevalence of diseases in the crater population in order to evaluate the impact of any future translocation.

As this disease outbreak was caused by multiple factors, it is necessary to reduce most man-made influences in this list of causes and establish thorough protocols for health monitoring for wildlife in the Ngorongoro Crater and the Serengeti Ecosystem.

[20] BIOMEDICAL SURVEY OF GIANT PANDAS IN CHINA *EX SITU*: OBSERVATIONS OF A STUNTED GROWTH SYNDROME, AND THE NEED FOR SEROEPIDEMIOLOGICAL STUDIES

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The giant panda (*Ailuropoda melanoleuca*) is endangered, and conserving the species depends on concerted efforts to preserve and restore its habitat and to establish self-sustaining *ex situ* populations. The *ex situ* giant panda population guards against extinction and plays a critical role in educating the public about the plight of wild counterparts and a diversity of unique Chinese wildlife. The giant panda in captivity also provides biologists and veterinarians the opportunity to study aspects of its special physiology and behavior, which will enhance our ability to protect the species in its natural setting and in captivity. Toward this end, the Chinese Association of Zoological Gardens invited the Conservation Breeding Specialist Group of IUCN's Species Survival Commission to lead a biomedical study of the *ex situ* giant panda population in China. The study was carried out during the breeding season (spring) from 1998 through 2000, and included 61 giant pandas from the Chengdu Research Base for Giant Panda Breeding, the Chengdu Zoo, Beijing Zoo, Chongqing Zoo and China Research and Conservation Center for the Giant Panda (Wolong Nature Reserve).

Methodologies for the survey were consistent across all three annual sessions and included a general physical examination (including measurement of weight and external anatomical structures), abdominal ultrasonography, reproductive evaluation (vaginal cytology, measurement of male external genitalia, and electroejaculation for sperm evaluation on select males), hematology and serum chemistry. Skin and blood samples were cryopreserved for genetic analysis. Animal histories and nutritional information were collected through interviews and survey forms.

Results from the biomedical survey indicated that multiple factors associated with behavior, nutrition, genetics, health and husbandry were limiting reproductive success of the giant panda *ex situ*. Two additional important factors that emerged from the study were (1) the identification of a group of giant pandas of unusually small size and other characteristics that constitute a 'stunted growth syndrome' (SGS), and (2) the need for studies to identify and characterize infectious diseases of potential threat to giant pandas both *ex situ* and *in situ*.

Giant pandas with SGS are abnormally small in stature and often have shorter limbs than normal animals. Affected individuals usually have immature genitalia, are nonreproductive and suffer chronic gastrointestinal disease. Pandas with SGS have ascites, but ascites is a frequent observation in otherwise apparently healthy giant pandas

in China. Tooth surfaces of pandas with SGS are consistently stained and pitted and show evidence of enamel dysplasia and/or hypoplasia. The teeth are also affected with increased and asymmetrical attritional changes. To date, nine giant pandas, all but one of which were born and raised at a single institution, displayed all or most of the signs of SGS. Two underdeveloped cubs with chronic diarrhea and dental abnormalities were born in August 2002 (at the same institution that raised the other eight), and may represent the most recent SGS cases.

Several potential etiologies have been proposed for SGS. These include primary nutritional deficiencies or imbalances, or nutritionally-induced gastroenteritis that may lead to abnormal growth and development. Systemic illness caused by an infectious agent or toxicosis during critical stages of development also may result in abnormal development. Dental abnormalities of giant pandas with SGS, for example, are similar to those observed in puppies after neonatal exposure to canine distemper virus (R.R. Dubielzig *et al., Vet Pathol.* 18:684-689, 1981). There is a critical need to study SGS and to identify the underlying factors of the condition. As a first step, a workshop is being planned at which an international group of veterinary specialists will examine the affected giant pandas and proceed with a differential diagnosis of SGS.

Vaccination protocols for giant pandas in China are varied, and effectiveness in terms of measuring antibody titers has not been evaluated. Those institutions holding giant pandas in China that vaccinate animals rely on a multivalent, modified live vaccine developed for dogs against canine corona, distemper, parvo and adenoviruses, rabies and sometimes *Leptospira interrogans*. The susceptibility of the red panda (*Ailurus fulgens*) to canine distemper is well established, but the incidence of distemper (either naturally occurring or vaccine-induced) in giant pandas is unclear. A preliminary serological study conducted on giant pandas in China's Wolong Nature Preserve suggested that giant pandas raised in captivity, as well as those recently rescued from the wild, had been exposed to canine distemper virus, parvovirus and other pathogens that can cause fatal or debilitating disease in non-domestic carnivore species (Mainka *et al.*, 1994. *J. Wildl. Dis.*, 30:86-89). The results of the study emphasizes the need for a comprehensive survey of infectious diseases in giant pandas in China in order to corroborate earlier findings and to expand our understanding of this issue.

To address this need, a seroepidemiological survey of *ex situ* giant pandas in China is currently underway through collaborations among Chinese giant panda breeding institutions, the China Wildlife Conservation Association, the Chinese Association of Zoological Gardens, the Smithsonian's National Zoological Park and Cornell University. As a first step in this project, 125 serum samples banked from giant pandas over the past five years will be assayed for a panel of nine common canine and feline infectious agents, including canine distemper, parvo, corona, adeno, herpes and parainfluenza viruses, *Neospora caninum*, *Toxoplasma gondii* and five serovars of *Leptospira interrogans*. The assays will be performed in a laboratory in China. Once the initial group of 125 samples has been analyzed (anticipated by the end of 2003), the project will proceed in the establishment of diagnostic and research laboratories in China that will monitor infectious diseases and vaccine efficacy in giant pandas (and, in the future, of other wildlife species as well).

Research of the Smithsonian's National Zoological Park in China is conducted in partnership with the China Wildlife Conservation Association/State Forestry Administration and is funded by Friends of the National Zoo.

[21] DISEASE AS A FACTOR LIMITING COLUMBIA BASIN PYGMY RABBIT RECOVERY

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The Columbia Basin pygmy rabbit was rediscovered in Washington in 1987 after it was thought to have been possibly extirpated. In 1997, six populations remained in Washington. Between 1997-2000, five of the six populations disappeared. Fire is believed to have been responsible for the disappearance of one population. The cause(s) for the disappearance of the remaining populations is unknown, although there is some evidence that sylvatic plague may have played a role. It is believed that fewer than 30 Columbia Basin pygmy rabbits remain in the wild.

Genetic analyses of pygmy rabbits from Oregon, Idaho, Montana, and the Columbia Basin of Washington suggest that the Columbia Basin rabbits have been isolated from other pygmy rabbit populations for thousands of years and are genetically distinct. The Columbia Basin pygmy rabbit was Federally listed as endangered in November 2001.

An emergency captive breeding program was initiated in May 2001. Eleven wild Columbia Basin pygmy rabbits were captured and translocated to Washington State University (WSU), followed by the capture and translocation of seven rabbits to the Oregon Zoo. A total of 15 live young were born in captivity during 2002 and nine of these survived to adulthood. At least five litters born during the 2003 breeding season have not survived.

The captive breeding program has been plagued by a number of health problems. Nine (6 wild-caught; 3 captive-born) rabbits have died of avian tuberculosis; coccidiosis has been responsible for the deaths of four captive-born kits; and captive-born rabbits from two litters have been born with missing metatarsal and/or metacarpal bones. The Oregon Zoo, WSU, and the Washington Department of Fish and Wildlife are pursuing investigations into the genetic, environmental, and management factors potentially responsible for these problems.

[22] RISK ASSESSMENT AS A TOOL TO EVALUATE HEALTH STATUS OF A SALVAGED HERD OF CAPTIVE WOOD BISON

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The Hook Lake Wood Bison Recovery Project is a co-operative wildlife conservation project with the principal aim of salvaging genetic diversity from a wild, free-ranging herd of wood bison *(Bison bison athabascae)* that is enzootic for bovine tuberculosis *(Mycobacterium bovis)* and brucellosis *(Brucella abortus)*. The project is based on a combination of techniques to propagate a captive herd of healthy bison that included: 1) orphaning of newborn wild-caught calves that may have been exposed to *B. abortus* and *M. bovis*; 2) field-testing of calves for maternal antibodies to brucellosis prior to entry in the isolation facility; 3) isolating calves in pairs to prevent potential spread of disease; 4) hand-rearing and prophylactic treatment of captive calves using a combination of anti-mycobacterial and anti-Brucella drugs; 5) serological testing of first time heifers and their captive-born calves at three days and four weeks post-calving, and 6) intensive whole-herd testing for both diseases and removal of any suspicious reactors. From 1996 to 1998, we captured a total of 62 calves. Presently 57 individuals comprise the founder herd with an additional 69 captive-born animals ranging in age from yearlings to four-year olds. To date there have been no cases of bovine tuberculosis or brucellosis in the captive herd; all founder animals have been tested using a combination of tests for brucellosis (Buffered Plate Antigen Test, Standard Tube Agglutination Test, Complement Fixation Test, Competitive Enzyme-Linked Immunosorbent Assay, and Florescence Polarization Assay) and tuberculosis (Caudal Fold Test and Florescence Polarization Assay). We defined an overall risk model for each disease that was a product of three risk probabilities – 1) prevalence of true infection at time of capture, 2) efficacy of prophylactic treatment regime, and 3) sensitivity of disease testing regime - summed across the three founder cohorts. We used recent data from Wood Buffalo National Park bison to estimate initial values for prevalence of disease in bison calves. We referred to published data on brucellosis in cattle and used a Beta (13, 298) distribution to represent the proportion of prevalence of true infection of calves at time of capture. With respect to tuberculosis, we used a Uniform (0.01, 0.02) distribution to estimate the proportion of calves infected via pseudo-vertical transmission through the dam's milk. For both animal health hazards, the efficacies of antibiotic and chemotherapeutic treatments were not included in this analysis, and therefore the overall probabilities indicated below may be actually lower. Our assessment of the disease-testing regime was based on one type of diagnostic test for each disease – the BPAT for brucellosis and the Caudal Fold Test for tuberculosis. We used a first-order Markov Model to estimate sensitivity of the testing regime with the criteria that all 57 founders had tested negative on a minimum of eight BPAT and Caudal Fold Tests respectively. Based on this risk model, the 95th percentile of the probability of at least one brucellosis infected bison being present given that all

bison have tested negative according to the testing regime, was less than 0.0002. The 95th percentile of the probability of at least one tuberculosis infected bison being present given that all bison have tested negative according to the testing regime, was less than 0.0003. We suggest that the risk estimates for the animal health hazards, *B. abortus* and *M. bovis*, are very low and negligible, respectively, which appear to lie within the Appropriate Level of Protection based on past and recent importation of animals into Canada. In addition, this represents a domestic risk assessment rather than international trade under the constraints of the Sanitary Phytosanitary Agreement of the World Trade Organization. Therefore the benefits of the Hook Lake Bison Recovery Project should be considered, especially, the reproductive capacity of the founding bison and their offspring and the unique and variable lineage of genetics this herd represents for conservation of wood bison in Canada.

[23] THE NBII WILDLIFE DISEASE INFORMATION NODE

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As part of the National Biological Information Infrastructure, the Wildlife Disease Information Node, hopes to create a resource that will provide information and links to information on wildlife health and wildlife-human-domestic animal disease interactions. The Node is developing a Web-based interface that hopes to create a mechanism to link existing databases that contain wildlife disease information, as well as a tool for on-line reporting of morbity and mortality events. This reporting system can also used by research and monitoring projects as a free toll to provide mapping capabilities for their project. The Node will also provide services for the wildlife disease community by providing discussion space, event calendars, and hosting of partner organization web sites.

[24] VETERINARY INVOLVEMENT IN CHELONIAN CONSERVATION PROGRAMS THROUGH THE TURTLE SURVIVAL ALLIANCE

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The Turtle Survival Alliance (TSA), an organization formed in 2001 in response to the Asian turtle extinction crises, is a joint initiative of the IUCN/SSC Tortoise and Freshwater Turtle and Conservation Breeding Specialist Groups. It is comprised of members from zoos and aquaria, universities, private breeders and hobbyists, commercial ventures and governmental organizations. The primary mission of TSA is to "*Develop and maintain an inclusive, broad-based global network of collections of living tortoises and freshwater turtles with the primary goal of maintaining Chelonian species over the long term to provide maximum future options for the recovery of wild populations*". In addition to supporting in-situ conservation programs, one of the basic goals of the TSA is to establish assurance breeding colonies to protect against extinctions. Animals for these programs can be found in zoos, private colonies, in rehabilitation facilities in range countries, and when confiscated by regulatory agencies.

Veterinary input to the TSA has been ongoing from its inception and consists of development and implementation of protocols for humane care of confiscated animals and for medical triage and treatment of large numbers of animals. One confiscation of animals during the winter of 2002 resulted in 3,000+ turtles and tortoises being imported in to the USA by the TSA in a one month period. Treatment protocols, diagnostic procedures, surgical interventions and necropsy findings developed in response to this provided templates for similar protocols for future confiscations. In addition, there are veterinary advisors for taxa that have been designated for intense management. The role of the advisors has been to gather and disseminate clinical, pathologic and parasitologic information and treatment protocols relevant to each taxus.

[25] THE ROLE OF WILDLIFE CENTERS AND REHABILITATORS IN WILDLIFE DISEASE MONITORING

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The traditional role of wildlife rehabilitation centers has been the treatment and release of healthy wildlife. However, recent studies of morbidity and mortality of wildlife, especially raptors, admitted to wildlife centers and universities have provided insight into the health status of wild populations. In addition, wildlife rehabilitation centers have been instrumental in identifying emerging diseases as well as monitoring ecosystem health. In order for these roles to be fully realized, more emphasis needs to placed on accurate record keeping, standardized health screening and more thorough clinical examinations, diagnostic tests and post mortem examinations. The Wildlife Center of Virginia (WCV) established a disease monitoring system in 1993. Since then, standardized health screening protocols have been developed based on Office International des Epizooties recommendations. Data such as species, date of admission, location by county, primary and secondary clinical syndromes, and primary and secondary morbidity and mortality categories are entered into an Epi Info database. These computer programs are produced by the Centers for Disease Control and Prevention and the World Health Organization and provide for easy form and database construction, data entry and analysis with epidemiologic statistics, maps and graphs. The programs are also compatible with other databases such as Microsoft Access and ArcView. This database has allowed us to identify significant temporal changes in animal admissions, and spatial clustering of clinical cases. Wildlife can also serve as early warning indicators or sentinels of disease outbreaks in humans and domestic animals. The wildlife rehabilitation community could play a critical role in filling gaps in wildlife disease monitoring and surveillance, and could dramatically increase sample sizes and expand geographic locations. We believe we have developed a model system that could be used by wildlife centers and universities that receive clinical wildlife cases to increase capacity to detect and monitor for emerging diseases such as West Nile Virus.

[26] PATHOLOGY OF NATIVE AND INTRODUCED REEF FISH IN HAWAII

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The bluestripe snapper, or taape, was introduced into Hawaii in the 1950s and has since become very abundant throughout the archipelago. As part of a health survey of reef fish in Hawaii, we necropsied 120 taape collected from various coastal areas south of Oahu and examined fish on histology for extraintestinal organisms. Forty seven percent of taape were infected with an apicomplexan protist evident mainly in the spleen, and less commonly, the kidney. Prevalence of this parasite increased with size of fish, and we saw no significant pathology associated with the organism. Twenty six percent of taape were also infected with epitheliocystis-like organism mainly in the kidney and, less commonly, the spleen. In contrast to the protist, affected fish mounted a notable inflammatory response to epitheliocystis-like organism that appeared to increase in severity with age. Prevalence of epitheliocystis increased with age but infection was not seen in fish greater than 26.5 cm fork length. The high prevalence of protistan infection in introduced taape prompts the concern that these organisms, along with epitheliocystis, have the potential to be transmitted to native reef fish. Given the impact of other introduced microbial organisms on native Hawaiian fauna, there is a clear need to assess which protistan and microbial pathogens are endemic to Hawaii and whether they negatively impact native reef fish that closely associate with taape.

[27] HEALTH MONITORING PROGRAMS ASSOCIATED WITH CONSERVATION OF THE GRAND CAYMAN IGUANA (*CYCLURA NUBILA LEWISI)*

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Rock iguanas (*Cyclura sp*), consisting of eight species, inhabit many islands in the Caribbean. The International Conservation Union's Species Survival Commission (IUCN/SSC) Iguana Specialist group (ISG) formerly the West Indian Iguana Specialist Group has developed strategies for protecting and bolstering the most critically endangered populations with a combination of field studies, protection of wild areas, captive breeding, head-starting juveniles with subsequent release, and ex-situ breeding programs.

In 1992 population studies of the Grand Cayman or Blue Iguana (*Cyclura nubila lewisi*) resulted in estimates that there were 100-200 wild animals left in the wild on west end of Grand Cayman island. A breeding and rearing facility was subsequently established at the Queen Elizabeth II Botanic Park. Breeding and release of 2-3 year old animals into the park has been conducted since 1996. In 2002 field studies revealed that the wild population, exclusive of the Botanic Park, has undergone a precipitous decline and now numbers only 10 – 25 isolated individuals living in a fragmented habitat. Considered functionally extinct in the wild, the captive program will ultimately be responsible for the survival of this species.

The general health program for the Grand Cayman iguanas consists of monitoring the health of the captive breeding animals, performing pre-release screening of head-started juveniles, and opportunistic sampling of free-ranging animals in the Botanic Park. Health screening includes physical exam, weights, measurements, collection of blood and feces or cloacal swabs. Laboratory analysis that are performed include: complete blood counts, plasma biochemicals, minerals and vitamin D analysis, cloacal or fecal bacterial culture, and fecal analysis for presence of cryptosporidium and other parasites,.

Health screening to date has not revealed any evidence of infectious disease. There is a low level endoparasitism in the adults that has not been seen in the juveniles. Additionally, *Salmonella sp*. has been cultured from feces of captive adults, but not juveniles.

[28] AN EPIZOOTIC OF SYSTEMIC COCCIDIOSIS (*CARYOSPORA CHELONIAE*) IN GREEN TURTLES (*CHELONIA MYDAS*) ALONG COASTAL NSW – A MARINE INDICATOR OF DROUGHT

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An epizootic of neurologic dysfunction and mortality in Green turtles (*Cheloniae mydas*) was identified along the east coast of NSW between 14th and 19th October 2002. Disseminated coccidiosis, characterised by the presence of necrosis and non-suppurative inflammation in the intestinal tract, renal interstitium, thyroid gland interstitium, and throughout the parenchyma of the brain, was associated with *Caryospora cheloniae*. Coccidia harvested from the intestinal tract were cultivated in filtered seawater and developed into the stellate sporulation pattern pathognomonic for *Caryospora cheloniae*.

During and shortly after the epizootic, a total of 13 green turtles, and four hawksbill turtles (*Eretmochelys imbricata*) was subject to gross and microscopic post mortem examination. Eleven of the subadult and adult green turtles had systemic coccidiosis. Affected turtles ranged between 28.4 and 105 kg, with straight carapace lengths ranging between 615 and 940 mm. These animals were feeding on sea grass beds in estuaries along coastal NSW.

Concurrent with the epizootic in green turtles were marine outbreaks of algal blooms attributed to *Trichodesmium erythraeum*. This alga was identified in large quantities in sea grass beds, and in the stomach content of the ill turtles. Stomach content from seven turtles and liver samples from five of the green turtles were analysed for the presence of several biotoxins. Hepatic microcystin concentrations ranging between 17.9 and 79.0 *u*g/kg were identified using an ELISA test.

All cases of *Caryospora cheloniae* associated mortality in green turtles that are logged in the Australian Registry of Wildlife Health have occurred in years with El Nino or drought conditions, which alter the sea grass ecosystem.

[29] BIGHORN SHEEP (*OVIS CANADENSIS*) DISEASES: A BRIEF LITERATURE REVIEW AND RISK ASSESSMENT FOR TRANSLOCATION

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Prior to European settlement in western North America, bighorn sheep (*Ovis canadensis*) were more widespread and abundant than they are today. The species arrived via the Bering land bridge approximately 70-100,000 years before the present (YBP) and slowly spread to occupy most mountainous regions of western North America from southern British Columbia and Alberta, Canada to the Cape of Baja California and northern Sierra Madre in Mexico. Based on fossil records, it is likely that bighorn sheep arrived in the southwestern United States at the end of the Pleistocene era approximately 9-12,000 YBP. It is clear that bighorn sheep underwent dramatic declines in both occupied area and numbers throughout their range in North America in the 3 decades prior to 1900. The most probable cause of declines in this era was the introduction of domestic sheep with a suite of diseases to which bighorn sheep were naïve. Subsequent to 1900, bighorn sheep population declines continued due to several causes including habitat fragmentation and degradation, unregulated harvest for trophies and subsistence, and competition with domestic livestock. One strategy to repatriate bighorn sheep populations is translocation of groups from healthy source populations to repopulate vacant historic habitat. Translocation is also used as a management tool to bolster populations that are below demographic objectives. Managers overseeing translocations need to be cognizant of the potential to introduce diseases when moving animals, and their potential impacts on indigenous wildlife or domestic livestock. To facilitate translocations and minimize disease risk, managers need to develop an understanding of diseases that play roles in bighorn sheep demographics, and develop methods to minimize any risk to bighorn sheep, other wildlife, and livestock. This is particularly important when managers move bighorn sheep between jurisdictions and across international boundaries (typically Canada to the U. S., and bi-directional from U. S. – Mexico). In this paper, we review several diseases of livestock and bighorn sheep and propose recommendations for health screening of bighorns to minimize disease risks to animals in the recipient area and to aid in reestablishing healthy bighorn sheep populations.

[30] EXPERIMENTAL INFECTIONS OF WHITE-TAILED DEER WITH *ANAPLASMA (EHRLICHIA) PHAGOCYTOPHILUM*, THE ETIOLOGIC AGENT OF HUMAN GRANULOCYTIC EHRLICHIOSIS

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Although approximately 1220 cases of human granulocytic ehrlichiosis (HGE) have been diagnosed in the United States since 1994, knowledge of the natural history of *Anaplasma phagocytophilum* (formerly *Ehrlichia phagocytophila*) remains incomplete. Because antibodies reactive to and PCR products identical with *A. phagocytophilum* have been found in wild white-tailed deer and because white-tailed deer are the main host for adult stages of *Ixodes scapularis*, the vector of HGE, we experimentally inoculated four white-tailed deer with a human isolate of HGE. Two deer served as negative controls. Infection dynamics were monitored by culture, PCR, serology and examination of granulocytes in Giemsa-stained whole blood smears. Cell culture and/or reverse transcriptase nested polymerase chain reaction (RT-nPCR) results from peripheral blood indicated that *A. phagocytophilum* circulated for at least 17 days in all deer. Moreover, the organism was consistently re-isolated from two deer at each of 4 sampling dates during a 23-day period between 16 and 39 DPI. Morulae were not observed in granulocytes, and there was no indication of clinical disease. These results confirm that white-tailed deer can support an *A. phagocytophilum* infection, with a resulting rickettsemia of sufficient duration to infect tick vectors. The relative role of white-tailed deer as competent reservoirs in the enzootic maintenance of *A. phagocytophilum* merits investigation.

[31] TUBERCULOSIS IN AFRICAN BUFFALO (*SYNCERUS CAFFER*): POPULATION EFFECTS OF A CHRONIC DISEASE

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The impact of chronic diseases on host vital rates is hard to measure in wildlife populations, and the effects of such diseases on population dynamics remain largely unknown. Bovine tuberculosis, caused by *Mycobacterium bovis*, is a chronic disease affecting many mammalian species. In African buffalo (*Syncerus caffer*), TB infections are usually manifested by characteristic caseous lesions in the lungs and associated lymph nodes. Here I present data from TB testing at Hluhluwe-Umfolozi Park, South Africa, that suggest that TB affects both fecundity and survival in African buffalo. Using a matrix population model, I show that the disease significantly decreases the population's growth rate; and therefore, its resilience to environmental and human-caused disturbances. The net effect of TB on buffalo population size depends on the interaction of disease with other regulating effects, and on the extent of environmental variation the population is exposed to.

[32] TRANSMISSION OF ZOONOTIC ISOLATES OF *GIARDIA* AND *CRYPTOSPORIDIUM* TO HARP SEALS

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Giardia and *Cryptosporidium* have recently emerged as potentially important parasites of marine mammals. It has been postulated that these coccidian parasites are entering the marine coastal areas though agricultural run-off and by municipal wastewaters which has been inadequately treated. Studies have identified *Cryptosporidium hominis* in an Australian Dugong and *Cryptosporidium* and *Giardia* in the feces of Californian sea lions. To date, only *Giardia* has been identified the feces of phocid species. Clinical signs of giardiasis and cryptosporidiosis in terrestrial mammals include severe diarrhea, abdominal cramps, nausea, and weight loss. These symptoms may persist for a few weeks or may evolve into a chronic reoccurring disease. Infection patterns, pathology, and the zoonotic potential of giardiasis and cryptosporidiosis in pinnipeds have not been studied, yet these diseases may have zoonotic and biological significance. The aim of this study was to follow the course of infection *Giardia* and *Cryptosporidium* in experimentally challenged pinnipeds. Twenty weaned harp seal (*Phoca groenlandica*) pups were captured in the Gulf of St. Lawrence and transported by helicopter and plane to Mont-Joli. Four seals served as pre-inoculation controls. Eight seals were orally inoculated with human infective strains of *G. duodenalis* (Assemblage A) cysts and *C. parvum* (genotype 2) oocysts. These 8 seals were housed in 2 salt-water tanks with 8 non-inoculated seals (experimental controls). The results demonstrated both *Giardia* and *Cryptosporidium* are capable of infecting harp seal pups through direct and indirect routes. This is the first report of cryptosporidiosis in phocids. This study also demonstrated indirect, water-borne transmission of *Giardia* and *Cryptosporidium* from experimentally inoculated to non-inoculated seals via fecal contamination of the tank salt water. These findings have significant impacts on public health, environment and wildlife conservation.

[33] MOLECULAR VARIATION IN THE VARIABLE LENGTH PCR TARGET (VLPT) AND 120-KDA ANTIGEN GENES OF *EHRLICHIA CHAFFEENSIS* FROM WHITE-TAILED DEER

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The white-tailed deer (WTD) is the principal reservoir for Ehrlichia chaffeensis, an obligate intracellular bacterium that causes human monocytotrophic ehrlichiosis (HME), the most important emerging tick-bone disease in the southeastern United States. Two surface expressed antigen genes of E. chaffeensis, the variable length PCR target (VLPT) and 120-kDa, have been shown to contain variable numbers of tandem repeats. In this study these two E. chaffeensis antigen genes were characterized for 12 culture isolates and 95 infected whole blood samples from WTD collected from 58 populations in 12 states. The VLPT and 120-kDa genes from infected WTD contained numbers of repeats similar to those reported from humans and ticks, although a new variant of the 120k-Da gene containing 5 repeat units was found in deer. Sequence analysis of the VLPT gene showed that E. chaffeensis from WTD contained more nucleotide variation than previously reported for infected humans or ticks and a novel amino acid repeat type unit. Unlike previous suggestions based on limited samples from humans and ticks, no geographic clustering was apparent as various repeat variants of E. chaffeensis from WTD were detected throughout the study area. In addition, single populations and individual deer were infected with multiple genetic variants of E. chaffeensis. This is the most extensive study of the genetic variation of the VLPT and 120-kDa genes of E chaffeensis and the first to examine genetic variation in E. chaffeensis from a nonhuman vertebrate host. Inclusion of reservoir-source organisms in the comparison of E. chaffeensis isolates provides a more comprehensive understanding of the intraspecific genetic variation of this pathogen.

[34] PATHOLOGY OF NATURAL WEST NILE VIRAL INFECTION OF RAPTORS IN GEORGIA

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Since its introduction to the United States in 1999, West Nile virus (WNV) has caused significant mortality in wild birds of many species. Although there has been significant emphasis on surveillance and transmission, particularly in corvids and other passerine species, there has been little focus on raptorial species or on basic pathology. As part of the West Nile surveillance effort, 260 raptors from Georgia were submitted postmortem for West Nile testing. These birds were necropsied and virus isolation was performed on heart, brain, and cloacal swabs. Histopathology and immunohistochemistry for West Nile virus were also performed. Of the 260 birds examined, 19 were positive for West Nile virus by virus isolation and 14 were positive by immunohistochemistry. Of these 19, 17 had histologic lesions consistent with West Nile viral infection. Of the remaining two, one had multiple penetrating traumatic injuries and the other had nonspecific evidence of disease. The most common histologic lesions associated with WNV infection were myocardial lesions (necrosis, lymphoplasmacytic to histiocytic inflammation, fibrosis) and lymphoplasmacytic encephalitis. Other lesions included hepatitis, myositis, lymphoid depletion in spleen and bursa, splenic and hepatic hemosiderosis, pancreatitis, and ganglioneuritis. Gross lesions included calvarial hemorrhage, myocardial necrosis, and splenomegaly. The majority of WNV positive birds were juvenile (12/19) and female (14/19); however, the majority of submitted birds were also juvenile (152/260) and female (145/260). Red-tailed hawks were most commonly affected, accounting for 8 of the 19 positives, although they comprised only 17% (44/260) of submissions. Also affected were sharp-shinned hawks (4/30), Cooper's hawks (3/63), a red-shouldered hawk (1/32), an osprey (1/5), a barred owl (1/18), and a great horned owl (1/16). Although 34 screech owls were submitted, none was positive for WNV. Barn owls, broad winged hawks, and an American kestrel were also negative for WNV, but these species were submitted in such low numbers (5, 2, and 1, respectively) that no conclusions can be drawn. Although birds were submitted and examined throughout the year, positive cases occurred only during the summer and late fall (July-November). West Nile viral infection accounted for 7% of mortality in 2001 (August to December) and 10% in 2002 (January to December). Therefore, West Nile appears to be a relatively stable and moderately low source of mortality in raptors in Georgia.

[35] EVIDENCE OF TICK-BORNE DISEASE AGENTS IN LONE STAR TICKS (*AMBLYOMA AMERICANUM*) FROM NORTHEASTERN GEORGIA

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White-tailed deer (*Odocoileus virginianus*) are a known or suspected reservoir host for at least three tick-borne disease agents: *E. chaffeensis*, the causative agent of human monocytic ehrlichiosis; *E. ewingii*, the agent of canine granulocytic ehrlichiosis and and a cause of human ehrlichiosis; and *Borrelia lonestari*, the putative agent of Southern Tick-Associated Rash Illness (STARI). Lone star ticks (LST; *Amblyomma americanum*) are known or suspected to transmit all three of these disease agents among white-tailed deer and to humans. Because northeastern Georgia has a high abundance of both LST and white-tailed deer, and at least one of these organisms, *E. chaffeensis*, is endemic in the area, we assayed individual LST collected during March through May from Clarke County, Georgia for these three organisms. From 2001 through 2003 a total of 200 ticks were dissected and midgut contents assayed by PCR using species-specific primers. Of 200 ticks tested, 19 (9.5%) harbored at least one of the three organisms. In 2001, 6.1% (3/49) and 4% (2/50) had DNA consistent with *E. chaffeensis* by amplification of a variable length PCR target (VLPT) and 16S rDNA gene, respectively; 4% (2/50) were positive by VLPT in 2002, and of 100 ticks tested in 2003, all were negative for *E. chaffeensis* by both PCR targets. DNA consistent with *E. ewingii* by amplification of 16S rDNA was not detected in 2001 ticks, but was detected in 4% (2/50) of ticks from 2002 and 7% (7/100) of ticks from 2003. One tick sampled in 2002 was co-infected with both *E. ewingii* and *E. chaffeensis*. PCR amplification of the flagellin gene revealed DNA consistent with *B. lonestari* in 4% (2/50) ticks from 2001, compared to 0% (0/50) in 2002 and 2% (2/100) in ticks sampled in 2003. *Borrelia* sp. spirochetes were also visualized by a fluorescent antibody test on the two PCR positive ticks from 2003. These results reconfirm the presence of *E. chaffeensis* and establish evidence *E. ewingii* and *B. lonestari* in questing adult LST from this area of Georgia, and provide evidence that at least two of the three organisms tested have been present in ticks over each of the previous three years, suggesting that WTD and people in this area may be exposed to all of these organisms.

[36] WEST NILE VIRUS IN AVIAN SPECIES OF GEORGIA

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West Nile virus (WNV), a flavivirus that in its most severe form causes fatal encephalitis in humans, wildlife, and livestock, was first detected in the southeastern United States (US) in the summer of 2000. As the amplifying hosts and vectors are abundant throughout the southeastern US, WNV has become a serious human and animal health risk in this area. The specific objective of this study was to identify common peridomestic avian species that are potentially involved in the epidemiology of WNV in the southeastern United States and to determine the utility of these species as potential indicators for this virus over the physiographic and land-use variation present in this area. Antibody prevalence rates by species were compared over the entire state of Georgia, within specific physiographic areas, and by land use categories. 2,714 avian serum samples from birds captured within 94 counties of the state of Georgia during the summer of 2002 were screened with serum-dilution plaque reduction neutralization tests. 146 of these screened samples were positive for flavivirus antibodies and endpoint titers were determined, with 141 samples testing positive for WNV. Birds with antibodies to WNV were found in each physiographic region of Georgia as well as in each land use type. Rock doves (Columba livia) and Northern cardinals (Cardinalis cardinalis) appear to be the best avian indicators of WNV in the state of Georgia based on ease of capture, distribution throughout the state, proximity to humans, high WNV antibody titers, as well as high WNV antibody prevalence in comparison to other avian species tested in Georgia.

[37] EVALUATION OF TISSUE CONTAMINANT CONCENTRATIONS AND SELECTED HEALTH PARAMETERS IN CARIBOU (*RANGIFER TARANDUS*) IN LABRADOR, CANADA

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Barren-ground and woodland caribou (*Rangifer tarandus*) are important species in many northern ecosystems and for Inuit and First Nations communities. Residents of Labrador Innu communities hunt caribou for subsistence primarily from the George River herd (GRH). There is a wide body of literature on various components and factors of health in caribou in Canada and elsewhere including body condition, parasite burdens and tissue contaminant levels. However, research into links between health parameters and potential influencing factors has been rare. Twenty-seven caribou from the GRH killed by Innu hunters in 2001 were evaluated for selected health parameters and tissue contaminant levels as part of a larger study on environmental contaminants, wildlife health and subsistence hunting by Labrador Innu. Regression analyses were carried out to assess the relationship between contaminant levels and health parameters including body condition and infection with the giant liver fluke *Fascioloides magna*. Overall, the sampled caribou appeared to be in adequate condition for the time of year and no clinically significant lesions were found. Levels of heavy metals, organochlorine pesticides and polychlorinated biphenyls in the caribou were within the ranges reported for caribou in Canada and no association was found between contaminant levels and body condition. The levels of renal Cd and Se were found to be significant predictors of *F. magna* abundance and mean intensity in the liver. Further investigation into this relationship in other ungulate populations endemically infected with *F. magna* may be warranted.

[38] LONG-TERM EFFECTS OF LOGGING ON PARASITE DYNAMICS IN AFRICAN PRIMATE POPULATIONS

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Emerging infectious diseases have raised global awareness of the potential impact that ecological change can have on biodiversity conservation and wildlife and human health. This study improves our understanding of this interplay by examining the effects of logging on disease dynamics in three primate species, the redtail guenon (*Cercopithecus ascanius*), the red colobus (*Procolobus badius*), and the black-and-white colobus (*Colobus guereza*). From August 1997 to August 2002, we collected 1,076 primate fecal samples to compare the prevalence and richness of gastrointestinal parasite infections of populations from logged and undisturbed forests in Kibale National Park, Uganda. Helminth eggs, larvae, and protozoan cysts were identified using sodium nitrate flotation and fecal sedimentation. Coprocultures and necropsies facilitated positive identification. Relative infection risk was quantified using a modified sedimentation technique, comparing densities of infective-stage parasites from 1 m^3 canopy and ground vegetation plots. Helminths recovered include *Trichuris trichuria, Oesophagostomum stephanostomum, Strongyloides fulleborni, Capillaria* sp., *Physoloptera* sp., *Enterobius* sp., *Coloboenterobius* sp., *Bertiella* sp., and *Dicrocelium lanceatum.* Protozoans recovered include *Giardia lamblia*, *Iodamoeba buetschlii*, *Chilomastix mesnili*, *Entamoeba histolytica*, and *Entamoeba coli.* The prevalence and richness of gastrointestinal parasite infections and the mean number of parasite species infecting individuals were significantly greater for guenons in logged compared to undisturbed forest, but these parameters did not vary between forests for either of the colobines. Infective-stage primate parasites were found at significantly higher densities in both canopy and ground vegetation plots from logged compared to undisturbed forest, demonstrating a greater infection risk for primates in logged forest. Our results suggest that frugivorous populations may be more effected by disease than folivores. This may explain why the abundance of frugivorous primate populations in logged forest at Kibale continue to decline 30 years after logging. Selective logging is a dominant habitat modification pattern throughout the tropics and understanding how this affects primate-disease dynamics is vital for designing effective conservation and management plans and improving our understanding of the interplay between ecological change and health.

[39] NATURAL AND EXPERIMENTAL WEST NILE VIRUS INFECTION IN FOUR RAPTOR SPECIES

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The extent of morbidity and mortality due to West Nile virus (WNV) in raptor species in the wild has been difficult to document, though at least 25 species of raptors have tested positive for WNV. WNV was blamed for numerous raptor deaths in the United States and Canada in 2002, though the effects of WNV infection in raptors remain poorly understood. We studied the effects of natural and experimental infections of WNV in four raptor species. American kestrels (*Falco sparverius*), great horned owls (*Bubo virginianus*), and barn owls (*Tyto alba*) were mosquito-infected, fed WNV-infected mice, or needle-inoculated, while six red-tailed hawks (*Buteo jamaicensis*) were naturally infected in the wild. We monitored all birds for clinical signs, tested tissues by virus isolation, and examined tissues for gross and microscopic lesions. Experimentally infected birds were evaluated for viremia, and oral and cloacal shedding of virus. Mosquito-infected kestrels and all great horned owls reached infectious level viremia from 1-5 days post-infection (DPI), while barn owls failed to reach infectious levels. Only mosquito-infected kestrels and mosquito- and needle-inoculated great horned owls shed infectious levels of virus from the oral cavity. Oral shedding was intermittent for barn owls at levels significantly lower than for other species, and they did not shed detectable levels of virus via the cloaca. Kestrels fed infected mice did not become detectably viremic or shed virus. Virus was isolated from spleen, kidney, skin, and eye of various birds. Histopathological findings varied among species and by method of infection. The most common histopathological lesions were mononuclear cell accumulations in liver, heart and kidney. A few birds had a more acute, severe disease condition represented by vasculitis and associated with tissue degeneration and necrosis. Results of this study have important implications concerning WNV infection in raptors, as well as the future management of raptor species.

[40] EVALUATION OF SUCCESS OF A SEA OTTER REHABILITATION PROGRAM AND IMPLICATIONS FOR POPULATION MANAGEMENT

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Despite the increased use of individual animal rehabilitation in attempts to mitigate damages and restore wildlife populations after catastrophic events, such as oil spills, there are no established criteria for assessing the effectiveness of rehabilitation programs. A southern sea otter (*Enhydra lutris nereis*) pup rehabilitation program was used as a case study to both evaluate the success of an orphan rehabilitation program and establish criteria for evaluating other similar programs from a population perspective. From July 1996 through January 2000, 21 rehabilitated and 16 free-ranging juvenile southern sea otters were surgically implanted with radiotransmitters and monitored in the wild for one year post-release/weaning in order to determine survival rates and mortality causes for the two groups. Seven veterinary and biological criteria were proposed for evaluating the wildlife rehabilitation program effectiveness. The sea otter rehabilitation program was judged favorably according to five of the seven criteria, including survival of individuals within the captive population, the completion of diagnostic testing prior to release, released animals having similar patterns of dispersal to wild counterparts, and similar rates of survival and reproduction of rehabilitated individuals in the wild relative to the source population. Specifically, no difference in survival was found between rehabilitated (n=21) and free-ranging juveniles (n=16), but survival of females from both groups was much lower than males ($P < 0.02$). The other two criteria, absence of abnormal behavior in the released animals and augmentation of the population with a corresponding positive growth rate, were not satisfactorily achieved. Only rehabilitated juveniles interacted with humans after release, possibly due to rearing strategy in captivity and the opportunity for contact via heavy recreational use by humans in the released sea otters' habitat. Other causes of failure/mortality were similar between the two groups. The release of 21 individuals from this program could not combat the negative and stagnant southern sea otter population growth during this study. These criteria provide valuable guidelines for the future development of sea otter rehabilitation programs and the evaluation of such programs' contributions to sea otter conservation. In addition, the poor survival of juvenile females in both groups has important demographic implications for recovery of the southern sea otter population.

[41] DYNAMICS OF RABIES AND STRIPED SKUNK (*MEPHITIS MEPHITIS*) POPULATIONS: AN INDICATION OF DISEASES AS POPULATION REGULATION MECHANISM

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Striped skunk (*Mephitis mephitis)* populations seemingly have high recruitment and turnover rates. In our study, we investigated cause-specific mortality and the survival rates of skunks in Saskatchewan. We also investigated the prevalence of rabies in the population. The survival rates of skunks can be categorized into fall (September 1 to November 30), winter (December 1 to February 27), spring (March 1 to May 31) and summer (June 1 to August 31) periods. Dead individuals were collected and sent to the Western College of Veterinary Medicine for necropsy to determine the cause of death. 141 (61 males, 80 females) skunks were captured and radio-collared in Willowbrook, Saskatchewan from 1999 to 2002. The prevalence of rabies in 1999 was 55% and it was significantly higher than in 2000, which was 17%, whereas in 2001 and 2002, no cases of rabies were reported. Accordingly, survival rates of skunks during the period have increased. Cause-specific mortality of skunks over the period has shifted from diseases including rabies to humans (e.g. shooting, poisoning etc) and predation. Our preliminary results suggest that diseases affect the survival rates of the population and it might be a potential regulating factor in the striped skunk population.

[42] FLEA COMMUNITIES AND SMALL MAMMALS IN PHILLIPS COUNTY, MONTANA: IMPLICATIONS FOR THE MAINTENANCE OF SYLVATIC PLAGUE

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Sylvatic plague is a disease that was introduced into North America ~1900 and has since become well established in native rodent populations in the western United States. Susceptibility to infection and disease varies widely among taxa. The etiologic agent of plague is the bacterium *Yersinia pestis* and most transmission between hosts occurs indirectly through the bite of an infected flea (Siphonaptera). In addition to their vectorial role, fleas may also act as reservoirs of plague following an epizootic which results in the die-off of preferred hosts. Rodent burrow systems provide hospitable environments where fleas infected with *Y. pestis* may survive and remain competent reservoirs/vectors for many months. In this sense, understanding flea ecology, abundance, and host relationships is central to understanding the mechanisms by which sylvatic plague is maintained in natural systems.

A total of 1,517 fleas were collected from small mammals and prairie dog burrows in Phillips County, MT during June – August 2002. Deer mice (*Peromyscus maniculatus*) and black-tailed prairie dogs (*Cynomys ludovicianus*) together represented 98.3% of all captured mammals. Overall prevalence of flea parasitism on deer mice and prairie dogs at initial capture was 54.0% and 75.2%, respectively. Flea loads ranged from 0 to 19 for deer mice and from 0 to 54 for prairie dogs. There was no difference in prevalence (percent with fleas) or intensity (number per "infected" host) of flea parasitism by sex or age for either species. Deer mice captured on prairie dog colonies had a higher prevalence of flea parasitism ($p = 0.004$) than those captured off prairie dog colonies. Flea intensity was also higher for deer mice captured on prairie dog colonies, although not significantly. However, there was no correlation between flea abundance on sympatric populations of deer mice and prairie dogs and there were no flea species common to both hosts. A review of the literature also shows that interspecific exchange of fleas between black-tailed prairie dogs and associated non-sciurid rodents is a rare event. Patterns of rodent and flea species diversity in relation to prairie dog colonies with and without a history of sylvatic plague activity in southern Phillips County will also be presented.

[43] PHENOTYPIC DIFFERENCES IN SEVERAL ISOLATES OF THE HOUSE FINCH STRAIN OF *MYCOPLASMA GALLISEPTICUM*

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Beginning in 1994, *Mycoplasma gallisepticum* (MG) spread as an epidemic through the populations of house finches (*Carpodacus mexicanus*) in eastern North America. Early in this epidemic approximately 60% of wild and captive house finches developed conjunctivitis, and mortality among these birds was high. Over the last few years, prevalence of the disease has decreased to 4-6% annually, and mortality among infected finches has also declined. These changes may be correlated to changes in the finches, the bacteria, or both. Early analysis on the house finch MG concluded that a single strain was responsible for the epornitic. However, nucleotide sequencing of a cytadhesion protein revealed sixteen different genotypes in the 55 house finch isolates evaluated, although one genotype represented over 70% of these isolates. The goal of this study was to determine if any phenotypic variation could be found between these genotypes that may be partially responsible for the changes we've seen in the host-parasite relationship over the last several years. To evaluate several measures of virulence in the house finch MG we developed an *in vitro* assay using house finch tracheal organ culture. We have determined that the house finch MG does bind to tracheal epithelial cells, both in natural infections and in cell culture, and that this attachment and colonization of the MG can lead to loss of cilial motility, a virulence mechanism common to most mycoplasmas. To measure cilial activity we use a simple colorimetric assay in which MTS tetrazolium salt is bioreduced into formazan in the presence of mitochondrial activity. The absorbance of formazan can be measured directly and is directly proportional to the number of living cells in the culture. With this assay we can now compare a number of isolates representing the different genotypes to determine whether their ability to alter cilial activity is different and these results will be presented at the meeting. We are also developing a quantitative PCR assay that will allow us to measure differences between isolates in their attachment rates and their rates of reproduction in house finch tracheal rings.

[44] CAUSES OF MORTALITY IN BOTTLENOSE DOLPHINS (*TURSIOPS TRUNCATUS*) STRANDED ALONG CENTRAL SOUTHWEST FLORIDA FROM 1985-2002

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From January 1, 1985 to December 31, 2002, 289 bottlenose dolphins stranded on the central southwest coast of Florida and were examined for cause of death and collection of biological data. These animals either stranded live and subsequently died, or were recovered dead. The animals were comprised of 120 females, 120 males and 49 animals of unknown gender. There were 94 animals that were partial carcasses or skeletal remains. Complete or partial post-mortem examinations were performed only on whole carcasses.

Of the 195 animals with post-mortem examinations performed, 47% of the animals were estimated to be less than 3 years of age base upon length (animals less than 210 cm). Cause of death was determined by gross post-mortem examination and/or histopathology findings. In 51% of the cases, cause of death could not be determined due to advanced decomposition (n=67) or to open diagnoses (n=33). The mortality categories of the remaining animals (n=95) included fisheries/human interaction (n=6), trauma of unknown origin (n=19), perinatal mortality (n=28), and natural diseases (n=42). Fisheries/human interactions included animals with entanglements that caused death by drowning, gastrointestinal obstructions/inanition and/or respiratory compromise. Trauma of unknown origin was characterized by lesions such as hemothorax; hemoperitoneum; pre-mortem soft tissue edema, bruising and hemorrhage; and pre-mortem rib and skull fractures. Perinatal mortality included animals that were less than 125 cm in length and had evidence of fetal folds, presence of umbilicus or umbilical stump, and no eruption of teeth. Natural diseases included bacterial and parasitic diseases, emaciation, reproductive complications (dystocia), and gastrointestinal foreign body obstructions/perforations.

[45] THE OCCURRENCE AND FOOD SAFETY IMPLICATIONS OF TRICHINELLOSIS IN MARINE MAMMALS

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Trichinellosis in arctic mammals is caused by *Trichinella nativa* (T2). The first marine mammal isolate was made in 1934 in polar bears and since that time, trichinellosis has been confirmed in walruses, bearded seals, ring seals and a single Beluga whale. Accumulating animal behavior and disease surveillance data have lead to a better understanding of the epidemiology of trichinellosis in marine mammals. Cannibalism is believed to be the primary mechanism for maintaining trichinellosis in polar bears and evidence is accumulating to suggest that an independent *Trichinella* cycle may occur in walruses. Seals and whales appear to be incidental hosts. The consumption of infected walrus meat is the most frequent cause of human trichinellosis in the arctic and is recognized as a significant food safety risk. A regional public health board in the Canadian arctic, in conjunction with the Centre for Animal Parasitology of the Canadian Food Inspection Agency, recently developed a diagnostic system to test the meat of harvested walruses before distribution for consumption. In order to evaluate the food safety risk in non-commercially prepared (country) foods prepared using *T. nativa*-infected meat, grey seals (*Halichoerus grypus*) were successfully infected with *T. nativa* and resultant muscle tissue was used to produce igunaq (aged/fermented meat), nikku (air dried raw meat) and sausage. These foods were bioassayed over time using mice and cats. Infective *T. nativa* larvae survived for at least 5 months in igunaq, nikku and raw frozen sausage, and in poorly cooked sausage. These data indicate a significant food safety risk for foods prepared using *T. nativa*-infected meat, and originate from the first experimental trichinellosis infection in seals.

[46] SUSCEPTIBILITY OF GREY SEALS TO *TOXOPLASMA GONDII* OOCYSTS, AND FOOD SAFETY IMPLICATIONS

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Despite several published reports of marine mammals naturally infected with *Toxoplasma gondii*, the source of infection and the effect on the health of these animals are unknown. The objectives of this study were to determine experimentally the susceptibility of seals *(Halichoerus grypus*) to *T. gondii* oocysts and the viability of stages that develop in seals, and to assess the infectivity of the parasite in country foods prepared with infected seal meat. Eight weaned seals approximately 4 weeks old were captured and raised in confinement tanks. Five weeks later, each of 4 seals was orally inoculated with 100 or 10,000 oocysts of *T. gondii* (VEG strain), and another 4 seals served as negative controls. Occasionally, mild behavioural changes were observed in infected seals, but not in control animals. Blood samples were collected weekly from all animals, and at 5 or 10 weeks post-inoculation seals and mice were killed. A modified agglutination test revealed the presence of antibodies to *T. gondii* in sera collected from inoculated seals and mice. No stage of the parasite was found on an extensive histological examination of sections collected from seal tissues, and immunohistochemical staining of numerous sections revealed a single cyst in one of the low-dose inoculated seals. Control mice inoculated with 10 oocysts became serologically and histologically positive to *T. gondii*. Cats fed brain or muscle tissues from inoculated seals had *T. gondii* oocysts in their feces within two weeks of feeding. However, cats fed infected seal meat products prepared by drying (nikku or jerky), fermenting (igunaq), or salting (sausage) and bioassayed in cats did not shed oocysts. This study provides experimental evidence that *T. gondii* oocysts can establish viable infection in seals, and supports the hypothesis that toxoplasmosis in marine mammals can be acquired from oocysts in surface water runoff and sewer discharge. Results also suggest that *T. gondii* might not survive processing conditions used in some country foods.

[47] DESCRIPTION OF THE PHOCINE DISTEMPER IN THE NETHERLANDS DURING 2002

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During 2002, high mortality of harbour seals (*Phoca vitulina*) occurred in northern European waters caused by phocine distemper virus (PDV). Because this was only the second PDV epidemic ever recorded, the objectives of this study were to provide a detailed description and to compare the epidemic with the previous one in 1988.The date and location of stranding of 2248 sick and dead seals (of a total of 3595 harbour seals counted in 2001) were provided by seal rehabilitation centres and coastal municipalities to a central reporting centre of the Ministry of Agriculture, Nature Management and Fisheries. Clinical signs were monitored in 19 PDV-positive seals that were accepted by the Seal Rehabilitation and Rescue Centre, Pieterburen. The carcasses of approximately 1400 seals were necropsied by trained personnel to obtain basic biological data, to describe gross lesions, and to collect tissue samples for various laboratory examinations, including RT-PCR for morbillivirus RNA. The sequences of P-gene and N-gene fragments of a selected number of positive samples were determined for phylogenetic analysis. Following the Dutch index case on the 16th of June, the weekly stranding rate increased rapidly, peaked in the fourth week of August and the third week of September, and declined to low numbers by November. Although both harbour seals and grey seals (*Halichoerus grypus*) occur along the Dutch coast, more than 99 % of stranded animals were harbour seals. Using standard length to estimate age class, 16 % were juvenile, 54 % were subadult, and 30 % were adult. Both sexes were equally represented. In the south of the Netherlands (Zeeland), the peak of mortality occurred a month later than in the north (Wadden Sea). The main clinical signs observed were respiratory distress (100%), conjunctivitis (70%), mucopurulent oculonasal discharge (55%), nervous signs, including absence of facial nerve reflexes, tremors or convulsions (50%) and diarrhoea (25%). The main gross lesions in freshly dead seals were pulmonary consolidation and subcutaneous, mediastinal, and subpleural emphysema. Infection with PDV was detected by RT-PCR in 155/313 (50 %) seals. Analysis of the P-gene and N-gene fragments showed that the PDV sequences from 2002 and 1988 had more than 97% identity, although they could be distinguished from each other. Above data indicate that PDV infection caused a propagating epidemic with high mortality in harbour seals, as in 1988, and that grey seals were either absent or less susceptible to the disease. The prevalence of nervous signs in the infected seals was much higher than reported in 1988. The observed low genetic divergence over a period of 14 years is an indication for the high genetic stability of PDV, and suggests that the PDV sequences from 1988 and 2002 may both originate from viruses circulating in the same geographical area. The repeated occurrence of PDV epidemics forms a substantial mortality factor for the northern European seal population, and needs to be considered in its management.

[48] IMMOBILIZATION OF FREE-RANGING MALE SOUTHERN SEA LIONS (*OTARIA BYRONIA*) WITH TILETAMINE-ZOLAZEPAM AND ISOFLURANE

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A total of 9 (nine) free-ranging sub-adult male southern sea lions (Otaria byronia) were immobilized between November 1999 (n=2) and January 2003 (n=7). Because physical restraint of such large animals (wt=300-400kg) is impracticable under field conditions, an injectable anesthetic was administered with a CO2 powered dart gun (Daninject®). Tiletamine-zolazepam (Telazol®) in doses of 1.19-1.67mg/kg (X= 1.38 mg/kg; n=9) was mixed in darts with atropine (17.14 – 20 mcg/kg; X= 18.36; n=7). Initial drug effects were observed at 3.14min (range 2-5min) and animals became recumbent at 8.66 min (range 2-13min). Isoflurane (5%) was administered via face mask to animals that were not sedate enough to be safely handled resulting in recumbancy in 2-3 minutes. Light anesthesia was achieved in seven animals, while two reached surgical anesthesia levels. Apneas were observed in these two animals, which had to be intubated for ventilatory support. Telazol immobilization was partially reversed with flumazenil at doses of 1mg flumazenil for every 25-30mg of zolazepam used. Additionally, doxapram was used to stimulate respiration in one animal at 0.331mg/kg. Effects of flumazenil were not always predictable and recoveries to mild sedation were seen at 60-135min (X=97.77min). Abnormal (stormy) recoveries were observed in three animals. Overall ratings of anesthesia were good or excellent in six cases and fair to poor in three. Physiological parameters monitored throughout procedures included temperature, respiration, heart rate and oxygen saturation. The anesthetic agents used were relatively safe and provided adequate sedation for the purpose of this study. However, quality and duration of anesthesia were unpredictable and apparently unrelated to telazol dosages used. In addition, supplementation of immobilizing agents might be required, as well as respiratory support. Therefore, field equipment suited for this purpose must be available at all times, complicating logistics.

[49] MARINE MAMMALS AND 'WILDLIFE REHABILITATION' PROGRAMS

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Wildlife rehabilitation involves rescue or capture, care and treatment of abandoned, orphaned, injured or sick wild animals. Wildlife that has been successfully treated may or may not be released to the wild depending on the nature of their injuries and ability to resume normal activities in a manner that does not jeopardize their survival nor place the public or other animals at risk. It is generally unknown whether treated and released wildlife have in fact been "rehabilitated". Existing or proposed legislation involving responsible agencies and international agreements pertaining to marine mammals and control of disease in Canada, and to some extent in the United States, is reviewed. Marine mammal stranding and mortality events, efforts at rescue, rehabilitation and release of stranded marine mammals, advantages and disadvantages for wildlife and humans, with potential consequences and costs involved are also provided. Prohibition or strict regulation of marine mammal rehabilitation if permitted in a jurisdiction is recommended including addressing transboundary issues of concern to national and international agencies reponsible for the conservation of marine mammals especially endangered or threatened species. Guidelines developed by the OIE and IUCN are useful and should be followed for the welfare of animals, control of disease, behavioural and genetic aberrations, and maintenance of healthy wild populations and the ecological integrity of the areas they inhabit.

[50] THE EFFECTS OF ENVIRONMENTAL CONTAMINANTS ON IMMUNE FUNCTION AND HEALTH IN FREE-RANGING PINNIPEDS IN ALASKA

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A number of marine mammal populations in Alaska have decreased in recent decades. The western stock of the Steller sea lion (*Eumetopias jubatus*) has undergone a severe decline resulting in listing as an endangered species. The northern fur seal (*Callorhinus ursinus*), with 80% of the world population breeding on the Pribilof Islands, has been designated as a depleted stock. The cause(s) of these population declines have not been discovered and several areas of investigation have yet to be thoroughly explored including the role of environmental contaminant exposure on health. Organochlorine (OC) contaminants have recently been identified to be present in the tissues of marine mammals in Alaska at concentrations higher than expected. Organochlorine contaminant exposures have been linked to immune suppression and reproductive dysfunction in marine mammals.

In field studies conducted during live-capture operations from 1995 to 2001, we investigated OC contaminant and mercury exposure along with the general health and development of immune function in juvenile northern fur seals and Steller sea lions. We optimized and validated multiple immune functional assays for use in these species, starting with the northern fur seal. These assays were then used to define each of the components of the immune system quantitatively and qualitatively in relation to age. Our approach included lymphocyte function assays [lymphoproliferative assays (specific T-cell and B-cell function), flow cytometry, IL-2 receptor expression, immunoglobulins, and specific antigen stimulation (B-cell function)]; and less specific white blood cell differential counts to demonstrate perturbations in leukocyte subpopulations and inflammatory/stress responses. These assays mainly utilized peripheral blood of free-ranging animals live-captured in Alaska, complemented with some captive animal validation.

By examining multiple cohorts of Steller sea lions from different stocks as well as repeat sampling of fur seals from birth to weaning, we documented baseline individual, stock, age-related, and stress-induced variation in responses in immune function in growing animals over time; thereby validating the use of these assays to assess the health of free-ranging otariids. We established reference ranges for normal leukocytes subpopulations for different age groups of free-ranging juveniles. Additionally, in Steller sea lions, we conducted expanded health surveys including serology, parasitology, bacterial cultures, viral cultures and viral PCR, fungal cultures, testing for Chlamydia by culture and PCR as well as detailed physical examinations.

These investigations detected significant correlations between OC exposure and impaired immune function at several levels including T-cell-mediated B-cell responses. Antibody production responses in fur seal pups to primary and secondary tetanus toxoid

vaccinations were negatively correlated to circulating blood levels of selected polychlorinated biphenyl congeners at the time of vaccination. Developmental age could not explain this effect. Responses to mitogen stimulation using lymphoproliferative assays in fur seals and Steller sea lions were negatively correlated to PCB levels but the effects of developmental age had an impact on these results in fur seals.

[51] PATHOLOGY, MICROBIOLOGY, AND CLUTCH VIABILITY OF FLORIDA AMERICAN ALLIGATOR (*ALLIGATOR MISSISSIPPIENSIS)* EMBRYOS AND NEONATES NATURALLY EXPOSED TO ORGANOCHLORINES

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In the last two decades, alligator populations in several Central Florida lakes have experienced a decreased hatching rate and increased neonatal mortality. Much of the research so far has focused on potential reproductive abnormalities and exposure to endocrine disrupting contaminants (EDCs). The lakes with the most documented problems include Lake Apopka, adjacent Emeralda Marsh Conservation Area, and Lake Griffin. Alligators from these sites have died from *in ovo* infections, and neonatal animals are often runted and poor doers. EDCs have been reported to contribute to immune system dysfunction in a number of animal species. It was hypothesized that exposure to EDCs via the yolk was contributing to immunosuppression in alligator embryos during development, resulting in increased susceptibility to infectious disease in the embryonic and perinatal periods. Several studies were conducted in the summer of 2000 to determine the causes of morbidity and mortality of alligator embryos and neonates, to determine the microflora to which the developing embryos were exposed, and to relate clutch viability with yolk organochlorine concentrations.

First, the egg microbiology and pesticide content of the yolks were studied. The hypothesis was that eggs exposed to pesticides would contain more bacteria from a lack of defenses to restrict bacterial growth, and that infections may arise in the embryos if they lacked in ovo defenses. Eggs were collected by the FFWCC from 4 lakes (Apopka, Griffin, Woodruff, and Emeralda). Lake Woodruff was the reference lake with little pesticide contamination. 64 samples from 35 clutches were submitted for organochlorine analyses. The viability for each clutch was compared to the results of the toxicologic analyses. Aerobic bacterial cultures were performed. There were over 60 bacterial species isolated including both Gram-positive and Gram-negative organisms. A mean of 4 aerobes was isolated per egg. The Gram-negative organisms *Stenotrophomonas maltophilia*, *Agrobacterium tumefaciens*, and *Pseudomonas aeruginosa* were the most frequently isolated. There was no evidence of a difference among lake sites or clutches in the total number of bacteria isolated. Fungal isolation was rare except in putrefied eggs. The presence of a few organisms was significantly higher in eggs with dead embryos than with viable embryos. *Pseudomonas* was isolated in 49% of the eggs with

dead embryos. *Serratia marcescens* and *Proteus vulgaris* were isolated from 14% of dead eggs.

For the necropsy study, the FFWCC collected over 4000 eggs from 8 different Florida lakes. The eggs were artificially incubated at 32ºC and produced over 2000 hatchlings. During the 4-week hatching period, the hatchlings were observed on a daily basis, and clinically ill or dead alligators were removed. Dead embryos that were in a relatively good state of preservation were also examined. The most common developmental defects in embryos included growth retardation, anasarca, and tail deformities. Embryos also had inflammation of the yolk sac and albumen, and pericarditis. The most common lesions in the neonatal animals euthanized or found dead because of severe clinical disease included ulcerative enteritis, bacterial hepatitis, suppurative pyelonephritis, suppurative adrenalitis, pyogranulomatous ventriculitis, interstitial pneumonia, and encephalomalacia. Thirty-nine bacterial species were isolated from the necropsied neonatal animals. The most frequent isolates were *Providencia rettgeri*, *Morganella morganii*, *Serratia marcescens*, *Stenotrophomonas maltophilia*, *Citrobacter freundii*, *Corynebacterium pseudogenitalium*, and *Listeria monocytogenes*. A subset of neonatal animals had neurologic disease including ataxia, wide-bases stance, circling, and a loss of righting reflex. Histologically, there was bilaterally symmetrical or unilateral encephalomalacia and necrosis in the telencephalon. Of the 31 brains from neurologic and non-neurologic alligators examined histologically for this study, necrosis was present in 6. Two of these were from a Lake Apopka clutch with low clutch viability and another was from a Griffin clutch with low clutch viability. None of the animals that hatched from this clutch survived more than a week. The neurologic lesions in these neonatal animals are similar to those that have been found in clinically neurologic adult alligators from Lake Griffin in recent years, although the cause(s) of this lesion in the neonates and adults may and may not be related. Possible causes for the necrosis include toxins, toxicants, thiamine deficiency, or other ischemic conditions.

It was hypothesized that clutches with eggs containing higher concentrations of organochlorines would have a decreased hatch rate. However, there did not appear to be a relationship between clutch hatch rates and the concentrations of these toxicants in the yolks. Animals from clutches with 70-90% hatch rates had as high or higher concentrations than animals with 0-20%. Several Lake Apopka eggs had yolk concentrations of DDE as high as 160 ppm, all of which had viable embryos at the time of sampling. Toxaphene concentrations in viable embryos were also high, up to 40 ppm. These studies provide evidence that infections play a role in embryonic and neonatal death, that neurologic disease of unknown cause may also play a role in mortality, and that embryonic mortality may not be directly related to contaminant exposure.

[52] COMPARISON OF CHOLINESTERASE LEVELS BETWEEN CAPTIVE AND WILD JUVENILE BURROWING OWLS (*AHTENE CUNICULARIA*)

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Saskatchewan's burrowing owl population is steadily declining due to habitat fragmentation and destruction, poor nestling survivability, exposure to pollutants and other reasons, which are not fully understood. Exposure to cholinesterase-inhibiting pesticides has been implicated in burrowing owl mortality. Toxic dosages may result in neurological effects, which are seen shortly after exposure, followed by death or apparent recovery. Low level exposure may result in no overt clinical signs. Sub-lethal effects of these agents have not been widely studied. Birds may lose weight, vocalize less, fail to defend territory, raise smaller broods, provide less food for chicks, exhibit abnormal behavior and be more susceptible to predation.

Over a two year period, plasma cholinesterase levels in 16 juvenile burrowing owls bred in captivity were compared to levels in 52 wild juvenile burrowing owls potentially exposed to organophosphate or carbamate pesticides, in order to document whether or not cholinesterase-inhibition (an indicator of pesticide exposure) had occurred. The mean cholinesterase level during the 2000 field season was 3526.9 umol/min/L in captive owls and 3668.6 umol/min/L in wild owls; there was no significant difference between these values. During the 2001 field season the mean cholinesterase level was 2929.9 umol/min/L in captive owls and 4026.9 umol/min/L in wild owls; the level in captive owls was significantly lower than in wild owls, which was the reverse of what was expected.

This study does not support the hypothesis that juvenile burrowing owls are exposed to sub-lethal amounts of cholinesterase-inhibiting pesticides while still in the burrow. Factors such as grasshopper population, and timing and amount of pesticide sprayed cannot be controlled and blood collection at different time periods may have yielded different results. However, it is difficult to access young burrowing owls for sampling once they have left the burrow.

This study was useful in demonstrating that it is possible to collect samples from live chicks opportunistically while they are still in the burrow. Sequential sampling over several weeks to monitor trends in cholinesterase levels may be of value for future studies.

[53] DIETARY, AND SPATIAL INFLUENCES ON CONTAMINANT LEVELS AND RELATED BIOLOGICAL EFFECTS IN TREE SWALLOWS (*TACHYCINETA BICOLOR*) IN POINT PELEE NATIONAL PARK, ONTARIO

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Point Pelee National Park, jutting into western Lake Erie, is Canada's second smallest national park. It is a fragmented landscape of former orchards and farmland, wetlands and Carolinian forest. It is internationally known as an important staging area for thousands of migrating birds during spring and autumn migrations, as well as a vital breeding ground for many species. The tree swallow, an aerial insectivore, was studied as a sentinel species here because of concerns about historic contamination of the park with organochlorine (OC) pesticides. Recently, 'hot spots' of these persistent compounds have been identified within the park. During two breeding seasons, 2001 and 2002, tree swallows were studied to examine food web transfer of these historically used environmental contaminants, and to determine biological impacts of such exposure.

Nest boxes were erected on contaminated and reference sites based upon recent soil residue analyses and historical land use. Bioindicators to evaluate contaminant impact were based upon reproductive, immunotoxicological, hepatic biotransformation, organ weights, tissue residues and dietary analyses. In year I, whole body tissue residues ranged from 3 - 107 ng/g ww for DDE, and 56 - 540 ng/g ww for ∑PCB. The *a priori* classification of sites did not agree with the contaminant levels found in tissues of the nestlings. We therefore reclassified areas based upon tissue residues. There were no significant differences for most of the reproductive endpoints. Nor were hepatic mass, T cell response to the mitogen, phytohaemagglutinin, or hepatic enzyme induction different between sites.

In year II, besides being the coldest spring on record for this area, further complications such as interspecific competition and predation resulted in the loss of most of the first clutches of the tree swallows. The results therefore are based upon the successful 2nd clutches. Tissue residues levels were considerably higher in the second year for both OCs (e.g., DDE 74 - 526 ng/g ww) and PCBs (∑PCB 154 - 1,367 ng/g ww). For all egg, clutch, and survival measures, differences between sites were modest and largely not significant. OC contamination produced no detectable physiological effects, but tissue PCB levels were positively associated with tarsus length ($p < 0.001$), relative hepatic mass ($p = 0.01$), and hepatic enzyme (EROD) induction ($p = 0.001$).

Dietary analyses of the invertebrate prey species being fed to the nestlings in 2002 allowed the comparison of contaminant residues in the nestlings with the relative amounts of aquatic, semi-aquatic and terrestrial insects in their diets. There was a close, significant and positive correlation between a diet high in terrestrial and semiaquatic invertebrates, and OC levels in the nestlings ($p = 0.008$). PCB levels in tissues of nestlings were well correlated ($r = 0.61$) with the proportion of the diet composed of aquatic insects, specifically mayflies (Ephemeroptera).

Differences in diet appear to explain much of the variation in contamination. In year II, the birds raised their young almost three weeks later in the season compared to year I. This timing corresponded closely with the peak emergence of aquatic invertebrates, which likely explains the three fold increase in tissue residues of PCBs in year II.

The reproductive and physiological endpoints varied little with contaminant status. However, given tree swallows are relatively hardy with respect to xenobiotic exposure, our findings serve as a warning to what might be occurring in more vulnerable species.

[54] VACUOLAR MYELINOPATHY OUTBREAKS IN MULTIPLE SPECIES AT A SOUTHEASTERN RESERVOIR

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Avian vacuolar myelinopathy (AVM) is a neurological disease that was first recognized as a cause of bald eagle (Haliaeetus leucocephalus) mortality in 1994 at DeGray Lake, Arkansas. In 1996, AVM was documented in American coots (Fulica americana) during a second major eagle mortality event at the same location. Since then, AVM has been found in other wild avian species and in additional southeastern reservoirs. The cause of AVM remains undetermined; however, it is hypothesized that a natural or man-made toxicant is responsible and bald eagles are exposed to the AVM agent via ingestion of affected coots. During October-March of 1998-2003, carcasses or tissues from 54 wild birds and mammals were submitted from Clarks Hill/Strom Thurmond Lake on the Georgia/South Carolina border to the Southeastern Cooperative Wildlife Disease Study for diagnostic testing. Avian vacuolar myelinopathy was confirmed or suspected as the cause of morbidity and mortality of 27 bald eagles, six American coots, 16 Canada geese (Branta canadensis), two great-horned owls (Bubo virginianus), and one killdeer (Charadrius vociferus). A beaver with signs of central nervous system disease was necropsied; however, autolytic changes precluded confirmation of vacuolar lesions in white matter of the brain. Active surveillance during the outbreaks yielded vacuolar lesions in 19-95% of coots collected but not in the brains of 10 beavers (Castor canadensis), four raccoons (Procyon lotor), and one gray fox (Urocyon cinereoargenteus) collected for the study. The outbreaks at this location from 1998 – 2003 represent the most significant AVM-related eagle mortality since the Arkansas epornitics as well as the first confirmation of the disease in members of Strigiformes and Charadriiformes.

[55] SALMONELLOSIS IN PASSERINE BIRDS IN ONTARIO, CANADA, 1996-2002

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Salmonellosis, caused by various phage types of Salmonella enterica, subsp. enterica, serovar Typhimurium, is the cause of sporadic winter mortality events in passerine birds. In Ontario, there have been 3 diagnosed epidemics of salmonellosis in passerine birds in recent years: 1998, 2000 and 2002. The principal species involved are northern finches, primarily Common Redpolls, that unpredictably appear in more southerly locations during the winter months. In 1998, in central Ontario, phage type (PT) 40 was isolated from the majority (5/7) of locations. In 2000, several phage types, including U284, were isolated from cases in mid-northern to northwestern Ontario. In 2001, few cases were reported, but PT U284 was found in a small group of cases from Kenora in northwestern Ontario. In the 2002 outbreak, which occurred over the broadest geographic area and likely involved the greatest number of birds, PT U284 was isolated from the majority (34/38) of birds tested. In addition to these epizootic events which occurred over a broad geographic area, there have been numerous more localized events in southern Ontario in which PT 160 has been recovered from affected house sparrows and a range of other species which commonly frequent bird feeders. In all of the these events, the most characteristic lesion has been a necrotizing ingluveitis.

[56] 2001 MARYLAND MORBIDITY EVENT INVOLVING MICROCYSTIN TOXICITY AND STEATITIS IN GREAT BLUE HERONS (*ARDEA HERODIAS*)

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During fall of 2001 the Maryland Department of Natural Resources (MD DNR), US Fish & Wildlife Service (FWS), and Tri-State Bird Rescue and Research (TSBRR) received reports of sick and dead birds. Three separate incidents documented morbidity and mortality in waterfowl, gulls and colonial waterbirds. Twelve captive waterfowl of various species died over a two week period at a nature conservation facility in Queen Anne's County; on Poplar Island in Talbot County, an estimated 100 birds of many species died; and in the third event, more than 9 great blue herons (GBH; *Ardea herodias*) were reported to be dead or dying from three counties. All events showed evidence of a connection with an algal bloom of *Microcystis*. This report will summarize findings of the GBH event.

The first GBH was found in distress on October 18, 2001. It was euthanized and sent to the National Wildlife Health Center, Madison, WI, for a post mortem examination. Subsequent live herons were taken to the TSBRR facility in Newark, DE for diagnostics and rehabilitation. Birds presented with clinical signs of emaciation, lethargy, inability to fly and an unusually hard abdomen. Upon admission to the rehabilitation facility the blood profiles revealed anemia, varying degrees of hypoproteinemia, and all birds were dehydrated**,** depressed, in lateral recumbency with profuse diarrhea. At least two birds were in respiratory distress. Due to poor prognosis, euthanasia was performed and necropsies were conducted on all birds.

Necropsies were performed at either TSBRR or at the MD DNR Cooperative Oxford Laboratory. Consistent necropsy findings in all birds included emaciation, decreased muscle mass, pale muscle color, fat atrophy, gastrointestinal parasitism, and excessive deposits of waxy, yellow fat in the abdomen, subcutis, and throughout the body cavity. Steatitis was diagnosed by the NWHC and later by TSBRR and MD DNR.

In mammals, steatitis is associated with a deficiency of Vitamin E and /or Selenium. Vitamin E and selenium have antioxidant properties and are essential to cell membrane integrity. Interaction between Vitamin E / Selenium and dietary unsaturated lipids likely play a role in the pathogenesis of steatitis though the exact mechanism is unknown. In birds, this condition is suspected to be caused by a diet high in rancid or oily fish containing polyunsaturated fats. Steatitis has been reported on the east and west coasts of the United States in several other bird species (black crowned night heron *Nycticorax nycticorax*, osprey *Pandion Haliaetus*, double crested cormorant *Phalocrocorax auritus*). A similar morbidity/ mortality event of great blue herons occurred in Queen Anne's County, MD in 1995 (no definitive cause was determined in this event).

[57] SHORT-TERM TRANQUILIZATION OF SANDHILL CRANES (*GRUS CANADENSIS*) USING TRIAZOLAM

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The early growth of crane species is very rapid, and as a result birds are prone orthopedic and soft tissue injuries to their long bones and joints. Captive bred whooping cranes (*Grus americana*) often require handling for treatment or testing, and injuries in this highly endangered species can be relatively common during these restraint periods. Injuries tend to occur during capture, or as birds struggle while being held. The use of an ultra short acting benzodiazepine tranquilizer (Triazolam, trade name Apo-Triazo, Apotex, Toronto, Canada) was investigated in sandhill cranes, a similar sized crane species, to test its efficacy in decreasing capture evading behaviours and struggling during handling.

An initial dose of 0.125 mg/kg was used, placed in a smelt and administered orally. Five individual birds were treated and observed for behaviour changes over the next nine hours initially at 15-minute intervals for the first three hours and then at one-hour intervals. Behaviour changes were subtle; the most common observations included a decrease in apprehension, less evasive movements and pacing and in one very tame bird, a slight ataxia. Effects were most marked from one to one and a half hours post administration, and appeared to last from one to two hours.

A total of 11 cranes were used for three separate double-blinded trials at three dosages: 0.1 mg/kg, 0.125 mg/kg and 0.15 mg/kg. In each trial, birds were randomly assigned into a non-treated control or a treated group. The assignment of the birds was blind to handlers and data collectors. Approximately one hour after administration of the drug, each bird was handled for a weight, blood sample, determination of heart rate and respiratory rate, and assigned a numeric score to represent ease of capture and handling. Fecals were collected on the day of treatment and the following two days for future corticosterone analysis. Trials were separated by at least a week to allow for complete excretion of the drug before any subsequent dosing.

While there was no significant difference in average respiratory rates between control and treated birds, the average heart rate at the 0.125 mg/kg dose (168 bpm) was significantly lower than that of control birds (238 bpm) (T-test: $t = 2.84$, degrees of freedom +9, $p = 0.019$). It was not immediately obvious to the handlers which birds were treated and the more apprehensive the personality of the bird, the less obvious the effects. In general treated birds did not evade capture as effectively, were not able to jump as high and once held with wings and legs folded did not struggle as much as control birds. Treated birds with calm personalities sometimes assumed a characteristic curved neck, head down position when held, and exhibited mild ataxia when released. Birds reacted to external stimuli fairly normally at all doses. Anecdotally evidence of a possible drug related amnesia was seen: treated birds appeared to be less apprehensive to the presence of their handlers on the day following a trial than did the control birds.

In conclusion, the use of Triazolam did appear to mitigate some of the behaviours associated with injuries in young canes. Treated birds could still respond to their environment, yet struggled less during catch-up and handling. Birds were able to

ambulate, to eat and drink immediately after release and there were no adverse effects noted in treated birds. Effects of the drug were mild and not always predictable, particularly in birds with an apprehensive temperament. While this drug shows some promise in cranes, further work may find that a higher dose, or a different tranquilizer offers more consistent results.

[58] TESTING RECOMMENDATIONS FOR WILD TURKEYS

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Wild turkeys (*Meleagris gallopavo*) are commonly transplanted into new areas; when this occurs it is necessary to ensure that they are disease-free. Unfortunately, ante mortem tests for diseases in wild turkeys present unique problems. Available tests were developed for use in domestic poultry and have not been validated for use in wild birds. False positive reactions have caused unnecessary delays and/or loss of transplant opportunities on many occasions.

Ideally, populations which may serve as source flocks should have 5-10 birds trapped and screened for diseases prior to the initiation of trapping for transplant (pre-transplant screening). If pre-transplant screening provides evidence that nonpathogenic species have infected the flock, it may provide reassurance to state veterinarians if, during pre-release testing, some turkeys test serologically positive.

Prior to release, testing will be required of every bird to be moved across state lines (pre-release testing). The agency conducting the transplant should consult the appropriate state of destination animal health personnel (state veterinarian) as well as the state wildlife health professional. They should review results of pre-transplant screening and should agree *a priori* what testing will occur, how the results will be interpreted, and if further diagnostics will be pursued should suspicious results arise. There is very little data about most diseases of concern to domestic poultry in wild turkeys, so making strong recommendations is difficult. Below is a summary of the diseases and tests we believe should be conducted:

Salmonella pullorum (Pullorum Disease) and *Salmonella gallinarum* (Fowl Typhoid). The Rapid Plate Agglutination (RPA) test is the most common test used, however, this test is not very specific and false positives may occur. The Salmonella Tube test (Standard Tube Agglutination test) may be more specific and dilutions of 1:25 and above should be used when conducting the test. The microagglutination test may be the most specific test and a final dilution of 1:40 should be considered positive; false positives can occur regardless of which test is used.

If some turkeys react on either serologic test during a pre-transplant screening, they should be necropsied and cultured. If some birds react during pre-release testing, and pre-transplant screening reveals that these are probably false positives, a case could be made for allowing release. If pre-transplant screening was not conducted, no serologic reactors should be released, and the state veterinarian may require culture of reactors before nonreactors from the area are released.

Mycoplasma gallisepticum (MG)

The Rapid Plate Agglutination test is often used to screen domestic flocks. However, this test can have specificity problems in wild turkeys. Hemagglutination inhibition (HI) tests are more specific, but more costly and time consuming. Additionally, HI tests are not as sensitive. Titers on HI below 1:40 should be considered

negative and those 1:80 and above considered positive. Titers of 1:40 should be considered suspects. Birds with suspect or reactive titers should not be transplanted unless they are cultured and proven negative. Other birds in the flock may be used if pre-transplant screening indicates the reactions are probably nonspecific, or if culture of reacting birds indicates nonspecific reactions. Enzyme-linked immunosorbent assays (ELISAs) are commonly used on commercial turkeys and have moderate to high sensitivity and specificity.

False positive reactions are possible with both serologic tests; therefore culture is the best technique to positively identify a wild turkey infected with MG. Birds testing seropositive should be necropsied, respiratory tract cultured. Alternatively, live birds may be sampled by culturing tracheal, choanal cleft, and cloacal swabs. If any birds are culture positive for MG, no birds from that flock should be transplanted. *Mycoplasma* spp.-specific PCR (Polymerase Chain Reaction) may be a better choice than culture as there is little problem with contamination, samples can be pooled to reduce costs, and it can be completed in 24 hours.

Mycoplasma meleagridis (MM)

Serologic testing and interpretation is identical to MG. False positives are much more common for MM than other *Mycoplasma* diseases; even PCR has given false positive results for MM. Infection with the nonpathogenic species *M. gallopavonis* may account for false positive tests; only wild turkeys from which MM has been cultured should be considered to be actually infected.

As with MG, all birds with suspicious MM titers should be necropsied and cultured, especially if pre-transplant screening was not conducted. Tissues to be cultured should include respiratory and reproductive tracts. Live birds may be sampled by culturing swabs of the trachea, choanal cleft, and vagina or phallus.

Mycoplasma synoviae (MS)

Serology is identical to MG and MM. False positives are less common with MS especially if the HI test is used. Turkeys with positive or suspicious serologic reactions will need to be necropsied and cultured to determine their actual status. Several foot and leg joints should be cultured even if they appear normal. Respiratory tract should be cultured as well. Tracheal and choanal cleft swabs may grow organisms. PCR may be more reliable for MS than for MM.

Newcastle Disease

Some serotypes of Newcastle disease virus (NDV) can cause serious outbreaks in domestic chicken flocks. Serologic surveys of wild turkeys have shown variable antibody levels; and there have been no reports of clinical disease or isolation of NDV from wild turkeys. Wild turkey populations may lack the frequent bird-to-bird contact found in domestic flocks required to transmit and maintain NDV. Routine testing for NDV is probably not warranted, but testing should be considered for turkeys from areas that have experienced NDV outbreaks in other species. The HI test has been used for NDV in wild turkeys, but specificity is unknown. Definitive diagnosis requires virus isolation from the respiratory or intestinal tracts.

Avian Influenza

Avian influenza (AI) virus can cause significant problems for the domestic poultry industry. However, the disease has not been documented in wild turkeys. Because AI does not appear to be a problem in wild turkeys, routine mandatory pre-release testing is probably not justified. But, testing should be considered for turkeys coming from areas that have experienced the disease in other species. A variety of serologic tests are available; specificity may be a problem in domestic species; thus definitive diagnosis in wild turkeys requires virus isolation from the respiratory or intestinal tract.

[59] THE CANADA DATABASE OF ANIMAL PARASITES (CDAP): MONITORING PARASITES IN WILDLIFE IN CANADA

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The Canada Database of Animal Parasites (CDAP) was established in 1998 at the Canadian Food Inspection Agency=s Centre for Animal Parasitology in Saskatoon. The database contains information on parasite occurrence and distribution in domestic animals and wildlife in Canada, assembled from published literature and provincial and private veterinary diagnostic laboratories across the country. CDAP is considered a passive surveillance tool. At present there are approximately 3000 records for 20 parasite genera within CDAP. The genera were selected because of their importance in animal or human health, food safety or trade. CDAP is a unique and convenient source of national parasite information.

At present, 15 of the 20 parasite genera included within CDAP contain records of occurrence in wildlife hosts: *Besnoitia*, *Cryptosporidium*, *Echinococcus*, *Elaeophora*, *Elaphostrongylus*, *Fasciola*, *Fascioloides*, *Giardia*, *Orthostrongylus*, *Parelaphostrongylus*, *Protostrongylus*, *Sarcocystis*, *Toxoplasma*, *Trichinella*, and *Umingmakstrongylus*. Of these genera, seven are potentially zoonotic. Currently, CDAP contains records of parasites in more than 100 wildlife species, including free-ranging animals and birds, ranched game animals, and animals in zoos.

The application of CDAP to monitoring and exploring parasitic infections in wildlife hosts in Canada will be discussed, using *Toxoplasma gondii* as an example.

[60] THE RISE OF URBAN FOXES AND THE ZOONOTIC PARASITE *ECHINOCOCCUS MULTILOCULARIS* – ECOLOGICAL AND EPIDEMIOLOGICAL ASPECTS OF AN URBAN PARASITE LIFE CYCLE

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In several European countries a distinct increase of red fox (*Vulpes vulpes*) populations has been observed. Due to fox invasion into urban areas, the small fox tapeworm *Echinococcus multilocularis* has gained this new habitat. As a possible consequence this development may have caused an increased infection risk for human alveolar echinococcosis – a severe human liver disease. It has been shown that due to a high anthropogenic food supply in the city of Zurich, Switzerland, urban foxes can achieve high population densities (9.8 – 11.2 adult foxes per km^2) and correspondingly can fulfil their nutritional requirements within small home ranges (females: mean 28.8ha, males: mean 30.8 ha). The resulting low spatial dynamics of urban fox populations is likely to be the reason for the occurrence of pronounced *E. multilocularis* prevalence changes within small distances. In the borderland between rural and urban habitat high fox population densities overlap with suitable habitats for voles. It has been shown that foxes prey significantly more on rodents in these areas compared to central urban areas. Furthermore, rodent prey composition differed significantly between the two zones: in the urban periphery fox stomachs contained relatively more voles but less murids, which are less susceptible to *E. multilocularis* infections than voles. Correspondingly, we found highest prevalences in the urban periphery where 67% of the foxes (final host) and 9.1% of the trapped voles (*Arvicola terrestris*, intermediate host) were infected. The urban periphery is intensively used by a broad public for recreational and other activities, and densities of domestic dogs, which can acquire the parasite by preying on infected voles, are high. Therefore, possible intervention strategies to lower the infection pressure should focus primarily on this urban periphery.

[61] UPDATE ON HEALTH, RECOVERY AND MANAGEMENT ISSUES FOR THE SOUTHERN SEA OTTER (*ENHYDRA LUTRIS NEREIS)*

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After several decades of steady slow growth the "threatened" southern sea otter populations along the California coast have failed to grow or have declined every year from 1995 through 2002. Postmortem examinations have revealed that from 40-45% of sea otters die of infectious diseases and/or parasites in most years, and adult mortality (rather than geriatric or neonatal mortality or reduced fecundity) appear to explain population performance. Protozoal encephalitis (due to *Toxoplasma gondii* or *Sarcocystis neurona)* and Acanthocephalan (*Profilicolis* spp) infestations, often manifested as peritonitis, are the two leading syndromes causing mortality, but bacteremias, systemic fungal infections, noninfectious diseases and various forms of trauma kill significant numbers of animals. In the late winter/spring of 2003 a mortality event resulted in the recovery of over 100 dead southern sea otters in a 4 month period (more than double the 10 year average). Findings from this event, which was recently declared an "unusual mortality event" (UME), will be discussed. It has also been hypothesized that exposure to contaminants or genetic.bottlenecking due to severe population reductions 200 years ago may make southern sea otters more susceptible to the pernicious effects of diseases. Efforts underway to examine both of these possibilities and to "connect the dots" between hypothetically reduced immune function and increased disease mortality, if such a connection exists, will be presented. With the recent publication of a Federal "Southern Sea Otter Recovery Plan" there will be increased emphasis on mitigation of negative impacts, but we are early in the process of considering management options for reducing various forms of pollution that may be harmful to sea otters and human health.

[62] NECROTIZING ENCEPHALITIS IN SWEDISH ARCTIC FOXES (*ALOPEX LAGOPUS*)

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The arctic fox (*Alopex lagopus)* is an endangered species in Scandinavia.Some of the individuals left in the wild were caught and kept at a breeding center for endangered species. Wild captured arctic foxes and their offspring developed encephalitis, with new cases occurring over a course of 9 years. Cases in adults appeared sporadically since 1994. Whole litters of puppies showed similar disease signs. Affected foxes displayed successively: apparent loss of smell and sight, changes in behaviour, fearfulness, unstable gait, loss of balance, walking in circles, biting objects, depression, apathy and somnolence. Clinical chemistry conducted in some of the foxes showed leukopenia, lymphopenia, and increased protein, glucose and leucocytes in the cerebrospinal fluid. Most of the affected foxes died or had to be euthanized up to one month after the onset of signs.

Pathology showed severe nonsuppurative meningoencephalitis with areas of necrosis. Inflammatory changes included multiple foci of gliosis and infiltration of macrophages and lymphocytes in the cerebral grey matter. In addition, there were prominent perivascular cuffs composed of lymphocytes, plasma cells and macrophages. A similar type of inflammatory cell infiltrate was present in the leptomeninges. Rarified areas contained debris mixed with macrophages, gitter cells, plasma cells, lymphocytes, numerous gemistocytes and binucleated or multinucleated reactive astrocytes. The histopathological changes were most pronounced in the olfactory bulbs and frontal lobes of the cerebrum, but involved also other areas of the cerebrum and brainstem and to a lesser extent the spinal cord. Degenerative changes and intracytoplasmic inclusions in the choroid plexus epithelium were observed in one of the foxes.

The cause of the disease has so far not been determined. Some of the known causes of encephalitis in foxes, such as *Toxoplasma gondii, Neospora caninum, Listeria monocytogenes*, canine distemper virus, rabies virus, adenovirus (fox encephalitis) and Borna disease virus have been excluded as etiological agents due to negative test results. Further investigations, including virus culture and various PCR tests, are presently in progress.

[63] INVESTIGATION OF A SEASONAL SEPTIC ARTHRITIS IN STRIPED SKUNKS (*MEPHITIS MEPHITIS*) ON CAPE COD

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A clinical syndrome characterized by septic arthritis in Striped skunks (*Mephitis mephitis)* has been observed annually in individuals presented to the Cape Wildlife Center during the months of April and May. Preliminary investigations to examine possible etiologies including aerobic bacterial infection, amyloidosis, and *Ehrlichia* or *Borrelia burgdorferi* infections have been previously carried out with negative results. Supported in part by a small grant received from the National Wildlife Rehabilitators Association, ongoing efforts to characterize and identify the etiology of this disease are described. Primary etiologies investigated include infection with Canine Distemper Virus or a *Mycoplasma* species.

[64] SUSCEPTIBILITY OF SALAMANDERS AND FROGS TO CHYTRID INFECTIONS

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The chytrid fungus, *Batrachochytrium dendrobatidis,* has been found in amphibians worldwide and is implicated in the declines of some species. While this fungus is capable of infecting a wide range of amphibian species, the results of infection are variable and are currently unpredictable. The likelihood of acquiring an infection, the severity of infection, and even the symptoms may differ among and within host species.

To understand the ecology of chytridiomycosis, we are studying this disease in Arizona amphibians. Here we report the results of experimental exposures in *Pseudacris triseriata* (Western Chorus Frog), *Rana yavapaiensis* (Lowland Leopard Frog), and *Ambystoma tigrinum nebulosum* (Arizona Tiger Salamander). *P. triseriata* is easily infected by chytrids in the lab, and shows high mortality at 10C, which corresponds to a field observation of an early spring massive mortality event. *A. tigrinum* is also easily infected with chytrids and can carry heavy infections that may persist for over a year, yet chytrid induced mortality is minimal. This finding is consistent with the absence of reported chytrid-related *A. tigrinum* die-offs. In contrast, a number of chytrid-associated mortalities have been reported for *R. yavapaiensis;* however, in the laboratory, *R. yavapaiensis* is difficult to infect, and infections tend to be light, clearing quickly with minimal mortality. Our data show that the outcome of infection in the laboratory may not reflect patterns of infection in the field, suggesting that the context of infection may play an important role in determining the outcome.

[65] STRESS-INDUCED IMMUNOSUPPRESSION IN RED-SPOTTED NEWTS: EFFECTS OF PHYSIOLOGICAL AND CHEMICAL STRESSORS

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Amphibian populations have suffered widespread declines and extinctions in recent decades. Although increased disease prevalence has been implicated at particular localities, the importance of human induced stress as a trigger for these outbreaks remains unclear. Given the potential role that disease may play, it is important to understand how different types of stress affect amphibians' ability to resist infections. To test how stressors could potentially affect the immune system of adult amphibians in a natural environment, we placed newts in enclosures within three ponds and gave them injections over the course of several weeks of either (1) corticosterone, the main glucocorticosteroid in amphibians that is important in the physiological response to stressful stimuli, or (2) Malathion, a common agricultural insecticide. We then measured lysozyme levels in the stomach and liver, white blood cell counts, spleen mass, serum complement activity, skin antimicrobial peptide activity, and the delayed-type hypersensitivity reaction to assess the state of the animals' innate and adaptive immune systems. These results could have implications for the types of pathogens and parasites that could evade host immune systems weakened by different types of stress.

[66] THE ECOLOGY OF A TIGER SALAMANDER RANAVIRUS

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Ambystoma tigrinum virus (ATV) causes recurrent epidemics in tiger salamander populations throughout North America. Larvae and metamorphs are highly susceptible to ATV in the laboratory and field, but virus has not been isolated from eggs. ATV is transmitted via direct contact (as early as four days post-exposure), feeding on infected tissues (e.g., cannibalism or scavenging), and in water with high viral titers.

ATV degrades quickly in pond water and dry mud, precluding long-term persistence in the environment, and alternate syntopic hosts are unknown. Other salamanders are susceptible to ATV, but it does not cause disease or enduring infections in experimentally challenged frogs (*Rana catesbeiana, R. pipiens, R. sylvatica, Pseudacris triseriata*) and fish (*Gambusia affinis, Lepomis cyanellus, Oncorhynchus mykiss*). However, ATV was re-isolated from Northern pike (*Esox lucius*) and walleye (*Stizostedion vitreum*) >20days after injections. The importance of infected fish in salamander disease dynamics is unclear because fish often extirpate amphibians through predation. Further, pike and walleye are often introduced into unsuitable habitats and quickly die. However, they may be important for long distance transport of ATV as they are both widely stocked game fish. Similarly, salamander larvae used as bait have probably dispersed ATV widely.
ATV tends to be lethal to salamanders within 2-3 weeks, although some individuals lose overt symptoms of infection (e.g., papules, lesions, edema) and survive indefinitely. In one experiment ATV was re-isolated from 40% of these survivors, suggesting that these are chronic rather than cleared infections. Chronic infections also occur in natural populations and appear to be the means by which ATV persists between epidemics.

[67] *ICHTHYOPHONUS*-LIKE INFECTION IN A MARBLED SALAMANDER (*AMBYSTOMA OPACUM*) AND A SHORT REVIEW OF MICROORGANISMS IN THE CLASS MESOMYCETOZOEA

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A wild caught marbled salamander died after 110 days in quarantine at a zoo. There had been no clnical signs and there were no gross post mortem findings. Histologically, the only significant finding was numerous encapsulated organisms, sometimes surrounded by a thin rim of granulomatous inflammation, in the interstitium of skeletal muscles. Groups of myofibers around these organisms were atrophied and sometimes degenerate. The organism was most similar to *Ichthyophonus*. *Ichythyophonus* and other related organisms have been mostly described in fish, however, there are a few reports in newts and other amphibians. Recent work places this type of organism in the Class Mesomycetozoea, a heterogeneous group of microorganisms at the animal-fungal boundary. These organisms are most often described in skeletal muscle, but they have been described in heart and viscera. They tend to elicit a granulomatous and eosinophilic response. Occasionally, they have been considered to be a significant factor in the death of an animal.

[68] FOOT-AND-MOUTH DISEASE AND DEER: REFLECTIONS ON THE 2001EPIDEMIC IN THE UK AND IMPLICATIONS FOR NORTH AMERICA

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No cases of foot-and-mouth disease (FMD) were confirmed in deer during the epidemic in the UK in 2001. However, early in the epidemic, there was serious concern that free-living and farmed deer might be playing a role in the transmission of FMD based upon a) the observation of clinical disease similar to FMD in farmed sika deer and free-living roe in the north west of England and neighboring parts of Scotland and b) the knowledge (from experimental studies in the 1970's) that the various species of deer in the UK are all susceptible to FMD. There is no compelling evidence to incriminate free-living deer in the transmission of FMD virus between farms in the 2001 epidemic in the UK despite their presence in the area of infected farms. However, complacency regarding the potential role of deer in any future FMD epidemic is unwise. Were outbreaks of FMD to occur in the USA (or in other countries with significant deer populations, such as New Zealand) deer might become involved. The clinical appearance and pathogenesis of FMD in the herding species of deer found in the UK (red, fallow, and sika) is similar to sheep. Experimental FMD in white tailed deer is also similar. Granted, nowhere in the world are deer marketed as extensively as live sheep are in the UK, but there is a lesson to be learned from the UK epidemic. In the UK, before February 2001, few, if any, would have predicted such an extensive epidemic involving sheep as potent "silent spreaders" of FMD virus in a country with sophisticated veterinary services. This paper illustrates the clinical appearance of FMD in deer experimentally exposed to infected livestock and points to the concerns of countries with large free-living species of deer were FMD to be confirmed in domestic livestock.

[69] WEST NILE VIRUS INFECTION IN REINDEER (*RANGIFER TARANDUS*)

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West Nile virus (WNV) is a member of the *Flaviviridae* family (genus *Flavivirus*), transmitted among bird populations by mosquitoes and incidentally infects mammals. First recognized in the United States in the New York City area in 1999, WNV has subsequently spread across the United States. Numerous cases of WNV-induced non-suppurative encephalomyelitis have been documented in horses since 1999. Here we describe non-suppurative encephalomyelitis in 4 reindeer (*Rangifer tarandus*) resulting from WNV infection. Clinical signs and lesions were similar to those described in horses with non-suppurative inflammation most common in the medulla oblongata and cervical spinal cord. Immunohistochemistry revealed WNV antigen within neurons and among mononuclear cell infiltrates. Nucleotide sequence of a 768 basepair region of the WNV E-glycoprotein gene revealed one nucleotide mutation, which resulted in an amino acid substitution from a serine to a glycine (position 227 of E-glycoprotein) when compared with the prototype WNV-NY99 strain (isolated from Bronx zoo flamingo 382-99).

[70] BRUCELLOSIS IN CAPTIVE ROCKY MOUNTAIN BIGHORN SHEEP (*OVIS CANADENSIS*) CAUSED BY *BRUCELLA ABORTUS* BIOVAR 4

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Nine (4 female, 5 male) captive, adult Rocky Mountain bighorn sheep (*Ovis canadensis*) contracted brucellosis caused by *Brucella abortus* biovar 4 as a result of natural exposure to an aborted elk (*Cervus elaphus*) fetus. Clinical signs of infection were orchitis, abortion, and perhaps death. Gross pathologic findings included enlargement of the testis and/or epididymis and yellow caseous abscesses and granulomas of the same. *Brucella abortus* biovar 4 was cultured in all sheep from a variety of tissues, including testes, mammary gland, and lymph nodes. All sheep tested were positive on a variety of standard *Brucella* serologic tests. This is the first report of brucellosis caused by *B. abortus* in Rocky Mountain bighorn sheep. It also provides evidence that sheep develop many of the manifestations ascribed to this disease and that infection can occur from natural exposure to an aborted fetus from another species. Wildlife managers responsible for sheep populations sympatric with *Brucella*-infected elk or bison (*Bison bison*) should be cognizant of the possibility of this disease in sheep.

[71] BRUCELLOSIS IN UGANDAN WILDLIFE: A PILOT STUDY

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In several Ugandan national parks there is an overlap of range used by traditional domestic stock and wildlife. Brucellosis is a recognised problem in both humans and livestock in Uganda. Results of several different serum antibody tests show prevalence between 9.6 and 38.5 percent of cattle and close to 100% of the goats in various regions of the country. Serological evidence of infection has been found in impala (*Aepyceros melampus*) and recently a single giraffe (*Giraffa camelopardalis*). Clinical (hygroma) and serological evidence of infection has been found in buffalo (*Syncerus caffer*). Up to 15% of people in some regions of the country have been found to have Brucella antibodies, with those processing carcasses and assisting at calving being at greatest risk.

Several of the lekking grounds of Uganda kob (*Kobus kob thomasi*) in the Queen Elizabeth National Protected Area (QEPA) several are located between the villages of Kasenyi and Hamukungu, which are themselves two of eleven communities within the park boundary. Kasenyi is primarily a fishing village but approximately one hundred goats live in close proximity with the residents. The people of Hamukungu village are of two ethnic groups, one fishermen, the other nomadic cattle herders. Approximately 1200 hundred of their cattle use grazing in the park (illegally) that overlaps with kob lekking grounds.

As the first part of a multi-year study we collected blood samples from goats at Kasenyi and from kob in the park. As part of other projects we also collected blood from two waterbuck and a warthog. All the blood samples were allowed to clot at ambient temperature for 30 minutes and then stored at 4^0C until subjected to laboratory processing with the Brucella card test, using known negative and positive bovine sera as standards. None of the samples collected from 10 goat or 12 kob reacted positively to the Brucella card test. Neither of the waterbuck samples, nor the warthog sample reacted positively to the Brucella card test.

[72] RFLP POLYMORPHISM IN *BRUCELLA* SP. BASED ON 3 NEWLY IDENTIFIED INSERTION SEQUENCES IN *BRUCELLA MELITENSIS* 16M

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A number of recent reports have described the isolation and characterization of *Brucella* strains from a wide variety of marine mammals such as seals, porpoises, dolphins, and a minke whale. These strains were identified as brucellae by conventional typing tests. However, their overall characteristics were not assimilable to those of any of the six currently recognized *Brucella* species and it was suggested that they comprise a new nomen species to be called *Brucella maris*. In the present study we analyzed DNA polymorphism based on newly identified Insertion Sequences (IS)-RFLP. Three IS in the genome of *Brucella melitensis* 16M were identified, when sequencing the cluster *rfb* coding for genes implicated in LPS biosynthesis. The IS*Bm1* is homologous of IS*869* of *Agrobacterium tumefaciens*, the IS*Bm2* is homologue of IS*511* of *Caulobacter crescentus* and the IS*Bm3* is homologous of IS*481* of *Bordetella pertussis*. The aim of this work was to test these IS as probes for *Eco*RI-RFLP in order to study *Brucella* polymorphism. We used *Brucella* reference strains and also strains isolated from different marine mammal species. The results of those RFLP were compared with IS*711*-based RFLP. The main results are: 1) RFLP based on IS*Bm1* differentiated the classical terrestrial mammal strains from the marine mammal strains. 2) RFLP based on IS*Bm1* differentiated *Brucella* strains isolated from cetacean and strains isolated from pinnipeds. 3) We didn't observe differences between all strains with IS*Bm2*-based RFLP but for B. canis. With IS*Bm3*-based RFLP, we differentiated *B. ovis* from all the other *Brucella* strains and *Brucella* strains isolated from pinnipeds showed a unique pattern. These results suggest that *Brucella* strains isolated from cetacean are different from *Brucella* strains isolated from pinnipeds and further support the classification of marine mammal brucellae in two species i.e., *Brucella cetacea* (strains isolated from cetacean) and *Brucella pinnipediae* (strains isolated from pinnipeds).

[73] PROTECTION AGAINST CHALLENGE-INDUCED ABORTION AND INFECTION BY *BRUCELLA ABORTUS* STRAIN 19 BY SINGLE CALFHOOD VACCINATION OF ELK (*CERVUS ELAPHUS*)

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Brucellosis in Greater Yellowstone Area (GYA) bison and elk has been a source of controversy and focus of the Greater Yellowstone Interagency Brucellosis Committee (GYIBC) for years. Brucellosis has been eradicated from cattle in the 3 states of Wyoming, Montana, and Idaho and all three states currently are classified as "brucellosis free" with regard to livestock. Yet free-ranging elk that attend feedgrounds in the GYA, and bison in Yellowstone and Grand Teton National Parks, still have high seroprevalence to the disease and are viewed as a threat to the state-federal cooperative national brucellosis eradication program. The GYIBC, formed of state and federal agencies involved in wildlife and livestock management in the 3 states, has committed to eventual elimination of the disease from wildlife. Management tools to control or eliminate the disease are limited; however, wildlife vaccination is one of the methods currently employed. We commenced a single-dose, calfhood vaccination, efficacy study of vaccine *Brucella abortus* strain 19 (S19) in elk in 1999.

In 2002 we presented preliminary findings of this study. Detailed information on methodology and serological response of elk to S19 vaccination can be found in WDA Proceedings 2002. The study was a controlled, challenge design, which included 44 control and 45 vaccinated pregnant elk (47 3.5 year old and 42 2.5 year old at breeding). We captured the elk as calves from areas without evidence of brucellosis. Serologic testing throughout the project did not indicate any prior exposure to the disease. Control elk remained seronegative until challenge and all vaccinated elk were negative before vaccination. We vaccinated elk intramuscularly with S19 or saline (2ml) for vaccinates and controls, respectively, in March of their capture year. Vaccine was procured from, and titrated by, Colorado Serum Company and consisted of 4.42×10^9 colony-forming-units (CFU) and 8.58×10^9 CFU per dose for 1999 and 2000 vaccinations, respectively. 100% of vaccinates responded serologically to vaccination. After breeding, pregnancy was determined by pregnancy specific protein B (PSPB) and ultrasound in January 2002. We challenged elk with 1×10^7 CFU of pathogenic *Brucella abortus* strain 2308 by bilateral intraconjunctival sac instillation on February 28, 2002. Following challenge, elk were placed in 3 pens of approximately 30 elk each containing equal numbers of control, vaccinate, 2 and 3 year olds.

Abortions occurred during March (2), April (22), May (29) and June (21), 2002. All live births occurred in May (11) and June (4). There was no difference in abortion rates among the 3 pens housing challenged elk, nor between the two age classes. With no pen or age effect, results for all vaccinates and all controls were combined for analysis. Abortion occurred in 42 controls (2 viable births) and 32 vaccinates (13 viable births). Vaccination significantly reduced abortion (Fisher exact, $p=0.002$). Using the control

group to estimate expected viable births in the vaccinate group without any vaccine effect, 26% of vaccinates expected to abort were protected by vaccine.

We cultured cows and fetuses/calves for *Brucella abortus*. If challenge strain *Brucella abortus* was found in either cow or fetus/calf, that cow/calf pair was considered infected. We found 43 of 44 controls (98%) and 42 of 45 (93%) vaccinates infected, a non-significant difference. Vaccine did not protect against infection. However, only 6 of 15 (40%) of viable births were infected whereas 68 of 74 (92%) of abortions were infected, though maternal infection occurred in many of the non-infected viable births and abortions.

The vaccine provided some protection, but the effect was minimal (26% protection against abortion). Peterson et al (1991), modeling brucellosis in Jackson bison, indicated that low vaccination efficacy had little impact on brucellosis prevalence, with a 24% efficacy stabilizing prevalence at 47% (starting at 61%) after 20 years of vaccinating. Gross et al (1998) modeled brucellosis prevalence change under a variety of conditions in elk. Over the 100-year time frame they modeled, this low efficacy did not eliminate brucellosis regardless of whether calves or both cows and calves were vaccinated. The model, however, predicted a reduction in disease prevalence by 40-50%. Combined with other treatments, such as test and slaughter, eradication might be achieved in 20-30 years. Others have argued that vaccination will simply not eliminate the disease and other controls will be necessary to significantly reduce prevalence (Enright and Nicoletti 1997). Enright, F. and P. Nicoletti. 1997. Vaccination against brucellosis. Pp 86-95, In Thorne, et al (eds), Brucellosis, bison, elk, and cattle in the greater Yellowstone area: Defining the problem, exploring solutions. National Symposium on Brucellosis, Pioneer Printing, Cheyenne, WY, 219pp. Gross, J. E, M. W. Miller and T.J. Kreeger. 1998. Simulating dynamics of brucellosis in elk and bison. Final report to USGS-Biological Resources Division, Laramie, Wyoming. 30 pages Peterson, M., W.E. Grant and D.S. Davis. 1991. Bison-brucellosis management: simulation of alternative strategies. *Journal of Wildlife Management.* 55(2):205-214.

[74] EPIDEMIOLOGY OF BOVINE TUBERCULOSIS IN HARVESTED ELK (*CERVUS ELAPHUS MANITOBENSIS*) NEAR RIDING MOUNTAIN NATIONAL PARK, MANITOBA, CANADA

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Riding Mountain National Park in Manitoba, Canada is a protected area of habitat that is home to many species of flora and fauna indigenous to a parkland and boreal forest ecology. Tuberculosis *(Mycobacterium bovis)* has been found in elk *(Cervus elaphus manitobensis)* that roam freely in the Park and surrounding agricultural areas, and the disease has spilled over into a number of nearby cattle herds. Federal and provincial wildlife and agricultural agencies have cooperated in a Wildlife Health Monitoring Project whereby animals that are shot by hunters or found dead are examined for gross evidence of bovine tuberculosis, and suspicious lesions are submitted for laboratory confirmation.

Of the 3273 animals listed in the database to the end of 2002, 10 of 1463 elk, 1 of 1079 white-tailed deer (*Odocoileus virginianus*), and none of 557 moose (*Alces alces*) were found to be culture positive for *M. bovis*. A descriptive epidemiological analysis of the elk was undertaken using Epi Info, a database and statistics program for public health professions (Centers for Disease Control, Atlanta, Georgia).

The age, sex, and geographic location of all elk harvested were examined to determine if bias existed in the hunter-shot sample. Four age groupings were assigned: Age Group 1 animals (42%) were < 3 yr of age; Age Group 2 animals (37%) were 3 yr to < 5 yr of age; Age Group 3 animals (16%) were 5 yr to < 8 yr of age; and Age Group 4 animals (4%) were 8 yr and older. There were no statistically significant differences in the age distributions of males and females harvested. The age distributions of harvested animals from one geographic area (Rural Municipality) to another were also within $\pm$ 10% of the Age Group means. However, there was a statistically significant preference for hunting males as compared to females. In the hunted population 62% were females, compared to 79% females in the live population as determined by classified aerial counts.

Although the overall prevalence of TB in harvested elk was low (< 1%) there were marked differences in age and sex distribution of positive animals. Males were 2.4 times as likely to be infected as females ($p= 0.14$), but this was primarily due to the dramatic disparity in risk that occurred in Age Group 3. Males in this group were 7.3 times more likely to be infected than females of the same age ($p=0.059$). This pattern was even more exaggerated near the west end of the Park where the disease seemed to cluster. None of 28 females in Age Group 3 were positive compared to 3 of 14 (21%) Age Group 3 males ($p=0.03$). These data indicate that TB is not evenly distributed in the elk population of Riding Mountain National Park. The disease clusters in the west end of the park and is most prevalent in mature, breeding-age males.

[75] BOVINE TUBERCULOSIS IN THE RIDING MOUNTAIN NATIONAL PARK REGION

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Bovine Tuberculosis (Mycobacterium bovis) was identified in the Riding Mountain National Park (RMNP) Region in domestic cattle in 1991 and in wild elk (Cervus elaphus) in 1992. Since then 10 additional cattle herds have been confirmed with Bovine Tuberculosis in the RMNP Region. This has resulted in over 1800 cattle being destroyed and over 45,000 cattle being tested in the area. In 1997, RMNP initiated a wildlife health surveillance program and since that time have examined 1286 elk heads from hunter-killed specimens of which nine have been confirmed positive with Bovine Tuberculosis. There have also been 486 white-tailed deer (Odocoileus virginianus) examined again from hunter-killed specimens, one of which was confirmed positive for Bovine Tuberculosis in 2001.

The presence of Bovine Tuberculosis in the elk and white-tailed deer populations in and around RMNP represent a significant threat to the region's cattle industry. Within RMNP the challenge involves weighing the impacts on the ecosystem due to the presence of a non-native disease with the potential impacts on the ecosystem from active management initiatives that may be required in an attempt to remove the disease.

[76] EXPERIMENTAL INFECTION OF REINDEER (*RANGIFER TARANDUS*) WITH *MYCOBACTERIUM BOVIS*: DIAGNOSTIC IMPLICATIONS

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Reindeer (*Rangifer tarandus*) are routinely tested for tuberculosis by the comparative cervical test (CCT, a measure of delayed type hypersensitivity) as outlined in the USDA Uniform Methods and Rules for the eradication of bovine tuberculosis. The CCT, however, has an apparent lack of specificity for tuberculosis surveillance in reindeer. Establishment of criteria for disposition of the CCT and other tests is hindered by a lack of confirmed *Mycobacterium bovis*-infected reindeer. Our objective was to evaluate and compare the CCT and an in vitro blood-based assay using experimentally infected reindeer of ~ 1 year of age. Treatment groups included: *M. bovis*-infected (n = 11, strain 95-1315 intratonsillarly), *M. bovis* BCG-vaccinated (n = 7, Pasteur strain subcutaneously), and naive reindeer (n = 12). CCT was performed 90 days after challenge or booster vaccination. Using USDA criteria, 11/11 infected reindeer were reactors; 4/7, 2/7, and 1/7 vaccinated reindeer and 1/12, 7/12, and 4/12 naïve reindeer were reactors, suspects, or considered negative, respectively. As early as 29 d postchallenge, mean mycobacteria-specific IFN-γ responses by mononuclear cells from *M. bovis*-infected reindeer exceeded ($P < 0.05$) those of both BCG-vaccinated and naïve reindeer. As with the CCT, positive IFN-γ responses [(i.e., no stimulation minus antigen stimulation > 0.1 optical density (Δ OD)] to crude mycobacterial antigens [i.e., *M. bovis* purified protein derivative (PPDb)] were also detected for a few naïve reindeer. ESAT-6 and CFP-10 are proteins expressed by tuberculosis complex *Mycobacteria spp.*, but not by *M. bovis* BCG and most other non-tuberculous *Mycobacteria spp*. With a cutoff value of 0.1 Δ OD, 11/11 infected reindeer were determined positive using PPDb, recombinant CFP-10, and recombinant ESAT-6:CFP-10 whereas 3/4, 1/4, and 1/4 naïve reindeer were determined positive using PPDb, CFP-10 or recombinant ESAT-6:CFP-10 antigens, respectively. As expected, stimulation of blood samples from BCG-vaccinated reindeer with either CFP-10 or recombinant ESAT-6:CFP-10 antigens did not induce IFN-γ production that exceeded the response to media alone. Concerning diagnosis of tuberculous reindeer, present findings indicate: (1) the current criteria for interpretation of the CCT results in numerous false positives, (2) a blood-based IFN-γ assay may prove useful for tuberculosis diagnosis, and (3) use of recombinant CFP-10 or ESAT-6:CFP-10 antigens enhances the specificity of the IFN-γ assay.

[77] MORTALITIES DUE TO MYCOBACTERIAL INFECTIONS IN WILD RED DEER (*CERVUS ELAPHUS*) IN BELGIUM

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Paratuberculosis caused by *Mycobacterium avium* subsp. *paratuberculosis* (Map) has recently been described in farmed red deer in Belgium. The infected animals were found in apparently healthy deer. Discrete gross pathology was limited to the gut associated lymphnodes. Histopathological changes compatible with mycobacterial infections were seen in the lymphnodes as well as in the ileal tissues. Ziehl-Neelsen staining gave consistently negative results in Map culture positive samples. All isolates were *IS900* positive.

Mortalities occured since then in the wild deer population, both in adult and young animals. The animals were severely emaciated, showing bloody diorrhea. Erosion and ulcers of the gastric and enteric mucosa were seen. All the animal were tested BVD negative. Ziehl-Neelsen staining on faeces and on mucosal smears gave consistently positive results, i.e. high numbers of acid-fast bacilli. Cultures on HEYM media yielded mycobacterial growth either in the absence and/or in the presence of Mycobactin. A duplex PCR for Differential Identification of *Mycobacterium bovis*, *M. avium* subsp. *avium* and *M. avium* subsp. *paratuberculosis* was applied on those bacterial cultures and gave always negative results for *M. bovis*. In line with the culture results, different types o6 duplex PCR results were obtained for *M. avium* subspecies: *M. a. paratuberculosis* positive and/or *M. a. avium* negative. Finally these results were validated by PCR results based on the IS*900* and IS*901* (IS*900+*, IS*901-;* IS*900-*, IS*901+;* IS*900+*, IS*901+*).

All together these results suggest that in wild deer, probably due to winterfeeding or to possible exposition to contaminated water or feedstuff, mortalities due to *M. a. avium*, *M. a. paratuberculosis* or dual infections may occur.

[78] FIRST ISOLATION OF *MYCOBACTERIUM BOVIS* FROM FREE-LIVING WILD BOARS (*SUS SCROFA*) AND RED DEER (*CERVUS ELAPHUS*) IN FRANCE

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Since 1965, the French bovine TB eradication plan in domestic cattle has been based on a test and slaughter program. This plan proved to be efficient and, with a herd prevalence of 0.01%, France was declared « officially free of bovine tuberculosis » by the European Commission in December 2000

Until that date, *Mycobacterium bovis* had never been found in free-living wildlife in France. However, in January 2001, a wild red deer, hunted-killed in a Norman forest (North West of France), presented typical lung and hepatic tuberculous lesions in which *Mycobacterium bovis* was isolated. Moreover, four cattle outbreaks had recently been reported in this area. These observations led us to undertake an epidemiological survey in the respective forest during the 2001-2002 hunting season.

Lung, liver, tonsil samples and retro-pharyngeal, hepatic and tracheo-bronchial lymph nodes were collected from 84 wild boars, 77 red deer and 38 roe deer and processed for mycobacterial culture. *M. bovis* was isolated from 24 wild boars (apparent prevalence = 28.5%) and 11 red deer (apparent prevalence = 14%) but none from roe deer. Most of them did not present visible macroscopic lesions at hunters´ first inspection. *M. bovis* isolation was confirmed by classical biochemical assays as well as by molecular biology tests (*pncA* + *oxyR* PCR).These results reveal the existence of a potential reservoir of *M. bovis* in wildlife that was unsuspected until now in France.

Genotyping by spoligotyping and different VNTR-PCRs was performed. The same genotype was found in the isolates obtained from wild boars, red deers and the four cattle outbreaks occurred two years before. Although a direct link between wild and domestic TB could be logically assumed, the moment and the origin of transmission remain unknown.

In order to face this situation, veterinary services took preventive measures in the infected area not only to avoid contamination of cattle and humans but also to reduce transmission among wildlife. The measures implemented were: to reduce wildlife densities by increasing hunting pressure, to ban artificial game feeding, to destruct killed animals viscera, to fence cattle pastures neighboring the forest, to inspect wild boars and deer carcasses and to reinforce TB cattle testing.

[79] INTRANASAL ADMINISTRATION OF SEDATIVE-TYPE DRUGS TO REDUCE STRESS IN ELK CAPTURED BY NET GUN

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This investigation examined the efficacy and benefit of sedative-type drugs administered by intranasal route to reduce handling stress in elk (*Cervus elaphus manitobensis*). Ninety-five free-ranging elk were captured by net gun in Riding Mountain National Park (Manitoba, Canada) during February 2002 and February-March 2003. A single-blind study was used to score degree of relaxation in 20 elk administered xylazine at 1.5 to 2.0 mg/kg estimated body weight by intranasal route, 26 elk administered xylazine (1 mg/kg) combined with Telazol® (1 mg/kg) by intranasal route, and 49 elk administered an equivalent volume of saline in the same manner (control elk). Elk in both treatment groups had higher relaxation scores than control elk suggesting they were more relaxed during handling. Some treatment elk also showed outward signs of sedation including lowering of respiratory rate and cessation of struggling. Serum biochemistry results indicated serum concentrations of cortisol and creatine kinase (CK) were lower in elk receiving xylazine than in control elk, whereas concentrations in elk receiving xylazine-Telazol® were intermediate between these groups. These results suggest stress and muscle injury was less in elk receiving sedative-type drugs, especially xylazine. In addition, anion gap and duration of time between capture and drug administration were positively correlated among elk receiving xylazine when controlling for variation in pursuit time, suggesting administration of xylazine as soon as possible following capture helped to reduce the progression of lactic acidosis during handling. Intravenous administration of yohimbine (0.15 to 0.20 mg/kg) or tolazoline (2.5 mg/kg) at the conclusion of handling quickly abolished sedative effects allowing elk to be released without concern of physical injury due to ataxia. Intranasal administration of xylazine would appear to be effective for reducing stress in situations where wild animals are being handled while physically restrained. The combination of xylazine and Telazol® may also prove effective at a higher dosage than used in this study.

[80] A PRELIMINARY REPORT ON THE USE OF THIOFENTANYL OXALATE FOR THE CAPTURE OF UGANDA KOB

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For capture of Uganda kob (*Kobus kob thomasi*) in the Queen Elizabeth National Protected Area (QEPA), Uganda, we used 1.5 ml and 3.0 ml Daninject™ darts fired either from a Daninject of a Teleinject™ dart projector. The range of the latter limited its use to animals that were no more than 15 meters from the operator.

All kob and impala were captured using the potent opioid Thiofentanyl oxalate (TF) (formerly known as A3080). Estimated doses between 50 and 60 microgram per kg were chosen based upon published information for weights of 50-60 for females and 90-110 kg for males. Two animals were captured with TF and the alpha2 adrenergic agonist Detomidine™ (D). In situations where animals could be safely weighed the actual doses were subsequently calculated. Naltrexone (Tr) (Trexonil™) was used as a specific opioid antidote for TF. Tolazoline (To) was used as a general antidote for Detomidine. Where possible animals were observed for periods up to 180 min. after capture and release.

Twelve kob were successfully captured. Ten of these were captured using only TF at doses ranging from 71.43 to 50 µg/kg of body weight (mean 55.2 µg/kg). Two others were captured using TF at 50 µg/kg together with Detomidine at 50 µg/kg. The time from injection to the first signs of drug effect ranged from 70 secs. to just under 178 secs. The time from injection to recumbency ranged from 80 secs. to 310 secs (mean 182 secs).

Trexonil (Tr) was administered at dose ratios of 20 mg Tr to 1 mg of TF, except in the first animal darted, when the dose was 100:1. Tolazoline (To) was given as an additional reversal agent at a dose rate of 3 mg/kg, administered half intravenously, half intramuscularly to animals that had been darted with the TF/D mixture. The time to standing after Tr administration ranged from 60 secs to 183 secs.

[81] WILD ELEPHANT (*ELEPHAS MAXIMUS MAXIMUS*) IMMOBILIZATION PROCEDURES FOR TREATMENTS – A REVIEW OF 27 CASES

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Elephant is the largest terrestrial animal in the world. The sub species *Elephas maximus maximus* is unique to Sri Lanka. It is estimated that 3000-3500 wild elephants inhabit in the country. As a result of existing intense human-elephant conflict, every two to three days an elephant and every one to two weeks, a human deaths are happened in the country. On an average 8 animals per month undergo medical treatment particularly for gun shot injuries.

Within the study period (1999-2001), 27 elephant tranquilizations were carried out in the Yala National Park (01), Udawalawa NP (05), Lahugala NP (02), Wasgamuwa NP (05), Minneriya NP (03), Kaudulla Reserve (04), Kantale (03) and Minneriya-Giritale Nature Reserve (03), and Sigirya Sanctuary (01 "Large Animal Immobilon" (etorphine hydrochloride + acepromazine maleate) and "Revivon" (diprenorphine hydrochloride) were the drugs used for elephant tranquilization and as antidote respectively. Dose rate of drug used was between 1.4-3.5ml per animal and the amount was determined by considering the body size, body condition, severity of wounds and physical fitness of the animal. Drug was given intramuscularly using a long range projector. Treatment has been done for the gun shot wounds (24), removal of nooses (04) and natural wounds (03). Darting sites were the back/thigh area (20), shoulder area (07) and below the knee joint (01).

Age of tranquilized animals ranged from 04-50 years. Male to female ratio of tranquilized animals was 20:7. Three animals died in the tranquilization operations due to complications with septicemia (02) and breathing obstruction (01). Induction period of the drug was around 10-15 minutes. Animals have fallen onto its sternum (05) and onto its side (22 - right 16 & left 6). Immediate actions were taken to turn the animals underwent sternal recumbency. Animals kept under immobilized condition for 20-50 minutes in order to carry out treatment. Animals were revived within 5-20 minutes after giving the antidote. Chase back the operators (02), standstill under partial sedation (17) and went away (05) were the behaviors expressed by revived animals.

It is difficult to measure progress of wild elephant treatment accurately, because of practical difficulty in post treatment monitoring. However compared to treatment without immobilization, the prognosis of elephants treated under immobilization is better since this clinical procedure allows intense wound management in wild condition. Further, immobilization is safer to the operators and animal itself, which is a humane way to treat wild elephants. Attending as early as possible for injured animals before they becoming weak is the major factor that determines the success of immobilization and following treatment.

[82] INTERACTIONS BETWEEN CLASSICAL SWINE FEVER VIRUS AND WILD BOAR (*SUS SCROFA*) IN FRANCE; TEN YEARS OF SURVEY: 1992-2002

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Classical Swine Fever (CSF) is a viral disease affecting wild and domestic swine worldwide. Outbreaks occurring in domestic pigs entail severe losses to pig farming industry: the virus is highly contagious and some strains can cause up to 90% mortality. Moreover, massive slaughtering is required to eradicate the disease then CSF-free countries restrict pig trade during outbreaks. As the only wild-living swine species in Europe, the European wild boar *Sus scrofa* is regarded as a potential reservoir of CSF. Cross-contamination is supposed to occur either through direct contact between wild boar and inappropriately restricted domestic swine or through introduction of infected feed, the CSF virus being able to survive in fomites and meat during several months. In recent years, several outbreaks of CSF occurred in European populations of wild boar. It was observed that epizootics have occurred in limited areas and usually have not shown extensive spatial propagation. Transmission amongst free ranging wild boars is supposed to occur mainly through direct contacts and less efficiently by indirect transmission which is regarded as a secondary process.

Factors determining disease propagation and time to extinction have been investigated using data from routine surveillance of an outbreak that occurred in France from 1992 up to 2001. In the Vosges Mountains (France) the here described CSF outbreak persisted nine years and is now considered extinct. The study area spreads over 3030 km² including 1180 km² of woodland During the nine years of survey, 215 spleens and 62 sera were collected and tested from wild boar that were found dead. During the same period 15,593 spleens and 8,013 sera were analyzed from hunted wild boar.

Inside the CSF infection focus, effect of population size, habitat fragmentation, distance from the primary starting point and finally density was examined both upon incidence (number of new cases per year) and persistence (duration of time with cases recorded). Incidence declined in space and was higher in large continuous forests compared to small remote ones. Persistence was positively correlated with population size and initial density; persistence was particularly long near the epicenter where population suffered a significant decline. It is speculated that incidence was affected by the spatial population structure and inhibited by habitat fragmentation. It is as well hypothesized that persistence was linked to population size and factors favoring a high birth rate.

[83] RETROSPECTIVE REVIEW OF DEMODECTIC MANGE AND DERMATOPHILOSIS IN SOUTHEASTERN WHITE-TAILED DEER *(ODOCOILEUS VIRGINAINUS*)

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Demodectic mange and dermatophilosis both produce similar gross lesions consisting of pustular skin lesions and alopecia in white-tailed deer (*Odocoileus virginianus*). Records of white-tailed deer submitted to the Southeastern Cooperative Wildlife Disease Study (SCDWS) from 15 southeastern states between 1971-2002 were examined for diagnoses of either demodectic mange or dermatophilosis. Demodectic mange and dermatophilosis comprised 0.97 % (n=16) and 0.77 % (n=12) of all diagnoses, respectively. Sixty-nine percent of the demodectic mange cases occurred in males with ages of affected deer ranging from 1.5 to 6.5 years. Dermatophilosis was observed more frequently (72%) in fawns and yearlings than adults but a gender predisposition was not apparent. Low sample sizes precluded determination of seasonal and geographical patterns for either disease. The submission of cases was influenced by many unknown factors and the data are undoubtedly biased, and as such may not accurately reflect patterns of these diseases in natural conditions.

[84] *LEISHMANIA* SPECIES ASSOCIATED WITH NON-SUPPURATIVE DERMATITIS IN RED KANGAROOS (*MACROPUS RUFUS*) IN AUSTRALIA'S NORTHERN TERRITORY

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Intracellular protozoa were identified upon microscopic examination of fixed tissues collected from several thickened, hairless and centrally ulcerated skin lesions found on the tail of a red kangaroo (*Macropus rufus*) maintained in a fauna park in Australia's Northern Territory, June 2000. Histological examination of the skin revealed hyperkeratosis, acanthosis, focal dermal necrosis, and extensive granulomatous dermatitis. Macrophages within the dermis contained multiple round cytoplasmic organisms with small, basophilic, eccentric nuclei. Electron microscopy was unsuccessful in further characterising the parasite, and fresh tissues were not available for PCR or culture.

When a second adult red kangaroo within the park developed similar lesions on the tail, biopsies of the lesions were collected and submitted for histopathological examination, protozoal culture and PCR, November 2002. H & E stained sections of the lesions illustrated a non-suppurative dermatitis, and small numbers of extracellular unicellular organisms. Culture of the lesion in two different media identified organisms morphologically consistent with *Leishmania* species. Polymerase chain reaction (PCR) testing and sequence analysis at three genetic loci for which the corresponding sequences for many *Leishmania* species are available, was conducted on ethanol fixed portions of the biopsies. The sequence results of two conserved coding regions of DNA (SSU rDNA, 5.8S rDNA) were highly indicative that the causative organism belongs to the genus *Leishmania*. Analysis of more variable region of DNA (rDNA ITS, mini-exon gene non-transcribed spacer) revealed sequences that differed from corresponding sequences known from *Leishmania* species.

This morphologic and molecular data supports a diagnosis of protozoal dermatitis in a red kangaroo caused by *Leishmania* species infection. This is the first known report of probable endemic leishmaniasis in Australia. Several other red kangaroos from the Darwin region have similar lesions and are currently under investigation.

[85] HERD HEALTH SURVEY AND ISOLATION OF A NEW PESTIVIRUS OF PRONGHORN (*ANTILOCAPRA AMERICANA*) IN WYOMING

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A survey of pronghorn (*Antilocapra americana*) herd health was undertaken at four sites in Wyoming, in the spring of 2000, 2001, and 2002. Seven to 20 pregnant female pronghorn per site were killed late in gestation each year for an ancillary study on reproductive success within each herd. For the study described here, all adult and fetal pronghorn were necropsied and serum/blood, fresh tissues, formalin-fixed tissues, feces, and parasites were collected. Serum from adult pronghorn was tested for evidence of exposure to a variety of ruminant viral and bacterial pathogens, virus isolation attempts were made on pooled fresh tissues from each adult and each fetus, gross lesions were recorded for all animals, formalin-fixed tissues were examined microscopically for significant lesions, and feces were examined by Baermann procedure and fecal flotation examinations. Additionally, abomasal contents were examined microscopically for parasites on 10 adult females from each site for each collection year. Gross necropsy findings, serologic survey results, some parasitology survey results, and virus isolation results will be presented here.

Gross lesions observed in the adult female pronghorn from two sites were minimal, and consisted of various lesions attributed to prior trauma (healed fractures, lacerations, abrasions) and evidence of prior bouts of pleuritis or pneumonia (fibrous adhesions between cranioventral lung lobes and the body wall). At the two other sites frequent abomasal parasitism was detected grossly (*Haemonchus contortus*) in 11/25 (44%) and 10/25 (40%) adult females sampled. Abomasal parasite counts for these two herds were low, compared to deer species (maximum = 120, range = 0 to 120, some counts pending). Several macerated or mummified fetuses were observed at two of the four locations each year.

In all four herds there was serologic evidence of significant exposure to a variety of pathogens. The two northernmost herds had high seroprevalence rates of exposure to epizootic hemorrhagic disease virus (EHDV) and bluetongue virus (BTV) in each of the three years of the study, while the central herd and southeastern herd were serologically negative for EHDV and BTV through all three years of the study. In all four herds almost all animals were seropositive for parainfluenza-3 virus, and there were a few animals in all herds seropositive for bovine viral diarrhea virus (BVDV) types 1 and 2 and border disease virus throughout all 3 years of the study. All animals tested were seronegative for Johne's disease, infectious bovine rhinotracheitis virus, bovine respiratory syncytial virus, five *Leptospira* serovars, and *Brucella abortus*.

During the 2002 collection, a novel pestivirus was isolated from two sets of fetuses collected at the southeastern site. Tissue preparations from each of the four individual fetuses inoculated on bovine and pronghorn cell cultures produced positive fluorescent antibody staining with an anti–BVDV antibody. The viral RNA from each of

these four isolates was amplified by reverse transcriptase polymerase chain reaction using primers that universally amplify the 5' untranslated region (UTR) of all known pestiviruses. Nucleotide sequence analysis of the PCR product obtained from each of these isolates indicates that all four fetuses were infected with the same virus. Phylogenetic analysis based on the sequence of the 5' UTR indicates that these isolates compose a separate group within the genus *Pestivirus* (distinct from BVDV, border disease virus, and classical swine fever virus). Of interest, a genetically identical novel pestivirus was isolated from a pronghorn at the far side of the state (southwest Wyoming) in 2000. The significance of this virus as a pathogen is unknown. Currently, we are characterizing the virus further, and we plan to perform additional field surveys and experimental live animal work to explore the significance of this virus to pronghorn individuals and populations.

[86] EFFECTS OF GNRH AGONIST (LEUPROLIDE) ON REPRODUCTION, BODY CONDITION, AND BEHAVIOR IN FREE-RANGING FEMALE WAPITI

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Overabundant wild ungulates have become a significant problem for natural resource managers in many areas of North America. Fertility control offers a potential solution for regulating the growth of these populations. A promising new approach to contraception involves a gonadotropin releasing hormone (GnRH) agonist (leuprolide). In controlled experiments with captive wapiti (*Cervus elaphus nelsoni)*, leuprolide was shown to be 100 % effective in preventing pregnancy and physiological and behavioral side-effects were minimal. In this study, we extend the evaluation of leuprolide, as a contraceptive agent, to free-ranging wapiti in Rocky Mountain National Park, Colorado. During August-September 2002, we immobilized 34 female wapiti, and fitted them with frequency specific transmitters. Seventeen animals received a 32.5 mg dose of leuprolide in a 120-day sustained release formulation and 17 a placebo formulation. We compared breeding behavior, daily activity patterns, pregnancy rates, and body condition of treated and untreated free-ranging wapiti. Preliminary results indicate that none of the leuprolide-treated females were pregnant while >80% of control females were pregnant in March-April 2003; confirmation of pregnancy status using pregnancy-specific protein B is pending. Additional study results are currently being analyzed and will be presented.

[87] SEROLOGICAL DIAGNOSIS OF *PARELAPHOSTRONGYLUS TENUIS* INFECTION IN MOOSE

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Meningeal worm or *Parelaphostrongylus tenuis* (Family : Protostrongylidae) is a known cause of neurological signs and death in moose, and infection can be diagnosed antemortem by a newly developed enzyme-linked immunosorbent assay (ELISA) which detects serum IgG antibodies directed against the excretory-secretory products of the third-stage larvae of *P. tenuis*. For the purpose of obtaining base-line serological information on moose in a *P. tenuis* endemic area, serum samples obtained from a total of 162 moose at the time of collaring were tested. These were made available from 2 different studies: 65 moose in the northeast part of Minnesota (47E 30' and 91E 25') collared between 2002 and 2003, and 97 moose in the northwest part of the state (48E 25' and 96E 15') collared between 1995 and 1998. Health status of the animals in the northwest was monitored and carcasses of dead animals examined; observations and presumed cause of mortality were recorded. In the northeast, the proportion of animals with antibodies to *P. tenuis* (% seropositive, ELISA Optical Density > 0.386) was 20.0% (n=65). Clinical signs were recorded for 4 animals which were eventually necropsied. Two were euthanized because of marked disease processes including inability to stand, complete lack of fear of humans or emaciation, and on postmortem examination adult *P. tenuis* worms were recovered from the heads, which correlated with positive serology. The other 2 also displayed clinical signs consistent with *P. tenuis* infection including emaciation or unsteady gait progressing to inability to stand, and both were seropositive even though no worms were recovered at postmortem. In the northwest group, 54 of the collared animals had died by 2000 when monitoring was discontinued, 34 animals were still alive, and 9 were missing. Recorded causes of death (3 categories) and initial anti-*P. tenuis* antibody titres were: (i) loss of body condition and likely disease process including *P. tenuis*, 46.7% seropositive (n=15); (ii) non-infectious, 14.8% seropositive (n=27); and (iii) possibly fluke-related (*Fascioloides magna)*, 25% seropositive, (n=12). Seven (20.6%) of the 34 animals still alive in 2000 were seropositive when bled 3-5 years earlier. Animals dying of a disease process where *P. tenuis* may be involved were more likely to be seropositive than those dying of non-infectious agents or where flukes may have played a role (P=0.04, Fisher=s Exact test; two tailed). Despite the lack of statistical Animals dying of disease process were more likely to be seropositive (46.7%) than surviving animals (20.6%) but this difference was not statistically significant (P=0.09; Fisher=s Exact test; two tailed) probably because of low sample size. From this preliminary study, the following conclusions can be made: (a) *P. tenuis* appears to play a

significant role in moose mortalities in northern Minnesota, (b) some animals survive for a number years despite *P. tenuis* seropositive status, although some of the seropositive animals may be expected to succumb to *P. tenuis* infection over time, (c) the serological test gave a higher estimate of exposure of moose to *P. tenuis* than past studies using traditional parasite recovery methods, (d) the serological test appears to be a worthwhile tool for the management of moose herds, (e) the test can now be used to directly investigate the hypothesis that moose survivorship in *P. tenuis* endemic areas is significantly associated with negative *P. tenuis* serology although a large sample size may be required because of the confounding effect of other causes of moose mortality.

[88] SURVEILLANCE AND MANAGEMENT OF CHRONIC WASTING DISEASE IN THE UNITS OF THE NATIONAL PARK SYSTEM

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Chronic wasting disease (CWD), a transmissible spongiform encephalopathy of deer and elk, has emerged as a significant wildlife management issue throughout the United States, including units of the National Park System (NPS). CWD has been confirmed in two national parks and numerous other NPS units are at significant risk due to proximity to areas with CWD. CWD has been diagnosed in elk and deer in Rocky Mountain National Park that is located within the historic or "enzootic" area of disease distribution in Colorado. In November 2002, CWD was detected in an elk collected at Wind Cave National Park, South Dakota. Subsequently, CWD positive mule deer were identified in the park. The National Park System is using general and targeted surveillance in deer and elk populations in areas where CWD has been found within or adjacent to a park. Additionally, live animal testing using tonsillar biopsy is being used as a management tool for deer populations in affected parks. On a Service-wide basis, cervid movements into or out of NPS units are strictly limited. All parks are encouraged to perform targeted surveillance, and to coordinate and cooperate with state wildlife management agencies. Surveillance and management actions performed within units of the NPS are guided by the NPS mission and management policies, including public involvement and environmental compliance processes.

[89] DEMOGRAPHIC PATTERNS OF CWD PREVALENCE IN A HIGH-DENSITY WHITE-TAILED DEER POPULATION

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Chronic wasting disease is a fatal disease of white-tailed deer (*Odocoileus virginianus*) associated with transmissible protease resistant prions. Since the discovery of chronic wasting disease in southern Wisconsin in 2002, more than 12,000 deer have been removed from the CWD affected area surrounding the three initial cases. All deer removed were tested for CWD by immunohistochemical staining of retropharyngeal lymph nodes or obex. In addition, sex, age, and harvest location were recorded for each deer as it was removed. Preliminary analysis from the first year of removals indicates that chronic wasting disease infection is more prevalent in males than in females. Testing of the remaining deer from the eradication zone is under way and will be completed and evaluated during summer 2003. We will report the prevalence of CWD in age and sex classes as well as the harvest vulnerability of CWD infected individuals in the demographic classes. In addition, several deer younger than 1 year of age have tested positive, including the youngest (5-6 months old) infected wild cervid to be reported. Much about CWD is unknown, but with the large sample of white-tailed deer from an infected area we will be able to form better hypotheses about how CWD occurs in a high-density population of free-ranging white-tailed deer.

[90] AN APPARENT DUPLICATION OF THE PRION PROTEIN CODING SEQUENCE IN MULE DEER (*ODOCOILEUS HEMIONUS*)

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The prion protein (PrP) gene sequence was determined from genomic DNA of 131 mule deer (*Odocoileus hemionus)* harvested during the 2001 hunter survey in the chronic wasting disease (CWD) endemic region of Wyoming. Ten recurring polymorphisms were found, manifested as heterozygous genotypes occurring at various frequencies at codons 20, 65, 131, 138, 139, 151, 156, 202, 225 or 247. The absence of homozygous genotypes at codons 138 (100% serine/asparagine), 139 (arginine^1/arginine^2) and 156 (asparagine^1/asparagine^2) suggested the possibility of a duplicate gene for the prion protein coding sequence in the mule deer genome.

We first obtained 5' flanking sequence for the mule deer open reading frame using a polymerase chain reaction (PCR) primer complementary to a partial LINE2a repeat element about 650 bases upstream of the protein coding sequence in published bovine and ovine DNA sequences. From the mule deer sequence thus obtained, another PCR primer was designed to amplify the coding sequence DNA from about 40 bases upstream, a primer site specific to only one gene. A downstream primer that binds beyond the termination codon of the protein was also obtained using the bovine and ovine sequences. We were thus able to amplify the complete open reading frame of one gene. When this was sequenced, the same animals that previously appeared heterozygous at codons 65, 138, 139, 151, 156 or 202 were homozygous, supporting the duplicate gene theory and allowing us to deduce its sequence by the differences between the two sequences.

Of the ten apparent polymorphisms found when both genes were sequenced together, six remain when the two genes are separated: four occur in this single gene (D20G, Y131Y, S225F, and I247I) and two in the duplicate gene (G65E, R151C). Codon 138 is homozygous for serine in the single gene, and therefore presumably homozygous for asparagine in the duplicate; codon 139 is homozygous for a different arginine (R) codon in each of the genes, likewise 156 is homozygous for different asparagine codons, and codon 202 for different threonine (T) codons. Of the single gene polymorphisms, only S225F results in an amino acid heterozygous condition within the mature polypeptide, as D20G is removed from the precursor protein as part of the signal sequence. We do not yet know if the duplicate gene is expressed, nor if it is what effect this might have on either susceptibility or resistance to CWD in mule deer. Because the single gene described here is found in the same DNA context as the prion gene in cattle and sheep whose complete *PRNP* gene region is known, and shares a high degree of similarity in the upstream region with elk (*Cervus elaphus*), fallow deer *(Dama dama)*, moose (*Alces alces*), and white-tailed deer (*Odocoileus virginianus*), it is likely to be a

functional gene. We do not yet know if a duplicate of the prion gene exists in any other species.

[91] SPATIAL DISTRIBUTION OF CHRONIC WASTING DISEASE IN FREE-RANGING WHITE-TAILED DEER IN WISCONSIN

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Chronic wasting disease (CWD) is a progressively degenerative and ultimately fatal condition in deer (Odocoileus spp.) and elk (Cervus elaphus) associated with transmissible protease resistant prion proteins. Since its discovery in white-tailed deer (O. virginianus) from Wisconsin in February 2002, over 40,000 deer have been sampled statewide to determine the extent of the affected area. Testing for presence of CWD was conducted by immunohistochemical staining of retropharyngeal lymph nodes and brain (obex) tissues at the Wisconsin Veterinary Diagnostic Laboratory in Madison, WI. For surveillance and management purposes, the state has been divided into three CWD zones: an approximately 1000 km2 "eradication zone" where CWD is known to be present; a "CWD management zone" that extends to approximately 65 km from the center of the eradication zone; and the "out-state" area consisting of the remainder of the state. None of > 21,000 deer sampled in the out-state zone tested positive for CWD. Sampling was sufficient in 42 of 48 sampling units in this zone to have > 90% probability of detecting CWD prevalence at 1% or greater. In the remaing 6 sampling units detection probability was 70-90%. Sampling intensity in the management zone was sufficient to detect CWD at 1% or greater prevalence with greater than 95% (4 units) and 99% (4 units) probability. In this zone, 6 of 5800 (0.1%) deer tested positive in three sampling units. Within the eradication zone, approximately 2% of the deer have tested positive. Initial spatial analysis of these data indicates the distribution of CWD in this area is heterogeneous, with a core area of higher prevalence surrounded by lower prevalence. We discuss the implications of this spatial distribution for our understanding of the epidemiology of CWD in white-tailed deer. The results of this massive sampling effort indicate the distribution of CWD may be limited to south-central Wisconsin, although further surveillance will be necessary in areas with insufficient sampling, areas adjacent to the known-affected area, and in areas deemed to be at high risk of becoming affected.

[92] STUDIES ON TSE (CWD, BSE, AND SCRAPIE) IN CERVIDS FROM GERMANY – PRELIMINARY RESULTS

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Since bovine spongiform encephalopathy (BSE) was first found in cattle from Germany in November 2000 and chronic wasting disease (CWD) has spread within North America, the question arises whether transmissible spongiform encephalopathies (TSE) might affect wild ruminants within Germany as well. Around 25,000 to 30,000 tons of venison originating from German game is consumed each year. Approximately 1.2 million cervids are hunted per year throughout Germany: specifically 1.1 million roe deer (*Capreolus capreolus*), 55,000 red deer (*Cervus elaphus elaphus*) and 42,000 fallow deer (*Cervus dama dama*). Until now, there is no information whether TSE exist in cervids from Germany or any other European country. However, it is theoretically possible that TSE (BSE, CWD or scrapie) could have been introduced to German cervid populations in the past. The main objective of our study is to determine the occurrence of TSE in cervids in Germany. Within a period of three years approx. 10,000 brain (obex region) and lymph node samples of indigenous cervid species from all over Germany shall be screened for TSE by Biorad ELISA (enzyme linked immunosorbent assay). Moreover, immunohistochemistry will be performed in at least 1000 brain and lymph node samples from German cervids. Because we are interested in several risk factors that may affect a possible transmission of BSE, CWD and scrapie we have set up a stratified sampling procedure. Most of the samples will be collected from free-living roe deer (n= approx. 6000), red deer (n= approx. 800) and fallow deer (n = approx. 500) that are older than 18 months. Special sampling effort is directed towards cervids that are living in regions where BSE has been diagnosed in cattle or scrapie in sheep and individuals that show clinical signs like cachexia or CNS disorders. One third of the samples (n= approx. 3000) will be collected from captive cervids, which could have been fed with BSE contaminated concentrates. CWD positive tissue is used for evaluating the specificity and sensitivity of various BSE rapid tests. Preliminary results show that none of several hundred brain samples originating from German cervids so far was tested positive for TSE by Biorad ELISA.

[93] EXPERIMENTAL INOCULATION OF TME, SCRAPIE, AND CWD TO RACCOONS (*PROCYON LOTOR*) AND THE UTILIZATION OF RACCOONS FOR STRAIN-TYPING OF UNKNOWN TSES IN THE UNITED STATES

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Raccoons (Procyon lotor) are omnivorous and their diet may include carrion. It is, therefore, possible that in the wild they may get exposed to carcasses of animals with transmissible spongiform encephalopathies (TSEs). To determine the susceptibility of raccoons to transmissible mink encephalopathy (TME), scrapie, and chronic wasting disease (CWD), each of these agents was inoculated intracerebrally into a group of 4 kits. Three uninoculated kits served as controls. All raccoons in the TME-inoculated group developed neurologic signs and were euthanized within 6 months post inoculation **(**PI). In the scrapie-inoculated group, 3 animals became sick and were euthanized between 18 and 22 PI. Although the fourth raccoon in this group did not show any clinical signs, it was euthanized at 24 months PI. At present, 3 years PI, all CWD-infected raccoons are alive and apparently healthy. At necropsy all clinically affected raccoons had extensive microscopic lesions of spongiform encephalopathy and protease-resistant prion protein (PrP^{res}) was detected in the CNS by immunohistochemistry and Western blot. These preliminary findings demonstrate that TME and scrapie can be transmitted to raccoons within 6 months and 2 years, respectively, whereas CWD cannot. Based on these incubation periods, it may be possible to differentiate these 3 TSEs. Such a laboratory model would be relatively simple, fast and inexpensive for strain-typing of unknown TSEs in the United States.

ABSTRACTS FROM POSTER PRESENTATIONS

[94] CLINICAL, PATHOLOGIC AND MOLECULAR CHARACTERIZATION OF POXVIRUS INFECTIONS IN TWO STELLAR SEA LIONS (EUMETOPIAS JUBATUS) IN ALASKA

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There are two genetically distinct populations of Steller sea lions (*Eumetopias jubatus*) in Alaska; the eastern and western stocks, that are listed under the Endangered Species Act as threatened and endangered, respectively. During the course of multidisciplinary studies on free-ranging, live-captured and released pups and juveniles, two animals were found to have skin lesions consistent with active poxvirus infection. Both of these animals were from Prince William Sound, a part of the western stock. Both were females in poor body condition at 2 and 5 months of age. Hematologic indices were within normal limits. In the two-month-old animal, raised, umbilicated and occasionally ulcerated nodules were present primarily on the foreflippers. In the second animal, similar lesions were present over extensive areas of the body. These lesions were biopsied, one half placed in 10% buffered formalin, the other half frozen on dry ice, then stored at -70 º C. Histologically, the lesions were very similar and consistent with poxviral infection, including epithelial cell proliferation with nodule formation in the dermis and presence of intracytoplasmic inclusion bodies within these epithelial cells. Viral cultures were unsuccessful. Skin biopsies were analyzed by polymerase chain reaction for poxvirus DNA using two sets of consensus primers that target highly conserved regions within the DNA polymerase and the DNA topoisomerase genes of capripoxvirus and swinepox virus and yield fragments of 543-bp and 344-bp, respectively. Both fragments were sequenced and their deduced amino acid sequences were aligned and compared to homologous sequences of other Chordopoxvirinae available in the GenBank database. The homologous nucleotide sequences derived from the skin lesions of both animals were identical. The deduced amino acid sequence of the Steller sea lion poxvirus DNA polymerase gene fragment was aligned and compared with homologous sequences of camelpox, capripox, smallpox, swinepox and vaccinia viruses and their identity varied between 71.7 and 77.2 percent. When the DNA topoisomerase amino acid sequence was aligned and compared to the homologous sequence of the same viruses the identities varied between 75.4 and 79.8 percent. These results indicate that the Steller sea lion poxvirus involved in these clinical cases is a member of the Chordopoxvirinae subfamily of poxviruses.

[95] ULTRASTRUCTURAL STUDIES ON THE LIFECYCLE AND PATHOLOGY OF *BATRACHOCHYTRIUM DENDROBATIDIS*, THE AMPHIBIAN CHYTRID

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Batrachochytrium dendrobatidis, the cause of chytridiomycosis, was apparently introduced to Australia in the 1970s. Mass mortality of amphibians in Australia due to chytridiomycosis resulting in population declines in high altitude sites was associated with first arrival of *B. dendrobatidis* in areas. Six frog species have disappeared since 1979 and four of these are now listed as extinct. Chytridiomycosis was listed as a "key threatening process" by the Australian government in 2002 (http://www.ea.gov.au/biodiversity/threatened/ktp/frog-fungus.html), and a national threat abatement plan is in preparation. Death in susceptible experimental animals usually occurs between 18 and 48 days after exposure, although the mechanism of pathogenicity remains unclear. The lifecycle of *Batrachochytrium dendrobatidis* is relatively simple: there is a motile, waterborne, infective zoospore for dispersal and a stationary zoosporangium for amplification that can occur within epidermal cells. We used a range of microscopy methods to examine stages of the life cycle in culture and in frog skin. Methods included transmission electron microscopy with conventional methods as well as high pressure freezing and freeze substitution, and scanning electron microscopy on critical point dried, bulk-frozen and freeze-fractured material. Although chytridiomycosis is an emerging disease, *B. dendrobatidis* has adaptations that suggest it has long been evolved to live within cells in the dynamic tissue of the stratified epidermis, supporting the theory that it has recently spread from amphibian hosts in a restricted distribution. Sporangia form in superficial cell layers in the *stratum granulosum* and develop at a rate that coincides with the maturing of epidermal cells. At the skin surface fungal discharge tubes open outwards to release zoospores into the environment. Up to three sporangia were observed within the one cell. Ultrastructural pathology was assessed in the skin of two frogs. The cycle of sloughing of keratinized cells was disrupted with multiple layers of dark keratinized cells accumulating before cleaving from the underlying cells resulting in loss of the entire thickened *stratum corneum*. Hyperkeratosis appeared to be due to a hyperplastic response and also as sporangia seemed to initiate early cell death and keratinization. The epidermis may be much thicker or thinner than normal, possibly reflecting differences in the balance between the germination of epidermal cells, the increased rate of keratinization and the loss of the superficial layer. A zone of condensed fibrillar cytoplasm occurred intracellularly in a roughly lamellar arrangement around some sporangia. This may protect the fungus and could explain the lack of effect of topical antifungal drugs. Dissolution of uninfected cells and vesiculation occurred in and above the *stratum basale*.

[96] AURAL ABSCESSATION IN FREE-LIVING EASTERN BOX TURTLES (*TERRAPENE CAROLINA*): A PATHOLIC AND EPIDEMIOLOGIC DESCRIPTION

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A recent study found aural abscessation to be a significant cause of morbidity and mortality in free-living Eastern box turtles (*Terrapene carolina*) of Virginia. Though its etiology remains unknown, hypovitaminosis A has been suggested based on a similar lesion occurring in captive chelonian fed diets deficient in vitamin A. This hypothesis was supported by significantly greater body burdens of organochlorine compounds (a reported disruptor of vitamin A metabolism), and a non-significant rend toward lower serum and vitamin A levels found in free-living box turtles with this lesion. The tympanic epithelium was evaluated in twenty-seven turtles (ten with aural abscessation and seventeen without) in order to describe the pathology of this lesion. Pathologic changes to the tympanic epithelium of turtles with aural abscessation included: hyperplasia, squamous metaplasia, hyperemia, cellular sloughing, desquamation, granulomatous inflammation, and bacterial infection. These changes were more severe in turtles with aural abscesses as opposed to those without, and were greater in tympanic cavities containing an abscess as opposed to one without (when the lesion was unilateral). The epidemiology of fifty cases of aural abscessation in free-living eastern box turtles admitted to the Wildlife Center of Virginia (WCV) from 1991 to 2000 was also evaluated using multivariable logistic regression. County human population density, year and season of admission, weight, and gender were not associated with an increased risk for box turtles developing aural abscesses. Spatial location of cases (based on county) was analyzed for clustering by Grimson's method. Counties with cases of aural abscessation were not randomly distributed, but rather were clustered into two multi-county regions. The histopathologic changes in turtles with aural abscessation and the epidemiologic profile are consistent with a syndrome that may involve hypovitaminosis A and/or environmental organochlorine compound exposure, and is being further investigated.

[97] CULTURE AND SEROLOGIC SURVEY FOR *MYCOBACTERIUM AVIUM* SUBSP. *PARATUBERCULOSIS* INFECTION AMONG SOUTHEASTERN WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*)

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From July 1998 through October 2002, radiometric culture (ileocecal lymph node, mesenteric lymph node, and feces) and serologic testing using an enzyme-linked immunosorbent assay (ELISA) were used to survey white-tailed deer (*Odocoileus virginianus*) from the southeastern United States for infection by *Mycobacterium avium* subsp. *paratuberculosis* (*Mptb),* the causative agent of paratuberculosis (Johne's disease). *Mptb* was isolated from the ileocecal lymph node of 1 of 313 deer (0.3%) originating from 63 populations in Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, South Carolina, Tennessee, and West Virginia. Six deer (2%), all from different populations, had ELISA results above a 0.25 sample-to-positive (S/P) cut-off value, but none of the ELISA reactors originated from the population from which the single *Mptb* isolation was made. The six ELISA reactors were seronegative when tested by agar gel immunodiffusion (AGID). These data indicate that white-tailed deer currently do not constitute a broad regional reservoir for *Mptb*; however, further study is warranted to clarify the significance, if any, of infected deer to the epizootiology of paratuberculosis on a local scale. Validation of ELISA or some other serologic assay for use wildlife would markedly enhance *Mptb* surveillance among wild populations and would be a powerful tool for clarifying any role of wild species in epidemiology of paratuberculosis.

[98] SURVEY OF THE BACTERIAL FLORA OF TREE- AND GROUND-NESTING DOUBLE-CRESTED CORMORANTS (*PHALACROCORAX AURITUS*) IN PRINCE EDWARD ISLAND, CANADA

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Malpeque Bay, Prince Edward Island (PEI), Canada, contains two islands with breeding colonies of Double-crested cormorants (DCC). One is a tree-nesting colony of approximately 4,000 pairs. The other is a ground-nesting colony of approximately 300 pairs on a treeless island 3 km to the south. The latter colony shares the island with large numbers of Herring Gulls (*Larus argentatus*) and a lesser number of Great Black-backed Gulls (*L. marinus*).

The objective of this survey was to determine the prevalence of carriers of potentially pathogenic bacteria among 2-3-week-old DCC nestlings in the tree-nesting colony (2001 breeding season) and, specifically, of *Salmonella* sp. and *Erysipelothrix rhusiopathiae* in the ground-nesting colony (2002 breeding season). Serotypes, phage types, biotypes, and antibiotic resistance analysis were performed on significant isolates.
In 2001, cloacal and pharyngeal swabs were taken from 100 nestlings. Potentially pathogenic bacteria recovered included 208 isolates of *Escherichia coli* and 22 isolates of *Campylobacter* spp. No *Salmonella* spp. was isolated. In 2002, cloacal swabs were obtained from 50 nestlings at the ground-nesting colony on Little Courtin Island. Sixteen isolates of *Salmonella* spp. were recovered from 13 birds.

The detailed bacteriological results of this survey show that, at least with respect to pathogenic species, bacteria isolated from these DCC differ in several respects when compared to isolates of human and livestock origin. None of the *Campylobacter jejuni* isolates match those found in cattle, swine, chickens or turkeys in Canada and have very little in common with isolates of human origin. In contrast to findings of a survey of Salmonella isolates of agricultural and human origin in PEI, drug resistance among the *Salmonella* isolates from DCC was very low. Finally, 87% of the *E. coli* isolates from DCC were sensitive to all 12 antibiotics tested. Resistance to cephalothin and enrofloxacin was 0%, and resistance to gentamicin was only 0.5%. In contrast, isolates from domestic poultry are frequently resistant to ampicillin, cephalosporins, enrofloxacin, gentamicin and tetracycline.

The results of our study suggest that DCC on PEI do not serve as amplifiers of bacterial contaminants associated with human and agricultural activities, that they are currently not a source of drug resistant plasmids, and that they are unlikely to contribute to a drug resistance pool among enterobacteriaceae in water sources and the environment.

[99] DISEASES OF WILD BIRDS IN FLORIDA: AN ANALYSIS AND OVERVIEW

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Florida has a rich and diverse avian fauna. There are 457 species in the state and of these, 446 are native and 11 have been introduced or have expanded their ranges into Florida recently. A large number and variety of diseases, parasites, and other morbidity and mortality factors have been recognized in wild birds of Florida. Some information is known for 311 (68%) of the 457 species, although the amount and quality of such information is variable. We have no information on the other 146 species, most of which are perching birds, shorebirds, waterfowl, gulls, terns, or oceanic birds that are transients or rare or uncommon statewide. The low relative abundance and lack of study of these birds in Florida may account for the absence of data on them. Other species such as Wood Ducks, Mottled Ducks, Wild Turkeys, Northern Bobwhites, and Mourning Doves have been well studied, probably because of their abundance and economic importance. The federal and state survival status (endangered, threatened, or species of special concern) of some species, such as Common Loons, Brown Pelicans, Sandhill Cranes, Whooping Cranes, Bald Eagles, and other raptors, has resulted in increased research on them. At least 153 infectious and parasitic disease agents are potentially shared or exchanged between wild birds, various exotic caged birds, poultry, and other domestic animals in Florida and at least 49 are zoonotic.

[100] PATHOLOGIC FINDINGS IN SEVENTEEN BLACK MARSH TURTLES, *SIEBENROCKIELLA CRASSICOLIS*

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In December of 2001, thousands of confiscated Asian turtles of 12 different species were diverted to the United States for rescue and rehabilitation under the supervision of the Turtle Survival Alliance (TSA). The TSA is a joint venture of several conservation organizations established by the World Conservation Union Species Survival Commission. On January 16th, 2002, 63 critically ill Black Marsh turtles (*Siebenrockiella crassicolis*), were allocated to the Wildlife Clinic at Tufts University (North Grafton, MA, 01569, USA) for treatment by a student volunteer rescue team. Forty-three (68%) were returned 3 months later for placement in captive breeding facilities. Basic medical and husbandry skills were employed with surprising success in these turtles. All mortalities (20 turtles) occurred during the first 13 weeks. Postmortem examinations of 17 turtles were performed at the Wildlife Clinic; histologic examinations and occasional ancillary tests (bacterial isolation, culture and antibiotic sensitivity) were performed at the Connecticut Veterinary Diagnostic Laboratory of the University of Connecticut (Storrs, CT 06269). The 17 Black Marsh turtles examined were invariably affected by several concurrent conditions. The most frequent lesions involved the skin, brain, mouth, upper respiratory tract, lungs and gastrointestinal tract.

Necrotizing stomatitis, pharyngitis, and/or tracheitis (n=9) and formation of thick diphtheric pseudomembranes were associated with bacteria (n=8) or fungal hyphae (n=1). Bacterial lesions including pneumonia (n=10), coelomitis (n=6), encephalitis (n=2), oophiritis (n=3), hepatitis (n=1) and endocarditis (n=1) were common findings. Bacterial cultures of the lung (n=1) and liver (n=2) of turtles with pneumonia, coelomitis and hepatitis identified *Aeromonas hydrophila*, a bacterium commonly found in the water column. *Citrobacter freundii* was isolated from the lung of a turtle (n=1) with granulomatous pneumonia and concurrent pulmonary mineralization. These bacteria could potentially have gained entry through the deep epidermal ulcerations present in the plantar and/or palmar surfaces of all the turtles examined or through the respiratory mucosa. Miracidium-filled trematode eggs caused interstitial pneumonia (n=2) and granulomas in the liver (n=3), spleen (n=3), stomach (n=1) and intestine (n=2). The eggs most likely belonged to Spirorchid trematodes who are frequent inhabitants of the vasculature of chelonians, although no adults were found to confirm the species. Necrotizing gastritis (n=4) and enteritis (n=2) with myriads of bacteria were also present in the turtles. Several cases of meningoencephalitis were associated with the presence of unidentified organisms resembling protozoans (n=3). Amyloidosis of hepatic, splenic and renal arterioles was observed (n=1) in concurrency with pneumonia, coelomitis and oophiritis and thus probably resulted from chronic inflammation.

Metastatic calcification of epithelial and endothelial basement membranes, collagen and muscle of multiple organs, including the lungs (n=9), stomach (n=6), trachea (n=4), dermis (n=2), aorta (n=1) and skeletal muscle (n=1) was a common finding. Although some of the turtles affected with metastatic calcification had concurrent renal

disease (n=8), many had no visible renal lesions. Dehydration during their confinement prior to rehabilitation and the wide range of infectious/inflammatory processes that afflicted these turtles were probably important factors in the multisystemic calcification.

[101] EXPERIMENTAL CROSS-SPECIES TRANSMISSION OF CHRONIC WASTING DISEASE TO DOMESTIC LIVESTOCK AT THE NATIONAL ANIMAL DISEASE CENTER: AN UPDATE

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To determine the transmissibility of chronic wasting disease (CWD) to cattle and sheep, 13 calves and 8 lambs were inoculated intracerebrally with brain suspension from mule deer ($CWD^{mule\ deer}$) naturally affected with CWD. Both investigations are currently in-progress. The cattle experiment was started in 1997. Within 5½ years post inoculation (PI), 10/13 cattle either died or were euthanized. Five of these were positive for prion disease (by Western blot and immunohistochemistry), but did not reveal obvious histologic changes indicative of spongiform encephalopathy (SE). The remaining 3 cattle are alive and apparently healthy. The ovine experiment is 4 years PI and so far 2 sheep (both QQ) have been euthanized. Only 1 had histopathologic lesions of SE which were indistinguishable from lesions of sheep scrapie. The brain was positive for prions. Six remaining sheep (2 QQ and 4 QR) are apparently healthy. These preliminary findings demonstrate that $CWD^{mule\ deer}$ can be transmitted to cattle and sheep and that diagnostic techniques currently used for BSE surveillance would also detect CWD in cattle and sheep should it occur naturally.

[102] TRANSMISSION OF SHEEP SCRAPIE TO ELK (*CERVUS ELAPHUS NELSONI*) BY INTRACEREBRAL INOCULATION

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To determine the transmissibility of scrapie to Rocky Mountain elk (*Cervus elaphus nelsoni*), 6 elk calves were inoculated intracerebrally with brain suspension from sheep naturally affected with scrapie. Two others were kept as uninoculated controls. A preliminary report of this study was published previously. During the first 2 years post inoculation (PI), 3 animals died or were euthanized because of injuries or infection other then spongiform encephalopathy (SE). In years 3 and 4 PI, 3 other elk died after brief terminal neurological episodes. Necropsy of these animals revealed moderate weight loss but no other gross lesions. Microscopically, characteristic lesions of SE were seen throughout the brains and spinal cords and these tissues were positive for PrP^{res} by immunohistochemistry (IHC) and Western blot. Also, scrapie-associated fibrils (SAF) were observed by negative stain electron microscopy in the brains of elk with neurologic signs. PrP^{res} and SAF were not detected in inoculated elk necropsied during the first 2 years or in the 2 control animals. Retrospective sequencing of gene encoding PrP in affected elk revealed MM or LM at codon 132. These findings confirm that intracerebral inoculation of sheep scrapie agent results in SE with accumulations of PrP^{res} in the CNS of elk and that this condition cannot be distinguished from chronic wasting disease (CWD) of elk with currently available diagnostic techniques.

[103] WILD RODENT ASSOCIATED BARTONELLA: A GROUP OF NEW AND EMERGING BACTERIA IN SASKATCHEWAN, CANADA

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The genus *Bartonella* is comprised of Gram-negative bacteria that parasitize mammalian red blood cells. Many recently described *Bartonella* species that cause human and animal diseases are associated with animal reservoirs including, *B. vinsonii subsp. berkhoffii* which has been isolated from coyotes (*Canis latrans*), and *B. elizabethae, B. grahamii* and *B. vinsonii subsp. auropensis* which have been isolated from a variety of wild rodent species.

Interest in wild rodents as reservoir hosts for *Bartonella* led to studies that found numerous isolates of *Bartonella* in many different rodent species. The majority of these rodent associated *Bartonella* isolates have not yet been associated with human or animal diseases but by deliberately scrutinizing reservoir species we might be given an early warning about potential disease-causing agents. Indeed, isolates of *Bartonella* from the blood of a California ground squirrel (*Spermophilus beecheyii*) were identical to *Bartonella washoensis* isolated from a human patient with fever and myocarditis and there is serological evidence that a rodent associated *Bartonella* was responsible for febrile illness in humans in New Mexico.

The objectives of this preliminary project were to determine if *Bartonella* occurs in wild rodents, specifically Richardson's ground squirrels (*Spermophilus richardsonii*) and deer mice (*Peromyscus maniculatus*), in Saskatchewan and genetically compare any *Bartonella* isolates found with isolates of *Bartonella* that have been found elsewhere.

Blood samples were collected from 86 wild rodents at 6 sites around Saskatoon. All sites had bacteremic animals with prevalence rates ranging from 17% to 100% for deer mice and from 36% to 46% for ground squirrels. Seven sequence variants of *Bartonella* were isolated from rodents around Saskatoon, three different isolates from *P. maniculatus*, and 4 different isolates from *S. richardsonii*. The 4 isolates from *S. richardsonii* have not been previously identified. Our isolates most closely resemble isolates from related species in the southwestern United States.

[104] GENOTOXIC EFFECTS OF FLARE GAS EMISSIONS IN RICHARDSON'S GROUND SQUIRRELS (*SPERMOPHILUS RICHARDSONII*)

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Many pollutants have the potential to interact with cellular DNA, resulting in genetic damage. While the techniques used to assess genetic damage are finding increasing use in wildlife toxicology, they are only beginning to be applied to field studies of wild mammals. Polycyclic aromatic hydrocarbons (PAHs) are a well-known group of genotoxicants associated with the petroleum industry. The combustion of excess natural gas at oil and gas field facilities (a process known as flaring) results in the atmospheric release of PAHs and other hydrocarbons at thousands of locations in the oil patch of Western Canada. This study was intended to evaluate the genotoxic potential of exposure to flare gas emissions using a widely distributed indigenous small mammal, Richardson's ground squirrel (*Spermophilus richardsonii*), as a model. Two methods of evaluating genetic damage were used. The comet assay, which detects DNA strand breaks, and flow cytometry to measure DNA variability, were performed on peripheral leukocytes collected from live-trapped ground squirrels at 21 sites in Alberta and Saskatchewan. The trap locations were chosen to represent a range of potential exposure to flare gas emissions, based on proximity to oil and gas field facilities and industry data on the volume of gas flared at each site. The comet assay involves detection of DNA fragments which, on electrophoresis, migrate from the nuclear core at a distance inversely proportional to fragment size, providing an index of the amount of DNA strand breaks. Flow cytometry measures variation in DNA content among cells from abnormal distribution caused by irreversible chromosomal damage. Since genotoxic effects are ultimately manifested as tumours and disease, results of comet assay and flow cytometry were combined with histopathological examination of selected tissues to enhance the prediction of toxic effects by encompassing a range of genetic processes, from first exposure to changes which become fixed to the disease outcome. The relationships between these endpoints of genetic damage in ground squirrels and chronic exposure to flare gas emissions at the field sites were determined.

[105] SCREENING FOR ZOONOTIC BACTERIA IN CERVID FECAL SAMPLES

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The annual outcome of game meat originating from moose (*Alces alces)*, red deer (*Cervus elaphus*) and roe deer (*Capreolus capreolus*) in Norway is approximately 7,000 metric tons, or approximately 1.5 kg pro capita. This meat is not always treated optimally with regards to hygienic principles, and faecal contamination of the carcasses must be regarded as very likely during field slaughtering. To a great extent, the meat is consumed by the hunters and their families, hence, it is not usually subject to any meat inspection control. Wild animals also constitute a risk for contamination of drinking water sources.

Salmonella, *Campylobacter* and verocytotoxic *E. coli* (VTEC) are all important bacterial enteropathogens in humans, in Norway with an increasing importance according to yearly numbers of reported cases. Screenings of meat products have revealed the presence of antibiotic resistant indicator organisms (i.e. *E. coli* and enterococci), particularly in pork and poultry products. The aim of the present study was to examine the occurrence of *Salmonella*, *Campylobacter*, VTEC and antibiotic resistance of indicator organisms in faecal samples from moose, red deer and roe deer killed during hunting in Norway.

In charge of the Health Surveillance Program for Cervids (HSP), faecal samples were collected during the hunting seasons from red deer in 2001, and from roe deer and moose in 2002. Samples from a total of 468 animals were examined for verocytotoxic *E. coli*, first by immunomagnetic separation of potentially pathogenic O-serovars (serovars O26, O103, O145, O111 and O157), cultivation on selective plates, and then agglutination of susceptible colonies with O-specific sera. All serologically positive strains were examined by PCR for the gene-sequences *stx* (shigatoxin) and *eae* (intimin). Samples from 458 animals were tested for *Salmonella*, by pre-enrichment in phosphate buffered peptone water, selective enrichment in Rappaport-Vassiliadis soya peptone broth, and plating out on red violet bile agar plates and bromthymol blue lactose, sucrose agar. Colonies were isolated and tested biochemically and serologically for confirmation. *Campylobacter*-analyses were carried out in 120 animals (roe deer and moose) by direct cultivation on *Campylobacter* blood free selective agar (Oxoid CM 739) supplemented with cefoperazone, amphotericin B and teicoplanin (Oxoid SR 174). Presumptive *Campylobacter* colonies were confirmed by phase-contrast microscopy. The different species were identified by phenotypic assays, including growth pattern at 42ºC, catalase production and hippurate hydrolysis. From 50 samples from each cervid species, the indicator bacterial species *E. coli* and *Enterococcus faecalis* or *E. faecium* were attempted isolated by incubation on bromthymol blue lactose, sucrose agar plates and Slanetz & Bartley enterococcus agar plates (44°C), respectively. Typical enterococcus colonies were confirmed by a negative catalase reaction, and *E. faecium* and *E. faecalis* were identified by PCR. Presumptive *E. coli* colonies were sub-cultured on blood agar and confirmed by the indole test. Antibiotic resistance patterns were tested in a MIC-test

based on dilutions in microtiter plates. *Escherichia coli* and *E. faecium* / *E. faecalis* were tested against 14 and 13 different antimicrobials, respectively.

[106] HUMAN DIMENSIONS IN CHRONIC WASTING DISEASE MONITORING

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Chronic wasting disease (CWD), a transmissible spongiform encephalopathy (TSE) of cervids, is rapidly spreading from the original endemic area of Colorado and Wyoming. Over the past two years, CWD has spread to 11 new states including Wisconsin and Illinois. As it spreads closer to the southeastern United States, state wildlife agencies in the region are preparing protocols to monitor and manage the disease. In general, these protocols are being developed for the biologists and law enforcement officers who will be observing or testing white-tailed deer in the field. Roles for the general public, however, are often overlooked in these emerging protocols. Realizing the importance of public involvement, the Florida Fish and Wildlife Conservation Commission (FWC) created a management and monitoring plan that actively engages the public in monitoring CWD in Florida.

The FWC's CWD surveillance protocol is based on traditional active and passive techniques observed in many states and countries today. However, a critical component of our protocol is an emphasis on public education and involvement. Letters and press releases explaining revised cervid importation laws and surveillance programs have targeted captive cervid owners, hunters and non-hunters alike. FWC biologists are working with hunters on private and public lands to obtain samples for our active and passive surveillance programs. And our CWD hotline not only provides relevant, factual information to citizens regarding questions or comments about CWD, but also enables us to rapidly respond to public sightings of unhealthy deer – an important part of our passive surveillance program.

During the first year of implementation, we tested 603 active, 19 passive and 12 captive herd samples. As of May 2003, results include 79% (478/603) of the active surveillance samples, 95% (18/19) of the passive surveillance samples and 75% (9/12) captive samples submitted. Five samples were unsuitable for testing; none of the suitable samples tested positive.

These results, although preliminary, indicate that CWD has not found its way into Florida. However, continuation of a surveillance program involving cooperation with the public is integral to the monitoring and management of this deadly disease.

[107] PRELIMINARY EVALUATION OF CARCASS DISPOSAL METHODS FOLLOWING AN OUTBREAK OF ANTHRAX IN NORTHERN BISON

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Anthrax *(Bacillus anthracis)* is endemic to free ranging wood bison *(Bison bison athabascae)* herds in northern Canada. The most recent outbreaks of the disease occurred in Wood Buffalo National Park in summers 2001 and 2002 and resulted in at least 100 and 91 bison mortalities respectively. In August 2002 during routine aerial surveillance for anthrax, we found 12 bison carcasses in the Hook Lake area of the Slave River Lowlands, Northwest Territories. All 12 animals were mature bulls and had been dead for at least several weeks as evidenced by their advanced stage of decomposition and extensive disarticulation due to scavengers. Through implementation of an emergency response plan, we coordinated the disposal of these carcasses and used a modified Before and After Control Impact (BACI) design to evaluate field carcass disposal and decontamination methods at seven carcass sites. We used a circular plot design to randomly sample surface soil and vegetation within a 3 – 5 meter radius around each carcass before and after an incineration and/or decontamination treatment event. We incinerated carcasses with various fuel types used singly or in combination including i) a kerosene-based jet fuel with a fuel thickener additive (Petrol Jel™), ii) dried wood from standing dead spruce trees *(Picea* spp.*)*, and iii) coal. We used a 10% formaldehyde solution (10% v/v) to decontaminate carcass sites either before or after incineration. Surface soil and vegetation samples (n = 340) were stored in freezers and then shipped to the Defence Research and Development Canada BL-3 Laboratory in Suffield and stored at room temperature prior to processing. One of us (DCD) analyzed the samples by culturing bacterial isolates with selective PLET medium for the presence of viable *B. anthracis* spores. Based on our experiences and observations on bison carcass incineration and decontamination, we suggest the following: 1) the use of gelled fuel is not a viable stand-alone technique for incinerating entire bison carcasses in the field; 2) coal burns hotter and longer than either wood or gelled fuel, and is easily transportable from a central staging area, 3) application and burning of fuel (solid or liquid) on top of a carcass is not effective and it is critically important to ensure adequate air flow underneath a carcass either by elevating the carcass &/or applying solid fuel below the carcass to facilitate complete incinerations in the field with minimal disturbance; and 4) the combination of carcass incineration and decontamination with formaldehyde had the strongest effect on reducing anthrax spore contamination at a site.

[108] IMMUNOLOGIC RESPONSES AND EFFICACY OF HAND OR BALLISTIC VACCINATION OF BISON (*BISON BISON*) WITH *BRUCELLA ABORTUS* STRAIN RB51

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The prevalence of brucellosis in bison within Yellowstone National Park in the United States has raised concerns in regards to possible transmission to domestic livestock. In a series of studies conducted at our laboratory, we have characterized immunologic responses of bison calves to hand vaccination with *Brucella abortus* strain RB51 (SRB51), or ballistic vaccination with a commercially available biobullet in which a SRB51 pellet has been placed. In addition, the influence of ballistic delivery on immunologic responses was evaluated by comparing responses of bison ballistically inoculated with SRB51 to responses of bison in which a SRB51 biobullet was surgically placed intramuscularly. Immunologic responses evaluated included: antibody responses, proliferative responses by purified peripheral blood mononuclear cells (PBMC) to killed SRB51, interferon- production by PBMC, nitrous oxide production, and flow cytometric analysis of proliferating PBMC subsets. Antibody responses of hand and ballistic vaccinates were similar. However, cell-mediated responses tended to be greater in hand vaccinates as compared to ballistic vaccinates. Bison were pasture bred as 3 years and pregnancy status and days in gestation determined by rectal palpation. Based on rectal palpation data, bison were challenged between 170 and 180 days gestation by placing 1 x 107 CFU of *Brucella abortus* strain 2308 bilaterally on the conjunctiva. Following abortion or full-term parturition, bison cows and calves were euthanized and tissues collected for bacteriologic and histologic evaluation. Although hand vaccination and single ballistic vaccination reduced the incidence of abortion or fetal infection when compared to nonvaccinates, the ability to recover the challenge strain from maternal tissues at necropsy did not differ between vaccine and control treatments. Our data suggests that immunologic responses of bison to vaccination with RB51 differ dependent upon the route of vaccine delivery. Our data also suggests that vaccination of bison with RB51 can reduce the incidence of abortion and risk of transmission, and may be of value as a management tool in a *Brucella*-infected bison herd.

[109] SHARED FEED AS A MEANS OF DEER TO DEER TRANSMISSION OF *MYCOBACTERIUM BOVIS*

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To determine the ability of experimentally inoculated white-tailed deer (Odocoileus virginianus) to transmit Mycobacterium bovis to naïve deer through the sharing of feed, 4 deer were intratonsillarly inoculated with 4 x 105 (colony forming units) CFU of M. bovis. On a daily basis, feed not consumed by inoculated deer after approximately 8 hours, was offered to 4 naïve deer maintained in a separate pen where direct contact, aerosol transmission, or transmission through personnel were prevented. After 150 days, naïve deer were euthanized and examined. All naïve deer had lesions consistent with tuberculosis and M. bovis was isolated from various tissues. The most commonly affected tissues were the lung, tracheobronchial lymph nodes and mediastinal lymph nodes. This study demonstrates the potential for indirect transmission of M. bovis through the sharing of feed. Artificial feeding of deer in regions where M. bovis infection is endemic should be avoided, as both direct and indirect transmission through sharing of feed is enhanced.

[110] THE IMPACT OF URBANIZATION ON THE INTESTINAL PARASITIC COMMUNITY OF RED FOXES (*VULPES VULPES*) IN THE CANTON OF GENEVA, SWITZERLAND

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Red foxes (*Vulpes vulpes*) are infected by a wide range of intestinal helminths, which can potentially occur in humans, hence referred as zoonotic parasites. Whereas nematodes, especially hookworms (*Uncinaria stenocephala, Ankylostoma caninum*) and ascarids (*Toxocara canis, Toxascaris leonina*), and some species of *Taenia* are widespread in foxes as well as in domestic carnivores (dogs and/or cats), one typically fox-infesting cestode species, *Echinococcus multilocularis* (E.m.), even if it might be harboured at low prevalence by dogs and cats, is of particular health concern. Indeed, since the late eighties, modifications in fox behavioural habits have led to a recent phenomenon of colonization of cities in continental Europe, by so-called urban foxes, therefore considered as potential vectors of parasitic zoonoses. Also described in Switzerland, it resulted in epidemiological and ecological studies on red foxes, and in particular in the city of Geneva, an agglomeration of over 300'000 inhabitants.
Red foxes found dead or shot were collected since 1998, in the canton of Geneva. Post mortem examination was performed and parasitic analyses were based on the intestinal Sedimentation and Counting Technique (SCT). The canton of Geneva was divided into three areas of different urbanization levels (rural, residential and urban areas), with regards to the human density, using the Kernel method.

The intestinal parasitic community of 159 foxes was dominated by four groups of helminths: hookworms (prevalence of 79.2%), mainly *Uncinaria stenocephala*; ascarids, *Toxocara canis* (72.3%); the small fox-tapeworm, *E. multilocularis* (43.4%); and other taeniids, Taenia spp. (38.8%). While no visible effect of the level of urbanization appeared on hookworm prevalence or abundance, rural foxes were significantly more often infected with *Toxocara canis* (prevalence of 77.3%) and with Cestodes (prevalence of 50.8% for E.m., 50.0% for Taenia spp.) than urban foxes (prevalences of 57.9%, 21.1% and 18.4%, Chi-square, p=0.0375, p=0.0029 and p=0.0084 respectively), residential area's prevalences being close to those from the rural area. Their abundances also differed significantly between foxes from rural and urban areas, *T. canis* and E.m. burden being higher in rural foxes (Mann-Whitney, p=0.0365 and p=0.0013 respectively) whereas *Taenia* spp. burden being higher in urban foxes (Mann-Whitney, p=0.0353). Another 90 carcasses are about to be analysed, to increase the statistical qualities of the sample.

Rodents with regards to the habitat urbanization and their landscape ecology and spatial distribution seem to play a major role in fox-taeniids epidemiology, as they are intermediate hosts in their biological cycles. Furthermore, they are described as paratenic hosts in *Toxocara canis* cycle. Whether predation, rodents infestation rates, and rodents spatial distribution are likely to explain these parasites distribution along an increasing

gradient of urbanization leads to further investigations, notably on foxes diet and parasitic analyses on rodents.

[111] IMMUNE RESPONSES OF WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*) TO *MYCOBACTERIUM BOVIS* BCG VACCINATION

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The objective was to evaluate the cellular immune response of captive white-tailed deer (*Odocoileus virginianus*) to live *Mycobacterium bovis* bacille Calmette Guerin (BCG) vaccination. In vitro proliferative and interferon-γ responses to *M. bovis* purified protein derivative (PPD) were detected beginning 9 days postvaccination. Responses to *M. avium* PPD, however, generally exceeded responses to *M. bovis* PPD. Interferon-γ responses to *M. avium* PPD were not detected prior to vaccination or by non-vaccinated deer, indicating that vaccination with *M. bovis* BCG boosted prior quiescent *M. avium* sensitization. Both $CD4^+$ and γδ T cells from vaccinated deer proliferated in response to *M. bovis* PPD stimulation. Intradermal administration of *M. bovis* PPD resulted in significant ($P < 0.05$) increases in skin thickness of vaccinated deer beginning 24 hrs postinjection. Early reactions (i.e., 24 hrs postinjection) were characterized by edema and minimal mononuclear cell infiltration whereas later reactions (i.e., 72 hrs postinjection) were more typical of delayed type hypersensitive reactions. Upon in vitro activation with pokeweed mitogen, CD44 expression (i.e., mean flourescence intensity) increased and CD62L expression decreased on lymphocytes from deer regardless of vaccination status. Likewise, *M. bovis* PPD stimulation of lymphocytes from vaccinated deer resulted in increased CD44 fluorescence and decreased CD62L fluorescence. In summary, these findings demonstrate the potential of an IFN-γ-based assay to detect mycobacterial sensitization of white-tailed deer, the presence of cross-reactive *M. avium* responses that may confound *M. bovis* diagnosis, and expected alterations in CD44 and CD62L expression upon in vitro stimulation of white-tailed deer lymphocytes.

AUTHOR INDEX[1]

[1] All presenting authors have their names underlined.