PRESENTATION SCHEDULE

MONDAY, JUNE 5

WELCOME AND OPENING COMMENTS

8:00-8:30 TERRY KREEGER, Conference Chair

8:30-8:50 JACK NECKELS, Superintendent, Grand Teton National Park

8:50-9:10 CLARENE LAW, Wyoming State Representative

WILDLIFE DISEASE ASSOCIATION AND AMERICAN ASSOCIATION OF WILDLIFE VETERINARIANS: CUTTINGE EDGE SPEAKER

9:15-10:05 **(1) BOVINE TUBERCULOSIS IN THE KRUGER NATIONAL PARK.** ROY G. BENGIS AND DEWALD F. KEET

10:05-10:15 **WYOMING WONDERS.** MARK GOCKE

10:15-10:30 **BREAK**

BOVINE TUBERCULOSIS SYMPOSIUM
Moderator: John Fischer

10:30-10:45 **(2) BOVINE TUBERCULOSIS IN MICHIGAN – AN UPDATE.** STEPHEN M. SCHMITT, SCOTT D. FITZGERALD, COLLEEN S. BRUNING-FANNNATHAN, NATHAN ZAUEL, AND DALE E. BERRY

10:45-11:00 **(3) DEER TO DEER TRANSMISSION OF *MYCOBACTERIUM BOVIS*.** MITCHELL V. PALMER AND DIANA L. WHIPPLE

11:00-11:15 **(4) SURVIVAL OF *MYCOBACTERIUM BOVIS* ON FEEDS USED FOR BAITING WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*) IN MICHIGAN.** DIANA L. WHIPPLE AND MITCHELL V. PALMER

11:15-11:30 **(5) TRANSMISSION OF *MYCOBACTERIUM BOVIS* FROM EXPERIMENTALLY INFECTED WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*) TO CATTLE THROUGH INDIRECT CONTACT.** DIANA L. WHIPPLE AND MITCHELL V. PALMER

11:30-12:30 **LUNCH**

BRUCELLOSIS AND OTHER DISEASES OF CERVIDS
Moderator: E. Tom Thorne

12:30-12:45 **(6) BRUCELLOSIS IN ELK (*CERVUS ELAPHUS*) OF EASTERN IDAHO.** MARK L. DREW

12:45-1:00 **(7) ELK (*CERVUS ELAPHUS NELSONI*) BRUCELLOSIS SURVEILLANCE IN WESTERN WYOMING.** SCOTT G. SMITH, E. TOM THORNE, WALTER E. COOK, HANK EDWARDS, TERRY KREEGER

1:00-1:15 **(8) LESIONS AND TISSUE COLONIZATION SITES OF *BRUCELLA ABORTUS* IN ABORTED BISON FETUSES, BISON CALVES, AND ADULT FEMALE BISON FROM YELLOWSTONE NATIONAL PARK.** JACK C. RHYAN, THOMAS GIDLEWSKI, THOMAS J. ROFFE, KEITH AUNE, L. MICHAEL PHILO, AND DARLA R. EWALT

1:15-1:30 **(9) EFFICACY OF *BRUCELLA ABORTUS* STRAIN RB51 VACCINE IN CAPTIVE ELK: A PRELIMINARY REPORT.** TERRY J. KREEGER, WILLIAM H. EDWARDS, WALTER E. COOK, STEVEN C. OLSEN, AND PHILIP H. ELZER

1:30-1:45 **(10) IMMUNE RESPONES AND EFFICACY OF *BRUCELLA ABORTUS* STRAIN RB51 VACCINE IN BISON (*BISON BISON*).** STEVEN C. OLSEN AND M.V. PALMER

1:45-2:00 **(11) BRUCELLOSIS IN DOMESTIC PIGS AND WILDBOARS DUE TO *BRUCELLA SUIS* BIOVAR 2 IN FRANCE.** B. GARIN-BASTUJI, J. HARS, D. CALVEZ, M. THIEBAUD, AND M. ARTOIS

2:00-2:15 **(12) PATHOGENESIS OF CHRONIC WASTING DISEASE IN ORALLY EXPOSED MULE DEER (*ODOCOILEUS HEMIONUS*): PRELIMINARY RESULTS.** ELIZABETH S. WILLIAMS AND MICHAEL W. MILLER

2:15-2:30 **(13) CHRONIC WASTING DISEASE IN ELK (*CERVUS ELAPHUS NELSONI*) HELD IN A CWD ENDEMIC FACILITY.** ELIZABETH S. WILLIAMS, WALTER E. COOK, HANK EDWARDS, TERRY KREEGER, AND SCOTT SMITH

2:30-2:45 **(14) MECHANISMS FOR CHRONIC WASTING DISEASE TRANSMISSION: CLUES FROM INFORMATION-BASED COMPARISON OF COMPETING TRANSMISSION MODELS.** MICHAEL W. MILLER

2:45-2:55 **WYOMING WONDERS.** MARK GOCKE

2:55-3:15 **BREAK**

DISEASES OF CERVIDS
Moderator: Todd Cornish

3:15-3:30 **(15) PREVALENCE OF *TOXOPLASMA GONDII* ANTIBODIES IN CARIBOU (*RANGIFER TARANDUS*) AND MUSKOX (*OVIBOS MOSCHATUS*) SERA FROM NORTHERN CANADA.** BRETT ELKIN, SUSAN KUTZ, AND J.P. DUBEY

3:30-3:45 **(16) ATTEMPTS TO TREAT VERMINOUS PNEUMONIA AND ASSOCIATED HAIR LOSS IN FREE RANGING BLACK TAIL DEER.** P. BRIGGS HALL AND LOUIS C. BENDER

3:45-4:00 **(17) A MULTI-AGENCY ATTEMPT TO RECOVER WOODLAND CARIBOU.** P. BRIGGS HALL AND JON A. ALMACK

4:00-4:15 **(18) CHARACTERIZATION OF COPROANTIGENS OF *OSTERTAGIA* SP. INFECTIONS IN FARMED RED DEER.** T. QURESHI, C. SANTRICH, R. E. LABES, M. TAYLOR, M. L. CROSS, AND C. G. MACKINTOSH

4:15-4:30 **(19) A RETROSPECTIVE SURVEY OF THE OCCURRENCE OF NEOPLASIA IN DEER IN THE SOUTHEASTERN UNITED STATES.** C.F. QUIST AND J.C. LANG

4:30-4:45 **(20) GIANT LIVER FLUKE IN ELK ISLAND NATIONAL PARK (ALBERTA): A RECENT CHANGE IN STATUS.** M.J. PYBUS

4:45-5:00 **(21) FATAL SYSTEMIC ADENOVIRUS INFECTION IN A CAPTIVE MOOSE CALF (*ALCES ALCES*).** ELIZABETH S. WILLIAMS, HOWARD LEHMKUHL, TERRY KREEGER, CAROL HEARNE, JAQUELINE CAVENDER, AND HANA VAN CAMPEN

TUESDAY, JUNE 6

8:00-8:10 **ANNOUNCEMENTS**

STUDENT RESEARCH RECOGNITION AWARD

8:10-8:30 **(22) INVESTIGATION OF FRANKLIN'S GULL (*LARUS PIPIXCAN*) MORTALITY IN RELATION TO THE INITIATION OF AVIAN BOTULISM AMONG WATERFOWL AT EYEBROW LAKE, SASKATCHEWAN.** CATHERINE SOOS AND GARY WOBESER

TERRY ADMUNSON PRESENTATION COMPETITION
Moderator: Ellis Greiner

8:30-8:45 **(23) CROSS-PROTECTION BETWEEN EPIZOOTIC HEMORRHAGIC DISEASE SEROTYPES 1 AND 2 IN EXPERIMENTALLY INFECTED WHITE-TAILED DEER.** JOSEPH K. GAYDOS, ELIZABETH W. HOWERTH, VICTOR F. NETTLES, AND DAVID E. STALLKNECHT

8:45-9:00 **(24) THE DISTRIBUTION OF *ECHINOCOCCUS GRANULOSUS* (CESTODA: TAENIIDAE) CYSTS IN MOOSE POPULATIONS: THE ROLE OF PREDATION BY WOLVES.** DAMIEN O. JOLY AND FRANCOIS MESSIER

9:00-9:15 **(25) EFFECTS OF BACK-MOUNTED RADIO TRANSMITTERS WITH A SUBCUTANEOUS ANCHOR IN MALLARD DUCKLINGS.** KAREN L. MACHIN

9:15-9:30 **(26) GENETIC VARIATION IN EPIZOOTIC HEMORRHAGIC DISEASE VIRUS ISOLATES COLLECTED FROM THE SOUTHEASTERN UNITED STATES: 1978-1998.** M.D. MURPHY, N.J. MACLACHLAN, E.W. HOWERTH, AND D.E. STALLKNECHT

9:30-9:45 **(27) PREVALENCE OF POTENTIAL ZOONOSES IN FERAL AND PET DOMESTIC CATS FROM RANDOLPH COUNTY, NORTH CAROLINA.** FELICIA B. NUTTER, MICHAEL K. STOSKOPF AND JAY F. LEVINE

9:45-10:00 **(28) HOST SUSCEPTIBILITY TO EXPERIMENTAL MYCOPLASMA INFECTION IN HATCHLING AMERICAN ALLIGATORS (*ALLIGATOR MISSISSIPPIENSIS*) EXPOSED TO ENDOCRINE DISRUPTING CONTAMINANTS.** LAUREN J. RICHEY, TRENTON R. SCHOEB, MARY B. BROWN, TIMOTHY S. GROSS, AND PAUL A. KLEIN

10:00-10:10 **WYOMING WONDERS**

10:10-10:30 **BREAK**

10:30-10:45 **(29) PRION PROTEIN IN THE VAGOSYMPATHETIC TRUNK AND PANCREAS OF MULE DEER NATURALLY INFECTED WITH CHRONIC WASTING DISEASE.** CHRISTINA J. SIGURDSON, TERRY R. SPRAKER, MICHAEL W. MILLER, BRUNO OESCH, KATHERINE I. O'ROURKE, AND EDWARD A. HOOVER

10:45-11:00 **(30) EXPERIMENTAL INOCULATION OF AMERICAN OPOSSUMS (*DIDELPHYS AZRAE)* WITH *MYCOBACTERIUM BOVIS*.** KELLY L. BUTLER, SCOTT D. FITZGERALD, DALE BERRY, STEVEN CHURCH, WILLIE M. REED, JAMES SIKARSKIE, AND JOHN B. KANEENE

CAPTURE AND IMMOBILIZATION
Moderator Kristin Charlton

11:00-11:15 **(31) REVERSIBLE IMMOBILIZATION OF FREE-RANGING SVALBARD REINDEER, NORWEGIAN REINDEER AND WOODLAND CARIBOU: A COMPARISON OF MEDETOMIDINE-KETAMINE AND ATIPAMEZOLE IN THREE SUBSPECIES OF *RANGIFER TARANDUS*.** JON M. ARNEMO, RONNY AANES, NIGEL A. CAULKETT, W. JAMES RETTIE, AND JERRY C. HAIGH

11:15-11:30 **(32) SURVIVAL AND IMPROVED MOOSE (*ALCES ALCES)* IMMOBILIZATION WITH CARFENTANIL AND XYLAZINE.** THOMAS J. ROFFE, KENNETH W. COFFIN, AND JOEL BERGER

11:30-11:45 **(33) ANESTHESIA OF PRONGHORN USING A-3080 OR A-3080 PLUS XYLAZINE.** TERRY J. KREEGER, WALT COOK, CLAUDE A. PICHÉ AND THOMAS SMITH

11:45-12:00 **(34) EVALUATION OF THE EFFECTS OF TRANQUILIZATION AND OTHER FACTORS ON MORBIDITY AND MORTALITY IN WILD-CAUGHT PRONGHORN ANTELOPE (*ANTILOCAPRA AMERICANA)* DURING TRANSLOCATION.** JULIE A. SMITH, THOMAS J. ROFFE, DONALD S. DAVIS, AND PHILIP H. ELZER

12:00-1:00 **LUNCH**

PROBLEMS OF CAPTIVE WILDLIFE
Moderator: Cindy Driscoll

1:00-1:15 **(35) MORTALITY IN CAPTIVE PRONGHORN ANTELOPE: A TEXAS EXPERIENCE.** JOHN F. EDWARDS, DONALD S. DAVIS, BARBARA C. LEWIS, FERNANDO RAMIRO-IBANEZ, LYNNE CHITTENDEN, THOMAS J. ROFFE, JULIE A. SMITH, AND PHILIP H. ELZER

1:15-1:30 **(36) KEEPING ARABIAN TAHR (*HEMITRAGUS JAYAKARI*) IN CAPTIVITY-VETERINARY REVIEW.** JACOB M. MWANZIA

1:30-1:45 **(37) "MALADAPTATION SYNDROME" REVISITED.** PETER DASZAK AND ANDREW A. CUNNINGHAM

PREDATORY MAMMALS

1:45-2:00 **(38) EVALUATION OF CABERGOLINE AS A REPRODUCTIVE INHIBITOR IN COYOTES (*CANIS LATRANS*).** THOMAS J. DELIBERTO, AMY E. SEGLUND AND BRUCE KIMBALL

2:00-2:15 **(39) EVIDENCE OF NATURALLY OCCURRING *EHRLICHIA CHAFFEENSIS* INFECTION IN COYOTES FROM OKLAHOMA.** A. ALAN KOCAN, G. CROWDER LEVESQUE, L. WHITWORTH, G.L. MURPHY, S.A. EWING, AND R.W. BARKER

2:15-2:30 **(40) SEROLOGIC SURVEY FOR CANINE CORONA VIRUS IN WOLVES (*CANIS LUPUS*) FROM INTERIOR ALASKA, 1994-1999.** RANDALL L. ZARNKE, JIM EVERMANN, JAY M. VER HOEF, MARK E. MCNAY, RODNEY D. BOERTJE, CRAIG L. GARDNER, LAYNE G. ADAMS, BRUCE W. DALE, AND JOHN BURCH

2:30-2:45 **(41) RETROSPECTIVE STUDY OF DYSPLASTIC AND NEOPLASTIC LESIONS OF THE BILIARY SYSTEM IN THE CAPTIVE POPULATION OF BLACK-FOOTED FERRETS (*MUSTELA NIGRIPES*).** STÉPHANE LAIR, KAY G. MEHREN, ELIZABETH S. WILLIAMS, AND IAN K. BARKER

2:45-2:55 **WYOMING WONDERS**

2:55-3:15 **BREAK**

PREDATORY MAMMALS
Moderator: Rick Brown

3:15-3:30 **(42) HEPATOCELLULAR CARCINOMA IN A POLAR BEAR (*URSUS MARITIMUS*) AND A GRIZZLY BEAR (*URSUS ARCTOS*).** HOWARD STEINBERG, WAYNE I. ANDERSON, KATHRYN COYLE, AND ANNETTE GENDRON-FITZPATRICK

3:30-3:45 **(43) MORBILLIVIRUS AND CALICIVIRUS ANTIBODIES IN POLAR BEARS (*URSUS MARITIMUS*) F'ROM SVALBARD.** MORTEN TRYLAND, ANITA HUOVELAMEN, HANNELE TAPIOVAARA, ANDREW E. DEROCHER, OYSTEIN WUG, AND ALBERT D. M. E. OSTERHAUS

3:45-4:00 **(44) EFFECT OF MORTALITY DUE TO VEHICULAR COLLISION ON FLORIDA'S BLACK BEAR POPULATIONS.** MARK W. CUNNINGHAM, J. WALTER McCOWN, THOMAS H. EASON, DAVID S. MAEHR, TERRY GILBERT, AND DONALD J. FORRESTER

4:00-4:15 **(45) INFECTION WITH *HELICOBACTER* SPP. IN FREE RANGING LYNX (*LYNX LYNX*) AND RED FOXES (*VULPES VULPES*) IN SWEDEN.** TORSTEN MÖRNER, CAROLINE BRÖJER, MARIE-PIERRE DEGIORGIS, DOLORES GAVIER-WIDÉN, HANS-OLOF NILSSO, AND TORKEL WADTSRÖM

NONCERVID UNGULATES

4:15-4:30 **(46) HEALTH EVALUATION OF MONGOLIAN GAZELLES (*PROCAPRA GUTTUROSA*) ON THE EASTERN STEPPES OF MONGOLIA.** SHARON L. DEEM, MICHAEL J. LINN, GEORGE SCHALLER, BADAMJAVIN LHAGVASUREN, HYAMSUREN, KIRK OLSON, ELLEN DIERENFELD, AND WILLIAM B. KARESH

4:30-4:45 **(47) AN UPDATE ON ANTHRAX IN WILDLIFE IN THE KRUGER NATIONAL PARK.** ROY G BENGIS

WEDNESDAY, JUNE 7

8:00-8:15 **ANNOUNCEMENTS**

TOXICOLOGY
Moderator: Mike Ziccardi

8:15-8:30 **(48) COMPARISON OF HEALTH STATUS BETWEEN MUSKRATS (*ONDATRA ZIBETHICA*) FROM HABITATS CONTAMINATED VS. NONCONTAMINATED WITH HIGHWAY RUNOFFS.** J. D. BORUCINSKA AND J. TRETTEL JR.

8:30-8:45 **(49) DECLINE OF THE SOUTHERN SEA OTTER (*ENHYRDA LUTRIS NEREIS*): FURTHER EVIDENCE OF CONTAMINANTS AND DISEASES FROM NON-POINT SOURCES.** DAVID A. JESSUP, MELISSA CHECHOWITZ, JACK AMES, MIKE HARRIS, KAREN WORCESTER, AND DAVID M. PARADIES

8:45-9:00 **(50) THE USE OF GELDANAMYCIN TO PREVENT THE TOXIC EFFECTS OF INTERNAL OIL EXPOSURE ON REPRODUCTION OF MINK (*MUSTELA VISON*) AS A MODEL FOR SEA OTTERS.** JONNA A. K. MAZET, KIRSTEN V.K. GILARDI, DEANA L. FRITCHER, AND RICHARD J. AULERICH

9:00-9:15 **(51) OILED WILDLIFE REHABILITATION: SCIENTIFIC EVALUATION OF SURVIVAL AND BEHAVIOR.** SCOTT H. NEWMAN and JONNA A. K. MAZET, RICK T. GOLIGHTLY, AND JAY HOLCOMB

9:15-9:30 **(52) SEIZURES CAUSED BY DIELDRIN TOXICITY IN A LITTER OF RED FOX (*VULPES VULPES*) KITS.** CATHERINE M. BROWN

9:30-9:45 (**53) DOSE-TITRATION AND SAFETY OF LUFENURON FED TO CAPTIVE WHITE-TAILED PRAIRIE DOGS (*CYNOMYS LEUCURUS*).** KEVIN T. CASTLE, MARGARET A. WILD, AND S. CRAIG PARKS

9:45-10:00 **(54) DIAGNOSIS, MAGNITUDE, AND REMEDIATION OF LEAD EXPOSURE AMONG WILD BIRDS AND MAMMALS AT A FIREARMS TRAINING FACILITY.** WILLIAM R. DAVIDSON, LYNN A. LEWIS, JOHN R. FISCHER, ROBERT H. POPPENGA, AND KATHLEEN A. MORGAN

10:00-10:15 **(55) A CHARACTERIZATION OF BREVETOXIN IN TISSUES OF MANATEES (*TRICHECHUS MANATUS LATIROSTRIS*) IN FLORIDA.** SCOTT D. WRIGHT AND THERESA CODY

10:15-10:30 **(56) FACIAL CLEFTS IN NORTHERN LEOPARD FROG TADPOLES (*RANA PIPIENS*) FROM WISCONSIN.** CAROL U. METEYER, D. EARL GREEN, AND KATHRYN A. CONVERSE

10:30-10:40 **WYOMING WONDERS**

10:40-11:00 **BREAK**

AVIAN DISEASES
Moderator: Christina Sigurdson

11:00-11:15 **(57) WEST NILE VIRUS, A NEW EMERGING DISEASE OF AMERICAN WILDLIFE.** ROBERT G. MCLEAN, SONYA RENEE UBICO, NICHOLAS KOMAR, NICHOLAS A. PANELLA, LINDA C. GLASER, DOUGLAS D. DOCHERTY, LOUIS SILEO, WALLACE R. HANSEN, MICHAEL D. SAMUEL, WARD B. STONE, AND DOUGLAS E. ROSCOE

11:15-11:30 **(58) WEST NILE VIRUS-ASSOCIATED MORTALITY IN BIRDS FROM NEW JERSEY**. DOUGLAS E. ROSCOE, ROBERT KENT, FAYE E. SORHAGE, WAYNE CRANS, ROBERT MCLEAN, LINDA GLASER, DOUGLAS DOCHERTY, WALLACE HANSEN, NICHOLAS KOMAR, AND ROBERT S. LANCIOTTI

11:30-11:45 **(59) AN AVIAN WEST NILE VIRUS EPORNITIC AT A ZOOLOGICAL PARK.** PAUL P. CALLE, TRACEY S. MCNAMARA, BONNIE L. RAPHAEL, ROSANDRA M. MANDUCA, MICHAEL J. LINN, TRACY L. CLIPPINGER, ELIZABETH M. RUSH, ROBERT A. COOK, GEORGE V. LUDWIG, KEITH E. STEELE, JOE MANGIAFICO, JONATHAN F. SMITH, MICHAEL J. TURELL, RANDAL J. SCHOEPP, AND TOM LARSEN

11:45-12:00 **(60) HEALTH RISK ASSESSMENT: A COMPONENT OF THE SITE SELECTION FOR A REINTRODUCED EASTERN MIGRATORY POPULATION OF WHOOPING CRANES (*GRUS AMERICANA*).** JULIE LANGENBERG

12:00-12:15 (**61) DIAGNOSIS OF DISSEMINATED VISCERAL COCCIDIOSIS IN CRANES USING PCR.** M. JOHNSON

12:15-1:15 **LUNCH**

AVIAN DISEASES
Moderator: Thijs Kuiken

1:15-1:30 **(62) TOWERKILL MORTALITY OF MIGRATING PASSERIFORMS: AN "EMERGING DISEASE"?** DONALD J. FORRESTER, WILLIAM R. EVANS, AND R. TODD ENGSTROM

1:30-1:45 **(63) SPATIAL AND TEMPORAL DISTRIBUTION OF AVIAN VACUOLAR MYELINOPATHY IN AMERICAN COOTS (*FULICA AMERICANA*) IN THE SOUTHEASTERN UNITED STATES.** JOHN FISCHER, ROBERT LONG, DAVID GREGORY, KAREN ROWE, MARK CLARK, BUDDY BAKER, CHRISTOPHER YEE, JAMES OZIER, PHILLIP SPIVEY, THOMAS AUGSPURGER, AND KAREN GAINES

1:45-2:00 **(64) EARED GREBE MORTALITY AT THE SALTON SEA: REVIEW OF FINDINGS AND NEW STUDIES.** J. CHRISTIAN FRANSON, LYNN H. CREEKMORE, CAROL U. METEYER, TONIE E. ROCKE, MARK J. WOLCOTT, AND TAHNI JOHNSON

2:00-2:15 **(65) MORTALITY OF THE COMMON LOON (*GAVIA IMMER*) IN NEW ENGLAND, 1988 TO 1999.** INGA F. SIDOR, MARK A. POKRAS, AND ROSE MICONI

2:15-2:30 **(66) DISTRIBUTION OF DUCK VIRUS ENTERITIS VIRUS DURING ACUTE INFECTION IN NATURALLY INFECTED DUCKS.** ELIZABETH W. HOWERTH, MOLLY MURPHY, BRADD C. BARR, CHARLOTTE QUIST, AND NANCY J. THOMAS

2:30-2:45 **(67) *MYCOPLASMA GALLISEPTICUM* FROM EVENING GROSBEAKS (*COCCOTHRAUSTES VESPERTINUS*) AND PINE GROSBEAKS (*PINICOLA ENUCLEATOR*) WITH CONJUNCTIVITIS IN QUEBEC, CANADA.** IGOR MIKAELIAN, DAVID H. LEY, R. CLAVEAU, AND M. LEMIEUX

2:45-3:00 **(68) SYSTEMIC *ISOSPORA*-LIKE COCCIDIOSIS IN A NORTHERN ORIOLE (*ICTERUS GALBULA*).** JEAN A. PARÉ AND SANDRA R. BLACK

3:00-3:15 **(69) MODELING AVIAN POX IN HAWAII.** CHARLES VAN RIPER, III, SANDRA G. VAN RIPER, AND WALLACE R. HANSEN

3:15-3:30 **(70) RESPIRATORY DISTRESS IN AN ADULT SHORT-EARED OWL.** CHRISTINE FIORELLO AND JOHN GLIATTO

3:30-5:00 **POSTER SESSION**

(71) CAUSES OF MORTALITY OF THE PUERTO RICAN PARROT (*AMAZONA VITATTA*). LAURIE A. BAETEN AND F. JOSHUA DEIN

(72) EXPERIMENTAL INFECTION OF MONTANA HOUSE FINCHES (*CARPODACUS MEXICANUS*) WITH THE HOUSE FINCH STRAIN OF *MYCOPLASMA GALLISEPTICUM*. KRISTY R. FARMER, SHARON R. ROBERTS, AND GEOFFREY E. HILL

(73) EVALUATION OF *BRUCELLA ABORTUS* VACCINE STRAIN RB51 IN PREGNANT REINDEER, A SAFETY STUDY. SUE HAGIUS, JOHN BLAKE. JULIA BEVINS-DUCE, GERHARDT SCHURIG, TODD FULTON, AND PHILIP ELZER

(74) PREVALENCE OF NEURONAL LIPIDOSIS (NEURONAL VACUOLATION) AND ANAL SAC CAPILLARIASIS IN RACCOONS (*PROCYON LOTOR*) FROM TWO GEOGRAPHICAL LOCATIONS IN THE USA. AMIR N. HAMIR AND JUDI STASKO

(75) THE FLUORESCENCE POLARIZATION ASSAY AND OTHER SEROLOGICAL ASSAYS FOR THE DETECTION OF ANTIBODIES TO *BRUCELLA ABORTUS* IN BISON IN SERUM AND WHOLE BLOOD. D. GALL, D.O. JOLY, P. SMITH, K. NIELSEN, L. FORBES, P. ELZER, AND S. OLSEN

(76) SURVEY FOR *BORRELIA* SPECIES AMONG RESERVOIR ANIMALS CAPTURED IN FORESTED AREAS OF GREATER METROPOLITAN CHICAGO. M. M. PICKEN AND R.N. PICKEN

(77) MECHANISMS OF SELENIUM-INDUCED TERATOGENESIS AND EMBRYOLETHALITY: OXIDATIVE STRESS. KATHY M. ORSTED, M. F. RAISBECK, D. A. SANCHEZ AND R. S. SIEMION

(78) AN EPIZOOTIC HEMORRHAGIC DISEASE OUTBREAK IN NEW JERSEY. DOUGLAS E. ROSCOE, DANIEL FERRIGNO, THOMAS R. BRIGGS, JANE E. HUFFMAN, AND DAVID STALLKNECHT

THURSDAY, JUNE 8

8:30-8:45 **ANNOUNCEMENTS**

OTHER MAMMALS
Moderator: Tom DeLiberto

8:45-9:00 **(79) DIVERSITY AND ECOLOGY OF BARTONELLA INFECTIONS IN RODENT COMMUNITIES.** MICHAEL Y. KOSOY, KENNETH L. GAGE, AND MARY EGGLESTON

9:00-9:15 **(80) PRESUMPTIVE PULMONARY MYCOPLASMOSIS IN CAPTIVE VANCOUVER ISLAND MARMOTS (*MARMOTA VANCOUVERENSIS*).** S. R. BLACK

9:15-9:30 **(81) POPULATION HEALTH CONCERNS FOR LOWLAND GORILLAS: ADDRESSING THE KNOWLEDGE GAP.** WILLIAM B. KARESH AND SHARON L. DEEM

9:30-9:45 **(82) PATHOLOGY AND MORTALITY IN THE SOUTHERN SEA OTTER (*ENHYDRA LUTRIS NEREIS)* POPULATION AS A RESULT OF PARASITIC INFECTIONS.** MURRAY D. DAILEY, KARL A. MAYER, AND MELISSA CHECHOWITZ

9:45-10:00 **(83) BASELINE HEALTH VALUES IN SEA OTTERS.** KRISTA D. HANNI, JONNA K. MAZET, FRANCES M. D. GULLAND, JIM ESTES, MICHELLE STAEDLER, MICHAEL J. MURRAY, AND DAVID A. JESSUP

10:00-10:10 **WYOMING WONDERS**

10:10-10:30 **BREAK**

OTHER MAMMALS
Moderator: Fleicia Nutter

10:30-10:45 **(84) LIFE CYCLE OF *OTOSTRONGYLUS CIRCUMLITUS* (METASTRONGYLOIDEA: CRENOSOMATIDAE) OF PHOCID SEALS.** L. N. MEASURES

10:45-11:00 **(85) *SARCOCYSTIS FALCATULA* DEVELOPMENT IN ITS NATURAL HOSTS.** E. C. GREINER, S. LUZNAR, R. PORTER, M. E. HEMENWAY, P .E. GINN, AND J. B. DAME

11:00-11:15 **(86) GENERALIZED, PRURITIC DERMATITIS, POSSIBLY ASSOCIATED WITH A HYPERSENSITIVITY REACTION, IN HAND-RAISED JUVENILE OPOSSUMS (*DIDELPHUS VIRGINIANA*).** CATHERINE M. BROWN

11:15-11:30 **(87) GENETICS OF NATURAL DISEASE RESISTANCE IN BIGHORN SHEEP.** KAREN M. RUDOLPH, TRICIA L. HOSCH, JOE W. TEMPLETON, AND DAVID L. HUNTER

11:30-11:45 **(88) *PARELAPHOSTRONGYLUS ODOCOILEI* AND *PROTOSTRONGYLUS STILESI* IN DALL'S SHEEP: PREDISPOSING FACTORS FOR MORTALITY?** SUSAN KUTZ, ALASDAIR VEITCH, EMILY JENKINS, BRETT ELKIN, ERIC HOBERG, MANUEL CHIRINO-TREJO, LYDDEN POLLEY, AND TRENT BOLLINGER

11:45-12:00 **(89) A MODEL FOR INVESTIGATING THE DEVELOPMENT, TRANSMISSION, AND RESPONSE TO CLIMATE CHANGE OF PROTOSTRONGYLID PARASITES ON THE ARCTIC TUNDRA.** SUSAN KUTZ, LYDDEN POLLEY, AND ERIC HOBERG

12:00-1:00 **LUNCH**

OTHER MAMMALS
Moderator: Catherine Soos

1:00-1:15 **(90) NOTOEDRIC MANGE IN WESTERN GRAY SQUIRRELS FROM WASHINGTON**. TODD E. CORNISH, MARY J. LINDERS, SUSAN E. LITTLE, AND W. MATTHEW VANDER HEAGEN

1:15-1:30 **(91) PERSISTENCE AND SAFETY OF PARENTERALLY DELIVERED IOPHENOXIC ACID AS A SEROMARKER IN BISON (*BISON BISON*).** STEVEN J. SWEENEY, THOMAS J. ROFFE, KENNETH W. COFFIN, MARK L. DREW, AND ROBERT H. POPPENGA

ORGANIZATIONS, PROGRAMS, INVESTIGATIONS, AND METHODS

1:30-1:45 **(92) SEROLOGY FOR SELECTED VIRUSES, BACTERIA, AND PROTOZOA IN FREE-RANGING ANACONDAS (*EUNECTES MURINUS*) IN VENEZUELA.** PAUL P. CALLE, JOHN THORBJARNARSON, WILLIAM HOLMSTROM, WILLIAM B. KARESH, JESÚS RÍVAS, AND MARÍA MUÑOZ

1:45-2:00 **(93) DEVELOPMENT OF A FISH AND WILDLIFE HEALTH PROGRAM FOR THE MARYLAND DEPARTMENT OF NATURAL RESOURCES.** CINDY P. DRISCOLL, SUSAN KNOWLES, BRENDA KIBLER, BRETT COAKLEY, KELLY GREENHAWK, AND TRICIA LITWILER

2:00-2:15 **BREAK**

ORGANIZATIONS, PROGRAMS, INVESTIGATIONS, AND METHODS
Moderator: Lauren Richey

2:15-2:30 **(94) NATURAL HISTORY STRATEGIES OF MICROORGANISMS CAUSING DISEASES OF WILDLIFE.** RICHARD G. BOTZLER AND RICHARD N. BROWN

2:30-2:45 **(95) WATEFOWL HEALTH AND MANAGEMENT: THE FIRST WILD*PRO* MODULE.** F. JOSHUA DEIN, SUZANNE I. BOARDMAN, AND DEBRA BOURNE

2:45-3:00 **(96) WILDLIFE HEALTH CAPACITY BUILDING IN SOUTH AMERICA.** MARCELA M. UHART, WILLIAM B. KARESH, AND SHARON L. DEEM

3:00-3:15 **(97) RADIOGRAPHS - AN ESSENTIAL TOOL FOR FORENSIC INVESTIGATIONS INVOLVINGWILDLIFE MORTALITY INVESTIGATIONS.** RICHARD K. STROUD AND RHODA RALSTON

3:15-3:30 **(98) ALBERTA'S DRAFT IMPORT PROTOCOLS FOR GAME FARM CERVIDS: A WORK IN PROGRESS.** M. J. PYBUS AND D. K.ONDERKA

3:30-3:45 **CLOSING REMARKS AND ADJOURN**

(1) BOVINE TUBERCULOSIS IN THE KRUGER NATIONAL PARK.

ROY G. BENGIS, and DEWALD F. KEET, Veterinary Investigation Centre – Kruger National Park, South Africa.

INTRODUCTION

Bovine tuberculosis (BTB) was first diagnosed in the Kruger National Park (KNP) in an emaciated, moribund, 2 year old buffalo bull, in 1990. This disease had not previously been confirmed in this Park, although a single case of Mycobacteriosis (uncultured – thought to be avian) was described in an impala by De Vos in 1967.In addition, during a total pathology survey of 100 buffalo by Basson *et al* in 1966, two cases of non-fungal pyogranulomatous lymphadenitis were described in which no acid-fast organisms could be demonstrated.

In intensive follow-up surveys following the detection of the confirmed index buffalo case, bovine tuberculosis was found to be widespread in the southern buffalo herds in the KNP. Retrospective circumstantial evidence indicates that BTB probably entered across the southern boundary of the KNP in the late 1950's, from heavily infected cattle herds on several border farms in that area.

Heightened awareness during other *ad hoc* necropsies, succeeded in detecting BTB in several "spill over" species, including baboons, lions, cheetahs, greater kudus and a leopard. This paper describes the clinical and necropsy features in each species, as well as important and pertinent epidemiological aspects and determinants.

BOVINE TUBERCULOSIS IN AFRICAN BUFFALO (*Syncerus caffer)*

African buffalo are highly gregarious wild bovids which occur on the African savannahs in herds, frequently numbering in their hundreds. In the Kruger National Park, there are approximately 100 different herds distributed through this 20,000 square kilometer Park, and the average herd size is 270.

Clinical Signs and Necropsy Findings

In general, most infected buffalo are asymptomatic until advanced / miliary disease develops. In these advanced cases, progressive emaciation and persistent coughing are characteristic signs. On necropsy, most lesions are present in the lymph nodes of the head, tonsils as well as the lungs and associated thoracic nodes. This lesional pattern is indicative of an aerosol/ droplet mode of transmission. The lung lesions are poorly encapsulated with minimal calcification, indicating that buffalo are naïve hosts with a less effective immune response. The lesions progress to caseation followed by cavitation with liquifaction, making advanced cases highly infectious. Occasionally, lesions are found in the mesenteric nodes (probably organisms coughed up and swallowed), peripheral lymph nodes and other distal sites.

Epidemiological Aspects

BTB in buffalo appears to have no sexual predilection, and there is an age related increase in prevalence. In the KNP random sampling detected herd prevalences between 1% and 67%, with the highest prevalence in the southern region of the Park, and a definite decreasing gradient northwards. The facts that buffalo are highly susceptible gregarious bovids, and that transmission occurs via aerosol/droplet infection, in a density dependent , nose to nose situation, make buffalo an ideal maintenance host in this African ecosystem.

BOVINE TUBERCULOSIS IN GREATER KUDU (*Tragelaphus strepsiceros)*

Kudu are gregarious browsers, but herds seldom number more than 12 individuals. Herds are generally made up of adult cows, yearlings and calves, and are visited by males during the early winter rut. Males may be solitary, or join up to form small bachelor herds. Bovine tuberculosis has previously been described in greater kudu in the Eastern Cape Province of South Africa (Paine and Martinaglia, 1928, ; Thornburn and Thomas,1940), where, the disease was associated with the kudu sharing range with BTB infected cattle. The first case of bovine tuberculosis in greater kudu in the Kruger National Park was diagnosed in 1996. Subsequently, a total of nine cases have been confirmed, but a further 20 suspect cases have been reported by field staff and tourists.

Clinical Signs and Necropsy Findings

The kudu is the only African species in which BTB can easily be diagnosed at a distance. The obvious “mumps like” swellings involving the lymph nodes of the head and neck are easily visible, and frequently fistulous tracts are present, which drain the abscessed parotid lymph nodes. The lymphoid tissues of the head appear to be the site of primary infection, and it appears that the infection thereafter spreads to the lungs and other distal sites. Initially, the lung lesions are well circumscribed and have a raised plaque-like appearance, but in advanced cases, miliary disease of the lungs and pleura are seen. Lesions have also been detected in the mesenteric lymph nodes, liver, spleen and long bones. In advanced cases, coughing , emaciation and blindness has be seen.

Epidemiological Aspects

It would appear that horizontal transmission of infection between kudu is related to the development of open fistulas draining infectious material from the head nodes. Kudu are mainly browsers and in the dry months will frequently be seen pushing their heads into bushes and trees (often thorn trees of the genera *Acacia and Xiziphus)* and feeding on the remaining available green leaves. During these activities, leaves, branches and thorns become contaminated with pus, which in turn may scarify the delicate skin of the ears, resulting in primary lesions developing in the parotid lymph node of a herd cohort. Similarly, kudu also frequently have micro-scarifications of the pharyngeal and esophageal mucosae, suspected to be due to browsing of thorn trees, and following ingestion of contaminated browse, these may act as ports of entry for infection of the tonsils, retropharyngeal, mandibular or cervical lymph nodes.

Kudu with advanced pulmonary lesions are also probably highly infectious via the aerosol/droplet route.

Kudu would thus appear to have some maintenance host potential and in this scenario, infection would probably smoulder at a low prevalence, in this relatively low density species. They may however be an important source of incidental infection for lions, leopards and cheetahs.

BOVINE TUBERCULOSIS IN CHACMA BABOONS (*Papio ursinus)*

Both human and bovine tuberculosis are commonly encountered in captive primates, but are generally rare in free-ranging primates. A single troop of chacma baboons was found to be infected in the KNP. It is postulated that some members of this troop originally became infected by feeding on a tuberculous carcass in the veld, or scavenging tuberculous material from a nearby post mortem facility, prior to incineration. Subsequently, horizontal transmission occurred via aerosol and oral routes within the confines of a deserted pumphouse on the Skukuza railway bridge, which was opportunistically used as "safe" sleeping quarters by this troop. A disease prevalence of 50% was found in this troop, and the disease proved to have a fulminating course in baboons.

Clinical Signs and Necropsy Findings

Clinical signs included depression, emaciation and coughing. On necropsy, the spleen and mesenteric lymph nodes were consistently affected, but the lung lesions were the most severe, and comprised of multifocal to confluent granulomas, with caseation, liquifaction and cavitation present. Focal circumscribed lesions (tubercles) were also seen in the kidneys, liver, vertebrae and peripheral lymph nodes.

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Epidemiological Aspects

When the opportunistic sleeping quarters was closed down, and the troop forced to sleep in trees, the disease disappeared from the troop as infected members died or were destroyed. A survey, six months later revealed no further cases in the troop, and no "spill over" into neighboring troops.

BOVINE TUBERCULOSIS IN CARNIVORES

In September 1995, the first case of BTB in a lion in the Kruger National Park was recorded. This was also the first recorded case of BTB in a free-ranging lion. The disease has however been recorded in captive lions as well as other captive felids fed on condemned *M. bovis* infected cattle carcasses. In the same year, BTB was also recorded for the first time in a free-ranging cheetah, and in August 1998, the first case of BTB in a free- ranging leopard was confirmed.

Since the first case was detected in lions, more than 50 cases have been confirmed, mainly from the southern and south central areas of the KNP, which corresponds to the buffalo high and medium TB prevalence areas.

Clinical Signs and Necropsy Findings

The clinical signs in all three carnivore species include emaciation, weakness, poorly healing skin lesions,- and in a significant number of lions, hind-limb lameness and ocular lesions were observed.

On necropsy, the most consistent lesions are found throughout the lymphatic system, especially in the mesenteric lymph nodes, peripheral lymph nodes and head lymph nodes. The lesions in these nodes are both inflammatory and cavitational, but no caseation or calcification is present. In several lions and in two cheetahs well defined pulmonary nodules have been identified, but once again, on section these lesions donot show any caseation or calcification. These lesions consists of a consolidated inflammatory mass which are frequently cavitated, and contain small amounts of glistening mucoid pus. The macroscopic appearance of BTB in these felids does not look anything like the classical TB lesions seen in hoofstock and primates**.** A significant number of lions also develop tuberculous periostitis and / or osteomyelitis, frequently involving the proximal tibia. Panopthalmia was also seen in some cases. Tuberculous lesions were also seen in the mammary tissue of a few individuals.

Epidemiological Aspects

It would appear that these carnivores become infected by killing or scavenging on tuberculous buffalo or kudu. The primary infection route is probably by ingestion, but aerosol infection is a possibility while in the act suffocating an infected prey animal by biting through the airway or muzzle. Horizontal transmission between lion pride members (with pulmonary lesions) may occur while growling /snarling as they compete with each other over a carcass, or when violent interactions result in skin wounds caused by contaminated teeth or claws. It is however, unlikely that TB infection could be maintained in carnivores in the absence of an infected prey species, and therefore carnivores are probably incidental, “dead end” hosts.

Discussion

The bovine tuberculosis outbreak in the Kruger National Park is probably the most threatening disease to enter this ecosystem since the decimating rinderpest epidemic of 1898 – 1902. However, unlike rinderpest, BTB is a chronic, slowly progressive disease that is not highly visible, and most infected individuals are asymptomatic until the disease reaches an advanced stage. Sensitive and specific *ante mortem* tests are only available for a few species, and this constitutes a regulatory dilemma in translocation exercises. This huge free-ranging potentially infectious source is also a nightmare for Veterinary authorities in a country with an active and effective BTB eradication scheme for cattle. The current available options to attempt to contain, control and in the long term eradicate BTB from the KNP are both drastic and controversial, and are considered to be morally and ecologically unacceptable by many conservationists. These options include: The large-scale slaughter out of all infected buffalo herds followed by re-introduction of BTB-free buffalo at a later date.

The creation of a buffalo exclusion *zona sanitaire* to separate the southern infected herds from the northern, relatively clean herds, and to extirpate any infected herds in the north. This may or may not include the erection of a double fence across the entire Park. The possibility of a southward roving buffalo-free zone (unfenced) with natural re-colonization from the north has also been considered.

The two important questions central to this highly emotive issue are: Whether other maintenance hosts exist in the ecosystem, which would complicate this buffalo directed eradication campaign. Will BTB impact on the biodiversity of the Park. Can one afford to wait and see.

The single long-term intervention and hope that would be acceptable to all parties would be the development of an effective vaccine that could be aerosolized in the proximity of buffalo herds (by crop-spraying helicopter), or the development of an antigenically suitable, self replicating recombinant vaccination agent, that could be used to infect and immunize the herds.

(2) BOVINE TUBERCULOSIS IN MICHIGAN – AN UPDATE.

STEPHEN M. SCHMITT, Wildlife Disease Laboratory, Rose Lake Wildlife Research Station, Michigan Department of Natural Resources, East Lansing, Michigan 48823; SCOTT D. FITZGERALD, Animal Health Diagnostic Laboratory and Department of Pathology, College of Veterinary Medicine, Michigan State University, East Lansing, Michigan 48824; COLLEEN S. BRUNING-FANN, Veterinary Services, Animal and Plant Health Inspection Service, United States Department of Agriculture, Holt, Michigan 48842; NATHAN ZAUEL, Animal Industry Division, Michigan Department of Agriculture, Lansing, Michigan 48909; and DALE E. BERRY, Bureau of Infectious Disease Control, Michigan Department of Community Health, Lansing, Michigan 48909.

Since 1994, the State of Michigan has recognized a problem with bovine tuberculosis (TB), caused by *Mycobacterium bovis*, in the wild white-tailed deer from a six-county area in northeastern lower Michigan. The disease has been found in other wildlife species and, in 1998, in domestic cattle. Recognizing the potential economic and public health consequences of *M. bovis* to the state, the governor has issued orders to eradicate *M. bovis* from the state's deer population. Unfortunately, the situation is unique in that there have never been reports of self-sustaining bovine TB in a wild, free-ranging cervid population in North America. There are no existing control programs for TB in wild deer, and there is much about TB in deer that is currently unknown. Scientists, biologists, epidemiologists, and veterinarians that have studied this situation have concluded that the most logical theory is that the supplemental feeding and baiting (the practice of hunting deer over feed) of wild deer serves to congregate deer, therefore contributing to the spread of TB. Supplemental feeding and baiting have been banned since 1998 in the area where bovine tuberculosis has been found with the intention of reducing the spread of TB between deer and eventually eliminating this disease from the wildlife, therefore completing the eradication. In addition, deer densities are being reduced through hunting with unlimited antlerless permits available during the 1998 and 1999 hunting seasons. Prevalence rates of bovine tuberculosis in free-ranging Michigan white-tailed deer have been declining since 1997.

(3) DEER TO DEER TRANSMISSION OF *MYCOBACTERIUM BOVIS.*

MITCHELL V. PALMER and DIANA L. WHIPPLE, Bacterial Diseases of Livestock Research Unit, National Animal Disease Center, Agricultural Research Service, USDA, 2300 Dayton Avenue, Ames, IA 50010.

In 1994 a free-ranging white-tailed deer (*Odocoileus virginianus*) in Michigan was diagnosed with tuberculosis due to *Mycobacterium bovis*. Subsequent surveys in northeast Michigan have identified the first known epidemic of tuberculosis in white-tailed deer. Information is lacking on the pathogenesis and transmissibility of *M. bovis* infection in white-tailed deer. In order to determine the efficiency with which deer transmit tuberculosis to each other, and the routes by which such transmission occurs we exposed non-inoculated deer to experimentally inoculated deer. Eight deer were experimentally inoculated by intratonsilar instillation of $2x10^8$ CFU of *M. bovis*. Eight non-inoculated deer were introduced 21 days after inoculation. Deer were housed in pens l5O $feet^2$ in size such that 2 in-contact deer were penned with 2 experimentally inoculated deer. Each pen had a single source of water, hay, and pelleted feed. Sixty-nine days after introduction, all in-contact deer developed delayed type hypersensitivity reactions to *M. bovis* PPD as determined by the comparative cervical test. One hundred and twenty days after inoculation all experimentally inoculated deer were removed. One hundred and fifty nine days after introduction, 4 in-contact deer were euthanized and examined and 4 new non-inoculated deer were housed with the remaining original in-contact deer such that 4 new in-contact deer were housed with 4 original in-contact deer. One hundred days after introduction, all new in-contact deer had developed delayed type hypersensitivity to *M. bovis* PPD. At 180 days after introduction of new in-contact deer all deer were euthanized and examined. All in-contact exposed deer developed tuberculosis. Lesions were most commonly seen in the lung, tracheobronchial and mediastinal lymph nodes. Experimentally inoculated deer were shown to shed *M. bovis* in nasal secretions, saliva, feces, and urine. In-contact infected deer also shed *M. bovis* in nasal secretions and saliva. Hay and pelleted feed were found to contain *M. bovis* at multiple times throughout the experiment. This study shows that tuberculous deer efficiently transmit *M. bovis* to other deer in close contact. Lesion distribution in in-contact exposed deer suggests aerosol transmission as a likely means of infection, however, contamination of shared feed must also be considered. Body fluids containing *Mycobacterium bovis* may become aerosolized or directly contaminate feed, both of which may be sources of infection for other susceptible hosts.

(4) SURVIVAL OF *MYCOBACTERIUM BOVIS* ON FEEDS USED FOR BAITING WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*) IN MICHIGAN.

DIANA L. WHIPPLE, Bacterial Diseases of Livestock Research Unit, National Animal Disease Center, United States Department of Agriculture, Agricultural Research Service, Ames, IA 50010; MITCHELL V. PALMER, Bacterial Diseases of Livestock Research Unit, National Animal Disease Center, United States Department of Agriculture, Agricultural Research Service, Ames, IA 50010.

Free-ranging white-tailed deer (*Odocoileus virginianus*) in northeast Michigan are recognized as a wildlife reservoir of tuberculosis caused by *Mycobacterium bovis.* Generally, animals become infected with *M. bovis* by inhalation of aerosolized organisms or by ingestion of organisms that are present in feed and water. *Mycobacterium bovis* has been isolated from saliva, nasal secretions, and tonsilar swab samples of experimentally infected white-tailed deer. Therefore, it is possible for infected deer to shed organisms in oral secretions and contaminate feed and water, which would then serve as a source of infection for other animals. Baiting of deer, which is allowed in Michigan, creates a situation where several deer eat from the same pile of feed and may contribute to transmission of tuberculosis. The purpose of this study was to determine how long *M. bovis* survives on various feeds when stored at different temperatures. The feeds examined were alfalfa hay, shelled corn, sugar beets, apples, carrots, and potatoes. Feeds were held at 75° F, 46° F, and 0° F for 2 hours, 1 day, 2 days, 3 days, and 7 days and 2 weeks, 3 weeks, 4 weeks, 8 weeks, 12 weeks, and 16 weeks. *Mycobacterium bovis* was isolated from all feeds stored at all temperatures for 7 days. At 46° F, *M. bovis* survives on all feeds except carrots for at least 12 weeks and at 0° F, it survives on all feeds for at least 16 weeks. Other experiments will be conducted to further examine the role of baiting in transmission of tuberculosis.

(5) TRANSMISSION OF *MYCOBACTERIUM BOVIS* FROM EXPERIMENTALLY INFECTED WHITE-TAILED DEER (*ODOCOILEUS VIRGINIANUS*) TO CATTLE THROUGH INDIRECT CONTACT.

DIANA L. WHIPPLE, Bacterial Diseases of Livestock Research Unit, National Animal Disease Center, United States Department of Agriculture, Agricultural Research Service, Ames, IA 50010; MITCHELL V. PALMER, Bacterial Diseases of Livestock Research Unit, National Animal Disease Center, United States Department of Agriculture, Agricultural Research Service, Ames, IA 50010.

Mycobacterium bovis has been isolated from herds of cattle in northeast Michigan where there is a wildlife reservoir of disease in free-ranging white-tailed deer (*Odocoileus virginianus*). Results of DNA fingerprinting indicate that the deer and cattle are infected with the same strain of *M. bovis*. The purpose of this study was to determine if cattle can become infected with *M. bovis* through indirect contact with experimentally infected white-tailed deer. Three groups of four deer were inoculated with 7×10^5 *M. bovis* by instillation of the organisms into the crypt of the palatine tonsil. After two weeks, pens where the deer were housed were topically disinfected and three groups of three six-month old calves were introduced into the barn. Each group of deer was paired with a group of calves. Deer were given excess feed and hay and allowed access to it for several hours. The deer were then moved to a holding pen and the calves were moved to the pen that had been occupied by the deer without cleaning the pen. The calf pens were cleaned and the deer were then moved to the clean pens. This process was repeated daily for 80 days. All of the deer were euthanized by day 91 of the experiment and all had extensive lesions of tuberculosis. On day 77, all of the calves were skin tested using the comparative cervical skin test and were classified as reactors. Results of the interferon gamma assay (BOVIGAM[7])were positive for three of the calves on day 28 and were positive for all nine calves on day 56. Calves were necropsied beginning on day 177 and examined for lesions of tuberculosis. Gross and microscopic lesions were observed and *M. bovis* was isolated from all calves. All calves had lesions in the lung or associated lymph nodes. One calf had lesions in the medial retropharyngeal lymph node. Results of this study show that calves can become infected with *M. bovis* through indirect contact with experimentally infected white-tailed deer. Additional studies will be conducted to determine if cattle can become infected through contact with only the feed that has been offered to experimentally infected deer. We also plan to determine the minimum infectious dose needed for infection of calves by the oral route.

(6) BRUCELLOSIS IN ELK (*CERVUS ELAPHUS*) OF EASTERN IDAHO.

MARK L. DREW, Wildlife Health Laboratory, Idaho Department of Fish and Game, Caldwell, ID 83607.

Brucellosis, due to *Brucella abortus*, is endemic in bison (*Bison bison*) and elk (*Cervus elaphus*) the Greater Yellowstone area located Wyoming, Montana and Idaho. Beginning in 1998, elk were sampled in eastern Idaho and tested for brucellosis. In 1998, 64 elk were tested in 3 locations; 11 (17.2%) were seropositive. In 1999, 111 elk were tested in 1 location; 64 (57%) were seropositive. A total of 23 adult female and 9 calves were euthanized and samples taken for culture. Eleven (48%) of the 23 adult females were culture positive and three of 9 (33%) of the calves born to these cows were culture positive. Two biovars of *Brucella abortus* were isolated from these animals, biovar 1 (n=5) and biovar 4 (n=9). In 2000, 49 elk were tested in two locations; 20 (40.8%) were seropositive. All seropositive animals were euthanized and samples taken for culture, but results are still pending. In addition, sampling kits were sent to elk hunters in controlled hunt zones in several parts of Idaho. In the known infected area, 1000 kits were sent out in 1998 and 900 in 1999. In 1998, a total of 183 kits were returned with 173 useable samples; 10 samples were seropositive (6.8%). A total of 56 kits were returned in 1999 with 46 useable samples; 4 samples were seropositive (9.5%). Based on the data collected to date, brucellosis is present in a high percentage of the elk in a small area of eastern Idaho. The presence of *B. abortus* biovar 4 in a large proportion of these animals makes the situation in Idaho unique from that in Wyoming. Management efforts have concentrated on reducing populations of elk, reducing winter feeding operations, and mandatory vaccination and testing of cattle herds in the area.

(7) ELK (*CERVUS ELAPHUS NELSONI*) BRUCELLOSIS SURVEILLANCE IN WESTERN WYOMING.

SCOTT G. SMITH, Wyoming Game and Fish Department, Pinedale, WY 82941; E. TOM THORNE, Wyoming Game and Fish Department, Cheyenne, WY 82006; WALTER E. COOK, HANK EDWARDS, TERRY KREEGER, Wyoming Game and Fish Department, Research Laboratory, Laramie, WY 82071.

Brucellosis (*Brucella abortus)* has been documented in elk throughout Wyoming's portion of the Greater Yellowstone Area. The highest prevalence is reported in elk that utilize supplemental feeding areas during winter months. The Wyoming Game and Fish Department (WGFD) uses two surveillance techniques to assess the distribution and prevalence of brucellosis exposed elk, establish priorities for management efforts, and evaluate management activities.

Elk that utilize supplemental winter feed areas are surveyed by live trapping individuals in permanent corral traps. Blood is collected by jugular venipuncture while elk are free-standing in chutes. Elk that are not fed during winter months are surveyed through the collection of hunter-killed blood samples. Hunters who have been issued antlerless elk permits are mailed a blood sampling kit prior to opening of the season. Sampling kits contain a blood tube, instructions, and postage-paid mailer. Samples are mailed directly to the WGFD Wildlife Disease Laboratory, Laramie, WY for testing. Sera are tested for *Brucella abortus* antibodies using standard procedures of U.S. Department of Agriculture/Animal and Plant Inspection Service. Tests include standard plate agglutination test (SPT), buffered *Brucella* antigen rapid card test (BBA), rivanol test (Riv), and complement fixation test (CFT). Elk sera reacting to two or more tests are considered positive. In addition, a competitive enzyme linked immunosorbent assay (cELISA) test is used to identify vaccine induced titers.

Between 1971 and 1999, over 3000 adult female elk were tested at 18 supplemental feeding areas. Seroprevalence for fed elk over the 29 year period averaged 34%. From 1990 to 1998, over 2900 adult female elk were tested in non-fed areas. Seroprevalence for these elk averaged 2%. These data indicate elk that winter on native ranges without supplemental winter feed do not maintain brucellosis at high levels thus management efforts have been focused on herds with winter feeding. Since 1985, remote delivery of strain 19 vaccine to juvenile elk is one management tool used to address brucellosis in supplementally fed elk.

(8) LESIONS AND TISSUE COLONIZATION SITES OF *BRUCELLA ABORTUS* IN ABORTED BISON FETUSES, BISON CALVES, AND ADULT FEMALE BISON FROM YELLOWSTONE NATIONAL PARK.

JACK C. RHYAN, U.S. Department of Agriculture, Animal and Plant Health Inspection Service, Veterinary Services, National Wildlife Research Center, 4101 Laporte Avenue, Fort Collins, Colorado 80521; THOMAS GIDLEWSKI, U.S. Department of Agriculture, Animal and Plant Health Inspection Service, Veterinary Services, National Veterinary Services Laboratories, P.O. Box 844, Ames, Iowa 50010; THOMAS J. ROFFE, U.S. Department of the Interior, U.S. Geological Survey, Biological Resources Division, Box 173220, Bozeman, Montana 59717; KEITH AUNE, Montana Department of Fish, Wildlife and Parks, Research and Technical Services Bureau, Box 173220, Bozeman, Montana 59717; L. MICHAEL PHILO, U.S. Department of Agriculture, Animal and Plant Health Inspection Service, Veterinary Services, Western Region, 9439 Owl Way, Bozeman, Montana 59718; and DARLA R. EWALT, U.S. Department of Agriculture, Animal and Plant Health Inspection Service, Veterinary Services, National Veterinary Services Laboratories, P.O. Box 844, Ames, Iowa 50010.

Between February 1995 and June 1999, specimens from seven aborted bison fetuses or stillborn calves and their placentas, two additional placentas, three dead neonates, one 2-week-old calf, and 35 juvenile and adult female bison from Yellowstone National Park were submitted for bacteriologic and histopathologic examination. One adult animal with a retained placenta had recently aborted. Serum samples from the 35 juvenile and adult bison were tested for *Brucella* antibodies. Twenty-six bison, including the cow with the retained placenta, were seropositive, one was suspect, and eight were seronegative. *Brucella abortus* was isolated from three aborted fetuses and associated placentas, an additional placenta, the 2-week-old calf, and 12 of the seropositive female bison including the animal that had recently aborted. The organism was recovered from numerous tissue sites from the aborted fetuses, placentas and 2-week-old calf. In the juvenile and adult bison, *B. abortus* was most often isolated from supramammary lymph nodes (ten bison), retropharyngeal lymph nodes (eight bison), and iliac lymph nodes (seven bison). Cultures from the seronegative and suspect bison were negative for *B. abortus*.

Lesions in the *B. abortus*-infected, aborted placentas and fetuses consisted of necropurulent placentitis and mild bronchointerstitial pneumonia. The infected 2-week-old calf had bronchointerstitial pneumonia, focal splenic infarction, and purulent nephritis. The recently-aborting bison cow had purulent endometritis and necropurulent placentitis. Immunohistochemical staining of tissues from the culture positive aborted fetuses, placentas, 2-week-old calf, and recently-aborting cow disclosed large numbers of *B. abortus* in placental trophoblasts and exudate, and fetal and calf lung.

A similar study with the same tissue collection and culture protocol was done using six seropositive cattle from a *B. abortus*-infected herd in July and August, 1997. Results of the bison and cattle studies were similar.

(9) EFFICACY OF *BRUCELLA ABORTUS* STRAIN RB51 VACCINE IN CAPTIVE ELK: A PRELIMINARY REPORT.

TERRY J. KREEGER, WILLIAM H. EDWARDS, WALTER E. COOK, Wyoming Game and Fish Department, 2362 Highway 34, Wheatland, WY 82201; STEVEN C. OLSEN, Zoonotic Disease Research Unit, National Animal Disease Center, Agricultural Research Service, U. S. Department of Agriculture, 2300 Dayton Ave., Ames, IA 50010; PHILIP H. ELZER, Department of Veterinary Science, Louisiana State University, 111 Dalrymple, Baton Rouge, LA 70803.

Free-ranging elk (*Cervus elaphus*) calves were trapped at the National Elk Refuge, Jackson, Wyoming and transported to the Sybille Wildlife Research Unit in Wheatland, Wyoming in 1998. Elk were divided into three groups. Group 1 (n = 16) received a single calfhood vaccination of 10^{10} CFU *B. abortus* strain RB51; Group 2 (n = 14) received a calfhood vaccination of 10^{10} RB51 followed by a booster dose of 10^{10} RB51 one year later; Group 3 (n = 17) served as non-vaccinated controls. All elk seroconverted to RB51. Elk were bred in the fall of 1999 and all were determined pregnant based on pregnancy-specific protein analysis. Elk were challenged on March 15, 2000 with 10^{7} CFU strain 2308 administered intraconjunctivally in a split dose. Elk began aborting on April 15. As of this abstract preparation, more than 50% of all elk in all three groups have aborted. Based on these preliminary results, RB51 vaccine does not appear to protect elk against brucellosis induced abortion.

(10) IMMUNE RESPONES AND EFFICACY OF *BRUCELLA ABORTUS* STRAIN RB51 VACCINE IN BISON (*BISON BISON*).

STEVEN C. OLSEN and M.V. PALMER, Bacterial Diseases Research Unit, USDA, ARS, National Animal Disease Center, Ames, IA 50010.

Brucellosis is caused by an intracellular bacteria and the disease is characterized by abortion and fetal losses. *Brucella abortus* is known to be prevalent in bison populations within Yellowstone National Park (YNP) and Grand Teton National Park in the U.S., and Mackenzie Bison Sanctuary in Canada. In a series of studies we have vaccinated bison heifer calves with *B. abortus* strain RB51 (SRB51) and characterized persistence of the vaccine strain, immune respones after vaccination, and vaccine-induced protection against experimental challenge with virulent *B. abortus* during pregnancy. SRB51 can be recovered from the lymph node draining the site of vaccination (superficial cervical) in some bison for up to 18 weeks after vaccination. Vaccinated bison do not appear to shed SRB51 from mucosal surfaces after vaccination. When compared to nonvaccinates, bison vaccinated with SRB51 have antibody responses that peak between 4 to 6 weeks after vaccination and dissipate by 18 weeks after vaccination. Proliferative respones of peripheral blood mononuclear cells to killed SRB51 bacteria are greater than nonvaccinates at 12 weeks after vaccination. Bison were pasture bred, pregnancy confirmed by rectal palpation, and pregnant bison intraconjunctivally challenged with 1 x 10^7 CFU of virulent Brucella abortus strain 2308 at 180 days gestation. Dams and calves were euthanized within 24 hours after parturition or abortion and samples obtained for histologic and bacteriologic examination.

(11) BRUCELLOSIS IN DOMESTIC PIGS AND WILDBOARS DUE TO *BRUCELLA SUIS* BIOVAR 2 IN FRANCE.

B. GARIN-BASTUJI, AFSSA Alfort, OIE/FAO Reference Laboratory for Brucellosis, BP 67, France – 94703 Maisons-Alfort cedex; J. HARS, Office National de la Chasse, Direction Recherche et Développement, 8 Impasse Champ Fila, France - 38320 Eybens; D. CALVEZ, AFSSA Alfort, OIE/FAO Reference Laboratory for Brucellosis, BP 67, France – 94703 Maisons-Alfort cedex; M. THIEBAUD, AFSSA Alfort, OIE/FAO Reference Laboratory for Brucellosis, BP 67, France – 94703 Maisons-Alfort cedex; M. ARTOIS, AFSSA Nancy, BP 9, France - 54220 Malzéville.

Brucellosis, a bacterial zoonosis still common in domestic ruminants in southern Europe, had disappeared from French breeding pig population since 1981. From 1993 to 1999, 17 outbreaks were identified in open-air pig units in 16 *départements. Brucella suis* biovar 2, which had been previously identified only in brown hares in France, has been isolated from 15 of these outbreaks. In all cases high levels of antibodies and a high proportion of animals presenting clinical signs were observed. In 1993, *B suis* biovar 2 *was* also isolated from the testes of a hunted wild boar suffering from orchitis. Since that date, surveys performed in wild boars, hunted or road casualties, in several *départements* (Ardennes, Charente, Cher, Creuse, Eure, Côte d'Or, Moselle, Tarn, Yonne), gave an important rate of anti-Brucella serological reactions. Prevalence rates varied from 20 to 35% according to the region or the test used (RBTP, CFT, or ELISA). Due to the lack of specificity of brucellosis serological tests in pigs, bacteriological examinations were performed simultaneously on the spleen, in four of these surveys. The results confirmed the presence of *B suis* biovar 2 in about 10% of animals, while *Yersinia enterocolitica* O:9, a micro-organism known as the most implicated in serological cross-reactions, was never isolated. Since open-air pig farms and wild boar populations are increasing from year to year in France (350 % increase in 10 years for wild boar population), and since the occasionnal presence of wild boars within the farm and obvious crossed reproduction have been evidenced in several cases, the involvement of wild boars in the emergence of porcine brucellosis in France is highly suspected. However, since the true prevalence rate of brucellosis in hares is not known, their potential complementary role should be investigated. Anyway, this new form of porcine brucellosis raises the question of the opportunity of developing open-air pig breeding in the absence of possibility to isolate the farmed animals from contact with rapidly extending wild boar populations.

(12) PATHOGENESIS OF CHRONIC WASTING DISEASE IN ORALLY EXPOSED MULE DEER (*ODOCOILEUS HEMIONUS*): PRELIMINARY RESULTS.

ELIZABETH S. WILLIAMS, Department of Veterinary Sciences, University of Wyoming, Laramie, WY 82070; and MICHAEL W. MILLER, Colorado Division of Wyoming, Fort Collins, CO 80523.

Twenty approximately 5 month old mule deer fawns (*Odocoileus hemionus*) were obtained from a chronic wasting disease (CWD)-free herd and orally exposed to 5 g of pooled brains from mule deer with CWD. Inoculated deer were observed for development of clinical signs of CWD and two principles and two control deer (from the CWD-free herd) were euthanized sequentially from 3 months to 28 months postinoculation. PrP^{res}, the abnormal isoform of the prion protein, was detected in cervical lymph nodes by immunohistochemistry at 3 months postinoculation; distribution of PrP was widespread in lymphoid tissues by 6 months postinoculation and remained so throughout the study. Control tissues were negative. PrP^{res} was first detected in the brain at the lateral aspect of the parasympathetic vagal nucleus in the medulla oblongata 6 months postinoculation. Spongiform encephalopathy was found in this nucleus at 16 months postinoculation. Development of spongiform encephalopathy coincided with early manifestations of clinical chronic wasting disease.

(13) CHRONIC WASTING DISEASE IN ELK (*CERVUS ELAPHUS NELSONI*) HELD IN A CWD ENDEMIC FACILITY.

ELIZABETH S. WILLIAMS, Department of Veterinary Sciences, University of Wyoming, Laramie, WY 82070; WALTER E. COOK, HANK EDWARDS, TERRY KREEGER, Wyoming Game and Fish Department, Research Laboratory, Laramie, WY 82071; and SCOTT SMITH, Wyoming Game and Fish Department, Pinedale, WY 82941.

Fifty-seven female elk calves were captured on the National Elk Refuge, Teton County, Wyoming in February, 1995 and transported to the Sybille Wildlife Research and Conservation Education Unit near Wheatland, Wyoming. Chronic wasting disease (CWD) is endemic in deer and elk in this facility. Chronic wasting disease has never been diagnosed in the Jackson Herd and tests of over 700 harvested adult elk have been negative. The elk calves were involved in a study of the efficacy of *Brucella abortus* strain RB51 vaccine; 33 were vaccinated by hand or ballistic implant. They were held in groups according to vaccination status in paddocks that had been used to house cervids for many years. These elk were not known to have had direct contact with elk or deer with CWD, though fence-line contact was possible. Between 19 and 28 months after moving into the facility, five elk developed clinical CWD. These animals had spongiform encephalopathy and were positive for accumulation of PrP^{res} in the brain by immunohistochemistry. As part of the vaccination trial protocol, the remaining elk were necropsied 27 to 31 months after moving to Sybille. Three of these elk had spongiform encephalopathy and were positive for PrP by immunohistochemistry and an additional three elk were positive by immunohistochemistry alone. Of the 54 elk that lived for 1 year or more after introduction into the research facility, 11 (20%) developed clinical or subclinical CWD within 31 months. Exposure to the CWD prion apparently was only from environmental sources or possible fence-line contact with affected cervids.

(14) MECHANISMS FOR CHRONIC WASTING DISEASE TRANSMISSION: CLUES FROM INFORMATION-BASED COMPARISON OF COMPETING TRANSMISSION MODELS.

MICHAEL W. MILLER, Colorado Division of Wildlife, Wildlife Research Center, Ft. Collins, CO.

Chronic wasting disease (CWD), a transmissible spongifom encephalopathy (TSE), occurs naturally in both captive and free-ranging deer (*Odocoileus* spp.) and elk (*Cervus elaphus nelsoni*). Although CWD is clearly a transmissible disease, mechanisms underlying its natural transmission remain undescribed. I used information-based model selection methods to compare four models for CWD transmission in mule deer (O. hemionus): maternal (dam-offspring) transmission; direct (lateral) transmission without latency; direct transmission with latency; and indirect (environmental) transmission with latency. Cumulative numbers of CWD cases forecast by each model were compared to an epidemic curve from a naturally-infected captive mule deer herd (n = 84 individuals) maintained by the Colorado Division of Wildlife between June 1992 and May 2000. I constrained parameter ranges and obtained maximum likelihood estimates of relevant parameters, then compared performance of respective models using Akaike's information criterion adjusted for small sample size (AICc). Of the models compared, the indirect transmission with latency model was strongly supported by epidemic data from captive mule deer (wr = 0.883). The direct transmission with latency model was only marginally supported by observed data (DAICc = 4.05, wr = 0.117), and the other two models enjoyed essentially no support from the data (wr * 3*10-7). Based on these findings, there appears to be a strong possibility that indirect transmission of CWD via contaminated environments occurs. It follows that indirect transmission should be considered in developing disease management strategies for both captive and free-ranging cervids.

(15) PREVALENCE OF *TOXOPLASMA GONDII* ANTIBODIES IN CARIBOU (*RANGIFER TARANDUS*) AND MUSKOX (*OVIBOS MOSCHATUS*) SERA FROM NORTHERN CANADA.

BRETT ELKIN, Wildlife & Fisheries Division, Government of the NWT, 600, 5102 - 50 Ave, Yellowknife, NT. X1A 3S8; SUSAN KUTZ, Department of Veterinary Microbiology, Western College of Veterinary Medicine, University of Saskatchewan, 52 Campus Drive, Saskatoon, SK S7N 5B4; and J.P. DUBEY, Parasite Biology and Epidemiology Laboratory, Livestock and Poultry Sciences Institute, United States Department of Agriculture, Agricultural Service, Beltsville, MD 20705-2350.

ABSTRACT: Prevalence of antibodies to *Toxoplasma gondii* was examined in muskoxen (*Ovibos moschatus*) and barrenground caribou (*Rangifer tarandus*) from northern Canada. A total of 153 caribou serum from 5 separate herds and 204 muskox sera from 3 geographically distinct regions were tested by the modified agglutination test (MAT). Antibodies were found in 44 (28.2%) of the caribou sera with MAT titers of 1:25 in 11, 1:50 in 24, and >1:500 in 9. Seven of 9 caribou with MAT titers of >1:500 were adult females from the 3 mainland caribou herds. No antibodies were detected in the Dolphin and Union herd from south Victoria Island, and only 1 of 24 caribou from the north Baffin Island population had detectable antibodies. In the muskoxen, antibodies were found in 15 (7.4%) of the animals with MAT titers of 1:25 in 3, 1:50 in 8, 1:200 in 2, 1:400 in 1, and 1:800 in 1. The 4 muskoxen with MAT titers of 1:200 or greater were adult females from the mainland population near Kugluktuk. Serological reactors were found primarily in mainland populations of both species, with significantly lower prevalence in island populations. The only wild felid found in northern Canada that could serve as an intermediate host is the Canadian Lynx (*Lynx canadensis*), which overlaps in range with the mainland caribou and to some degree with the mainland muskox populations, but not the island populations of either species. The epidemiology and clinical significance of toxoplasmosis in free-ranging populations are unknown, though *T. gondii* has been shown to cause clinical disease in captive and free-ranging arctic ungulates including muskoxen and reindeer. *Toxoplasma gondii* is an important zoonotic agent, and the potential public health implications of its presence in these important country food species needs to be evaluated further.

(16) ATTEMPTS TO TREAT VERMINOUS PNEUMONIA AND ASSOCIATED HAIR LOSS IN FREE RANGING BLACK TAIL DEER.

P. BRIGGS HALL, Washington State Department of Fish and Wildlife. 16018 Mill Creek Blvd, Mill Creek WA 98012-1296; and LOUIS C. BENDER, Washington Department of Fish and Wildlife, 2950 Ley Road, Cle Elum, WA 98922-8880.

Since 1996, black tail deer (*Odocoileus hemionus columbianus*) in western Washington have been observed suffering from weakness and hair loss. This condition is now observed in most of the low lying elevation areas of western Washington. The primary cause appears to be a low grade pneumonia caused by high numbers of a *Parelaphostrongylus* larvae and, secondarily, a heavy louse infestation. Black tail fawns injected with ivermectin responded by growing new hair and gaining weight within several weeks of treatment Attempts at treating several localized populations of free-ranging deer by offering ivermectin medicated pellets were ineffective because deer did not eat the pellets. Molasses, crack corn, apples in various forms, salt, and other attractants have been added to the medicated pellets to try to induce deer to ingest them.

(17) A MULTI-AGENCY ATTEMPT TO RECOVER WOODLAND CARIBOU.

P. BRIGGS HALL, Washington Department of Fish and Wildlife, 16018 Mill Creek Blvd, Mill Creek WA 98012-1296; and JON A. ALMACK, Washington Department of Fish and Wildlife, 12645 Sullivan Lake Rd, Metaline Falls WA 99153-9711

The woodland caribou (*Rangifer tarandus caribou*) is the most endangered large mammal in the lower 48 states. In 1996 the population was estimated to be 34 individuals. A multi-agency effort supported by the Washington Department of Fish and Wildlife, Idaho Department of Fish and Game, U.S. Fish and Wildlife Service, U.S. Forest Service, and British Columbia Wildlife Branch was mounted to augment the exhisting population. Over a three year period 43 animals were captured in the Prince George region of British Columbia and transported to the Selkirk Mountains. Poachers, grizzly bears, mountain lions and high rates of calf mortality have all combined to prevent the desired population increase. At this time the fate of the woodland caribou in the lower 48 states remains uncertain.

(18) CHARACTERIZATION OF COPROANTIGENS OF *OSTERTAGIA* SP. INFECTIONS IN FARMED RED DEER.

T. QURESHI, C. SANTRICH, Department of Veterinary Pathobiology, School of Veterinary Medicine, Purdue University West Lafayette, IN 47907; R. E. LABES, M. TAYLOR, M. L. CROSS, and C. G. MACKINTOSH, AgResearch, Invermay Agreicultural Centre, Private Bag 50034, Mosgiel, New Zealand.

Deer farming constitutes an important agricultural based economy in New Zealand and in many other parts of the world, including the USA. *Ostertagia*-type species are the most abundant gastrointestinal nematodes of farmed red and wapiti deer in New Zealand, although of lesser importance than the lungworm, *Dictyocaulus viviparus*. Sub-clinical infections of *Ostertagia*-type worms are important because they cause significant losses in production. Diagnosis of gastrointestinal parasites in deer is unreliable as neither fecal egg counts nor plasma pepsinogen correlate to worm burdens. The objective of this study is to develop a coproantigen ELISA test that correlates with worm burdens, and this paper reports on the analysis of excretory/sectretory proteins (ESP) from adult *Ostertagia*-type sp. isolated from New Zealand red deer (*Cervus elaphus*). Patent infections of *Ostertagia*-type species were established in parasite-free red deer. Adult parasites were isolated and cultured in the laboratory to obtain ESP. These ESP were characterized using gel electrophoresis (SDS-PAGE) and western blot analysis. SDS-PAGE analysis of the ESP indicated several bands, of which 4 were identified on western blot analysis using sera from experimentally infected deer. Rabbit anti-ESP polyclonal antibodies recognized several bands, the 51 kD band was consistently found in samples from infected deer. A sandwich ELISA test, to detect coproantigens, was developed. This test had higher OD values for samples from infected deer. Data on the use of this test to identify coproantigens of *Ostertagia* sp. from experimentally infected deer will be presented.

(19) A RETROSPECTIVE SURVEY OF THE OCCURRENCE OF NEOPLASIA IN DEER IN THE SOUTHEASTERN UNITED STATES.

C.F. QUIST AND J.C. LANG. Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, University of Georgia, Athens, GA 30602.

With the exception of cutaneous fibromas, neoplasia in deer is considered to be a rare occurrence that has limited documentation in the literature. A retrospective study was performed using the Southeastern Cooperative Wildlife Disease Study records to document the occurrence of neoplasia in deer. Two databases were examined. One database consisted of diagnostic case records of 1135 deer submitted for necropsy between 1975 and 2000. This database provides qualitative data on the types of tumors that have been diagnosed in deer by SCWDS diagnosticians. The second database consists of apparently normal deer collected during herd health surveys, which gives a more accurate indication of the true incidence of neoplasia in deer.

The vast majority of the deer were necropsied were white-tailed deer (*Odocoileus virginianus*) or related subspecies. Based on diagnostic case records, approximately 3% of over 1100 deer necropsied between 1975 and 2000 were diagnosed with some form of neoplasia. The most commonly reported tumors were cutaneous fibromas. Herd health records indicate that cutaneous fibromas, which are virally induced, occur throughout the southeastern United States. Most of these benign lesions were restricted to the skin, but on rare occasions neoplastic nodules were found in subcutaneous tissue and muscle. Other benign tumors found in deer included lipomas, papillomas, and hemangiomas. Malignant tumors included squamous cell carcinomas, lymphosarcomas, brain tumors, and several types of adenocarcinomas.

(20) GIANT LIVER FLUKE IN ELK ISLAND NATIONAL PARK (ALBERTA): A RECENT CHANGE IN STATUS.

M.J. PYBUS, Alberta Natural Resources Service - Fish and Wildlife, 6909-116 Street, Edmonton, Alberta T6H 4P2.

In an on-going survey of giant liver fluke (*Fascioloides magna*), livers of 166 ungulates from Elk Island National Park (EINP) in central Alberta were assessed. Samples were examined opportunistically incidental to ongoing park management and research projects between 1987 and March 2000. A total of 77 elk (*Cervus elaphus*), 50 moose (*Alces alces*), 15 white-tailed deer (*Odocoileus virginianus*), 15 plains bison (*Bison bison*), 8 wood bison (*Bison bison athabascae*), and 1 mule deer (*Odocoileus hemionus)* was examined. The park is bisected by a four-lane highway with 3m high game fences on each side of the road and around the park perimeter. Giant liver flukes were not found in any of the samples (13 elk, 18 moose, 8 wood bison, 5 white-tailed deer) collected south of the highway. Of the remaining samples, overall prevalence of *F. magna* was 59% of 59 elk, 17% of 30 moose, and 1 of 7 whitetails. However, prevalence (94%, n=18) and intensity (37 $\pm$28, range 2-84) of infection in elk was significantly higher in 1999/2000 than in 1988 to 1995 (prevalence 44%, intensity 4 $\pm$ 4, range 1-13, n=41). The increase was reflected equally across yearlings and adults as well as males and females. Giant liver fluke is now enzootic at relatively high levels throughout the northern portion of the park. Management programs for controlling flukes in elk translocated from EINP may need to be reassessed.

(21) FATAL SYSTEMIC ADENOVIRUS INFECTION IN A CAPTIVE MOOSE CALF (*ALCES ALCES*).

ELIZABETH S. WILLIAMS, Department of Veterinary Sciences, University of Wyoming, Laramie, WY 82070; HOWARD LEHMKUHL, National Animal Disease Center, Ames, IA 50010; TERRY KREEGER, Wyoming Game and Fish Department, Research Laboratory, Laramie, WY 82071; CAROL HEARNE, JAQUELINE CAVENDER, and HANA VAN CAMPEN, Department of Veterinary Sciences, University of Wyoming, Laramie, WY 82070.

Severe pulmonary edema and hemorrhagic typhlocolitis resulted in the acute death of a recently captured 6 month old moose calf at the Wyoming Game and Fish Department's Sybille Wildlife Research and Conservation Education Unit near Wheatland, Wyoming. Histologically there was massive pulmonary edema associated with acute vasculitis and the presence of large intranuclear inclusion bodies in endothelium. Similar vasculitis and viral inclusions were present in the submucosa and muscularis of the cecum and colon. Viral particles, typical of adenovirus, were observed by negative stain electron microscopy in tissues from the calf. Adenovirus was isolated in culture on white-tailed deer umbilical endothelial cells. This virus was identical by serologic and restriction enzyme analysis to adenoviruses isolated from black-tailed and mule deer (*Odocoileus hemionus columbianus, O. hemionus hemionus*) and white-tailed deer (*O. virginianus*) from California and Iowa, respectively. The clinical and pathologic features of systemic adenoviral infection in this moose calf were consistent with the lesions described in adenoviral hemorrhagic disease in black-tailed deer in California. No other cervids at Sybille developed a similar disease and the source of the virus for the moose calf is not known.

(22) INVESTIGATION OF FRANKLIN'S GULL (*LARUS PIPIXCAN*) MORTALITY IN RELATION TO THE INITIATION OF AVIAN BOTULISM AMONG WATERFOWL AT EYEBROW LAKE, SASKATCHEWAN.

CATHERINE SOOS and GARY WOBESER, Department of Veterinary Pathology, Western College of Veterinary Medicine, University of Saskatchewan, 52 Campus Drive, Saskatoon, Saskatchewan, S7N 5B4, Canada.

Over the last several years, extensive mortality of immature Franklin's gulls (*Larus pipixcan*) had been observed prior to, or coincident with, the occurrence of botulism outbreaks in waterfowl on several marshes in prairie Canada. The presence of numerous gull carcasses in a marsh may initiate a botulism outbreak by providing substrate within which *Clostridium botulinum* type C can proliferate and produce toxin. A three-year study was designed, with the objective of defining the causes, extent, and significance of Franklin's gull mortality in relation to the occurrence of botulism at Eyebrow Lake, Saskatchewan. Here we present results of the first year of study. From May to August, 1999, a 1.76 km^2 area overlapping the gull colony was intensively monitored using standardized transect procedures. Detailed information on colony distribution, nesting, hatching, and mortality was collected. Extensive mortality of immature gulls occurred, beginning soon after hatching, and extending to fledging. Mortality peaked just prior to the onset of waterfowl botulism, and botulinum toxin was detected in maggots feeding on gull carcasses. The most common conditions diagnosed in immature gulls included bacteremia with *Staphylococcus aureus*, emaciation, dehydration, anemia, generalized lymphocytic apoptosis, severe ulcerative pododermatitis secondary to schistosome larval penetration, severe ulcerative dermatitis of the head secondary to trauma, coccidiosis, and small intestinal parasitism. Less commonly diagnosed were salmonellosis, adenovirus infection, renal coccidiosis, and botulism. The head and foot lesions were the most likely portals of entry for *S. aureus* bacteremia since bacterial cultures of these lesions consistently resulted in large numbers of *S. aureus* in pure culture. At present, it is unknown whether the mortality observed in juvenile Franklin's gulls is significant to the gull population itself; however, the potential for extensive gull mortality to initiate avian botulism outbreaks in waterfowl under the appropriate environmental conditions is substantial.

(23) CROSS-PROTECTION BETWEEN EPIZOOTIC HEMORRHAGIC DISEASE SEROTYPES 1 AND 2 IN EXPERIMENTALLY INFECTED WHITE-TAILED DEER.

JOSEPH K. GAYDOS, ELIZABETH W. HOWERTH, VICTOR F. NETTLES, and DAVID E. STALLKNECHT, Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, The University of Georgia, Athens, Georgia 30602.

Since the discovery of epizootic hemorrhagic disease virus (EHDV) in 1955, serotype 2 (EHDV-2) has been the virus most often isolated during hemorrhagic disease epizootics in white-tailed deer in the eastern United States. In 1999, an unprecedented widespread outbreak of serotype 1 (EHDV-1) caused considerable deer mortality in the eastern United States. Deer surviving infection with EHDV-2 are protected from disease when challenged again with EHDV-2. During the EHDV-1 outbreak we did not know if deer populations with high herd immunity against EHDV-2 would be protected against disease caused by EHDV-1 infection. To test the hypothesis that deer previously infected with EHDV-2 would be protected against disease caused by subsequent EHDV-1 infection, we experimentally infected eleven serologically negative white-tailed deer fawns with a low-virulence strain of EHDV-2 and subsequently challenged them with EHDV-1. Two naïve deer also were challenged with EHDV-1. All 13 deer became viremic. Signs of clinical disease were mild or absent in deer previously infected with EHDV-2, where as both naive deer became moribund and were euthanatized 10 days post infection. Prior infection with EHDV-2 appears to provide some protection against clinical disease caused by subsequent EHDV-1 infection, but it does not prevent these animals from acting as amplifying hosts.

(24) THE DISTRIBUTION OF *ECHINOCOCCUS GRANULOSUS* (CESTODA: TAENIIDAE) CYSTS IN MOOSE POPULATIONS: THE ROLE OF PREDATION BY WOLVES.

DAMIEN O. JOLY and FRANCOIS MESSIER, Department of Biology, University of Saskatchewan, 112 Science Place, Saskatoon, SK S7N 5E2 Canada.

Echinococcus granulosus is a tapeworm primarily found in Canidae, with intermediate hosts in the family Cervidae. Heavy infection by *E. granulosus* may predispose moose (*Alces alces*) to increase risk of predation by wolves (*Canis lupus*). Theory predicts that parasite induced vulnerability to predation (PIVP) will be reflected in the frequency distribution of parasites in the host population. Specifically, PIVP will reduce the degree of aggregation of parasites in a host population. We tested for an effect of PIVP by comparing the distribution of *Echinococcus granulosus* in moose in areas of low, moderate, and high levels of wolf predation rates. Parasite aggregation was significantly lower in areas with high predation rates and wolf densities, suggesting that heavy infection by *E. granulosus* predispose moose to predation by wolves. This increase in predation rate may exacerbate the role of wolves in regulating moose populations.

(25) EFFECTS OF BACK-MOUNTED RADIO TRANSMITTERS WITH A SUBCUTANEOUS ANCHOR IN MALLARD DUCKLINGS.

KAREN L. MACHIN, Department of Veterinary Physiological Sciences, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK S7N 5B4.

Waterfowl broods are difficult to monitor because of high mobility and low visibility. More accurate estimates of duckling survival may be obtained by radio-marking ducklings within the brood, however, transmitters may have deleterious effects. Most studies report few effects of transmitters but ducklings in these studies were raised in ideal conditions. Thus, I examined the effects of radio transmitters on mallard ducklings (Anas platyrhynchos) raised in outdoor pens, exposed to natural environmental conditions. Ducklings were hatched and raised by brood hens in outdoor pens at St. Denis Wildlife Refuge, Saskatchewan. In 1997, 36 day-old ducklings (5 broods) were divided randomly into 2 matched groups, with half the ducklings in each brood receiving transmitters and half the ducklings not receiving a transmitter but were handled for the same amount of time. In 1998, 154 day-old ducklings (21 broods) were divided randomly into 3 groups with one third of the brood receiving a transmitter, one third a sham surgery and one third control. In 1997, duckling mortality in the transmitter group (29%) was significantly greater than in the control group (5%). In 1998, 37% of transmitter ducklings died, whereas 8% sham ducklings and 4% control ducklings died. Duckling mortality in the transmitter group was significantly greater than in the control group but mortality did not differ between control and sham groups. Results also suggest that back-mounted radio transmitters have sublethal effects on duckling growth and behaviour. Researchers should be cautious when interpreting duckling survival using radio transmitters.

(26) GENETIC VARIATION IN EPIZOOTIC HEMORRHAGIC DISEASE VIRUS ISOLATES COLLECTED FROM THE SOUTHEASTERN UNITED STATES: 1978-1998.

M.D. MURPHY, Department of Pathology, College of Veterinary Medicine, University of Georgia, Athens, GA 30602; N.J. MACLACHLAN, Department of Pathology, Microbiology and Immunology, School of Veterinary Medicine, University of California, Davis, CA 95616; E.W. HOWERTH, Department of Pathology, College of Veterinary Medicine, University of Georgia, Athens, GA 30602; and D.E. STALLKNECHT, Southeastern Cooperative Wildlife Disease Study, University of Georgia, Athens, GA 30602.

Epizootic Hemorrhagic Disease Virus (EHDV), the causative agent of highly variable disease in wild and domestic ruminants, is endemic in the southeast with outbreaks of severe clinical disease occurring in white-tailed deer periodically. EHDV-specific antibodies in deer peak in late summer to early fall, concordant with the activity of the potentiating vector, *Culicoides* spp. A member of the Orbivirus subgroup of the Reoviruses, the EHDV genome is comprised of ten double-stranded RNA fragments, encoding three non-structural and seven structural proteins. The high error rate of RNA proofreading enzymes, in concert with the potential for segment reassortment indicates that genetic variation in EHDV is quite likely. The contributing factors to genetic variation in EHDV are currently unknown. The goal of this study is to determine the effects of time and geographic space on genetic variation in EHDV isolates collected from southeastern white-tailed deer, over a twenty-year period. Data pertaining to the genetic variation seen in the gene encoding a highly conserved protein (NS3), and the gene encoding the neutralizing epitope from the virion surface (VP2) will be presented, and several hypotheses generated by this data will be discussed.

(27) PREVALENCE OF POTENTIAL ZOONOSES IN FERAL AND PET DOMESTIC CATS FROM RANDOLPH COUNTY, NORTH CAROLINA.

FELICIA B. NUTTER, Department of Clinical Sciences, College of Veterinary Medicine, North Carolina State University, Raleigh, NC, 27606; MICHAEL K. STOSKOPF, Department of Clinical Sciences, College of Veterinary Medicine, North Carolina State University, Raleigh, NC, 27606; and JAY F. LEVINE, Department of Farm Animal Health and Resource Management, College of Veterinary Medicine, North Carolina State University, Raleigh, NC, 27606.

Consensus about the management of feral cats is required for effective action, yet uncertainty about the ramifications of different control strategies can make such agreement difficult. Valid concerns have been raised about the possible public health risks posed by managing established feral cat colonies with surgical sterilization. This project evaluates the potential role of feral cats as reservoirs of infectious diseases for humans, and compares the prevalence of exposure to infectious diseases in feral and pet domestic cats. In conjunction with a project examining population dynamics of neutered and intact feral cat colonies, over 150 feral cats have been trapped and biological samples collected. A geographically matched sample of 100 pet cats presented to local practitioners for routine preventative medicine exams serves as the reference population. Blood and fecal samples have been tested for evidence of exposure to *Toxoplasma gondii*, *Cryptosporidium* sp., *Giardia*, *Bartonella* sp., and rabies. Prevalence of feline leukemia virus and feline immunodeficiency virus will be evaluated as potential contributing or confounding factors. Chi-squared and Wilcoxon rank sum tests will be used to evaluate associations between disease prevalence and putative explanatory factors such as age and environment. Odds ratios and risk factors will be evaluated for an age-class and gender matched subsample. Documenting and comparing the prevalence of exposure to potentially zoonotic pathogens in feral and pet domestic cats is an important step in thoroughly evaluating management and control strategies for feral cat colonies.

(28) HOST SUSCEPTIBILITY TO EXPERIMENTAL MYCOPLASMA INFECTION IN HATCHLING AMERICAN ALLIGATORS (*ALLIGATOR MISSISSIPPIENSIS*) EXPOSED TO ENDOCRINE DISRUPTING CONTAMINANTS.

LAUREN J. RICHEY, Department of Pathobiology, College of Veterinary Medicine, University of Florida, Gainesville, FL 32611; TRENTON R. SCHOEB, Department of Pathobiology, College of Veterinary Medicine, University of Florida, Gainesville, FL 32611; MARY B. BROWN, Department of Pathobiology, College of Veterinary Medicine, University of Florida, Gainesville, FL 32611; TIMOTHY S. GROSS, USGS-BRD-Florida Caribbean Science Center, Gainesville, FL 32653 and Department of Physiological Sciences, College of Veterinary Medicine, University of Florida, Gainesville, FL 32610; and PAUL A. KLEIN, Department of Pathology, University of Florida, Gainesville, FL 32610.

Evidence of alterations to the immune system, such as decreased antibody responses and lymphoid organ atrophy, have been found in a previous study of hatchling American alligators (*Alligator mississippiensis*) from contaminated Lake Apopka in Florida. In addition, a decline in the juvenile population and increased neonatal mortality have been observed on several Florida lakes in the last 20 years. One potential cause of the increased neonatal mortality could be increased susceptibility to infectious disease. The objective of this study was to determine whether hatchling alligators exposed *in ovo* to endocrine disrupting contaminants (EDCs) have decreased resistance to infectious disease. Although other measures of host resistance were examined during this study, the ultimate measure of immunosuppression is the host response to infectious disease, involving a number of coordinated defense mechanisms. The first group of study animals consisted of alligators hatched from eggs taken from an uncontaminated reference site that were exposed *in ovo* to 1, 5, or 25 ppm DDE or to DMSO vehicle control. The second group of animals consisted of eggs taken from contaminated and reference Florida lakes; these alligators were naturally exposed to environmental contaminants. The eggs were artificially incubated, and the hatchlings were maintained in captivity until 3 months of age. Host susceptibility to infectious disease was tested using a species of mycoplasma isolated from a natural outbreak of disease in captive alligators. Hatchlings were inoculated intravenously with 10^6 colony forming units (CFU) per animal, following preliminary dose response experiments. Animals were observed daily for signs of lameness or swelling of joints. Body weights were obtained weekly. Animals were sacrificed 4 weeks post-inoculation. Colony counts from meningeal and pericardial swabs and blood samples were determined by standard dilution and plating, using SP4 broth and agar media. No significant differences (at $p<0.05$) were found between the DDE-treated groups and controls or between the lake groups in numbers of footdays of swelling or lameness, changes in body weight, or numbers of bacteria isolated. Lesions noted on gross and histologic examination included fibrinosuppurative pericarditis and epicarditis, meningitis, encephalitis, polyarthritis, synovitis, fascitis, conjunctivitis, and gastritis.

(29) PRION PROTEIN IN THE VAGOSYMPATHETIC TRUNK AND PANCREAS OF MULE DEER NATURALLY INFECTED WITH CHRONIC WASTING DISEASE.

CHRISTINA J. SIGURDSON, Department of Pathology, Colorado State University, Fort Collins, CO 80523; TERRY R. SPRAKER, Colorado State University Veterinary Diagnostic Laboratory, Fort Collins, CO 80523; MICHAEL W. MILLER, Colorado Division of Wildlife, Fort Collins, CO 80526; BRUNO OESCH, Prionics AG, University of Zürich 44J30, CH-8057 Zürich, Switzerland; KATHERINE I. O'ROURKE, Animal Disease Research Unit, U.S. Department of Agriculture, Pullman, WA 99164; and EDWARD A. HOOVER, Department of Pathology, Colorado State University, Fort Collins, CO 80523.

Chronic wasting disease (CWD) of deer (*Odocoileus hemionus, O. virginianus*) and elk (*Cervus elaphus nelsoni*) belongs to a group of fatal neurodegenerative diseases known as transmissible spongiform encephalopathies or prion diseases. The transit route of CWD prion protein from the initial exposure site, likely the alimentary tract, to the central nervous system is unknown. We detected the abnormal isoform of the prion protein, PrP^{res}, in peripheral nerves of six naturally infected deer by immunohistochemistry (IHC) using the 6H4 and 99/97.6.1 monoclonal antibodies against PrP. Course, granular stain denoting PrP^{res} was present in the vagosympathetic trunk (n=6) and in pancreatic islet cells. No PrP^{res} staining was evident in peripheral nerves of 12 control deer. These findings suggest that (1) PrP^{res} axonal transport occurs centrifugally or centripetally as one route of organ invasion, and (2) prion protein may enter the nervous system directly from ganglia or nerve fibers in the alimentary tract.

(30) EXPERIMENTAL INOCULATION OF AMERICAN OPOSSUMS (*DIDELPHYS AZRAE)* WITH *MYCOBACTERIUM BOVIS.*

KELLY L. BUTLER, Animal Health Diagnostic Laboratory and Department of Pathology, Michigan State University, East Lansing, MI 48824; SCOTT D. FITZGERALD, Animal Health Diagnostic Laboratory and Department of Pathology, Michigan State University, East Lansing, MI 48824; DALE BERRY, Michigan Department of Community Health Tuberculosis Laboratory, Lansing, MI 48909; STEVEN CHURCH, Michigan Department of Community Health Tuberculosis Laboratory, Lansing, MI 48909; WILLIE M. REED, Animal Health Diagnostic Laboratory and Department of Pathology, Michigan State University, East Lansing, MI 48824; JAMES SIKARSKIE, Department of Small Animal Clinical Sciences, College of Veterinary Medicine, Michigan State University, East Lansing, MI 48824; and JOHN B. KANEENE, Population Medicine Center, Department of Large Animal Clinical Sciences, College of Veterinary Medicine, Michigan State University, East Lansing, MI 48824.

Michigan has an endemic population of tuberculosis (*Mycobacterium bovis*)-infected white-tailed deer located throughout the Lower Peninsula of the state. Surveillance for spread of the disease into additional species has been ongoing since 1995. Although surveillance has detected spread into several carnivore species, no native opossums have been found to be culture-positive for the organism. The foundation for carnivore and omnivore surveillance involves the idea that the species studied are known to gather in agricultural settings and to commonly feed on organic materials (viscera, feed, and/ or fecal matter) that may harbor microorganisms. Ingestion of contaminated materials would allow for considerable transmission if these animals can in fact become infected with, or carry, the bovine tuberculosis agent and shed microorganisms in distant sites. No reports of *M. bovis* in American opossums exist to our knowledge. Carnivore/ omnivore species which have had positive cultures for *M. bovis* thus far in Michigan include the raccoon, coyote, bobcat, black bear, and red fox, although several other species including the badger, skunk, grey fox, and opossum have also been included in surveillance submissions. In New Zealand, the free-ranging brushtail possum has been implicated as a primary reservoir host for the spread of *M. bovis* infections into domestic cattle and deer, and wildlife populations. Lesions and routes of transmission in this species are well defined. This study, the first of its kind in Michigan, set to prove or disprove the related species, the American opossum, as a similarly important and dangerous reservoir host for *M. bovis*.

Wild-caught, native-Michigan opossums were obtained for the study. The project divided experimental animals equally into three exposure groups. Four animals were fed 1×10^5 CFU of *M. bovis* organisms obtained from a cultured case of an infected Michigan white-tailed deer, and four animals were injected intramuscularly with the same concentration of organisms. Four animals were housed separately as controls. Half of the animals in each group were sacrificed at approximately 30 days post-infection and the remaining half were sacrificed after approximately 60 days. Rectal swabs were obtained from all animals on the day prior to exposure, and at 24 hours post-inoculation. These swabs were cultured for *Mycobacteria* to determine whether fecal shedding of organisms had occurred and to rule-out the potential of established carriers. Weights of the animals

were recorded every 2 weeks throughout the experiment and at necropsy. Organ weights were recorded at necropsy, and multiple tissues were collected, fixed in formalin, and assessed histologically, both with routine hematoxylin and eosin, and acid-fast staining, and tissue samples were submitted for mycobacterial isolation and identification procedures.

Gross lesions attributable to various internal parasitic infections were detected in animals in all exposure groups. Hepatomegaly and splenomegaly were noted in both groups of inoculated opossums. Two of four intramuscularly inoculated animals had purulent to granulomatous myositis and cellulitis detectable both grossly and histologically near injection sites. Light microscopic examination revealed numerous acid-fast bacilli in special-stained tissue sections obtained from these areas. The presence and presentation of histologic lesions varied considerably between and within groups of inoculated opossums. Fecal and tissue culture results documented organism shedding and active mycobacterial infection in both inoculation-route groups, although not consistently in either group. The findings suggest American opossums as potential carriers of the bovine tuberculosis agent, although as a species they appear less susceptible to fulminant tuberculosis than the brushtail possum, and as such, are not likely as important a reservoir host.

(31) REVERSIBLE IMMOBILIZATION OF FREE-RANGING SVALBARD REINDEER, NORWEGIAN REINDEER AND WOODLAND CARIBOU: A COMPARISON OF MEDETOMIDINE-KETAMINE AND ATIPAMEZOLE IN THREE SUBSPECIES OF *RANGIFER TARANDUS*.

JON M. ARNEMO, Department of Arctic Veterinary Medicine, The Norwegian School of Veterinary Science, NO-9292 Tromsø, Norway; RONNY AANES, Norwegian Polar Institute, NO-9170 Longyearbyen, Norway; ØYSTEIN OS, Wildlife Veterinary Consultants Inc., NO-2580 Folldal, Norway; NIGEL A. CAULKETT, Department of Anesthesiology, Radiology and Surgery, Western College of Veterinary Medicine, University of Saskatchewan, 52 Campus Drive, Saskatoon, SK, S7N 5B4 Canada; W. JAMES RETTIE, Department of Biology, University of Saskatchewan, 112 Science Place, Saskatoon, SK, S7N 5E2 Canada; and JERRY C. HAIGH, Department of Herd Medicine and Theriogenology, Western College of Veterinary Medicine, University of Saskatchewan, 52 Campus Drive, Saskatoon, SK, S7N 5B4 Canada.

Combinations of medetomidine (M) and ketamine (K) were evaluated in free-ranging Svalbard reindeer (*Rangifer tarandus platyrhynchus*), Norwegian reindeer (*R. t. tarandus*) and woodland caribou (*R. t. caribou*) as parts of ecological studies from 1993 to 1999. All trials were carried out in winter. The drugs were administered by dart syringe injection. Svalbard reindeer were approached on foot and darted in the heavy muscles of the shoulder or thigh from 15-25 meters, Norwegian reindeer were darted from a helicopter, while woodland caribou were darted either from a helicopter or from a hide. The mean (SD) effective immobilizing doses of M + K in two groups of adult female Svalbard reindeer were 0.113 (0.009) mg M/kg + 2.26 (0.19) mg K/kg (group 1, n = 10) and 0.215 (0.043) mg M/kg + 1.08 (0.21) mg K/kg (group 2, n = 10). All Svalbard reindeer were weighed. The mean induction time of group 1 was significantly shorter than that of group 2 ($p < 0.05$, Mann-Whitney U-test), 6.5 (3.2) versus 14.3 (10.6) minutes, respectively. In Norwegian reindeer (8 adult males, 21 adult females), the mean (SD) effective immobilizing doses were 0.206 (0.036) mg M/kg + 1.03 (0.18) mg K/kg based on estimated body mass. No significant difference in mean induction times between males and females were found. However, animals with optimal hits (shoulder or thigh) (n = 16) had a significantly shorter mean induction time than animals with suboptimal hits (abdomen or flank) (n = 13), 5.6 (2.2) versus 11.1 (4.7) minutes, respectively. The mean (SD) effective immobilizing doses in woodland caribou (n = 10) were 0.237 (0.043) mg M/kg + 2.3 (0.4) mg K/kg. Woodland caribou were either weighed or the body mass was estimated. The mean (SD) induction time was 7.9 (3.8) minutes (n = 8). In all three subspecies, inductions were calm and immobilized animals were in sternal or lateral recumbency. Major clinical side-effects were not seen although mild to moderate hypoxemia developed during immobilization. In Svalbard reindeer (pooled data), the mean (SD) rectal temperature was 39.0 (0.2) °C, the mean (SD) heart rate was 31 (6) beats/minute, the mean (SD) respiratory rate was 13 (3) breaths/minute and the mean (SD) SpO_2 was 88 (2) %. The corresponding values in Norwegian reindeer (pooled data) were 40.6 (0.8) °C, 47 (19) beats/minute, 17 (9) breaths/minute and 84 (11) %, and in woodland caribou (pooled data) 40.4 (0.4) °C, 47 (7) beats/minute, 15 (4) breaths/minute and 83 (8) %. For reversal, Svalbard reindeer and Norwegian reindeer received 5 mg atipamezole (A) per mg M while in woodland caribou a dose ratio of 3 mg A per mg M was used. In Svalbard reindeer, the dose of A was divided and given half

i.m. and half s.c., and the mean (SD) time from administration of A to the animals were on feet was 11.3 (5.7) minutes (pooled data). In Norwegian reindeer and woodland caribou, the dose of A was divided and given half i.v. and half i.m. and the mean (SD) times to standing were 3.7 (3.6) and 4.5 (3.5) minutes, respectively. We conclude that medetomidine-ketamine and atipamezole are good non-opioid alternatives for reversible immobilization in both Svalbard reindeer, Norwegian reindeer and woodland caribou. Different capture techniques make direct comparisons of effective dose levels between the three subspecies difficult, but induction times seem to be influenced by stress (helicopter chasing), injection site (hit quality) and ketamine dose.

(32) SURVIVAL AND IMPROVED MOOSE (*ALCES ALCES*) IMMOBILIZATION WITH CARFENTANIL AND XYLAZINE.

THOMAS J. ROFFE, KENNETH W. COFFIN, Biological Resources Division, U.S. Geological Survey, FWP Building, Montana State University, Bozeman, MT 59717; and JOEL BERGER, Program in Ecology, Evolution, and Conservation Biology (Ms-186), University of Nevada, Reno, NV 89512 and Wildlife Conservation Society, Box 340, Moose, WY 83012.

Previous work on moose chemical immobilization with potent narcotics (etorphine and carfentanil) have reported mortality of 6-19%. Even the most recently reported study using the same drug combination as we used in this report (carfentanil and xylazine) had a 6% mortality in moose within several days of immobilization. We used 3.3 – 3.6 mg of carfentanil combined with 50 – 60 mg xylazine on 36 adult moose that immobilized with one dart. Mean carfentanil and xylazine doses for adult moose was 3.52 mg (± 0.12) and 58.8 mg (± 3.2), respectively. Mean induction time was 4.39 min (± 1.9; range 1.83 – 8.83 min) and mean down time was 26.01 min (± 6.1; range 12.42 – 37.38 min). We antagonized carfentanil with 400 – 500 mg of naltrexone predominantly through intramuscular and subcutaneous routes. Yohimbine, 30 – 60 mg or 400 – 900 mg tolazoline was delivered intravenous to antagonize xylazine. Mean naltrexone to carfentanil ratio was 127:1. In total, we chemically immobilized 48 moose (42 adult cows, 1 immature male, 5 calves); only 1 moose died within 7 days of chemical immobilization. This mortality was attributed to excessively poor body condition and not immobilization. Our high success rate was presumably due to the use of naltrexone delivered both intramuscularly and subcutaneously, and to the low stress technique of ground darting. Retrospective analysis of our data on female body condition, suggests that female survival is diminished when below a body fat threshold and, as a consequence, some mortality is likely to arise due to poor condition per se and not as a direct result of immobilization. Our data demonstrate that moose can safely be chemically immobilized with carfentanil and xylazine from the ground with very low mortality rates provided care is taken to prevent potential renarcotization problems and stress is minimized.

(33) ANESTHESIA OF PRONGHORN USING A-3080 OR A-3080 PLUS XYLAZINE.

TERRY J. KREEGER, Wyoming Game and Fish Department, Wheatland, WY 82001; WALT COOK, Wyoming Game and Fish Department, Laramie, WY 82070; CLAUDE A. PICHÉ, Wildlife Pharmaceuticals Inc., Fort Collins, CO 80524; and THOMAS SMITH, F. E. Warren Air Force Base, Cheyenne, WY 82005

A-3080 is a potent synthetic opioid anesthetic being developed for use in wildlife. A-3080 was tested for safety and efficacy on free-ranging pronghorn (*Antilocapra americana*) inhabiting an Air Force base in southeastern Wyoming. Pronghorn were darted with pre-measured dosages of either 4.0 or 5.0 mg of A-3080 without (Group 1) or with (Group 2) the addition of 25.0 mg xylazine. Seventeen pronghorn were captured in Group 1 and 14 pronghorn in Group 2. There were no differences between Groups for capture times or physiological parameters ($P = 0.21$). Anesthetic induction was rapid for both Groups ($= 2.7 \pm 0.4$ min) as was recovery after antagonism ($= 0.7 \pm 0.07$ min). The actual A-3080 dosage administered was 0.10 ± 0.005 mg/kg; the actual xylazine dosage was 0.56 ± 0.03 mg/kg. Anesthesia in both Groups was characterized by muscle rigidity and rapid, shallow respiration. Twenty-five pronghorn were radio collared and survived at least 21 days after the capture operation. One adult male in Group 1 died during capture. A-3080 was considered more effective on pronghorn than other anesthetic regimens.

(34) EVALUATION OF THE EFFECTS OF TRANQUILIZATION AND OTHER FACTORS ON MORBIDITY AND MORTALITY IN WILD-CAUGHT PRONGHORN ANTELOPE (*ANTILOCAPRA AMERICANA)* DURING TRANSLOCATION.

JULIE A. SMITH, Department of Veterinary Clinical Sciences, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA 70803; THOMAS J. ROFFE, Biological Resources Division, CRO, USGS, Montana State University, Bozeman, MT 59717; DONALD S. DAVIS, Department of Veterinary Pathobiology, College of Veterinary Medicine, Texas A&M University, College Station, Texas 77843-4467; and PHILIP H. ELZER, Department of Veterinary Science, Louisiana State University, Baton Rouge, LA 70803.

Immobilization or sedation of pronghorn antelope has historically been a challenge and often unrewarding. Commonly used tranquilizers have unpredictable, and often negligible effects, in this species. When working with pronghorn, morbidity and mortality is best avoided by not administering any type of immobilizing agent and by simply restraining them. However, the stress of capture alone still results in a high incidence of mortality during translocation of this species. In an effort to identify an agent that could possibly reduce the stress of capture and transport in pronghorn, two tranquilizers were evaluated in a group of antelope that was being removed from a ranch in Wyoming, as part of a population control measure, and relocated to a facility in Texas.

One hundred and twelve (112) wild pronghorn antelope (*Antilocapra americana*) were captured in Wyoming and translocated to a research facility in College Station, Texas, on November 19, 2000. The antelope were herded by helicopter in groups of approximately 50 into a net-sided corral, hand captured and loaded onto one of three different cattle trailers. Seven animals each received either Haloperidol (Haldol®), a butyrophenone tranquilizer, or Perphenazine (Trilafon® enantate), a long acting phenothiazine tranquilizer, both routinely used in many wild species to alleviate captured-related stress, as they were loaded onto the truck. Following a nineteen-hour trailer transport to Texas, there was an overall 9.8 % acute captured-related mortality (defined as mortality from capture to unloading transport vehicles). This included 57.1% (4/7) of animals that received haloperidol, and 14.3% (1/7) in animals that received trilafon. Over the next 40 days there was a steady die-off, primarily caused by aspiration pneumonia related to capture stress.

Mortality was affected by length of time to capture, type of trailer arrangement, tranquilization and length of time on trailer. Administration of either tranquilizer did not reduce mortality.

(35) MORTALITY IN CAPTIVE PRONGHORN ANTELOPE: A TEXAS EXPERIENCE

JOHN F. EDWARDS, DONALD S. DAVIS, BARBARA C. LEWIS, FERNANDO RAMIRO-IBANEZ, LYNNE CHITTENDEN, Department of Veterinary Pathobiology, College of Veterinary Medicine, Texas A&M University, College Station, TX 77843-4467; THOMAS J. ROFFE, Biological Resources Division, CRO, USGS, Montana State University, Bozeman, MT 59717; JULIE A. SMITH, Department of Veterinary Clinical Sciences, Louisiana State University, College of Veterinary Medicine, Baton Rouge, LA 70803; and PHILIP H. ELZER, Department of Veterinary Science, Louisiana State University, Baton Rouge, LA 70803.

A group of 112 Pronghorn Antelope were removed from a ranch in Wyoming as part of a population control measure and were transported to Texas at the end of November for use in a study of the effects of vaccination of Pronghorn Antelope with two live *Brucella* vaccines (RB51 and strain 19). To limit physical handling, animals were vaccinated as they were removed from the transporting vehicles in Texas and were released into a secluded and screened pasture. For approximately one month, animals succumbed acutely to a necrotizing bronchopneumonia and pleuritis. Animals were necropsied, cultures were performed, and histology of lesions was studied. Only coliforms (primarily *E. coli*) and *Corynebacterium sp*. were isolated. The necrotizing nature of the pneumonia and the bacterial culture results suggested the problem involved an inhalation pneumonia. An occasional animal had lesions suggesting nutritional myopathy. Because of the association of myopathy of muscles of deglutition with aspiration pneumonia in cattle, a change in feed was suggested. Twenty days after their arrival, the feed and salt blocks were supplemented with selenium. One week after supplementation, losses due to pneumonia became infrequent. By that time, in the first 28 days of the study, over fifty percent of animals had died due to pneumonia. Only three more animals were lost to pneumonia between days 29 and 39 of the study when the pneumonia problem ceased. At study day 50 and following a heavy rain, animals began to develop pododermatitis and systemic lesions from which *Fusobacterium necrophorum* was isolated. Complete drainage of the pasture was not possible, and many animals persistently drank from an area of standing water rather than from their watering tanks. As the pasture dried, fifteen animals died of pododermatitis and septicemia. At study day 68, some animals began to develop lesions of and die from oral stomatitis with septicemia without foot lesions. Again, *Fusobacterium necrophorum* were cultured from oral and systemic lesions. Animals continued to die from pododermatitis or stomatitis with septicemia, and the entire collection essentially was lost by 120 days of captivity. Spontaneous, ecto and endoparasitic lesions were seen occasionally, and other incidental and coincident disease conditions were noted.

(36) KEEPING ARABIAN TAHR (*HEMITRAGUS JAYAKARI*) IN CAPTIVITY-VETERINARY REVIEW.

JACOB M. MWANZIA, Environment and Wildlife Management Section, Private department of His Highness Sheikh Zayed Bin Sultan Al Nayan, P.O. Box 77 Abu Dhabi, United Arab Emirates.

Three years of experience in managing Arabian tahr (*Hemitragus jayakari*), the most endangered Arabian mammal in Sir Bani yas island, united Arab Emirates, is reported. Problems arose with obstetrics, paediatrics, ecto-and endoparasites, and injuries resulting from flight and social aggression. Information is given on haematology, serum biochemistry, and immobilization.

(37) "MALADAPTATION SYNDROME" REVISITED.

PETER DASZAK, Institute of Ecology, University of Georgia, Athens, GA 30602, USA; and ANDREW A. CUNNINGHAM, Institute of Zoology, Zoological Society of London, Regent's Park, London NW1 4RY, UK.

Reptiles are an increasingly important component of conservation programs. However, an understanding of their diseases has lagged behind advances in the study of mammalian and avian diseases. Here, we reassess the term "maladaptation syndrome", first coined by Cowan (1968) as the cause of death for captive reptiles at Philadelphia Zoo, and still frequently used as a diagnostic term. Its broad definition of an "inability to adapt to the zoo environment" includes failure to eat, malnutrition and sepsis, loss of tissue integrity, oral, dermal and cloacal ulceration, enteric mucosal necrosis and ulceration, bacterial invasion of ulcers, parasitic enteritis, pneumonia, exhaustion and death, usually within 2 years of acquisition.

We present case studies to demonstrate that apparent "maladaptation syndrome" of reptiles can be due to a range of infectious diseases, exacerbated by poor or unsuitable husbandry. These cases include new and recently described parasitic diseases that were unknown at the time of Cowan's study. Data from a study of parasite prevalence in recently imported reptiles show that importation significantly perturbs host-parasite dynamics. Prevalence within captive populations is markedly elevated compared to wild populations, and this heightened prevalence persists for up to two years following importation. Data collected at the Zoological Society of London reveal high prevalence of microsporidia in captive reptiles with morphological similarities to a species described by Koudela *et al.* in 1998. This appears to represent a recent introduction into captive populations, and a rapid expansion in prevalence within a wide range of host species.

Recent work on reptile host-parasite ecology suggests that reptiles may be more dependent on behavioral and life history traits to avoid infection than mammals and birds. These behavioral tactics may be disrupted in captivity by higher than natural stocking densities, restricted enclosure size, unsuitable food or sanitation and other husbandry parameters. This scenario may be particularly important for captive reptiles, since the natural history of many species is poorly understood. In these cases, "maladaptation syndrome" reflects maladapted husbandry - not an inability of the animal to adapt to these conditions.

We propose that the term "maladaptation syndrome" should be avoided for the following reasons:

1) It fails to address the real problem with these cases; that of (often subtly) unsuitable husbandry.
2) Many new diseases and conditions described in reptiles since 1968 may explain some of the "maladapted" animals reported by Cowan (1968), and further novel diseases almost certainly await description.
3) The term is confusing since there are a vast array of adaptations and maladaptations in other, related, scientific disciplines, eg. evolutionary, psychosocial, medical and physiological maladaptation.

4) Citing “maladaptation syndrome” as a cause of death provides a false basis for therapeutic decisions and puts the onus on the animal to adapt to unsuitable conditions, rather than on the keeper to provide the correct husbandry for the animal’s requirements.

(38) EVALUATION OF CABERGOLINE AS A REPRODUCTIVE INHIBITOR IN COYOTES (*CANIS LATRANS*).

THOMAS J. DELIBERTO, AMY E. SEGLUND, USDA/APHIS/WS, National Wildlife Research Center, Predation Ecology and Behavior Project, Utah State University, Logan, UT 84322-5295; and BRUCE KIMBALL, USDA/APHIS/WS, National Wildlife Research Center, 4101 La Porte, Fort Collins, CO 80521-2154

The use of fertility control as a means for the resolution of predation problems has surfaced in recent years. Cabergoline ($C_{23}H_{27}N_5O_2$), a dopamine antagonist with a prolonged prolactin-lowering effect, has been found to be an effective abortifacient during the second half of pregnancy in the domestic dog, and red fox. Because it exists in an oral form and is relatively species specific, cabergoline may have potential use as a reproductive control agent for coyotes. The goal of our study was to evaluate the use of cabergoline as an effective abortifacient and provide insight into the reproductive physiology of the coyote. We examined the effect of three different dosages (50, 100, 250, 300, and 500 mcg) of cabergoline administered daily from day 38 - 45 of pregnancy. We also evaluated the 500 mcg dose administer on day 38 and 41 of pregnancy. Blood samples were collected weekly after the first observed tie, and continued until two weeks past the predicted or actual whelping date. The efficacy of cabergoline was evaluated on blood concentrations of prolactin and progesterone and whether females successfully whelped young.

(39) EVIDENCE OF NATURALLY OCCURRING *EHRLICHIA CHAFFEENSIS* INFECTION IN COYOTES FROM OKLAHOMA.

A. ALAN KOCAN, G. CROWDER LEVESQUE, L. WHITWORTH, G.L. MURPHY, S.A. EWING, and R.W. BARKER, Oklahoma State University, Stillwater, Oklahoma 74078.

A nested polymerase chain reaction assay was used to survey for the presence of *Ehrlichia chaffeensis*, *E. canis*, and *E. ewingii* DNA in blood samples from free-ranging coyotes from central and north-central Oklahoma. Fifteen of 21 coyotes (71%) examined were positive for *E. chaffeensis* DNA while none was positive for either *E. canis* or *E. ewingii*. Results suggest that *E. chaffeensis* infections are common in free ranging coyotes in Oklahoma and that this wild canid could play a previously unrecognized role in the epidemiology of human monocytotropic ehrlichiosis.

(40) SEROLOGIC SURVEY FOR CANINE CORONA VIRUS IN WOLVES (*CANIS LUPUS*) FROM INTERIOR ALASKA, 1994-1999.

RANDALL L. ZARNKE, Alaska Department of Fish and Game, 1300 College Road, Fairbanks, AK 99701-1599; JIM EVERMANN, Department of Veterinary Clinical Sciences, College of Veterinary Medicine, Washington State University, Pullman, WA 99164-6610; JAY M. VER HOEF, Alaska Department of Fish and Game, 1300 College Road, Fairbanks, AK 99701-1599; MARK E. MCNAY, Alaska Department of Fish and Game, 1300 College Road, Fairbanks, AK 99701-1599; RODNEY D. BOERTJE, Alaska Department of Fish and Game, 1300 College Road, Fairbanks, AK 99701-1599; CRAIG L. GARDNER, Alaska Department of Fish and Game, PO Box 355, Tok, AK 99780-0355; LAYNE G. ADAMS, US Geological Survey, Alaska Biological Sciences Center, 1011 East Tudor Road, Anchorage, AK 99503; BRUCE W. DALE, Alaska Department of Fish and Game, 333 Raspberry Road, Anchorage, AK 99518-1599; and JOHN BURCH, National Park Service, 201 First Avenue, Fairbanks, AK 99701

Wolves (*Canis lupus*) were captured during spring (March-April) and autumn (October-November) in three areas of Interior Alaska during 1994-1999. Sera were tested for evidence of exposure to canine corona virus (CCV) by means of a serum neutralization method. CCV is an enteric pathogen that often occurs in conjunction with canine parvovirus. Test results followed a unique pattern for two of the study areas. Results indicated that 0% of the 5-month-old pups and 35% of the adults sampled during the autumn period had been exposed to CCV. Tests on the spring samples revealed that 72% of the 10-month-old pups and 81% of the adults had been exposed. These results indicate that: 1) transmission occurs primarily during the winter, and 2) antibody levels decline rapidly. Impact(s) of this virus on the three wolf populations are unknown.

(41) RETROSPECTIVE STUDY OF DYSPLASTIC AND NEOPLASTIC LESIONS OF THE BILIARY SYSTEM IN THE CAPTIVE POPULATION OF BLACK-FOOTED FERRETS (*MUSTELA NIGRIPES*).

STÉPHANE LAIR, Toronto Zoo, 361A Old Finch Avenue, Scarborough, Ontario M1B 5K7 Canada and Dept. of Pathobiology, Ontario Veterinary College, University of Guelph, Guelph, Ontario N1G 2W1 Canada; KAY G. MEHREN, Toronto Zoo, 361A Old Finch Avenue, Scarborough, Ontario M1B 5K7 Canada; ELIZABETH S. WILLIAMS, Department of Veterinary Sciences, University of Wyoming, 1174 Snowy Range Road, Laramie, Wyoming, 82070; and IAN K. BARKER, Dept. of Pathobiology, Ontario Veterinary College, University of Guelph, Guelph, Ontario N1G 2W1 Canada; Present address (Lair): University of British Columbia, 6199 South Campus Road, Vancouver, B.C. V6T 1W5 Canada.

A retrospective study was conducted to evaluate the occurrence and clinical significance of anomalies of the liver in the captive population of Black-footed ferrets (*Mustela nigripes*). Clinical files, and *postmortem* reports from 184 adult ferrets that died during the first 12 years of the breeding program were reviewed. Archived materials from 116 of these ferrets were retrieved and examined. Pertinent information also was recovered from the studbook maintained at the USFWS National Black-Footed Ferret Conservation Center.

Intrahepatic cysts were seen in 65.8% of the adult ferrets examined. These cysts were filled with a clear fluid, did not communicate with the main biliary system, and ranged in size from microscopic to large coalescing structures structures almost completely replacing affected hepatic lobes. The immunostaining proprieties of the flattened lining epithelium are suggestive of a cholangiocellular origin. Intrahepatic biliary cysts often remained subclinical for several years, but occasionally were associated with clinical signs following rupture or secondary bacterial infection.

Cholangiohamartomatas, formed of disorganized clusters of mildly dilated cholangioles embedded in fibrous tissue, were detected in 14.8% of the sections of liver examined. These dysplastic structures were seen only in ferrets affected by biliary cysts. Clusters of undifferentiated progenitor cells (so-called Aoval cells@) were seen in 86.2% of the livers available for histologic examination. Based on their immunohistochemical proprieties these cells were believed to be precursors of biliary elements.

Cholangiocellular neoplasms were diagnosed in 20.1% of the adult ferrets that died between the establishment of the captive breeding population and 1998. These neoplasms, which were always associated with biliary cysts, were either classified as biliary cystadenomas (prevalence of 7.6%) or biliary cystadenocarcinomas (prevalence of 12.5%) according to their cytologic features and degree of invasiveness. These tumors were mainly characterized by poorly defined intracystic or pericystic neoplastic proliferations, budding from the epithelial lining. Abdominal carcinomatosis and distant metastases (lungs and lymph nodes) were seen in approximately half of the cases of cystadenocarcinoma. Most cystadenocarcinomas were associated with clinical signs, with abdominal distention by ascitic fluid being the most common antemortem finding.

Multivariate logistic regression was used to investigate the relationship of host and environmental variables as factors associated with development of biliary cysts and cholangiocellular neoplasms in black-footed ferrets. Age at death was the only factor that was positively associated with the probability of been affected by biliary cysts and biliary neoplasms. The odds of having biliary cysts or biliary cystic neoplasms increased by five times and two times, respectively, for each additional year of life. The prevalence at death of biliary cysts and cystic biliary neoplasms in our study progressively increased with age, reaching 100% and 60% respectively in animals over 8 years old. Cholangiohamartomas and clusters of progenitors cells were also more prevalent in older ferrets. Sex predisposition, environmental and reproductive predisposing factors, patterns of Mendelian inheritance, and familial clustering were not detected for the biliary cysts and biliary neoplasms.

The presence of both benign and malignant cellular phenotypes in most cases of biliary cystadenocarcinomas, and their overall histological similarity to cystadenomas, strongly suggest that they are an extension of the same pathological process. The most striking feature of these tumors was their close association with biliary cysts; neoplastic growths clearly originated from the epithelial lining of these cysts. The presence of cholangiohamartomata and of clusters of proliferating biliary progenitor cells suggests a possible disruption in the homeostasis of the cholangiocellular elements leading to the development of biliary cysts and derived tumors. The theory that would best explain the very high occurrence and broad distribution of biliary cystic diseases in this population is based on a multifactorial model involving both genetic and environmental factors. In that circumstance, this population of ferrets might have a genetic defect associated with a low disease-threshold, or an unusual sensitivity of the biliary elements to environmental "cystogenic" stimuli.

Biliary cysts and biliary cystic neoplasms were recognized as the cause of death in 1.6%, and 11.4%, respectively, of the adult ferrets of this population. With the exception of two cases, all these fatalities involved post-reproductive animals. Therefore, the impact of biliary diseases on the captive propagation of this species is limited. Since life expectancy of free-ranging Black-footed ferrets usually does not go beyond four years, these cystic conditions of the biliary system probably represent an insignificant cause of mortality in the wild.

(42) HEPATOCELLULAR CARCINOMA IN A POLAR BEAR (*URSUS MARITIMUS*) AND A GRIZZLY BEAR (*URSUS ARCTOS*).

HOWARD STEINBERG, Department of Pathobiological Sciences, School of Veterinary Medicine, University of Wisconsin – Madison, Madison, WI 53706; WAYNE I. ANDERSON, Wyeth-Ayerst Research Laboratory, Drug Safety Evaluation Department, Chazy, NY 12921; KATHRYN COYLE, Department of Pathobiological Sciences, School of Veterinary Medicine, University of Wisconsin – Madison, Madison, WI 53706; and ANNETTE GENDRON-FITZPATRICK, Research Animal Resources Center, University of Wisconsin – Madison, Madison, WI 53706.

Hepatic neoplasia in bears is relatively uncommon, and when reported, is more frequently diagnosed as carcinoma originating in the biliary tree and not of primary hepatocellular origin. We present two cases of primary hepatocellular carcinomas in two species of bear.

CASE 1: A 23-year-old, female Polar Bear (*Ursus maritimus*) on exhibit at the Buffalo Zoological Gardens was presented to necropsy with a history of progressive weight loss. Gross lesions were restricted to the liver and consisted of multilobulated, 5-11 cm. diameter, tan & red masses with necrotic centers throughout all lobes. Histologically, the lobulated masses were well delineated and partially encapsulated by thin fibrous bands. Cells comprising the lobules were mildly pleomorphic with distinct cell borders, with occasional multinucleated and karyomegalic cells. Frequent (0-5/HPF) mitotic figures were present. Cells were arranged in both a trabecular and pseudoacinar pattern. The diagnosis of hepatocellular carcinoma with intrahepatic metastases was made.

CASE 2: A 37-year-old, male Grizzly Bear (*Ursus arctos*) on exhibit at the Milwaukee County Zoo was presented to necropsy with a history of progressive arthritis that had become refractory to anti-inflammatory therapy. Severe lesions of chronic degenerative joint disease were present bilaterally in scapulohumeral, humeroradialulnar, coxofemoral, and femorotibial joints. Additionally, a 12 x 8 x 8 cm., dark red, nodular mass was present in the right lateral liver lobe. Microscopically, the mass consisted of variably sized pools of blood separating and dissecting cords and sheets of large polygonal cells with abundant eosinophilic cytoplasm; central large round variably sized nucleus with stippled peripheral chromatin and prominent large nucleolus. Binucleate and multinucleated forms were present. Mitotic figures were present but uncommon. The diagnosis of hepatocellular carcinoma with pelioid features was made. An underlying cause in either case was not determined.

(43) MORBILLIVIRUS AND CALICIVIRUS ANTIBODIES IN POLAR BEARS (*URSUS MARITIMUS*) F'ROM SVALBARD.

MORTEN TRYLAND, Department of Arctic Veterinary Medicine, The Norwegian School of Veterinary Science, NO-9292 Tromso, Norway; ANITA HUOVELAMEN and HANNELE TAPIOVAARA, Department of Virology, National Veterinary and Food Research Institute, P. 0. Box 368, FIN-00231 Helsinki, Finland; ANDREW E. DEROCHER, Norwegian Polar Institute, NO-9296 Tromso, Norway; OYSTEIN WUG, Zoological Museum, University of Oslo, Sars gt. 1, NO-0562 Oslo, Norway; ALBERT D. M. E. OSTERHAUS, Department of Virology, Erasmus University, P. 0. Box 1738, 3000 DR Rotterdam, The Netherlands.

A survey of plasma from 275 polar bears from Svalbard revealed antibodies against morbillivirus in 20 individuals (7.2%) in a virus neutralization test using canine distemper virus (CDV) as antigen. Antibodies against calicivirus were found in 4 individuals (1.5%) in a virus neutralization test with a feline strain (ATCC VR529) as antigen. No antibodies against phocid herpesvirus 1 (PhHV-1; virus neutralization test) or rabies virus (RFFIT method) were detected. The polar bears were captured during the period from 1990 to 1998 by remote injection of a drug filled dart fired from a helicopter (bears > 1 year) or by hand injection (bears <1 year) of the drug Zoletil® (5-10 mg/kg of body mass). The population of polar bears in the Svalbard area has increased since it became protected in 1973, but information on diseases in this and other populations of feral polar bears is scarce. Morbillivirus is previously shown to be endemic among polar bears in the Bering, Chukchi and East Siberian seas (seroprevalence 35.6%), and also among many marine mammal populations, including seals in the Barents Sea. Calicivirus and herpesvirus are also found to be endemic in many marine mammal populations in the arctic, the latter also among harp and hooded seals from the Greenland and Barents seas. On the contrary, marine mammals are only accidentally infected with rabies, since only two cases, one polar bear and one ringed seal (*Phoca hispida*), have been reported. The polar bear at Svalbard are exposed to and infected by morbillivirus and calicivirus, probably through their prey species, and they are responding immunologically by producing antibodies. The lack of antibodies against PhHV-1 may indicate that this virus is not present among the prey species of polar bears in this area, which is mainly ringed and bearded seals (*Erignathus barbatus*), or that PhHV-1 is not able to infect polar bears. We have at this stage no indication that morbillivirus or calicivirus cause disease among polar bears.

(44) EFFECT OF MORTALITY DUE TO VEHICULAR COLLISION ON FLORIDA'S BLACK BEAR POPULATIONS.

MARK W. CUNNINGHAM, Florida Fish and Wildlife Conservation Commission, Wildlife Research Laboratory, Gainesville, FL 32601; J. WALTER McCOWN, Florida Fish and Wildlife Conservation Commission, Wildlife Research Laboratory, Gainesville, FL 32601; THOMAS H. EASON, Florida Fish and Wildlife Conservation Commission, Tallahassee, FL 32399; DAVID S. MAEHR, Department of Forestry, University of Kentucky, Lexington, KY 40546; TERRY GILBERT, Florida Fish and Wildlife Conservation Commission, Tallahassee, FL 32399; DONALD J. FORRESTER, Department of Pathobiology, College of Veterinary Medicine, University of Florida, Gainesville, FL 32610.

Human population growth and development have resulted in the fragmentation of Florida black bear (*Ursus americanus floridanus*) populations and an increased threat of anthropogenic mortality. Since 1976 black bear mortality in the state has been monitored by the Florida Fish and Wildlife Conservation Commission (formerly Florida Game and Fresh Water Fish Commission). Of 897 recorded non-hunting mortalities, 787 (87.7%) were due to vehicular collision. Yearly totals have risen from a low of 0 in 1977 to a high of 91 in 1998. This rise is likely due to a combination of increasing vehicular traffic, increasing bear populations, and greater reporting compliance. Males were 1.56 times more likely to be killed by vehicular collision than females. Cubs and bears of dispersing age also were killed more frequently than bears in other age classes. Seasonal mortality due to vehicular collision was greatest in May-July and October-December. Mortality due to vehicular collision is likely much higher as some bears are able to move off the road following collision and die unobserved. Similarly, results of necropsies and field observations indicate that a substantial number of bears suffer injuries from vehicular collision but survive. Of radio-instrumented bears in Florida, vehicular collision accounted for 6 of 16 (37.5%) recorded mortalities, equaling illegal kills as the major cause of mortality in this group. Population fragmentation and individual mortality due to vehicular collision has likely had a substantial impact on some Florida black bear populations. The Chassahowitzka population has lost at least 23 bears to vehicular collision since 1980, 9 (39%) of which were adult females. Currently the Chassahowitzka population numbers <15 adults. Preliminary results of microsatellite DNA analysis of the Chassahowitzka population indicates virtually complete isolation from larger sympatric populations and a significantly decreased genetic variation.

(45) INFECTION WITH *HELICOBACTER* SPP. IN FREE RANGING LYNX (*LYNX LYNX*) AND RED FOXES (*VULPES VULPES*) IN SWEDEN.

TORSTEN MÖRNER, CAROLINE BRÖJER, MARIE-PIERRE DEGIORGIS, DOLORES GAVIER-WIDÉN, Department of Wildlife, National Veterinary Institute, SE-751 89 UPPSALA, Sweden; HANS-OLOF NILSSON and TORKEL WADTSRÖM, Department of Infectious Diseases and Medical Microbiology, University of Lund, Sölvegatan 23, SE-223 62 LUND, Sweden.

In 1983 unidentified curved bacilli, now named *Helicobacter pylori*, was discovered in the gastric epithelium in humans with active chronic gastritis. Infection with *Helicobacter* is now recognized as the most important cause of human gastritis and also identified as a pathogen for various other domestic, laboratory and zoo animals. The bacterium has also been demonstrated in a few species of wild birds. In wild mammals infections with *Helicobacter* spp or *Helicobacter* -like organisms have been demonstrated in carnivores like red foxes (*Vulpes vulpes*) and cheetah (*Acinonyx jubatus*). We examined the gastric mucosa and livers of 25 free ranging wild lynx (*Lynx lynx*) and four red foxes. The lynx were apparently healthy animals shot in the field and submitted to the National Veterinary Institute to be a part in a health-monitoring program. The red foxes were submitted because of skin lesions caused by *Sarcoptic scabiei.* No animal showed signs of gastritis or hepatitis. Specimens from the gastric mucosa and liver were investigated for the presence of *Helicobacter* by histology, by a 16S rRNA PCR test recognizing the genus *Helicobacter*, by culture and by Urease-test.

No *Helicobacter* or *Helicobacter* like organisms could be cultivated from any animal. In the 16S rRNA PCR test the gastric mucosa from 17 lynx were positive for *Helicobacter*. Seven of these were also positive on the urease test. All livers were negative. In three of the four red foxes investigated with the PCR test *Helicobacter* spp could be demonstrated in the gastric mucosa.

Several helical, coiled, corkscrew-like organisms closely resembling *H. felis* and *H. bizzozeronii* were observed by histological examination of the gastric mucosa. One 400 base-pair PCR-fragment from a lynx *Helicobacter* and two from fox *Helicobacter* were sequenced and compared using the EMBL database with other known *Helicobacter* spp. The fragment from the lynx was 96% homologous to *H. felis*. The PCR-fragments from the foxes showed 98% homology to *H. heilmanni* and 96% homology to *H. felis*, respectively.

The results indicate that infections with *Helicobacter* occur commonly in wild lynx and red foxes in Sweden. The pathogenicity of these organisms is however not known and has to be further evaluated.

(46) HEALTH EVALUATION OF MONGOLIAN GAZELLES (*PROCAPRA GUTTUROSA*) ON THE EASTERN STEPPES OF MONGOLIA.

SHARON L. DEEM, Field Veterinary Program, Wildlife Conservation Society, 2300 Southern Boulevard, Bronx, New York, 10460 USA; MICHAEL J. LINN, Pathology Department, Wildlife Conservation Society, 2300 Southern Boulevard, Bronx, New York, 10460 USA; GEORGE SCHALLER, International Programs, Wildlife Conservation Society, 2300 Southern Boulevard, Bronx, New York, 10460 USA; BADAMJAVIN LHAGVASUREN, Society for the Conservation of the Mongolian Gazelle and Institute of General and Experimental Biology, Mongolian Academy of Sciences, Ulaanbaatar-51, Mongolia; HYAMSUREN, Choibalsan Veterinary Hospital, Choibalsan, Mongolia; KIRK OLSON, International Programs, Wildlife Conservation Society, 2300 Southern Boulevard, Bronx, New York, 10460 USA; ELLEN DIERENFELD, Nutrition Department, Wildlife Conservation Society, 2300 Southern Boulevard, Bronx, New York, 10460 USA; and WILLIAM B. KARESH, Field Veterinary Program, Wildlife Conservation Society, 2300 Southern Boulevard, Bronx, New York, 10460 USA.

Mongolian gazelles (*Procapra gutturosa*), found throughout the Eastern Steppes of Mongolia with remnant populations in Southeastern Russia and Northeastern China, persist as the largest concentration of any large bodied mammal in Asia. Although there is an estimated 2 million Mongolian gazelles still present in Central Asia, little is known about this species' ecology, including their migratory routes. We know even less about their health status, even though intermittent disease epidemics associated with hundreds of thousands of gazelle mortalities have been recorded since the mid-1900s. Current threats to the long-term survival of the Mongolian gazelle include legal and illegal hunting, habitat loss that may result in changes in their historic migratory patterns, and the potential rise in livestock diseases due to recent political events. As part of an ongoing study of the ecology of the Mongolian gazelle, we are conducting a health evaluation of this free-ranging population. In November 1998 and 1999, blood and tissue samples were collected from 26 hunted gazelles during the annual legal harvest. In June 1999, blood samples were collected from 56 neonatal gazelles. Additionally, blood samples were collected from 25 domestic sheep and goats herded in this region. Specific objectives of this study are 1) to evaluate serologic and histologic evidence of infectious and parasitic agents within this population; 2) to monitor the prevalence of infectious and parasitic agents in this population over time; and 3) to compare these findings with domestic sheep and goats that graze along side the gazelles. In this talk, we will present preliminary findings from the first two years of this long-term study and comment on the findings in regard to potential disease interactions between domestic livestock and free-ranging gazelles in Mongolia.

(47) AN UPDATE ON ANTHRAX IN WILDLIFE IN THE KRUGER NATIONAL PARK.

ROY G BENGIS, Veterinary Investigation Centre, Skukuza, Kruger National Park, South Africa.

Epidemiological Aspects of Anthrax

During the period 1991 to 1999, four anthrax epidemics occurred in the Kruger National Park, which afforded an ideal opportunity to study this disease in free-ranging African wildlife populations in this ecosystem.

Several distinct epidemiological cycles appear to be operatiing during these epidemics, but these cannot be considered in isolation, because they augment and interrelate with each other.

A. The Kudu / blowfly cycle

Blowflies (Genera *Chrysomyia and Lucilia)* feed on body fluids of infected carcasses and once engorged they alight on leaves and branches in the nearby vicinity - usually at a height of 1-2 meters from the ground. The blowflies then defecate and regurgitate a "discard droplet" onto the surface of the leaves. Millions of anthrax organisms are present in these droplets, which are on the same height as the Kudu browse level. The Kudu ingest these infected leaves and the cycle is repeated. The blowflies also lay numerous eggs on these carcasses, which develop into maggots that eventually pupate in the surrounding soil. After maturation, these pupae then develop into adult blowflies, completing the cycle. Thus each carcass becomes a blowfly generator and population amplifier.

B. The Scavenger / water cycle

Water holes and drinking cribs become contaminated by vultures and hyaenas which have fed on infected carcasses and then bathe, drink, regurgitate and defecate in the water. Any wildlife species may become infected by this source, but buffaloes are particularly vulnerable because they utilise water holes in large numbers, and the last animals to drink (usually bulls) tend to physically climb into the cribs or wade into water pools. This results in stirring up the sediment (into which the spores have gravitated), thus placing the spores back into suspension, which facilitates ingestion. Water holes may also become infected by animals dying in or near them, - it should be remembered that animals infected with anthrax may become very thirsty and seek water because they are febrile. Many carcasses are generally found close to water holes and drainage lines.

C. The Direct carcass/Predator cycle

Although predators in general are less susceptible to the *Bacillus anthracis*, they are however highly vulnerable in that they feed on infected carcasses ingesting billions of organisms. In this way many lions, several leopards, jackals, and wild dogs became infected during the last epidemic. Most lions develop a massive cellulitis of the head which becomes grossly swollen. In these lions, anthrax bacilli are frequently not detectable in peripheral

blood, but were detectable in smears of the oedema fluid in the head tissues. In other lions, where acute death occurred with no swelling of the head, anthrax bacilli were readily detectable in peripheral blood smears. If predators survive their first few exposures to anthrax, they appear to develop a strong immunity.

D. Other epidemiological factors

1. Kudu are extremely important generators of infection because they develop very high bacterial counts in their blood. They also have very thin skins so their carcasses can easily be opened by even small predators and scavengers, exposing the bacilli to atmospheric oxygen and thus stimulating sporulation.

2. Buffalo, in contrast, generally have low bacterial counts in their blood. They have thick skins and their carcasses are not easily opened except by large predators. They therefore have a much lower spore generating potential.

3. Other species are purely incidentally infected with anthrax and are not important in the maintenance and spread of an outbreak. This is confirmed by analysis of the carcasses documented in these recent outbreaks where kudu and buffalo made up over 70% of all carcasses found.

4. Vultures have both positive and negative inputs in an anthrax outbreak.

a. Positive - they assist the field teams in locating carcasses. They also remove a lot of infectious material from the environment because their digestive processes are capable of destroying most vegetative forms of this organisms - so when a vulture feeds on a fresh carcass - most bacilli present in the meat are destroyed in the vultures gastro-intestinal tract.

b. Negative - when a vulture feeds on an older or opened carcass where sporulation has already occurred-, these resistant spores pass through the gastro intestinal tract of the vulture and their faeces are then infectious. Also blood present on their feet, beaks and feathers are infectious.

5. When anthrax reaches epidemic proportions in free-ranging wildlife, it passes through the population at risk in a wave-like front, with a high incidence of new cases occurring at the leading edge of the front. Behind the front, the number of cases progressively decreases, and after a few weeks, only isolated deaths are seen in the area through which the front has moved. The reason for this progressive decrease in incidence behind the front has not been elucidated, but is probably as a result of avoidance behaviour rather than the presence of an immune response in herbivores. In carnivores, if they survive their primary exposure to anthrax, a progressive immunity appears to develop making them resistant to infection during future exposure.

With the advent of the first good rains, the incidence of new anthrax cases decreases dramatically due to the physical rinsing off of leaves and washing of the environment plus the massive dilution of spores. The availability of numerous new water points as well as new plant growth, results in dispersal of the ungulate populations.

E. Control of Anthrax

It would appear from our experience that anthrax is difficult to control in a large area like the KNP. One can however contain it to some degree, and the measures used include:

a. Locating and burning or burying of most carcasses.

b. Remote inoculation of important rôle players, e.g. Kudu and buffalo as well as endangered species by means of bio-bullets or disposable darts.

c. Disinfection (where possible) of watering points.

d. Strategic veld burning.

F. Conclusion

Anthrax is an indigenous disease of wildlife. It is the only disease within the KNP ecosystem which must kill its hosts in order to propagate itself. There is no sexual predilection, with males and females equally affected. Young animals are less frequently affected.

Anthrax should be judged on its effect on animal populations in an ecosystem, and not on the individual mortalities. The analysis of several outbreaks in the KNP since 1959 have shown that Anthrax never destroys a total population or exterminates species. It appears to be a natural population regulatory mechanism. Only the rare and highly susceptible roan antelope has been endangered by anthrax, but this Park appears to be marginal habitat for this species.

We can live with this situation in the KNP with its large dispersed populations. The disease however has much more serious implications for game farmers on the KNP border, who have small areas, and limited game populations - often introduced at great cost.
This, plus the fact that anthrax remains a threat to domestic stock, and bearing in mind its zoonotic potential, control or containment should be attempted when a major epidemic occurs.

In summary, in the KNP ecosystem, anthrax appears to be primarily a disease of kudu. If one draws the analogy between anthrax and a brush fire, then the vulture is the match, the kudu are the grass which sustains and spreads the fire, and the other species are incidental bushes and trees which get burnt in the fire. This is substantiated by the fact that although kudu constitute only 3,7% of the total ungulate population of the KNP, they represented 42% of all positive anthrax carcasses found in 1990, 51,7% of all positive carcasses found in 1991, 62% of positive carcasses found in 1993, and 60% of all positive carcasses in 1999.

(48) COMPARISON OF HEALTH STATUS BETWEEN MUSKRATS (*ONDATRA ZIBETHICA*) FROM HABITATS CONTAMINATED VS. NONCONTAMINATED WITH HIGHWAY RUNOFFS.

J. D. BORUCINSKA and J. TRETTEL Jr., Department of Biology, University of Hartford, 200 Bloomfield Avenue, West Hartford, Connecticut 06117-1559, USA, Tel. (860) 768-4586, Fax; 860 768-5002, Email: borucinsk@uhavax.hartford.edu

This study was the first in a series of investigations designed to outline the long term effects of automobile derived contamination on the health of animals in their natural habitats. Histopathological lesions and body and organ weight indices were used as bioindicators of health status. Forty muskrats were collected by licensed trappers during February and March of 1998. One half of these were caught in ponds contaminated with automobile derived effluents in Connecticut, USA and the other half was caught in ponds located within agricultural and forested habitats in Vermont, USA. Muskrats were necropsied within 48 hours after death. Body weight (BW), weight of liver and spleen, and gross lesions were recorded for each animal. Samples for histopathology were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned at 5 ? m, and stained with hematoxylin and eosin (H&E). Histopathological lesions were described from blind-coded slides. Two tailed paired Student's T-tests were used to compare the BW, relative liver and spleen weights, and number of microscopic lesions between the two groups. The differences were considered to be significant at P ? 0.05. Mean BW in males and females from VT and CT were significantly different. No statistically significant differences were found in relative liver and spleen weights between the groups. Histological lesions included necrotizing encephalitis associated with *Frenkelia microti*, proliferative endarteritis and endocarditis, dacryoadenitis of Harderian glands, conjunctivitis, hypertrophy/hyperplasia of peribronchial lymphoid tissue, tracheitis and alveolitis, hepatocellular necrosis, hepatic cysticercosis, intestinal giardiasis, *Sarcocystis* sp. cysts in skeletal muscles, osseous metaplasia of bronchiolar cartilage, and a schwannoma and thyroid adenoma. The total number of observed microscopic lesions was 140 in Vermont, and 176 in Connecticut, and this number was significantly higher (P=0.01) in muskrats from Connecticut. In summary, our results indicate decreased BW and increased incidence of microscopic lesions in muskrats from habitats contaminated with gasoline derivatives. The evidence that the above changes are due to gasoline derivatives is only circumstantial, as no tissue concentrations of these chemicals were measured in this study. Further studies should explore the correlation of these two biomarkers with tissue levels of gasoline derivatives.

(49) DECLINE OF THE SOUTHERN SEA OTTER (*ENHYRDA LUTRIS NEREIS*): FURTHER EVIDENCE OF CONTAMINANTS AND DISEASES FROM NON-POINT SOURCES.

DAVID A. JESSUP, MELISSA CHECHOWITZ, JACK AMES, MIKE HARRIS, California Department of Fish and Game, Marine Wildlife Veterinary Care and Research Center, 1451 Shaffer Road, Santa Cruz, CA 95060; KAREN WORCESTER, Regional Water Quality Control Board, 81 Higuera St., Ste. 200, San Luis Obispo, CA 93401; and DAVID M. PARADIES, Bay Foundation of Morro Bay, 909 Santa Ysabel, Los Osos, CA 93402.

The approximately 12% decline of the southern sea otter since the spring of 1995 has been viewed with some alarm by conservationists and indeed, if present trends continue, this "threatened" population may be a candidate for "endangered" status within 2-3 years. A good deal of debate has centered on the role of infectious diseases and parasites, contaminants, nutrition and prey availability, net fishery interactions and other sources of mortality, as loss of adult animals rather than depressed recruitment appear to underlay the decline. Previous work suggests that contaminants (tributyltin) may predispose sea otters to dying from infectious diseases. Indeed mortality rates appear to follow years of higher than normal runoff and subsequent release of a variety of contaminants into the nearshore environment. Recently we collected typical sea otter prey items in Elkhorn Slough, an area with historic contaminant problems, and found that relatively high levels of organochlorine pesticides and their residues are still present. Rudimentary mapping of sea otter infections with toxoplasmosis show clustering of cases in areas with greater numbers of sewage outfalls into the ocean. From both ecological and regulatory perspectives, if nonpoint source pollution, whether via traditional pollutants (contaminants) or via "pathogen pollution", is killing sea otters or predisposing them to infectious diseases that subsequently kill them in numbers sufficient to threaten populations, there may be reason to call for regulatory and management actions.

(50) THE USE OF GELDANAMYCIN TO PREVENT THE TOXIC EFFECTS OF INTERNAL OIL EXPOSURE ON REPRODUCTION OF MINK (*MUSTELA VISON*) AS A MODEL FOR SEA OTTERS.

JONNA A.K. MAZET, Wildlife Health Center, School of Veterinary Medicine, University of California, Davis, California 95616; KIRSTEN V.K. GILARDI, Wildlife Health Center, School of Veterinary Medicine, University of California, Davis, California 95616; DEANA L. FRITCHER, Wildlife Health Center, School of Veterinary Medicine, University of California, Davis, California 95616; and RICHARD J. AULERICH, Department of Animal Science, Michigan State University, East Lansing, Michigan, 48824.

In order to determine whether early embryonic loss in oil-exposed mink can be attenuated by administration of a potential antidote, geldanamycin, we conducted a clinical trial involving female mink divided into 3 experimental groups: Group 1 received Bunker C fuel oil-contaminated feed (n = 19), Group 2 received oil-contaminated feed and the antibiotic geldanamycin (n = 19), and Group 3 served as controls (10 receiving uncontaminated feed, 10 receiving uncontaminated feed and geldanamycin). This clinical trial was devised not only to evaluate a potential treatment for reproductive impairment in oiled sea otters, but also to elucidate the mechanism by which chronic ingestion of petroleum polycyclic aromatic hydrocarbons (PAHs) leads to reproductive toxicity in mink.

We have previously shown that dietary exposure of mink to ecologically relevant levels of crude and fuel oils throughout the breeding season results in significant reduction in reproductive performance. While the mechanism by which this toxicity occurs is unknown, our results were similar to the pattern of early embryonic loss observed in mink fed other environmental contaminants (e.g. PCBs), and we do know that halogenated aromatic hydrocarbons (HAHs) like PCBs and dioxins act through the cellular Ah receptor (AhR) to cause adverse effects in multiple organ systems. It has been suggested that HAHs modulate reproduction in animals by binding to the AhR and triggering the production of the cytosolic protein Src-kinase, which in turn triggers some of the same signal pathways as do endogenous steroid hormones and growth factors, thereby disrupting the normal action of reproductive hormones. We hypothesized that PAHs may act on cells by the same mechanism as do HAHs, explaining embryonic loss in pregnant mink exposed internally to oil.

The number of live kits whelped per female was reduced in the oil-exposed group relative to the controls (3.1 and 4.6, respectively), and the litter size in the oil-exposed and geldanamycin treated group was intermediate (Group 2; 4.0 kits/female). The number of kits surviving to weaning was dramatically reduced in the oil-exposed group (1.1 kits/female) compared with the control group (4.0 kits/litter) with an intermediate weaning litter size in the geldanamycin treated group (2.4 kits/female). Our results indicate that the Src-kinase inhibitor, geldanamycin, may modulate the effects of oil exposure on the reproductive system of mink. This information is *prima facie* evidence that petroleum PAHs are reproductively toxic through the AhR. After further analysis of clinical and anatomic pathologic data, geldanamycin may be considered as a legitimate treatment for pregnant oil-exposed mustelids.

(51) OILED WILDLIFE REHABILITATION: SCIENTIFIC EVALUATION OF SURVIVAL AND BEHAVIOR.

SCOTT H. NEWMAN and JONNA A. K. MAZET, Wildlife Health Center, School of Veterinary Medicine, University of California, Davis, CA 95616; RICK T. GOLIGHTLY, Humboldt State University, Wildlife Department, Arcata, CA 95521; and JAY HOLCOMB, International Bird Rescue Research Center, 699 Potter Street, Berkeley, CA 94710.

Over the past 4 years, California's Oiled Wildlife Care Network (OWCN) has responded to numerous oil spills and cared for over 2000 birds. Rehabilitation of oiled wildlife has been controversial because previous research suggested that for some species commonly cared for in California, rehabilitated birds did not survive very long after release, and if individuals survived, they were unlikely to behave normally. Therefore, many biologists have felt that rehabilitation not contribute to population health or species survival. In order to investigate our survival rates and improve care, the OWCN recent conducted 2 post-release survival studies; a completed project on Western Gulls and an in-progress study on Common Murres. Results from the gull study and preliminary findings from the murre study suggest that birds survive longer than previously expected (at least 235 d for gulls and at least 90 days thus far for murres). Study gulls and murres used similar habitat to free-ranging healthy birds of the same species, and thus they may be contributing to overall population health. Other recent oil spill responses by the OWCN have also demonstrated long-term survival (over 3 years) for rehabilitated American Coots. The recent OWCN research findings suggest that oiled wildlife care can be successful, and an organized science-based response may contribute to populations of wildlife. Our successful oil spill rehabilitation efforts are dependent on well-organized rapid responses, availability of professionally trained response personnel, quick retrieval of oiled wildlife from the environment, and availability of pre-established and appropriately equipped care facilities. With further refinement of techniques in the future, oiled wildlife rehabilitation may be a valuable conservation tool for endangered aquatic birds and other sensitive species.

(52) SEIZURES CAUSED BY DIELDRIN TOXICITY IN A LITTER OF RED FOX (*VULPES VULPES*) KITS.

CATHERINE M. BROWN, Forest Preserve District of DuPage County, Willowbrook Wildlife Center, 525 S Park Blvd., Glen Ellyn, IL 60137.

Between May 17 and May 21, 1999, 4 Red Fox (*Vulpes vulpes*) kits were admitted to Willowbrook Wildlife Center with generalized seizures. All 4 individuals showed CNS depression, hypersalivation, urination and defecation, pale mucous membranes, and ataxia. Canine distemper and acute toxin ingestion were the primary differential diagnoses. Treatment consisted of IV diazepam as needed for seizure control, prophylactic oral administration of activated charcoal to reduce toxin absorption, and administration of IV fluids supplemented with dextrose. Blood work indicated mild regenerative anemias, hyperglycemia consistent with a post-ictal period, and slight increases in ALT and AST. Canine distemper titers were negative. The death of 2 foxes provided the opportunity for complete post mortems, which were unremarkable, and toxicology profiles were submitted. Dieldrin levels in the brain were found to be 10.66 and 12.32 ppm. The remaining kits were treated symptomatically and eventually released. Although low levels of organochlorines are persistent in the environment, the level of dieldrin in these kits indicates an acute exposure from a single source.

(53) DOSE-TITRATION AND SAFETY OF LUFENURON FED TO CAPTIVE WHITE-TAILED PRAIRIE DOGS (*CYNOMYS LEUCURUS*).

KEVIN T. CASTLE Colorado Division of Wildlife, 317 W. Prospect St. Fort Collins, CO 80521; MARGARET A. WILD, Colorado Division of Wildlife, 317 W. Prospect St. Fort Collins, CO 80521; and S. CRAIG PARKS, Novartis Animal Health, P.O. Box 26402, Greensboro, NC 27404.

Plague is a zoonotic disease that impacts populations of prairie dogs (*Cynomys* spp.) and other species, such as black-footed ferrets, that rely on them for food and shelter. *Yersinia pestis*, the etiological agent of plague, is transmitted primarily by the bite of an infective flea. Recently developed compounds used to control fleas in pet animals offer a promising alternative to insecticide dusts for the control of fleas in wild rodents. Lufenuron is a lipid-soluble insect growth regulator with ovicidal and larvicidal activity. Lufenuron is efficacious for controlling fleas in cats and dogs at blood concentrations above 50-100 parts per billion (ppb). A single oral dose of lufenuron has been shown to be effective in controlling the cat flea (*Ctenocephalides felis felis*) for at least 30 days in treated cats and dogs. To date, there have been no studies conducted to determine the duration of lufenuron blood concentrations in any prairie dog species. We compared lufenuron blood concentrations in white-tailed prairie dogs (*C. leucurus*) during periods of activity (nontorpid group) and hibernation (torpid group) during January-March 1999. We hypothesized that if high serum concentrations of lufenuron could be maintained over winter during hibernation or for > 1 mo in active prairie dogs, the compound may be effective for use in breaking the plague cycle. Thirty captive WTPD were fed 300 mg/kg lufenuron; half the animals were allowed to become torpid, while the other half were kept awake. All animals remained healthy throughout the 9 week study period. Prairie dogs in the active group gained weight, while those in the torpid group lost weight over the 9 weeks. Blood was drawn from each animal prior to dosing, one week after dosing, then every other week until week 9 post-dosing. Serum was harvested and tested by HPLC for lufenuron concentration. Blood lufenuron concentration did not differ between the groups one week post-dosing. Concentration in both groups decreased over time, but the concentration in torpid animals declined at a more gradual rate; after weeks 3, 5, and 7, lufenuron levels in torpid WTPD were significantly higher than levels in nontorpid WTPD. After nine weeks, blood levels were again similar, and had approached the limit of detection (10 ppb). Blood levels in nontorpid WTPD declined to <50 ppb after 3 weeks, while levels in torpid WTPD declined to <50 ppb after 7 weeks. Future studies will be required to determine efficacy of lufenuron in controlling fleas on WTPD. If effective blood concentrations are similar to dogs and cats, however, frequent dosing would be required to control flea numbers on prairie dogs and thus break the plague cycle.

(54) DIAGNOSIS, MAGNITUDE, AND REMEDIATION OF LEAD EXPOSURE AMONG WILD BIRDS AND MAMMALS AT A FIREARMS TRAINING FACILITY.

WILLIAM R. DAVIDSON, LYNN A. LEWIS, and JOHN R. FISCHER, Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, The University of Georgia, Athens, GA 30602; ROBERT H. POPPENGA, Laboratory of Large Animal Pathology and Toxicology, New Bolton Center, University of Pennsylvania, Kennett Square, PA 19348; and KATHLEEN A. MORGAN, Environmental and Safety Division, Federal Law Enforcement Training Center, U.S. Department of the Treasury, Glynco, Georgia 31524.

In May 1999, a yellow-rumped warbler (*Dendroica coronata*) and gray squirrel (*Sciurus carolinensis*) found at the Federal Law Enforcement Training Center (FLETC), Glynn County, Georgia were submitted to the Southeastern Cooperative Wildlife Disease Study for diagnosis of a central nervous system disease. Lead poisoning was diagnosed based on detection of 6.2 and 90 ppm lead in liver tissue of the warbler and squirrel, respectively. The case history indicated that additional wild birds and mammals occasionally had been found dead near the FLETC outdoor firearms range complex. In October 1999, 72 wild animals (35 birds and 37 mammals) comprised of 22 different species were collected from a 25 ha area surrounding the outdoor range complex to evaluate exposure to lead and other metals. Liver and kidney tissues were analyzed for lead and other metals, and gizzards/stomachs were examined visually and radiographically for ingested metal fragments. Twenty-four (33%) animals (11 species) had tissue lead levels >1.0 ppm, and 12 of these (6 species) had levels >2.0 ppm. Ingested lead fragments were detected in a brown thrasher (*Toxostoma rufum*) and two white-tailed deer (*Odocoileus virginianus*). In February 2000, seven yellow-rumped warblers and a solitary vireo (*Vireo solitarius*) found dead near the FLETC outdoor ranges also were diagnosed with lead poisoning. Lead fragments on the soil surface of four adjacent outdoor ranges are the principal source of lead exposure at this location. Prior to the recognition of lead poisoning of wildlife at this site, FLETC had begun a series of measures to reduce and eventually eliminate deposition of lead bullets in the environment. These include: 1) a major shift to use of non-toxic ammunition, 2) transferring most firearms training to new baffled ranges (4 side walls and semi-open top) with bullet recovery capabilities, and 3) remediation of existing outdoor ranges to remove existing lead fragments.

(55) A CHARACTERIZATION OF BREVETOXIN IN TISSUES OF MANATEES (*TRICHECHUS MANATUS LATIROSTRIS*) IN FLORIDA.

SCOTT D. WRIGHT and THERESA CODY, Florida Marine Research Institute, Florida Fish and Wildlife Conservation Commission, St. Petersburg, Florida 33701.

Brevetoxicosis has been implicated in the deaths of several marine organisms. They include fish, fish-eating birds, fish-eating marine mammals, sea turtles, and manatees. Brevetoxin is a product of the micro-algae *Gymnodinium breve*. This algae is one of several species that cause harmful algal blooms known locally as red tides. As a result of the investigation of an epizootic of manatees in 1996, brevetoxin was suggested as the most likely cause of the event. As part of the investigation, an immunoperoxidase (IP) assay was developed to visualize brevetoxin in manatee tissues. The advantage of this assay is that it allows the specific identification of brevetoxin and provides a visualization of the location of the toxin in fixed tissues. A disadvantage is that the assay is not quantitative aside from a subjective interpretation of stain intensity. Further, manatees likely encounter brevetoxin several times during their lives and survive. Therefore, the presence of the toxin, as revealed by the immunoperoxidase stain, is not diagnostic.

However, the IP assay can reveal much about brevetoxin in tissues. Preliminary results indicate that brevetoxin has an affinity for lymphocytic tissues and macrophages. It is also present in other tissues including renal tubules and microglial cells. There is also considerable background staining of granulocytes, connective tissues, and hemosiderin. We have performed the IP assay on manatee cases as far back as 1989. We have detected brevetoxin in manatees from around the state. In some cases, the intensity of staining is similar to the animals examined during the 1996 epizootic. However, we have also observed that not every manatee is positive. Additionally, we have found that brevetoxin is consistently found in certain tissues and not others. This consistent profile of staining provides clues on how manatees respond to exposure to brevetoxin, particularly at the lower non-lethal levels.

(56) FACIAL CLEFTS IN NORTHERN LEOPARD FROG TADPOLES (*RANA PIPIENS*) FROM WISCONSIN.

CAROL U. METEYER, D. EARL GREEN, and KATHRYN A. CONVERSE, USGS-BRD National Wildlife Health Center, 6006 Schroeder Rd. Madison, Wisconsin 53711.

Early in our investigations of malformations in the northern leopard frog it became clear that the malformations were the result of teratogenic events occurring during early developmental stages. With this insight, we began an investigation in 1998 examining tadpoles at sensitive stages of development. As part of this two-year investigation, tadpoles just beginning metamorphosis (Gosner stage 40 to 43) were collected and examined. Of the 28 tadpoles collected from one Wisconsin site over ten days, 15 had facial clefts involving degeneration and loss of the suprarostral cartilage. Approximately 2,200 tadpoles have been examined from 15 sites in 4 states and this malformation has not been seen elsewhere. Microscopic examinations of affected tissues showed degenerative changes consistent with apoptosis. Apoptosis is genetically programmed cell death critical to normal remodeling of tissues during development. Facial clefts have been documented in tadpoles of *Xenopus laevis* and *Rana temporaria* experimentally exposed to DDT as well as *Xenopus laevis* tadpoles exposed to corticosteroids. Experimental studies in mammals have also produced facial clefts (including cleft lip and cleft palate) by exposing the developing fetus to; vitamin A; jervine, a steroid alkaloid from the toxic plant *Veratrum californicum;* toxic levels of ethanol; methyl mercury; hyperthermia and others. This is the first report of spontaneously occurring facial clefts in free-living tadpoles.

(57) WEST NILE VIRUS, A NEW EMERGING DISEASE OF AMERICAN WILDLIFE.

ROBERT G. MCLEAN, USGS National Wildlife Health Center, 6006 Schroeder Rd., Madison, WI 53711-6223; SONYA RENEE UBICO, Atitlan Green & Wild EcoSearch, 5605 Champion Circle, Oregon, WI 53575; NICHOLAS KOMAR and NICHOLAS A. PANELLA, Division of Vector-Borne Infectious Diseases, Centers for Disease Control and Prevention, P.O. Box 2087, Fort Collins, CO 80522; LINDA C. GLASER, DOUGLAS D. DOCHERTY, LOUIS SILEO, WALLACE R. HANSEN, and MICHAEL D. SAMUEL, USGS National Wildlife Health Center, 6006 Schroeder Rd., Madison, WI 53711-6223; WARD B. STONE, New York Department of Environmental Conservation, Division of Fish, Wildlife, and Marine Resources, Wildlife Pathology Unit, 108 Game Farm Road, Delmar, NY 12054; and DOUGLAS E. ROSCOE, New Jersey Division of Fish and Wildlife, P.O. Box 400, Trenton, NJ 08625.

The introduction of West Nile Virus (WNV) into the Western Hemisphere resulted in a human epidemic of encephalitis during the summer of 1999 in the New York City (NYC) region. The mosquito-borne virus also caused extensive mortality in crows and infected a number of other bird species, including birds in zoological collections, in the affected area. The American crow (*Corvus brachyrhynchos)* emerged as a symbol of WNV activity in this region because of its apparent high susceptibility to WNV infection. Infected crows may have been responsible for the geographic expansion of WNV into adjoining counties and states. The local and state public health departments began using WNV positive crows to make public health decisions about human risk. Other bird species were also affected; nearly 35% of 700 bird carcasses, including 16 native species, were found positive for WNV.

Field investigations of the WNV epizootic in the NYC region during September 1999, revealed intense virus transmission among peri-domestic birds within the urban zone where human cases occurred. WNV antibody prevalence in birds was 51% in this zone compared to 7% in adjoining counties. Overall, 33% of 430 birds sampled circulated neutralizing antibodies to WNV. House sparrows, chickens, and geese were the most sensitive groups sampled, with more than 60% seroprevalence in each group. Serosurvey investigations conducted during October 1999 in areas surrounding the disease focus found a low prevalence of infection (0.6% Ab positive and 0.1% virus isolation) in 1032 birds of 69 species sampled at 21 sites in New York and New Jersey.

Enhanced surveillance for the detection of WNV outside the NYC region was subsequently established using crow mortality as an indicator of WNV activity. The USGS National Wildlife Health Center and the Centers for Disease Control collaborated with local, state and federal agencies to provide diagnostic assistance in testing dead wild birds and in sampling resident species of birds throughout the Atlantic coastal states to detect the presence of WNV. The only evidence of the virus outside of the New York City region was an infected crow in Baltimore, Maryland. Enhanced passive surveillance of dead birds and active surveillance of live birds will continue during 2000 in the Atlantic and Gulf coastal states to track this deadly virus for naive North American avifauna.

(58) WEST NILE VIRUS-ASSOCIATED MORTALITY IN BIRDS FROM NEW JERSEY.

DOUGLAS E. ROSCOE, New Jersey Division of Fish and Wildlife, P.O. Box 400, Trenton, New Jersey 08625; ROBERT KENT, Mosquito Control Program, P.O. Box 400, Trenton, New Jersey 08625; FAYE E. SORHAGE, New Jersey Department of Health and Senior Services, P.O. 369, Trenton, New Jersey 08625; WAYNE CRANS, Department of Entomology, Rutgers University, 180 Jones Avenue, New Brunswick, New Jersey 08901; ROBERT MCLEAN, LINDA GLASER, DOUGLAS DOCHERTY, and WALLACE HANSEN, US Geological Survey National Wildlife Health Center, 6006 Schroeder Rd., Madison, Wisconsin; and NICHOLAS KOMAR and ROBERT S. LANCIOTTI, Centers for Disease Control, P.O. Box 2087, Ft. Collins, Colorado 80522.

An outbreak of fatal West Nile virus (WNV) infection in humans, horses and birds was recognized late in the summer of 1999 in the New York metropolitan area. A merlin (*Falco columbarius*), found in a moribund state on September 17, 1999 in Cliffside Park, New Jersey on the west side of the Hudson River, died shortly after capture. The bird was slightly emaciated with moderate pectoral muscle atrophy and had encephalitis characterized by generalized gliosis. WNV was cultured from the brain at the National Wildlife Health Center (NWHC). This represented the first evidence of WNV in birds from New Jersey. The prevalence of WNV positive crows submitted to the NWHC and the Centers for Disease Control from New Jersey between September 17, 1999 and December 2, 1999 was 42% (73/172). Additional samples of brain, liver, lung, heart, spleen, kidney and serum from necropsies of 52 birds representing 28 species were submitted to NWHC for virus isolation. Of these species, American crows (*Corvus brachyrhynchos*), a blue jay (*Cyanocitta cristata*), a red-tailed hawk (*Buteo jamaicensis*), and a merlin were positive for WNV. WNV was also cultured from a stray kitten with neurologic signs. No human cases of WNV were detected in New Jersey despite enhanced surveillance and serological screening of 48 suspect cases. WNV was cultured from two pools of trapped mosquitoes (*Culex pipiens* and *Aedes vexans*) collected in Hudson County near where the merlin was found. *Ae. vexans* is the fresh floodwater mosquito and is the primary biting pest for humans in New Jersey. WNV was cultured from a pool of *Ae. vexans*, which had been collected on September 9, 1999. *Cx. pipiens*, an ornithophilic species, breeds in stagnant and polluted pools (ie. rain gutters, untended drains, discarded tires). Mosquito control involves an integrated program of surveillance through mosquito trapping, species identification, virus culturing and serologic monitoring of sentinel chicken flocks and trapped wild birds. Once problem areas are identified control measures are focused on larvae and include draining of small structures (tires, gutters, bird baths etc.), restoration of tidal flow in marshes through open marsh water management, stocking breeding waters with mosquito-eating fish, and the application of larvicides. Adulticides are used as a last resort. The mix of these control measures minimizes the need for large-scale pesticide applications. Monitoring of WNV-associated mortality in crows and other birds will play a significant role in the integrated approach to mosquito control and in assessing the impact of this exotic virus on native avifauna.

(59) AN AVIAN WEST NILE VIRUS EPORNITIC AT A ZOOLOGICAL PARK.

PAUL P. CALLE, TRACEY S. MCNAMARA, BONNIE L. RAPHAEL, ROSANDRA M. MANDUCA, MICHAEL J. LINN, TRACY L. CLIPPINGER, ELIZABETH M. RUSH, and ROBERT A. COOK, Wildlife Conservation Society, Bronx, NY 10460; GEORGE V. LUDWIG, KEITH E. STEELE, JOE MANGIAFICO, JONATHAN F. SMITH, MICHAEL J. TURELL, RANDAL J. SCHOEPP, and TOM LARSEN, United States Army Medical Research Institute of Infectious Diseases, Frederick, MD 21702.

During the summer and fall of 1999 there was an outbreak of human viral encephalitis in New York City, with the earliest human cases occurred in late July. [2] This was later determined to be from infection with a strain of West Nile virus (WNV) identical to a strain circulating in Israel in 1998, and represented the first documented occurrence of WNV in the Western Hemisphere. [3] Human cases were preceded by a WNV epornitic in free ranging birds (especially crows) in New York, with later involvement of birds in Connecticut and New Jersey. [1,3,4] Illnesses and deaths also occurred in horses in New York. [3] Subsequently, non-domestic captive birds in the collections of the Wildlife Conservation Society (WCS) also exhibited morbidity and mortality resulting from infection with WNV. [3,4]

The clinical signs in WCS collection birds were often nonspecific. Many were found dead with no premonitory signs. Several birds had neurologic abnormalities including abnormal head or neck posture, ataxia, tremors, circling, disorientation, unilateral or bilateral posterior paresis, and impaired vision. Generalized signs of illness included depression, anorexia, weakness, weight loss, and recumbency. Hematologic and biochemical changes were variable and nonspecific. Most clinically affected birds died but a few recovered.

The most prominent gross lesions included widespread hemorrhage of the calvarium, meninges, brain, spinal cord, heart, and GI tract; pale foci in the myocardium; hepatosplenomegaly; renal swelling and congestion with a pronounced lobular pattern; and diphtheritic intestinal membranes. Histologic lesions included meningoencephalitis with perivascular cuffing; necrotizing myocarditis; lymphoplasmocytic enterocolitis; pancreatitis; nephritis; necrotizing splenitis; hepatic coagulative necrosis; and widespread visceral congestion and hemorrhage. A provisional diagnosis of WNV infection in birds was based on the gross and histologic lesions. Virus isolation, electron microscopy, immunohistochemical staining, or in situ hybridization was used to confirm viral infection. [4]

Serologic testing of the WCS bird collection was performed to confirm infection of clinical cases, assess the extent of WNV exposure, and to investigate both the time of introduction and potential origin of the virus infecting the collection birds. Heparinized plasma or serum samples were tested by the plaque reduction neutralization test. Serologic results revealed that in addition to the clinically affected birds, many others had been asymptomatically infected, but none of the birds housed indoors had detectable WNV specific antibodies. The earliest serologic confirmation of WCS WNV infection was 9 August 1999 and the first WCS confirmed fatal case was 10 Aug 1999, after the

outbreak had already been recognized in free ranging crows and in humans. There was no serologic evidence that WNV had been present in the WCS collection prior to the summer of 1999. All six of the birds imported by the WCS in 1999 had completed the federally mandated 30 day quarantine at a USDA quarantine facility remote from the outbreak origin, and none had detectable WNV specific antibodies.

WCS halted shipments of birds to other institutions at the onset of the collection bird epornitic, before the cause of the outbreak had been identified. When shipments were resumed, only indoor birds without mosquito exposure or outdoor birds seronegative after the mosquito exposure season ended were approved for shipment. The source of WNV responsible for this outbreak is unknown but it is speculated that it may have entered the United States by way of an infected person, an illegally imported bird or domestic pet, or an unintentionally introduced virus-infected tick or mosquito. [3] WNV is documented to have persisted throughout the winter in both a bird and mosquitoes. It is therefore widely feared that there might be future, recurrent outbreaks of WNV infection in people, horses, and both captive and free-ranging birds in the Northeastern United States. In addition, there is a potential for dissemination of WNV to other parts of the country through the movements of infected people, captive or domestic mammals or birds, free-ranging migratory birds, or virus infected mosquitoes.

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(60) HEALTH RISK ASSESSMENT: A COMPONENT OF THE SITE SELECTION FOR A REINTRODUCED EASTERN MIGRATORY POPULATION OF WHOOPING CRANES (*GRUS AMERICANA*).

JULIE LANGENBERG, Wisconsin Department of Natural Resources, 101 S. Webster St. Box 7921, Madison, WI 53707-7921.

To fulfill recovery plan goals for the endangered whooping crane (*Grus americana*), the U.S./Canada Whooping Crane Recovery Team is planning a re-introduction effort to create an eastern U.S. migratory population of whooping cranes. Ultra-light aircraft will be used to teach captive-reared whooping cranes to migrate between a Wisconsin breeding site and a Florida wintering site. Because health problems have been limiting factors in the success of previous whooping crane reintroduction efforts, the selection process for the breeding and wintering sites and migratory pathway included health risk assessments. Information was gathered from the published literature, Federal and State wildlife health databases, and from field investigation of the sites. Health risks of known or potential importance to wild whooping cranes were considered, including: (1) infectious diseases such as avian cholera, botulism, alpha viruses such as Eastern Equine Encephalitis, and avian tuberculosis; (2) toxins such as lead, pesticides, mercury, mycotoxins, marine biotoxins, and oil; and (3) environmental hazards, such as power lines, excess water salinity, vehicular traffic, and predation. The summarized information was used by the Recovery Team in choosing the initial Wisconsin and Florida release sites, and to design disease monitoring and research plans for the 10 year re-introduction program.

(61) DIAGNOSIS OF DISSEMINATED VISCERAL COCCIDIOSIS IN CRANES USING PCR.

M. JOHNSON, Department of Pathobiology, College of Veterinary Medicine, University of Florida, Gainesville, FL 32610.

Disseminated visceral coccidiosis (DVC) is a disease characterized by the presence of disseminated lymphohistiocytic inflammatory lesions in sandhill cranes (*Grus canadensis*) and whooping cranes (*Grus americana*). The etiologic agent of DVC is a coccidian parasite of the genus *Eimeria*. Currently, diagnosis of this disease requires microscopic identification of the *Eimeria* parasite in tissue samples. However, microscopic identification of this parasite is often difficult due to the small numbers of organisms present or severe autolysis of field-collected specimens. A polymerase chain reaction (PCR) based assay was developed to detect *Eimeria* spp. DNA in frozen tissue samples from cranes known or suspected to have DVC. The PCR assay successfully detected *Eimeria* spp. DNA in tissue lesions known to contain coccidial organisms and also detected DNA in highly suspicious lesions in which organisms were not microscopically visible. Tissue samples that did not contain lesions consistent with DVC and tissue samples from uninfected control birds did not produce a positive result with the PCR assay. This work provides a useful diagnostic tool, the PCR assay, to confirm the presence of coccidian DNA in tissue lesions suspected to be the result of DVC.

(62) TOWERKILL MORTALITY OF MIGRATING PASSERIFORMS: AN "EMERGING DISEASE"?

DONALD J. FORRESTER, Department of Pathobiology, College of Veterinary Medicine, University of Florida, Gainesville, FL 32611; WILLIAM R. EVANS, P.O. Box 46, Mecklenburg, NY 14863; and R. TODD ENGSTROM, Tall Timbers Research Station, Tallahassee, FL 32312.

Casualties resulting from nocturnally migrating birds striking man-made structures (radio, TV, and cell-phone towers, lighthouses, smoke stacks, buildings, bridges, etc.) in various areas of North America have been studied and analyzed over the past 50 years. In the early 1980s it was estimated that more than a million birds died each year in the United States due to collisions with towers. It has been estimated that the annual losses in the late 1990s to tower impacts by songbirds in eastern North America was between 2 and 4 million birds. Most likely this figure will increase as more and more towers and tall buildings are constructed. In 1998 there were an estimated 75,000 man-made obstructions above 200 feet in height in the U.S. and another 100,000 are due to be built in the next 10 years. There are two ways in which birds are killed at communications towers. One is by "blind collision", i.e., when birds fly during times of poor visibility and fail to see a structure in time to avoid a collision. The other way in which they are killed is when there is fog or a low cloud ceiling and lights on the structure reflect off of water particles in the air, resulting in an illuminated area around the tower. When this happens the migrating birds tend to continue flying in the lighted area, since they can no longer navigate by the stars. They are killed when they collide with the tower or its guy wires or even when they collide with each other. Apparently lights do not attract birds from afar, but tend to hold birds that pass within a certain illuminated vicinity. Most of the mortality that occurs at tall towers is due to this second phenomenon, which has been called "the phototactic mechanism." In Florida there were 2,342 towers over 200 feet in height as of November 2, 1998. Of these 1,126 were between 200 and 299 feet high, 1,009 were between 300 and 499 feet, 134 were between 500 and 799 feet, and 73 were over 800 feet. The higher towers are the ones that cause the most mortality. Details obtained from a 28-year study of mortality of over 44,000 birds representing 189 species at a 1,010 foot TV tower in Leon County, Florida will be presented.

(63) SPATIAL AND TEMPORAL DISTRIBUTION OF AVIAN VACUOLAR MYELINOPATHY IN AMERICAN COOTS (*FULICA AMERICANA*) IN THE SOUTHEASTERN UNITED STATES.

JOHN FISCHER, ROBERT LONG, and DAVID GREGORY, Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, University of Georgia, Athens, GA 30602; KAREN ROWE, MARK CLARK, and BUDDY BAKER, Arkansas Game and Fish Commission, 31 Halowell Lane, Humphrey, AR 72073; CHRISTOPHER YEE, Texas A&M University, PO BOX 2174, Kingsville, TX 78363; JAMES OZIER and PHILLIP SPIVEY, Georgia Department of Natural Resources, 116 Rum Creek Road, Forsyth, GA 31029; THOMAS AUGSPURGER, U.S. Fish and Wildlife Service, PO Box 33726, Raleigh, NC 27636; and KAREN GAINES, Savannah River Ecology Laboratory, PO Drawer C, Aiken, SC 29802.

Since 1994, avian vacuolar myelinopathy (AVM) has been recognized as a cause of death of bald eagles (*Haliaeetus leucocephalus*) in the southeastern United States. The cause of AVM remains unknown despite extensive field and laboratory investigations. Lesions of AVM also occur in American coots, and it is suspected that eagles are exposed to the causative agent via ingestion of affected coots. The objectives of this study were to determine the geographic and temporal distribution of coots with AVM and to evaluate the relationship between clinical disease and microscopic lesions. During the 1998-1999 approximately 53,000 coots were observed for clinical signs of AVM at 28 sites in eight southeastern states and brains from 943 coots were examined microscopically. During the 1999-2000 migratory season, more than 117,000 coots at 36 sites in 13 states have been observed, and brains from more than 1,300 coots have been examined to date. Lesions of AVM were detected in coots at six locations in Arkansas, Georgia, North Carolina and South Carolina during the first year, and in coots at the same six locations during the second year plus two sites where coots had tested negative the first year. Brain lesions often were present in coots that were regarded as clinically normal. During the first year, samples were obtained from coots on multiple occasions at two sites and during the second year, serial sampling occurred at four sites. Affected coots were not found in October, but AVM lesions were detected in coots at the same sites in November. The severity of clinical disease in coots varied by location and year, and it appeared to peak in early December. At one Georgia site, AVM lesions were found in coots sampled monthly from November through March, although clinical signs were not observed after mid-December. Lesions of AVM were not detected in coots at the site in April. Results of this study indicate that AVM may be more widespread in the southeastern U.S. than previously suspected. Because the correlation between signs of neurologic disease and the presence of lesions is poor, collection of coots and microscopic examination of brain are necessary to determine whether AVM is present at a site.

(64) EARED GREBE MORTALITY AT THE SALTON SEA: REVIEW OF FINDINGS AND NEW STUDIES.

J. CHRISTIAN FRANSON, LYNN H. CREEKMORE, CAROL U. METEYER, TONIE E. ROCKE, and MARK J. WOLCOTT, U.S. Geological Survey, National Wildlife Health Center, Madison, Wisconsin 53711; and TAHNI JOHNSON, Salton Sea Authority, Calipatria, California 92233.

In late 1991 and early 1992, an undiagnosed disease occurred in eared grebes (*Podiceps nigricollis*) at the Salton Sea. Carcasses of 46,040 eared grebes were picked up and estimates of total mortality for this event were as high as 145,000, making it the largest epizootic in eared grebes ever documented. Since 1992, annual losses of eared grebes at the Salton Sea have been as high as several thousand birds. Affected grebes often exhibit unusual behaviors, such as congregating at freshwater drains, coming out on shore, and excessive preening. The feathers of affected grebes frequently appear disheveled or wet. From 1991 through early 2000, nearly 200 eared grebe carcasses from the Salton Sea were necropsied, but the primary cause of the die-offs remains unknown. No cause of mortality has been diagnosed in about one-half of the grebes examined, although a variety of pulmonary, hepatic, and skin lesions were noted in some of the carcasses. Diagnoses in the remainder of grebe carcasses have included avian cholera, emaciation of unknown cause, trauma, avian botulism, and other miscellaneous conditions. Salt toxicity and exposure to selected contaminants were investigated, but ruled out as primary causes of mortality. In the autumn of 1999, we initiated a new study of eared grebe mortality at the Salton Sea and will focus our efforts on avian cholera; avian botulism; physiological alterations and feather disruption; and, in collaboration with Wright State University, algal biotoxins.

(65) MORTALITY OF THE COMMON LOON (*GAVIA IMMER*) IN NEW ENGLAND, 1988 TO 1999.

INGA F. SIDOR, MARK A. POKRAS, and ROSE MICONI, Wildlife Clinic, Tufts University School of Veterinary Medicine, 200 Westboro Rd, North Grafton, MA 01536.

Findings are presented on 466 common loons (*Gavia immer*) found dead or moribund in New England between 1988 and 1999. Goals of this study were to identify reasons for mortality, quantify natural versus anthropogenic causes, and investigate their possible interaction. Cause of death varied by age group. Primary mortality in chicks (n =101) was from intraspecific loon trauma (28%) or other trauma (32%). Death in immature loons (n = 60) was primarily from aspergillosis (31%) and trauma (20%). Adult loon mortality differed significantly between breeding and wintering populations. Wintering adults (n = 91) were affected by trauma (15%), infection (15%) or unknown causes (29%), and had significantly poorer body condition than breeding loons. In breeding adults (n = 214) lead poisoning from ingested fishing weights was the greatest cause of death (49%). Loons dying from lead toxicity were in significantly better body condition than loons dying from other causes. Overall, direct anthropogenic factors accounted for 50% of all loon mortality in this study. Data suggest the presence of synergistic factors that combine to cause mortality, as has been found in previous studies. Because of this study's scope, these data appear to be a good representation of loon mortality in New England. Results highlight the importance of human influences in conservation and management of the common loon in New England.

(66) DISTRIBUTION OF DUCK VIRUS ENTERITIS VIRUS DURING ACUTE INFECTION IN NATURALLY INFECTED DUCKS.

ELIZABETH W. HOWERTH and MOLLY MURPHY, Department of Pathology, College of Veterinary Medicine, University of Georgia, Athens, GA 30602; BRADD C. BARR, California Animal Health and Food Safety Laboratory, University of California, Davis, CA 95616; CHARLOTTE QUIST, Athens Diagnostic Laboratory, College of Veterinary Medicine, University of Georgia, Athens, GA 30602; and NANCY J. THOMAS, National Wildlife Health Center, 6006 Schroeder Road, Madison, WI 53711.

Duck virus enteritis (DVE) virus is an alpha herpesvirus that infects a variety of waterfowl. Distribution of virus in various tissues during the acute phase of infection is poorly characterized. While it is still uncertain whether this virus has a latent state similar to other alpha herpesviruses, distribution of virus during the acute phase could give important clues as to possible sites of latency. Using a combination of immunohistochemistry, in situ hybridization, and PCR we studied the distribution of DVE virus during the acute phase of infection. Paraffin embedded brain and visceral organs from 10 ducks (muscovy, Pekin, mallard, and mallard hybrids) naturally infected with DVE virus were examined; 2 uninfected ducks served as controls. DVE viral antigen and viral DNA were detected in many different cell types in a variety of visceral organs, including the digestive tract, reproductive tract, kidney and its excretory ducts, liver, and spleen. In tubular organs, infected cells included epithelium, inflammatory cells, and fibroblasts. Hepatocytes and bile duct epithelium in liver, endothelial cells in kidney, and cells of the sheathed arterioles and macrophages in spleen contained virus. Serosal and mesenteric vessels were severely involved and virus was detected in endothelium, tunica muscularis, and adventitial cells. Cells in ganglia and nerves of the autonomic nervous system supplying the digestive system and other viscera contained DVE virus. While virus was not detected in brain by immunohistochemistry or in situ hybridization, viral DNA was detected in brain by PCR. During acute infection virus is widely distributed and the distribution of the virus is similar to other acute alpha herpesvirus infections. Distribution of the virus indicates that both fecal and urate shedding probably occur. Involvement of the reproductive tract suggests that virus infected eggs may be laid. Findings would support the possibility of DVE virus establishing latency in the nervous system. Hopefully the techniques developed for this study can be utilized to clearly prove that DVE virus becomes latent in nervous tissue of infected waterfowl.

(67) *MYCOPLASMA GALLISEPTICUM* FROM EVENING GROSBEAKS (*COCCOTHRAUSTES VESPERTINUS*) AND PINE GROSBEAKS (*PINICOLA ENUCLEATOR*) WITH CONJUNCTIVITIS IN QUEBEC, CANADA.

IGOR MIKAELIAN, Canadian Cooperative Wildlife Health Centre and Centre Québécois sur la Santé des Animaux Sauvages, Département de Microbiologie-Pathologie, Faculté de Médecine Vétérinaire, Université de Montréal, 3200 Sicotte, C.P. 5000, Saint-Hyacinthe, J2S 7C6, PQ, Canada; DAVID H. LEY, Department of Farm Animal Health and Resource Management, College of Veterinary Medicine, North Carolina State University, 4700 Hillsborough St., Raleigh, NC 27606; R. CLAVEAU, Clinique vétérinaire Claveau-Dubord-Blue, 1310 Bd du Royaume, Chicoutimi, G7H 5B1, PQ, Canada; and M. LEMIEUX, Ministère de l'environnement et de la faune, 605 de la Station, Shawinigan, G9N 1V9, PQ, Canada.

Mycoplasma gallisepticum conjunctivitis emerged in 1994 as a disease of free-flying house finches (*Carpodacus mexicanus*) in the Mid-Atlantic region of the United States and has since spread to house finches throughout their entire eastern range including south-eastern Canada. The disease is now endemic in this species and is clinically characterized by conjunctivitis, sinusitis and debilitation. A similar clinical disease has been observed in American goldfinches (*Carduelis tristis*) and the same apparent strain of *M. gallisepticum* has been isolated from affected birds of this species.

An outbreak of conjunctivitis affected evening grosbeaks (*Coccothraustes vespertinus*) and pine grosbeaks (*Pinicola enucleator*) in Quebec, Canada, during the winter of 1998-99. Observations at 13 feeding stations indicated that 1 to 30% of birds of these species had clinical signs consistent with mycoplasmal conjunctivitis. Sick birds were thin and had unilateral or bilateral catarrhal and lymphoplasmacytic conjunctivitis and rhinitis, and mucopurulent infra-orbital sinusitis. Samples of periorbital tissues and conjunctivas from affected evening and pine grosbeaks were positive for *M. gallisepticum* by polymerase chain reaction (PCR), and mycoplasmal organisms isolated in culture were identified as *M. gallisepticum* by direct immunofluorescence. Random amplified polymorphic DNA (RAPD) fingerprints of grosbeak *M. gallisepticum* isolates were essentially identical to RAPD fingerprints of isolates from house finches (*Carpodacus mexicanus*) and American goldfinches (*Carduelis tristis*).

These observations suggest that the 'finch strain' of *M. gallisepticum* is causally implicated in this recent outbreak of conjunctivitis in pine grosbeaks and evening grosbeaks in Canada. The effect of *M. gallisepticum* infection on pine grosbeak and evening grosbeak populations is difficult to estimate. Based on the large number of birds affected and the severity of the lesions, a negative population impact is expected. Persistence of the finch strain of *M. gallisepticum* in grosbeak populations is yet to be determined. However, clinical infection was not observed in grosbeaks after May 1999. This report and others indicate that at least five species of free-flying fringillid birds have experienced conjunctivitis associated with a single strain of *M. gallisepticum*. Some of these species may now constitute reservoirs for this pathogen. The potential for transmission of this *M. gallisepticum* strain to other wild bird species is undetermined and remains a concern.

(68) SYSTEMIC *ISOSPORA*-LIKE COCCIDIOSIS IN A NORTHERN ORIOLE (*ICTERUS GALBULA*).

JEAN A. PARÉ, Department of Surgical Sciences, School of Veterinary Medicine, University of Wisconsin, 2015 Linden Drive West, Madison, WI 53706; and SANDRA R. BLACK, Calgary Zoo, PO Box 3036, Station B, Alberta T2M 4R8, Canada.

A 51-gram adult male Northern oriole (*Icterus galbula*) died in the Aspen Woodland Aviary of the Calgary Zoo. The bird had sustained obvious crushing trauma, but what appeared like multiple minute translucent gelatinous cysts were observed on the serosal surface of viscera. Histologically, these cysts were conspicuous, roughly circular, cellular aggregates of homogeneous histiocyte-like cells. Such aggregates were particularly prominent in the spleen, testes, and lungs. Aggregates were also identified in the serosa of viscera, as well as in the bone marrow of the first cervical vertebral body. The nucleus of most of these histiocytic cells was indented or slightly displaced by intracellular organisms. The size and shape of these organisms were most consistent with protozoal zoites. In the sections of intestine, there were various stages of coccidial development within enterocytes. A diagnosis of systemic *Isospora*-like coccidiosis was made. This case report documents incidental systemic coccidiosis in the Icteridae.

(69) MODELING AVIAN POX IN HAWAII.

CHARLES VAN RIPER, III and SANDRA G. VAN RIPER, US Geological Survey, Forest and Rangeland Ecosystem Science Center, Colorado Plateau Field Station and Department of Biological Sciences, Box 5614 Northern Arizona University, Flagstaff, Arizona 86011; and WALLACE R. HANSEN, National Wildlife Health Laboratory, US Geological Survey, Biological Resources Division, Madison, Wisconsin 53711.

Analysis of free flying wild birds, coupled with field and laboratory experiments were conducted on the island of Hawaii in order to determine the epizootiology and potential impact of the introduced disease, *Poxvirus avium*, on forest birds. At 16 study sites, from sea level to tree line in mesic and xeric habitat, birds were captured from 1977-1980 and examined to determine the incidence and altitudinal distribution of pox-infected individuals. Laysan finch (*P. canatans*) were placed as sentinels and controls at each station for a 2-week period in order to document transmission of pox virus infections to native birds. We also documented distributions and activity cycles of potential avian pox vectors. Infected birds from the wild were brought into the laboratory in order to determine the course of infection in native versus introduced species. This study confirms that *Poxvirus avium* was present from sea level to tree line, concentrated in the mid-elevational ranges in the ecotonal area where vectors and native birds had the greatest overlap. Native forest birds were: (a) more susceptible to infection than were introduced species; (b) most likely to be infected during the wet season; and (c) found to have a lower prevalence of pox virus infections in xeric when compared to mesic forests. Temporal as well as elevational differences in wild bird incidence levels were apparent throughout the annual cycle. Avian pox virus probably did not reach epizootic proportions on Hawaii until after introduction of the mosquito and domestic birds in the 1800s, and since then has had a negative impact on the population dynamics of native forest birds. Today, coupled with avian malaria, avian pox is one of the major conservation concerns in Hawaii. This introduced disease is causing a continued decline of native birds and is restricting abundance, distribution, and recovery of native bird species in Hawaii.

(70) RESPIRATORY DISTRESS IN AN ADULT SHORT-EARED OWL.

CHRISTINE FIORELLO, Center for Environmental Research and Conservation, 1200 Amsterdam Avenue, Columbia University, New York, NY 10027; and JOHN GLIATTO, Department of Pathology, Tufts University School of Veterinary Medicine, 200 Westboro Road, North Grafton, MA 01536.

An adult female short-eared owl presented to the Tufts University Wildlife Clinic on 12 August 1999 with severe dyspnea. She had been brought into captivity as an adult nine years previously because of a wing injury that left her flightless; she was therefore at least 11 years old. She had a two-week history of inappetance that her owner had attributed to the heat. Upon presentation the bird was placed immediately into an oxygen cage. After 30 minutes in oxygen, a brief physical exam was performed. The owl was open mouth breathing with greater effort on inspiration. No abnormal sounds were ausculted. She was thin at 306 g and had slightly pale mucous membranes. The owl was treated with subcutaneous fluids and 50% dextrose on her oral mucous membranes. Her condition deteriorated rapidly with handling, so no further treatments or diagnostics were attempted. The next day she was anesthetized with isoflurane for diagnostic tests. Blood was taken for a complete blood count (CBC), chemistry profile, and *Aspergillus* titer. Lateral and ventro-dorsal radiographs, and a tracheal culture were taken. She was then given additional subcutaneous fluids and 8.8 mg of enrofloxacin intramuscularly and returned to the oxygen cage. Radiographs revealed diffuse fluffy white opacities throughout her thoracic air sacs and lungs. The differential diagnosis included aspergillosis, neoplasia, and bacterial pneumonia. Nebulization twice daily with clotrimazole in DMSO and saline was begun, and 15 mg of itraconazole once a day was added to her food. Despite aggressive antifungal and antibacterial treatment, she died two days after presentation. A complete necropsy was performed. The *Aspergillus* titer was negative, and the tracheal culture grew *E. coli* and *Proteus mirabilis*, both of which were sensitive to enrofloxacin. The CBC and chemistry profile were unremarkable. Grossly, the lungs were effaced by a mass consisting of grey-white to pink nodular tissue. Histologically, the lung tissue was diffusely involved. The cells of the mass had round to elongate hyperchromatic nuclei with scant or moderate amounts of cytoplasm; there were four to seven mitotic figures per high power field. Some tumor cells formed small solid spherical lobules separated from the surrounding tumor cells by a cleft-like space. These regions had a resemblance to immature glomerular tufts, and were bordered by a clear space resembling Bowman's space. The diagnosis was poorly differentiated tumor of mesenchymal cell origin. There was a small focus of similar cells in the kidney, which may have represented an unrelated blastemal nodule (remnant of metanephric blastema that did not develop into mature renal tissue) unrelated to the neoplasm, a metastatic lesion, or the primary neoplasm. One possible differential for this tumor is a nephroblastoma. Such a diagnosis would be extraordinarily unusual because of the age of the bird and the site of the tumor. Immunocytochemical studies were performed to obtain a definitive diagnosis. The results were somewhat equivocal, but not strongly supportive of a nephroblastoma. The tumor was positive for vimentin, indicating its mesenchymal origin, but negative for cytokeratin, an element found in most nephroblastomas. Although the final answer is unknown, this was an interesting case because neoplasms are rarely reported in raptors.

(71) CAUSES OF MORTALITY OF THE PUERTO RICAN PARROT (*AMAZONA VITATTA*).

LAURIE A. BAETEN and F. JOSHUA DEIN, USGS-BRD National Wildlife Health Center, 6006 Schroeder Road, Madison, Wisconsin 53711.

The Puerto Rican Parrot (*Amazona vittata)* was one of the first species listed under the U.S. Endangered Species Act and remains one of its most critical members. For over 30 years, efforts have focussed on the species life history, habitat conservation (Snyder et al. 1987, Wilson et al. 1994) and wild bird nest management. Starting in 1975, wild parrot eggs and chicks were brought in from the wild to develop a captive population. Currently there are approximately 100 parrots held in captive breeding facilities at Luquillo and Rio Abajo aviaries, Puerto Rico, under the stewardship of the U. S. Fish & Wildlife Service and the Puerto Rico Department of Natural and Environmental Resources. While significant progress has been made in habitat conservation, reproductive success has been low and/or variable in both the captive and wild populations.

As part of an ongoing effort to evaluate the captive flock health and management, an historical review of parrot mortality was undertaken. A total of 89 Puerto Rican parrot mortalities were documented in the medical records between February 1976 and August 1999. The majority of the carcasses were submitted to the National Wildlife Health Center or Southeastern Cooperative Wildlife Disease Study for post mortem evaluation. These consisted of 26 adult parrots held in captivity and 63 chicks that were wild caught or captive born.

Of the 30 adult (greater than 1 year of age) mortalities, a definitive cause of death was determined in 50% (15) of the cases. Death secondary to trauma was the most frequent diagnosis (7, 23%). Visceral gout was diagnosed in three cases (10%); one being a parrot less than two years of age with gout secondary to a nephroblastoma. Two other tumor types were noted in adult mortalities; a thyroid carcinoma was diagnosed in a 13 year old female, and a thymoma was an apparent incidental finding in a 5 year old male. Thyroid dysplasia was noted as an incidental finding in two other cases. Other miscellaneous causes of death included cardiovascular disease (2), hemorrhage (2) and peritonitis (1).

A group of 10 adult parrots have undetermined causes of death with similar histopathological lesions within the liver or liver/kidney. This group, presented from 1988 to 1997, will be reevaluated with the knowledge that has emerged in avian medicine in the past 10 years. Further testing of tissues and histopathological review are planned.

Fifty-nine chick mortalities were noted from 1981 thru the 1998 breeding season. Seventy-one percent (42) of these chicks died before 30 days of age. Twenty-seven percent (16) died before five days of age. Sixty-eight percent (40) were captive born chicks. Twenty-two percent (13) of these cases did not have post mortem evaluations available for review. Cause of death could not be determined in 12% (7) of the cases due to the condition of the carcass. Pneumonia was the most frequent cause of death in parrot chicks (29%, 17). Aspergillosis was the cause of mortality in 10% of the 59 cases (6). Malnutrition/starvation/neonatal death was found in 7% (4) of the cases. Trauma was noted as the cause of death in 7% (4) of the cases. Myiasis was found to contribute

to the cause of death in 2 chicks obtained from the wild. Scoliosis was noted in two chicks; one captive reared, the other wild born. Septicemia or omphalitis was noted in 2 cases.

Four fledgling (4 to 10 months of age) mortalities were evaluated. The cause of death for these four cases included: visceral gout, pneumonia with pericarditis, zinc toxicosis, and a myocardial rupture.

The primary objective in this investigation was to determine if there was a pattern in mortalities that could be identified as contributing to the poor population growth. Adult parrot losses have been minimal, with an average of only four losses per year. Only two of the etiologies have exhibited five or more mortalities. As the data indicates, the only disease pattern that predominates is the high prevalence of pneumonia in the chick population. As the majority of the chick mortalities and those with pneumonia appear to be in the same age bracket (less than 30 days of age), further evaluation of the contributing factors will be pursued. In addition, continued efforts in flock management will revolve around reproductive and nutritional evaluations.

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(72) EXPERIMENTAL INFECTION OF MONTANA HOUSE FINCHES (*CARPODACUS MEXICANUS*) WITH THE HOUSE FINCH STRAIN OF *MYCOPLASMA GALLISEPTICUM*.

KRISTY R. FARMER, SHARON R. ROBERTS, and GEOFFREY E. HILL, Department of Biological Sciences, Auburn University, Auburn, AL 36849.

Since 1994 an epidemic of mycoplasmal conjunctivitis has spread throughout the eastern population of the house finch (*Carpodacus mexicanus*), leading to a significant reduction in this population. Since 1995 we have monitored the impact of this new pathogen on the Auburn University house finch population. Data from an on-going serological survey of this population has documented a steady decline in the prevalence of conjunctivitis during the peak months of late summer and early fall. However, the annual prevalence has remained steady.

The infection has not yet been reported from house finch populations in the west and we would hypothesize that this population, like the eastern population, may be highly susceptible to the infection. To test this we have experimentally infected house finches from Montana with the house finch strain of *Mycoplasma gallisepticum* (MG). Twenty-four house finches from Montana were shipped to Auburn and quarantined for six weeks at the Auburn University aviary. All of the birds were negative for antibodies to MG when tested by the serum plate agglutination assay and MG could not be detected in any bird by PCR. We tested two methods of infection. Two flocks of eight birds each were infected by eye-drop and by dripping the inoculum into the trachea. One of these flocks was inoculated with an isolate from 1995 and the other flock was inoculated with an isolate from 1999. The eight remaining birds were exposed to a house finch with conjunctivitis trapped on the Auburn University campus. The sixteen birds infected via direct inoculation with both isolates developed conjunctivitis within 4-6 days post infection and all had seroconverted by 9-12 days post infection. All infections were confirmed by PCR using MG-specific primers. There were no differences between the 1995 isolate flock and the 1999 isolate flock. The eight birds exposed to the diseased Auburn house finch first showed sign of conjunctivitis 9 days post initial exposure, most were sick within eighteen days, however a few birds did not develop conjunctivitis until 25 days post exposure. Twenty-one of the twenty-four (88%) birds died during the twelve-week study. This is reminiscent of the eastern house finch population at the outset of the epidemic, demonstrating that western house finches are highly susceptible to infection with MG.

(73) EVALUATION OF *BRUCELLA ABORTUS* VACCINE STRAIN RB51 IN PREGNANT REINDEER, A SAFETY STUDY.

SUE HAGIUS, Department of Veterinary Science, LSU AgCenter, Baton Rouge, LA 70803; JOHN BLAKE and JULIA BEVINS-DUCE, Institute of Arctic Biology, University of Alaska-Fairbanks, Fairbanks, AK 99775; GERHARDT SCHURIG, College of Veterinary Medicine, Virginia Tech, Blacksburg, VA 24061; and TODD FULTON and PHILIP ELZER, Department of Veterinary Science, LSU AgCenter, Baton Rouge, LA 70803.

Brucellosis is a bacterial disease, which is endemic in most Alaskan reindeer herds, causing reproductive losses and lameness. *Brucella suis*, the causative agent, is also pathogenic to man. In Alaska it has been thought that eradication of brucellosis in reindeer herds was not feasible because of extensive range conditions and co-mingling with caribou. Therefore efforts to control this disease have been through vaccination utilizing a killed *Brucella suis* biovar 4 vaccine. However, using the currently available diagnostic tests which are based on serology, one cannot distinguish between vaccinates and field-strain infected reindeer. In an effort to overcome this problem, we propose using *Brucella abortus* RB51, an attenuated, rough vaccine (USDA official brucellosis vaccine for cattle), which when administered to ruminants does not cause seroconversion on the standard diagnostic tests for brucellosis. To test the safety of RB51 in reindeer, 22 mid-gestational pregnant females were subcutaneously injected with 1×10^9 colony forming units of the commercially available vaccine. Animals were monitored, and the appropriate bacteriological samples taken upon abortion or delivery. Fifty-five percent (12/22) of the reindeer aborted or had stillbirths; ten cows had live calves. Evaluation of tissue or swabs from both live and dead animals indicated a 68% (15/22) infection rate. Isolates were determined to be *B. abortus* RB51. This is the first report in ruminants where the attenuated vaccine strain RB51 caused significant losses in a vaccinated population; therefore, we would not recommend vaccination of pregnant reindeer at this point of gestation or with this dose of vaccine until further studies are performed.

(74) PREVALENCE OF NEURONAL LIPIDOSIS (NEURONAL VACUOLATION) AND ANAL SAC CAPILLARIASIS IN RACCOONS (*PROCYON LOTOR*) FROM TWO GEOGRAPHICAL LOCATIONS IN THE USA.

AMIR N. HAMIR and JUDI STASKO, National Animal Disease Center, Agricultural Research Service, United States Department of Agriculture, 2300 Dayton Road, Ames, IA 50010.

Recently, two previously unrecognized conditions, namely, neuronal vacuolation (neuronal lipidosis) and anal sac capillariasis were described in raccoons (*Procyon lotor*) from Oregon. Subsequent studies showed that both conditions had high prevalence rates (42% and 97%, respectively) in that state's raccoon population. Since raccoons in Oregon are not geographically isolated from raccoons in other parts of the country, it was suggested that a similar high prevalence of these conditions could be expected in raccoons from other areas of the USA. To test this hypothesis, we examined brains and anal sacs of additional raccoons from Oregon and Iowa for the presence of neuronal vacuolation (neuronal lipidosis) and anal sac capillariasis, respectively. During a 12-month retrospective study, 36 raccoons were examined from these two geographical locations. Microscopic evidence of neuronal lipidosis was seen in 75% and 0% from raccoons in Oregon and Iowa, respectively. Electron microscopic examinations of the vacuolated neurons revealed membrane bound lipid-like material within neuronal perikarya. Absence of neuronal lipidosis in raccoons from Iowa suggests that factors that induce the accumulation of lipid within neurons are not present in this state. These factors may be environmental or genetic. However, since the raccoons in Oregon are not restricted by any geographical boundaries, the possibility of a genetic etiology in raccoons from that state appears to be less likely. Anal sacs of raccoons from both locations were found infected with *Capillaria* spp., and these were seen in 93% of the animals. Although most anal sacs in the present study were heavily infected with the nematodes, impaction of the anal sacs, which has been previously reported with this condition, was not seen in any of the examined raccoons.

(75) THE FLUORESCENCE POLARIZATION ASSAY AND OTHER SEROLOGICAL ASSAYS FOR THE DETECTION OF ANTIBODIES TO *BRUCELLA ABORTUS* IN BISON IN SERUM AND WHOLE BLOOD.

D. GALL, Canadian Food Inspection Agency, Animal Diseases Research Institute, 3851 Fallowfield Road, Nepean, ON, K2H 8P9, Canada; D.O. JOLY, Department of Biology, University of Saskatchewan, 112 Science Place, Saskatoon, SK S7N 5E2, Canada; P. SMITH and K. NIELSEN, Canadian Food Inspection Agency, Animal Diseases Research Institute, 3851 Fallowfield Road, Nepean, ON K2H 8P9, Canada; L. FORBES, Canadian Food Inspection Agency, Animal Health Laboratory, 116 Veterinary Road, Saskatoon, SK, S7N 2R3, Canada; D. DAVIS, Department of Veterinary Pathobiology, Texas A & M University, College Station, TX 77843; P. ELZER, Department of Veterinary Science, Louisiana State University, Baton Rouge, LA 70803; and S. OLSEN, Bacterial Diseases Research Unit, National Animal Disease Center, Agricultural Research Service, Ames, IA 50010.

A number of serological tests were compared with the fluorescence polarization assay (FPA) for the detection of antibodies to *Brucella abortus* in bison (*Bison bison*) in serum and whole blood. The sensitivity and specificity of the FPA in the preliminary evaluation using serum from culture positive animals and animals from areas with no apparent clinical or epidemiological evidence of brucellosis were 92.1% (n = 38) and 99.4% (n = 2807), respectively. The sensitivity and specificity in a subsequent blind study using serum from culture positive animals and animals of known negative status, the FPA was 96.3% (n =54) and 97.6% (n = 160), respectively. The relative sensitivity and specificity for the whole blood FPA using the serum FPA as the standard was 97.4% (n = 38) and 100% (n = 36), respectively. In a double blind preliminary study conducted on bison vaccinated with *B. abortus* strain 19 (n = 3), the FPA could differentiate bison infected with *B. abortus* from bison vaccinated with *B. abortus* strain 19. In the preliminary and blind studies using reference sera, both the indirect immunoassay (IELISA) and the competitive immunoassay (CELISA) for the detection of antibodies in serum to *Brucella abortus* performed nearly as well as the serum FPA. The sensitivity and specificity of the IELISA were 100% (n = 38) and 96.2% (n = 1044), respectively, while the sensitivity and specificity of the CELISA were 92.1% (n = 38) and 98.4% (n = 1000), respectively. The sensitivity and specificity estimates in the blind study for the IELISA were 96.3% (n = 54) and 97.6% (n = 160)respectively, while the sensitivity and specificity estimates for the CELISA were 96.3% (n = 54) and 94.1% (n = 160), respectively. In comparison, the relative sensitivity and specificity of the IELISA and CELISA for detection of antibodies to *Brucella abortus* in serum in field studies using the serum FPA as the standard were lower than the FPA for the detection of antibodies to *Brucella abortus* in whole blood. The relative sensitivity and specificity of the IELISA were 94.7% (n = 38) and 88.9% (n = 36), respectively. The relative sensitivity and specificity of the CELISA were 94.7% (n = 38) and 97.2% (n= 36), respectively. The buffered antigen plate agglutination test (BPAT) and the complement fixation test (CFT) for detection of antibodies in serum were less sensitive and specific than the serum FPA, CELISA, IELISA or whole blood FPA. The FPA is a homogeneous assay that saves time and money. These attributes, together with its excellent sensitivity and specificity, make the FPAs an attractive test for the detection of antibodies to *Brucella abortus* in bison in both laboratory and field applications.

(76) SURVEY FOR *BORRELIA* SPECIES AMONG RESERVOIR ANIMALS CAPTURED IN FORESTED AREAS OF GREATER METROPOLITAN CHICAGO.

M.M. PICKEN and R.N. PICKEN, Loyola University Medical Center, Chicago, Illinois 60153.

Background: Lyme disease in the US is concentrated in three endemic foci: northeastern, upper mid-West and along the Pacific coast. The mid-Western focus of Lyme disease has expanded to include large parts of Wisconsin and Minnesota. Despite its proximity to the mid-Western focus, Illinois, so far, has not been considered an endemic focus. However, more recent data suggests that this situation may be changing. Here, we present the results of attempts to culture the Lyme disease agent(s) from rodents captured in both Cook and Lake Counties both of which are parts of the greater metropolitan Chicago area in Illinois.

Design: We investigated the rodent reservoir present in forested areas of suburban Chicago in order to determine the frequency of infection with the Lyme disease agent or agents by culture isolation of *Borrelia* spirochetes. We identified rodent isolates of *Borrelia* to the species level by genetic characterization. Further plans include studies of the potential human pathogenicity of such isolates using a rabbit model of Lyme disease.

Results: In total, 19 isolates were obtained over 3 years from NW Cook Co. and Lake Co. These were isolated from the white-footed mouse, *Peromyscus leucopus* (2 isolates), the meadow vole, *Microtus pennsylvanicus* (12 isolates), and the meadow jumping mouse, *Zapus hudsonius* (5 isolates). Two Chicago isolates, 96-182 and 96-255, have the same pattern of high molecular mass fragments as strain DN127 (PF type I). All other isolates, except strain 97-251E, have the same macrorestriction pattern as strain 25015 (PF type II). Strain 97-251E has a variant of this type (PF type III). The closely similar macrorestriction pattern findings (summarized in Table 1) indicate that all of the Chicago rodent isolates investigated to date belong to the species *B. bissettii*, which is the second most important group of *Borrelia* found in North America. The sequence data generated from the rrf (5S)-rrl (23S) intergenic spacer region (IGSR) of the ribosomal RNA gene cluster confirmed the identity of the Chicago isolates as *B. bissettii*. We further propose to investigate the potential human pathogenicity of the rodent *B. bissettii* isolates we have obtained by testing them in an animal model.

Conclusion: *Borrelia* are present in the rodent reservoir of forested areas in suburban Chicago. The strains of *Borrelia* isolated to date appear most similar to Californian (DN127) and New York (25015) tick isolates of *B. bissettii*, and are unlike our previous *Borrelia* isolates from NW Illinois and Wisconsin. This suggests that the isolates may be native to the region.

(77) MECHANISMS OF SELENIUM-INDUCED TERATOGENESIS AND EMBRYOLETHALITY: OXIDATIVE STRESS.

KATHY M. ORSTED, M. F. RAISBECK, D. A. SANCHEZ and R. S. SIEMION, Department of Veterinary Sciences, College of Agriculture, University of Wyoming, Laramie, WY 82070.

Selenium (Se) is widely recognized as teratogenic and embryotoxic in avian species, however, relatively little is known about the mechanism(s) by which it produces such defects. The experiments described in this paper were designed to test the hypothesis that the underlying mechanism of Se embryotoxicity is oxidative stress. Embryonated mallard (*Anas platyrhynchos*) eggs were treated with various concentrations of L-selenomethionine (Se met) by injection in ovo and the eggs incubated for 8 days. Embryos were monitored by candling and, after opening, terata were evaluated visually. The median embryo-lethal dose (LD50) was established as 0.07 ppm Se on a whole egg basis. Pretreatment of embryos with vitamin E decreased (non-significantly) the frequency of Se-induced terata in embryos treated with a LD50. Other embryos were challenged with a LD50, incubated, and then examined at 6 days age for several indicators of oxidative stress: reduced glutathione (GSH) concentration, thiobarbituric acid reactive substances (TBARS) and DNA fragmentation. Treated embryos contained significantly more thiobarbituric acid reactive substances (TBARS) than did controls. Total reduced glutathione (GSH) and reduced:oxidized (GSH:GSSG) ratios did not differ between controls and treated embryos, nor was there any evidence of DNA fragmentation. The response to vitamin E and increased TBARS in Se-treated embryos support the involvement of oxidative stress in avian selenosis.

(78) AN EPIZOOTIC HEMORRHAGIC DISEASE OUTBREAK IN NEW JERSEY.

DOUGLAS E. ROSCOE and DANIEL FERRIGNO, New Jersey Division of Fish and Wildlife, P.O. Box 400, Trenton, New Jersey 08625; THOMAS R. BRIGGS and JANE E. HUFFMAN, East Stroudsburg University, Department of Biological Sciences, East Stroudsburg, Pennsylvania 18301; and DAVID STALLKNECHT, Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, University of Georgia, Athens, Georgia 30602.

An outbreak of epizootic hemorrhagic disease (EHD) in wild and captive white-tailed deer (*Odocoileus virginianus*) began on or about August 27, 1999 in Salem County, New Jersey and continued until the final case, confirmed through virus isolation, was found on October 19, 1999 in Burlington County. Only three outbreaks of EHD have been documented in deer from New Jersey. One involved 700 deer in northeastern New Jersey in 1955, another with 1000 deer losses in northwestern New Jersey in 1975, and the current epizootic. All of these were characterized by the serotype 1 EHD virus. Nine of 10 captive white-tails in one compound died of EHD in the 1999 epizootic. The survivor had been observed shivering, limping and frequently lying down. It had erosions on the underside of the tongue, but no obvious lesions of the feet to account for the limping. A serum sample from that deer was positive for EHD/BT antibodies using agar gel immunodiffusion. EHD virus serotype 1 was isolated from the blood. A fawn, which died of EHD, and came from the same compound, had no detectable antibody. A serologic survey was performed at mandatory deer check stations during the fall of the1999-2000 deer season. Blood samples from 290 white-tailed deer from two epizootic regions and one negative control area were collected on paper strips and later eluted and tested by agar gel immunodiffusion. These data were used together with hoof sloughing and field investigated deer deaths to delineate the spacial extent of the hemorrhagic disease epizootic. Based on a statewide grid system of deer management units (14 square miles/unit) the main epizootic impacted the deer in 224 square miles of marsh, tidal meadows and upland field along four major river drainages in southwestern New Jersey. Twenty one percent (15) of the 70 deer sampled at the deer check stations in the epizootic area had seroconverted to EHD (13) and/or had hoof sloughing (5) suggestive of prior EHD exposure. The deer kill in the area for the 1999-2000 season was down 10 to 24% in the face of a general statewide increase of 25%.

(79) DIVERSITY AND ECOLOGY OF BARTONELLA INFECTIONS IN RODENT COMMUNITIES.

MICHAEL Y. KOSOY, KENNETH L. GAGE, and MARY EGGLESTON, DVBID, NCID, CDC, Fort Collins, CO 80522; and JAMES E. CHILDS, DVRD, NCID, CDC, Atlanta, GA 30333.

Ecological and bacteriological observations of *Bartonella* spp. associated with rodents demonstrated abundant and diverse communities of these bacteria in different regions of the U.S.A. The *Bartonella* isolates were obtained from the blood of rodents collected in Arizona, Colorado, Florida, Georgia, New Mexico, Nevada, North Carolina and Wyoming. The prevalence of the infection varied from 0% in Phoenix, Arizona, to 84% in La Plata County, Colorado. *Bartonella* bacteria were found in 12 rodent genera (24 species): *Dypodomys* (1), *Mus* (1), *Neotoma* (3), *Ochrotomys* (1), *Onychomys* (1), *Orizomys* (1), *Peromyscus* (6), *Rattus* (2), *Reithrodontomys* (1), *Sigmodon* (1), *Spermophilus* (2), and *Tamias* (4). The highest prevalence of infection at a given site was always among the most commonly captured species of rodent in the community. Sequence analysis of the citrate synthase gene in > 1,000 isolates originated from rodents demonstrated 58 genetically different variants of *Bartonella* clustered into 14 phylogenetic clades. The level of sequence homology between clusters varied from 89 to 94%, and the degree of homology among variants within clusters was > 97%. Multiple species of *Neotoma*, *Peromyscus*, *Rattus* and *Tamias* harbored unique variants of *Bartonella* that were identical or closely related members of the same phylogenetic group. An experimental study indicated that laboratory-raised cotton rats and white-footed mice could only develop bacteremia if the strain used for inoculation was obtained from the same rodent species or from a phylogenetically close species. These data strongly suggest host-specific relations between *Bartonella* spp. and rodents. *Bartonella* infections in rodents from the western U.S. were less prevalent compared to those observed in rodents in the southeastern U.S., but the diversity of *Bartonella* spp. among the western rodents was higher. Immunological comparison of sera obtained from laboratory mice inoculated with the 30 strains of *Bartonella* demonstrated a high degree of specificity to the homologous antigen. However, screening of rodent populations by indirect fluorescent antibody test against a variety of *Bartonella* antigens produced either negative results or very low antibody titers. Isolates of *Bartonella* spp. were obtained from 18 of 31 embryos and 7 of 19 neonates from bacteremic females of two rodent species. These finding suggested the possibility of vertical transmission among natural rodent hosts. Preliminary data suggest that rodent fleas may be important in the transmission cycle of *Bartonella* spp. among rodents.

(80) PRESUMPTIVE PULMONARY MYCOPLASMOSIS IN CAPTIVE VANCOUVER ISLAND MARMOTS (*MARMOTA VANCOUVERENSIS*).

S. R. BLACK, Calgary Zoo, P.O. Box 3036, Station B, Calgary, AB Canada T2M 4R8; and S. RAVERTY, Animal Health Centre, B.C. Ministry of Agriculture and Food, 1767 Angus Campbell Rd., Abbotsford B.C. Canada V3G 2M3.

The Vancouver Island marmot, an alpine rodent confined in range to the south central mountainous areas of Vancouver Island, British Columbia is one of the most critically endangered mammals in the world, with less than one hundred animals remaining in the wild. The population of these marmots has been declining precipitously over the last decade, while the factors influencing this decline have remained speculative. Natural mortality during hibernation has been high, and several animals were live trapped and moved to captive facilities during the summers of 1997-1999. This has been one part of a comprehensive recovery plan for the species, intended to increase knowledge of the natural history of the animals, and to attempt captive propagation. While the majority of the animals have adapted well to captivity, there have been six mortalities in three years, four at the Calgary Zoo and two at the Toronto Zoo.

The first loss at the Calgary Zoo was a mature female with a heavy parasite load, bronchopneumonia and a necrotising hepatitis; this animal died within three days of capture in September of 1998. The second animal, also in 1998 was a male young of the year, found dead 10 days post capture with a perforated caecal ulcer and associated peritonitis.

Captures occurred earlier in the season in 1999, and included a female and her six offspring. It required several days to trap the entire group, with the mature animal brought in last. The two smallest littermates failed to gain weight compared to their siblings: one died 17 days post capture after a three day illness and necropsy findings included a perforated duodenal ulcer and peritonitis; the other was found dead 23 days after arrival at the zoo with presumptive bronchopneumonia.

Histopathology was conducted on all four animals. While the proximate cause of death in two animals is attributed to the duodenal and caecal lesions, in three of four marmots, a bronchointerstitial pneumonia was consistently observed. The extent of pulmonary involvement varied considerably between animals and within examined sections, however within more severely affected lungs there was moderate to marked multifocal peribronchiolar lymphoid hyperplasia. Adjoining airways exhibited mild to moderate hyperplasia of the respiratory epithelia with mild transmural lymphocytic infiltrate. Throughout the intervening parenchyma, alveolar lumina were variably ectatic and filled with dense accumulations of histiocytes with abundant, foamy to vacuolated cytoplasm.

Aerobic bacterial cultures of lungs were inconsistent between the four animals and culture for *Mycoplasma* was unrewarding. *Chlamydia* culture was negative and viral culture on one specimen was similarly unrewarding. Serology for Hantavirus, Sendai virus and *Mycoplasma pulmonis* were negative, however polymerase chain reaction for consensus *Mycoplasma* sp. was positive in frozen lung tissue from two animals. Vitamin

A and E levels (liver) in all animals appeared within the normal range for other species. Trace mineral analysis was also performed and all values were within accepted normals.

Based on the PCR results and the microscopic lesions, a diagnosis of pulmonary mycoplasmosis was adduced. The natural history of *Mycoplasma* infections is generally chronic and multifactorial. It is difficult to determine if this disease is a contributing factor to the decline of Vancouver Island Marmots in the wild. There has been a change in the habitat of many marmots as logging practices impact their range. Dispersal of marmots, exposure to novel animal species and dissemination of this putative pathogen may relate to this environmental change. Further investigations into the role of *Mycoplasma* spp. infection in this critically endangered population will be pursued.

(81) POPULATION HEALTH CONCERNS FOR LOWLAND GORILLAS: ADDRESSING THE KNOWLEDGE GAP.

WILLIAM B. KARESH and SHARON L. DEEM, Field Veterinary Program, Wildlife Conservation Society, 185th St. and Southern Blvd., Bronx, NY 10460.

Presently, only scant information is available on the health status of free-ranging populations of western lowland gorillas (*Gorilla gorilla gorilla*). Unlike eastern lowland gorillas (*G. g. graueri*) and mountain gorillas (*G. g. beringei*), lowland gorillas are distributed over a large geographic region and are found in areas with very limited human contact. In areas of the Central African Republic, Republic of the Congo, and Gabon, programs geared towards the development of lowland gorilla eco-tourism as well as long-term ecological and behavioral research are currently underway by local governments and several organizations, including WWF, ECOFAC, EDG, WCS, and CIRMF. The current lack of baseline information makes it impossible to evaluate changes in wildlife health, and the effects of conservation, research, or development programs on wildlife health. Concurrently, project staff awareness of potential health concerns for free-ranging western lowland gorillas has risen. The reasons for the increasing concern include reports of the disappearance of gorillas from vast areas of Gabon following the last Ebola virus outbreaks, increased recognition of *Treponema* sp. infections in gorillas and humans in the region, the widespread decline of human vaccination and health care programs in the region over the last two decades, and the shift to more multi-disciplinary backgrounds and approaches of project managers and staff.

The Field Veterinary Program of the Wildlife Conservation Society is developing and implementing an integrated approach to protecting the health of lowland gorillas by partnering with several projects and organizations. Our goal is to establish rational preventive health care programs and health monitoring systems. The approach includes: 1) guideline and policy development to mitigate the impact of visiting tourists, 2) guidelines and human preventive medicine programs for project staff, 3) gorilla health assessment and monitoring programs tailored for the different needs and capabilities of the ongoing projects, and 4) training of project staff.

(82) PATHOLOGY AND MORTALITY IN THE SOUTHERN SEA OTTER (*ENHYDRA LUTRIS NEREIS)* POPULATION AS A RESULT OF PARASITIC INFECTIONS.

MURRAY D. DAILEY, The Marine Mammal Center, Marin Headlands, Sausalito, CA 94965; KARL A. MAYER, Moss Landing Marine Laboratories, Moss Landing, CA 959039; and MELISSA CHECHOWITZ, Marine Wildlife Veterinary Care and Research Center, California State Department of Fish and Game, Santa Cruz, CA 95060.

Since 1995 when the southern sea otter population numbered 2,377, it has dropped 440 animals to its lowest count (1,937) in 1999. The number of dead otters recovered along the Central California coast has risen from 90 in 1955 to over 200 in 1999. Over 40 per cent of these mortalities are caused by infectious diseases and the most frequent of these are acanthocephalan parasites of the genus *Profilicollis* (Thomas et al., 1996).

From October 1997 to July 1999, 63 otter carcasses from 15 locations were examined for helminth parasites and their potential role in mortality assessed. Three species of trematodes (*Microphallus pirum, M. nicolli, Plenosoma minimum*) and 4 species of acanthocephalans (*Corynosoma enhydra, Profilicollis altmani, P. kenti, P. major*) were recovered from the intestine and body cavities of 59 (93%) of the carcasses examined. Pathology caused by trematodes was limited to host reaction from trapped eggs in the mucosa. A total count of all acanthocephalans recovered was carried out in a subsample of 10 carcasses from 8 locations. A total of 20,030 worms were recovered (8,610 *C. enhydri,* 11,420 *Profilicollis* spp.) with peritonitis from intestinal perforations causing death occurring in 6 (60%). Perforations resulting in peritonitis was caused only by those members of the genus *Profilicollis* (*P. altmani* 56%, *P. kenti* 34%, *P. major* 9%), while *C. enhydri* appears to be nonpathogenic.

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(83) BASELINE HEALTH VALUES IN SEA OTTERS.

KRISTA D. HANNI and JONNA K. MAZET, Wildlife Health Center, University of California, Davis, CA 95616; FRANCES M. D. GULLAND, The Marine Mammal Center, Marin Headlands-GGNRA, Sausalito, CA 94965; JIM ESTES, USGS-BRD, Marine Sciences, University of California, Santa Cruz, CA 95060; MICHELLE STAEDLER and MICHAEL J. MURRAY, Monterey Bay Aquarium, 886 Cannery Row, Monterey, CA 93940; and DAVID A. JESSUP, Marine Wildlife Veterinary Care and Research Center-DFG, 1451 Shaffer Rd, Santa Cruz, CA 95060

Due to concerns about sea otter health, hematological and/or serum chemistry values and exposure to a panel of marine and terrestrial pathogens were determined from blood collected during ongoing studies for 77 free ranging southern (*Enhydra lutris nereis*), 72 Alaskan (*E. l. kenyoni*), and 50 rehabilitated southern sea otters. Most blood and serum chemistry values fell within published normal ranges. For adults in California (free ranging) and Alaska, significant differences were found for gender (Na, cholesterol) and location (calcium, phosphorous, total bilirubin, and blood urea nitrogen, creatinine, and glucose). For free ranging Californian animals, significant differences were found for age (globulin, alkaline phosphatase, ALT, cholesterol, creatinine, BUN, RBC, HGB, HCT, lymphocytes). Differences were observed for free ranging versus rehabilitated pups for albumin, glucose, RBC, Hgb, HCT, neutrophils, lymphocytes, and eosinophils. Prevalence of exposure to calicivirus, *Brucella*, and *Leptospira* varied by location. The naivete of these populations to some pathogens (e.g., morbillivirus) has important implications for their susceptibility to disease outbreaks. These baseline health values and seroprevalence results are of significant interest to wildlife veterinarians and managers and are of critical importance in the development of long-term conservation plans for sea otter populations.

(84) LIFE CYCLE OF *OTOSTRONGYLUS CIRCUMLITUS* (METASTRONGYLOIDEA: CRENOSOMATIDAE) OF PHOCID SEALS.

L. N. MEASURES, Fisheries and Oceans Canada, Maurice Lamontagne Institute, P.O. Box 1000, 850 Route de la Mer, Mont-Joli, Qc Canada G5H 3Z4.

The lungworm, *Otostrongylus circumlitus*, found in the bronchi and bronchioles of phocid seals can be obstructive causing verminous pneumonia. Prevalence in infected free-ranging seals ranges from 10 to 89% with infections involving predominantly young-of-the-year or age = 0 animals. Mean intensities reported from stranded and hunted animals vary from 9 – 58. Laboratory work has demonstrated that invertebrates are not required in the life cycle as intermediate hosts. First-stage larvae passed in the faeces of infected seals are infective to fish and develop to the third stage which is infective to seals. In grey seals and harbour seals third-stage larvae cross the stomach wall and migrate via the circulatory system to the lungs where young adults are found as early as 10 d post-infection (dpi). The pre-patent period is 31 - 35 dpi and larvae are observed in the faeces. Clinical signs observed are vomition within 15 minutes of infection, coughing of blood as worms exit the pulmonary artery and crackling and wheezing sounds at auscultation of the thorax.

(85) *SARCOCYSTIS FALCATULA* DEVELOPMENT IN ITS NATURAL HOSTS.

E. C. GREINER, S. LUZNAR, R. PORTER, M. E. HEMENWAY, P .E. GINN, and J. B. DAME, Department of Pathobiology, University of Florida, Gainesville, FL 32611.

This study was conducted to determine the longevity of sarcocysts in the natural intermediate host, potential crowding effect on sarcocyst development, and the length of shedding of sporocysts by opossums. Experimental infections of *Sarcocystis falcatula* were produced by feeding brown-headed cowbirds harboring sarcocysts to non-sporocyst-shedding opossums. A series of brown-headed cowbirds were fed 200 sporocysts and two were necropsied at 2 week intervals along with an uninfected control. A piece of cowbird thigh and breast muscle tissue were formalin fixed and sectioned for histological examination from all cowbirds to confirm the presence of sarcocysts. All inoculated birds harbored sarcocysts after 6 weeks PI and the sarcocysts increased in size the entire time. Carcasses from 40 weeks PI group were fed to an uninfected opossum and it began shedding sporocysts 8 days later. Another series of cowbirds was inoculated with 1000, 500, 100, 10 or 0 sporocysts (n = 12) and were necropsied at 60 days post infection (DPI). The number of sporocysts in the inoculum did influence the number and the size of the resultant sarcocysts. The more sarcocysts which developed (from larger inocula), the smaller in diameter they were. Opossums continued to shed until they were euthanised and the longest time was 203 days.

(86) GENERALIZED, PRURITIC DERMATITIS, POSSIBLY ASSOCIATED WITH A HYPERSENSITIVITY REACTION, IN HAND-RAISED JUVENILE OPOSSUMS (*DIDELPHUS VIRGINIANA*).

CATHERINE M. BROWN, Forest Preserve District of DuPage County, Willowbrook Wildlife Center, 525 S Park Blvd., Glen Ellyn, IL 60137.

Since August of 1997, 14 orphan juvenile Virginia opossums (*Didelphus virginiana*) being hand-raised at Willowbrook Wildlife Center, have developed a generalized, pruritic dermatitis. These individuals show mild to moderate, diffuse, cutaneous inflammation characterized by erythema, minimal exudate, pruritis and thinning fur. Scrapings for external parasites were negative. Skin biopsies show mild hyperkeratosis with intermittent accumulations of mast cells and plasma cells. Hair follicles exhibit an increased number of telogen phase elements. This chronic cutaneous irritation often occurs in association with underlying allergic disorders such as atopy (Type-I allergic reactions). Many of these animals have responded over a period of 2-3 weeks to systemic treatment with Sulfadiazene/Trimethoprim or third generation cephalosporins and topical therapy with a keratolytic, antimicrobial shampoo. However, 4 individuals were euthanized due to a non-specific "failure to thrive" and a single individual died unexpectedly. A Type-I hypersensitivity reaction, possibly to a *Staphylococcus* spp., is hypothesized and the possibility of an underlying metabolic or stress disorder is discussed.

(87) GENETICS OF NATURAL DISEASE RESISTANCE IN BIGHORN SHEEP.

KAREN M. RUDOLPH and TRICIA L. HOSCH, Wildlife Health Laboratory, Idaho Fish and Game, PO Box 25, 600 S. Walnut Ave., Boise, Idaho 83707; JOE W. TEMPLETON, Department of Veterinary Pathobiology, College of Veterinary Medicine, Texas A&M University, College Station, Texas 77843-4467; and DAVID L. HUNTER, Turner Enterprises, Inc., 401 Mill Street, P.O. Box 190, Gallatin Gateway, Montana 59730.

Considerable effort has been devoted to the identification and characterization of pathogenic microorganisms involved in bighorn sheep pneumonia epizootics as well as the development of experimental vaccines specific for pathogenic *Pasteurella* spp. strains. A novel genetic approach to disease susceptibility in bighorn sheep is being explored to complement current disease control approaches. Natural disease resistance is the inherent capacity of a previously unexposed animal to resist disease when challenged by pathogens, and is transmitted from parent to offspring. Studies have demonstrated the importance of natural disease resistance in elk and bison to diseases including brucellosis and tuberculosis. To determine whether bighorn sheep have a natural resistance to *Pasteurella* spp., polymerase chain reaction (PCR) primers from domestic sheep were used to amplify Nramp product from bighorn sheep genomic DNA. A microsatellite assay was then employed to identify three Nramp alleles in 42 samples tested. One allele was very common (41/42) while two others were less common (3/42 and 7/42). Future experiments include a bacteriocidal assay to determine which of the three alleles may be protective in bighorn sheep, determination of the prevalence of the protective allele in wild bighorn sheep populations, and a captive bighorn sheep challenge trial.

(88) *PARELAPHOSTRONGYLUS ODOCOILEI* AND *PROTOSTRONGYLUS STILESI* IN DALL'S SHEEP: PREDISPOSING FACTORS FOR MORTALITY?

SUSAN KUTZ, Department of Veterinary Microbiology, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK, Canada S7N 5B4; ALASDAIR VEITCH, Department of Resources, Wildlife and Economic Development (DRWED), Government of the Northwest Territories (GNWT), Norman Wells, NT, Canada XOE 0V0; EMILY JENKINS and BRETT ELKIN, DRWED, GNWT, Yellowknife, NT, Canada, X1A 3S8; ERIC HOBERG, Biosystematics and National Parasite Collection Unit, United States Department of Agriculture, Agricultural Research Service, Beltsville, MD 20705-2350; MANUEL CHIRINO-TREJO, LYDDEN POLLEY, and TRENT BOLLINGER, Canadian Cooperative Wildlife Health Center, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK, Canada S7N 5B4.

Collections of 43 Dall's sheep (*Ovis dalli dalli*) fecal samples from the northern Mackenzie Mountains, Northwest Territories, Canada during July to October, 1997, demonstrated a high prevalence and high fecal larval counts of *Protostrongylus* sp. [74%, 0.2-700 larvae per gram (LPG)] and of dorsal-spined protostrongylid larvae (77%, 0.2-967 LPG). Subsequent examinations of four sets of lungs from hunter-killed sheep confirmed the presence of adults, eggs, and larvae of *P. stilesi,* and full post mortem examinations of six ewes (November 1998 and April 1999) revealed adult *Parelaphostrongylus odocoilei* in the skeletal muscles, and eggs and larvae consistent with *P. odocoilei* in the lungs. Moderate to severe pulmonary pathology was associated with both protostrongylid species. Examination of the initial 43 fecal samples by modified Wisconsin technique revealed the eggs from the genera *Marshallagia*, *Nematodirus*, *Trichuris*, *Skrjabinema*, oocysts of at least two species of *Eimeria*, and eggs from the family Trichostrongylidae. In addition, eggs of *Skrjabinema* were found in one of the six ewes.

Subsequently, from June 1999 to April 2000, six dead and two sick sheep, representing the first reported cases from this area in over 30 yr of commercial outfitting, were found at four locations in the northern Mackenzie Mountains. Post mortem examinations were performed on three of the dead sheep. Two adult ewes, found dead in June and July, had severe, subacute to chronic, fibrino-purulent broncho-pneumonia; ewe #1 was septicemic. Although fecal protostrongylid larval counts were low, gross and histological lesions along the dorso-caudal borders of the diaphragmatic lung lobes of both ewes were consistent with *Protostrongylus* sp. and histological lung lesions consistent with *P. odocoilei* infection were present. A pure culture of *Arcanobacterium pyogenes* was obtained from lungs of ewe #1 and a mixed population of *Mannheimia* ("*Pasteurella*") *granulomatis*-like, *Escherichia coli,* and *A. pyogenes* from lungs of ewe #2. Lung and tracheal tissue from both ewes were negative on immunohistochemistry for *Haemophilus somnus*, *Mannheimia haemolytica*, parainfluenza virus type 3, bovine herpes virus type 1, bovine respiratory syncytial virus, and bovine viral diarrhea. The third post mortem was on a 10 mo old female lamb that died while running across a meadow in April. Severe, multifocal, petechial and ecchymotic pulmonary and skeletal muscle hemorrhages were the major findings. The tentative cause of death was exercise induced, acute respiratory failure. No bacteria were isolated from the lungs on aerobic and anaerobic culture; histological results are pending. First-stage dorsal-spined larvae (probably *P. odocoilei*;

187 LPG) and few *Protostrongylus* sp. (8 LPG) were present in the feces. In September three additional dead adult sheep (two rams, one ewe), with no evidence of predation, were reported from one location but were not recovered for examination. Also, in September, two rams with respiratory signs were observed at a separate location. One of the rams was shot and the small piece of lung submitted cultured positive for *A. pyogenes*.

The underlying cause and population effects of these mortalities are unknown. However, a possible predisposing factor is the pulmonary compromise caused by *P. stilesi* and *P. odocoilei.* Mixed infections with these two parasites in a single host species are unique. Their possible synergistic effects, together with bacteria and possibly viruses, on individual hosts and host populations, warrant further consideration.

(89) A MODEL FOR INVESTIGATING THE DEVELOPMENT, TRANSMISSION, AND RESPONSE TO CLIMATE CHANGE OF PROTOSTRONGYLID PARASITES ON THE ARCTIC TUNDRA.

SUSAN KUTZ and LYDDEN POLLEY, Department of Veterinary Microbiology, Western College of Veterinary Medicine, University of Saskatchewan, 52 Campus Drive, Saskatoon, SK S7N 5B4; and ERIC HOBERG, Biosystematics and National Parasite Collection Unit, United States Department of Agriculture, Agricultural Service, Beltsville, MD 20705-2350.

Protostrongylid parasites are important pathogens in many North American ruminants and have been reported with increasing frequency in hosts at arctic and subarctic latitudes. Development and survival of these parasites in the environment are intimately linked to climatic conditions. In the north, where global warming is predicted to be the most pronounced, the patterns of development, transmission, geographic distribution and host range, may be profoundly influenced by climate change. Using *Umingmakstrongylus pallikuukensis,* a protostrongylid lungworm in muskoxen (*Ovibos moschatus),* we have developed a model for investigating the development and availability of a protostrongylid parasite in the environment. This model incorporates both laboratory and field data. The threshold temperature (8.5 C) and degree-day (DD) requirements (167 DD) for development of *U. pallikuukensis* from first to third-stage (L3) larvae were established using the slugs *Deroceras laeve* in the laboratory. The patterns of larval development in *D. laeve* were then investigated under field conditions near Kugluktuk, Nunavut, Canada, in 1997 and 1998.

Development in the field corresponded with that which would be predicted based on soil surface temperatures and threshold temperature and degree-day requirements. Development to L3 occurred within 4-6 wk in slugs infected on or before 17 July, larvae in slugs infected after this date (1997 experiments only) did not mature to L3 before the end of September. With a constant rate of infection during the summer, the number of L3 in slugs peaked in August and then significantly decreased, possibly because of larval emergence. Although these results suggest that transmission rates of *U. pallikuukensis* to the definitive host may be highest during late summer, year-round transmission, linked to emergence of L3 on the vegetation, is possible.

Increased environmental temperatures coincidental with global warming could result in a higher peak of L3 in slugs, occurring earlier in the summer, and lasting later into the fall. The effects of increased temperatures on availability of emerged L3 are less predictable. Thus, under conditions of global warming, the perhaps seasonally restricted "windows for transmission" may expand, resulting in amplification of parasite populations in both intermediate and definitive hosts over time. Ultimately, this may result in the emergence of parasite-induced disease as a factor influencing individual hosts and host populations.

This model for larval development of *U. pallikuukensis* could be applied to other protostrongylids, other geographic regions, or other climatic conditions to predict larval development. It is, therefore, a very important tool for monitoring and predicting alteration in patterns for development and transmission of helminths and biotic responses of host-parasite systems to global climate change.

(90) NOTOEDRIC MANGE IN WESTERN GRAY SQUIRRELS FROM WASHINGTON.

TODD E. CORNISH, Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, University of Georgia, Athens, GA 30602 (present address: Wyoming State Veterinary Laboratory, Department of Veterinary Sciences, University of Wyoming, 1174 Snowy Range Road, Laramie, WY 82070); MARY J. LINDERS, University of Washington, Wildlife Science Group, Box 352100, Seattle, Washington 98195-2100; SUSAN E. LITTLE, Department of Medical Microbiology and Parasitology, College of Veterinary Medicine, The University of Georgia, Athens, Georgia 30602; and W. MATTHEW VANDER HEAGEN, Washington Department of Fish and Wildlife, 600 Capitol Way North, Olympia, Washington 98501.

Notoedric mange, caused by the mite *Notoedres centrifera* (syn. *douglasi*), has been reported in a variety of squirrel species, including eastern and western gray squirrels and fox squirrels. Typical cases are self-limiting and affect individual animals or small groups of animals; however, one report details a large outbreak of notoedric mange that caused significant morbidity and mortality in the Eldorado National Forest of California in 1921. Here we report a similar outbreak of notoedric mange that significantly impacted a local population of western gray squirrels in Klickitat County, Washington. Sixty-eight squirrels were trapped for a home range and habitat use study from February 1998 to July 1999. None of the nine squirrels initially trapped (February and March 1998) had mange lesions. Beginning in August 1998 and continuing through July of 1999 squirrels from three different trap sites were captured with lesions consistent with notoedric mange. In total 33/59 (56%) squirrels captured or recaptured had mange lesions ranging from mild to severe, 14/33 (42%) affected squirrels are known to have died, and only four breeding females are known to have survived the outbreak. The diagnosis of notoedric mange was confirmed by histopathology and examination of mites recovered from skin scrapings from two squirrels submitted to the Southeastern Cooperative Wildlife Disease Study. Potential predisposing factors for this mange outbreak include a mast crop failure leading to nutritional stress and increased dispersal and emigration and possible iatrogenic transmission during initial trapping and handling.

(91) PERSISTENCE AND SAFETY OF PARENTERALLY DELIVERED IOPHENOXIC ACID AS A SEROMARKER IN BISON (*BISON BISON*).

STEVEN J. SWEENEY, THOMAS J. ROFFE, and KENNETH W. COFFIN, Biological Resources Division, U.S. Geological Survey, Bozeman, MT 59717; MARK L. DREW, Wildlife Health Laboratory, Idaho Department of Fish and Game, Caldwell, ID 83605; and ROBERT H. POPPENGA, Department of Pathobiology, School of Veterinary Medicine, University of Pennsylvania, Kennett Square, PA 19348.

We investigated the safety and effectiveness of iophenoxic acid (IA) as a candidate seromarker in nine captive subadult, male bison (*Bison bison*). A biomarker that is safe, dose-responsive, and relatively long-lived could be used in a variety of ecological and management-related studies requiring a marking system that is not externally visible. Our objectives were to verify whether parenterally administered IA could be detected in bison serum; to characterize the relationship between IA dosage and serum concentration; to estimate serum IA persistence and half-life; and to establish whether IA treatment would adversely affect bison clinically or hematologically.

We suspended IA in sterile water (100 mg/ml) and injected bison intramuscularly at dosages of 1.1 mg/kg, 2.1 mg/kg, and 4.1 mg/kg (3 bison per dose). Sterile saline was injected into the opposite limb as a negative control for site reaction. We collected blood (30 ml) by jugular venipuncture immediately before IA treatment, 18 hours after treatment, weekly through week 8, every other week through week 12, and at four-week intervals through week 24. Whole blood and serum samples were submitted for complete blood count and clinical chemistry. Serum IA concentrations were analyzed by direct assay using high performance liquid chromatography. We monitored bison for swelling at injection sites, lameness, and behavioral changes.

Serum IA concentrations were significantly elevated in all bison within 18 hours of treatment and remained elevated throughout the study. IA levels declined at an average weekly rate of 1.4%, yielding a predicted mean serum half-life of 48 weeks. Although there was a direct, positive relationship between estimated IA dosage and initial serum concentration, IA levels were highly variable within groups. We found no evidence of gross physical or behavioral changes associated with IA treatment in bison. Packed cell volume and serum creatinine levels increased in all animals, but changes in these parameters did not relate to IA dosage and appeared to be seasonal. We tentatively conclude that IA is safe for parenteral delivery at the dosages used in bison, and that it shows promise as a seromarker for large ungulates.

(92) SEROLOGY FOR SELECTED VIRUSES, BACTERIA, AND PROTOZOA IN FREE-RANGING ANACONDAS (*EUNECTES MURINUS*) IN VENEZUELA.

PAUL P. CALLE, JOHN THORBJARNARSON, WILLIAM HOLMSTROM, and WILLIAM B. KARESH, Wildlife Conservation Society, Bronx, New York 10460-1099; and JESÚS RÍVAS and MARÍA MUÑOZ, Profauna, Ministerio del Ambiente y de Los Recursos Naturales, Renovables, Caracas, Venezuela.

Reptiles may harbor viral, bacterial, and protozoal pathogens asymptomatically or serve as reservoirs of infection for man, domestic species, or other reptiles. Potential pathogens include Venezuelan (VEE), Eastern (EEE) and Western (WEE) Equine Encephalitis viruses; Vesicular Stomatitis virus (VS); ophidian paramyxovirus; *Leptospira* spp.; and ophidian cryptosporidia.

In March 1992, during a study of the biology and conservation of free-ranging anacondas (*Eunectes murinus*) in the Venezuelan Llanos (seasonally flooded savannas), health assessments and surgical implantation of radio transmitters were performed. Blood was collected, processed in the field, and serum and heparinized plasma samples frozen at -70 ^{o} C until analyzed. Samples from ten anacondas (five male and five female) examined at the ongoing study site (Hato El Cedral, a cattle ranch) were tested for antibodies to selected viruses, bacteria, and protozoa. Testing for VEE, EEE, WEE, and ophidian paramyxovirus titers was by hemagglutination inhibition. Titers for VS viruses Indiana and New Jersey were determined for seven (4 female and 3 male) by serum neutralization. The microscopic agglutination test was used to determine titers to 19 *L. interogans* serovars. Titers from eight (5 female and 3 male) were determined by enzyme immunoassay for ophidian cryptosporidia.

All had negative titers of 10 for EEE and WEE. VEE titers for nine anacondas were 10; one had a titer of 20 that probably is a nonspecific response. All anacondas tested were negative for antibodies to VS Indiana and New Jersey; ophidian paramyxovirus; and ophidian cryptosporidia. Five had low titers (1:100 or 1:200) against one to four *L. interogans* serovars (*L. icterohaemorrhagiae/copenhageni*, *L. autumnalis*, *L. bratislava*, *L. ictero./ictero.* or *L. kennewicki*); these are probably nonspecific responses.

None of these anacondas had elevated antibody titers to VEE, WEE, EEE, VS, ophidian paramyxovirus, or ophidian cryptosporidia. The low *Leptospira* titers were nonspecific, may indicate cross-reaction with the nonpathogenic saprophyte *L. biflexa*, and are not diagnostic for exposure to a pathogenic *Leptospira* spp. The results of this survey suggest that these anacondas do not serve as significant reservoirs for selected arboviruses, ophidian paramyxovirus, pathogenic *Leptospira* sp., or ophidian cryptosporidia at this location.

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(93) DEVELOPMENT OF A FISH AND WILDLIFE HEALTH PROGRAM FOR THE MARYLAND DEPARTMENT OF NATURAL RESOURCES.

CINDY P. DRISCOLL, SUSAN KNOWLES, BRENDA KIBLER, BRETT COAKLEY, KELLY GREENHAWK, and TRICIA LITWILER, Maryland Department of Natural Resources, Oxford, MD 21654.

To address the many fish and wildlife health concerns in the state of Maryland, the Department of Natural Resources formed the Fish and Wildlife Health Program in 1999. While fish health investigations had received some funding in the state in previous years, a more comprehensive program incorporating wildlife was needed. A veterinarian was hired in January 1999 and 3 biologists were assigned to help design and implement the project. A fourth biologist was added in September 1999 and an experienced computer programmer was enlisted to assist the program. The framework of the program consists of five elements - Emergency Response, Monitoring, Research, Outreach, and Database Development. To begin development of the program biologists throughout the state were contacted to ascertain the health needs for the various species in Maryland. Selected species were identified as well as specific disease problems. The first year incorporated extensive activity in all five areas: emergency responses to fish health issues such as Harmful Algal Blooms (*Pfiesteria piscicida*), marine mammal and sea turtle strandings, West Nile Virus and Epizootic Hemorrhagic Disease outbreaks; monitoring for vian Vacuolar Myelinopathy, baseline health of black bear and white tailed deer; research into the cause(s) of striped bass ulcerative skin lesions, grant development for investigation of nuisance species (nutria); development of outreach materials and presentations to promote fish and wildlife health investigations, representation on interagency and university health task forces; development of a comprehensive database. Services of the Fish and Wildlife Health Program are in constant demand. The challenge of the next few years will be in limiting our projects to essential state issues concerning the natural resources of Maryland.

(94) NATURAL HISTORY STRATEGIES OF MICROORGANISMS CAUSING DISEASES OF WILDLIFE.

RICHARD G. BOTZLER and RICHARD N. BROWN, Department of Wildlife, Humboldt State University, Arcata, CA 95521.

One of our goals, as instructors of upper-division courses covering the principles of wildlife diseases, is to provide students with a conceptual framework to facilitate their understanding of the maintenance, transmission, and potential for management of wildlife disease agents. Natural history strategies commonly are used to describe macroparasites, such as arthropods and helminths, as well as the Protista. While a natural history approach to understanding bacteria and viruses is less easily categorized into a conceptual framework, such an approach would help promote an ecological understanding of diseases. We propose the following natural history classifications for bacteria and viruses.

Bacteria

Natural history strategies:

- Directly transmitted bacteria
  - No carrier state – clinical illness necessary
  - Interspecific carrier state – sub-clinical illness typical
  - Intraspecific carrier state – sub-clinical illness typical
- Indirectly transmitted bacteria
  a. Transmission via a second parasite
     - Vector-borne
     - Helminth-borne
  b. Transmission via non-parasitic organisms or the environment
     - Bacteria reproducing in soil, water, or dead organic matter
- Bacteria that persist in the environment without reproducing

Viruses

Natural history strategies:

- Directly transmitted viruses
  - No carrier state – clinical illness necessary
  - Intraspecific carrier state – sub-clinical illness typical
  - Intraspecific carrier state – sub-clinical illness typical
- Indirectly transmitted viruses
  - vector-borne viruses
  - viruses that persit in the environment

(95) WATEFOWL HEALTH AND MANAGEMENT: THE FIRST WILD*PRO* MODULE.

F. JOSHUA DEIN, USGS National Wildlife Health Center, 6006 Schroeder Road, Madison, WI 53711; and SUZANNE I. BOARDMAN and DEBRA BOURNE, Wildlife Information Network, The Royal Veterinary College, Royal College Street, London, NW1 0TU, UK.

There is frequently a need by professionals to rapidly have access to a wide range of reference information to solve an acute problem, but there is not time, nor access, for an exhaustive search of printed material. In addition, many situations require knowledge of facts and procedures in fields ancillary and remote to ones own, which makes information retrieval for decision making more difficult. While databases, both on-line and library/CD-ROM based, may contain these details, accessibility and ease of use limit their benefits in many situations.

WILDPro takes an innovative to approach to information management by offering a broad range of technical data in text and image form, all hyperlinked to allow information to be found through different entry points and paths. Links are provided to ensure that important related data is highlighted. Procedural flow-charts are also available to guide searchers though complex and/or unfamiliar tasks.

While this concept is applicable to many subjects and disciplines, we have chosen waterfowl for the first module to be fully developed. This is currently available on-line at the Wildlife Information Network WWW site (http://www.wildlifeinformation.org) and contains information on biology, disease, infectious and non-infectious agents, environment and habitat, diagnostic, investigative and veterinary procedures, as well as documents on “best practices” and guidelines. Portions of the NWHC’s Field Manual to Wildlife Diseases are also included.

(96) WILDLIFE HEALTH CAPACITY BUILDING IN SOUTH AMERICA.

MARCELA M. UHART, Field Veterinary Program, Wildlife Conservation Society, 14 de julio 430, (7000) Tandil, Pcia. de Buenos Aires, Argentina; and WILLIAM B. KARESH and SHARON L. DEEM, Field Veterinary Program, Wildlife Conservation Society, 185th St. and Southern Blvd., Bronx, NY 10460.

During the past few years, the Wildlife Conservation Society's Field Veterinary Program has been directing part of its international efforts to strengthening local capacity for studying and addressing wildlife health issues. Our base of involvement has been oriented towards providing wildlife health services to ongoing research projects by collaborating with local field biologists and veterinarians. Working with local researchers, we have provided hands-on assistance in the restraint and handling of wild animals, including marine mammals in Argentine Patagonia, brocket deer, peccaries and tapirs in the Bolivian Chaco, and small carnivores in diverse landscapes ranging from the Argentine pampas to Amazon forest and Yungas in Bolivia. Samples for health evaluations were collected in all of these studies, and are currently being analyzed in order to establish baseline health information needed for the management of these populations. Local veterinarians, veterinary students, biologists and parabiologists are trained during these projects to continue with proper sample collections.

Expanding on original health studies of Magellanic and Humboldt penguins, we have collected samples from various seabird species being handled for biotelemetry studies, including cormorants and giant petrels from the Atlantic coastlines of Patagonia and the Pacific Peruvian shores, for the evaluation of disease and contaminant exposure. Conservation research teams in South America are increasingly consulting us regarding the application of capture techniques, to review sample collection protocols, to provide advice and training on proper necropsy procedures, and to advise on animal welfare concerns for threatened and endangered species.

Beginning in 1999, we have signed cooperative agreements with several universities to initiate training programs for young local veterinarians with limited access to foreign educational centers. These training programs include classroom courses and practical learning in the field, thus assisting national research groups while acquiring hands-on experience. These courses have begun in Argentina, and we have secured funding to expand these efforts to other countries, such as Colombia, Bolivia and Peru. It is our goal to continue enhancing local capabilities to better deal with wildlife health and population management issues in this highly biodiverse region. We hope that we can encourage other wildlife health professionals from around the world to lend their expertise and would like to facilitate participation by linking field projects in South America with intellectual resources and advanced capabilities available in other countries. There is no shortage of important work to be done, only a shortage of participants and resources to back them.

(97) RADIOGRAPHS - AN ESSENTIAL TOOL FOR FORENSIC INVESTIGATIONS INVOLVINGWILDLIFE MORTALITY INVESTIGATIONS.

RICHARD K. STROUD and RHODA RALSTON, National Wildlife Forensic Laboratory, 1490 East Main St., Ashland, Oregon 97520.

Radiographs are essential tools in the investigation of forensic wildlife cases. The radiograph becomes evidence as it not only assists the pathologist with assessing the cause of death but also documents findings in a graphic manner for court presentation. A series of unique radiographs from cases processed at the National Wildlife Forensic Laboratory will be presented to illustrate interesting cases which have been encountered.

(98) ALBERTA'S DRAFT IMPORT PROTOCOLS FOR GAME FARM CERVIDS: A WORK IN PROGRESS.

M. J. PYBUS, Alberta Natural Resources Service - Fish and Wildlife, 6909-116 Street, Edmonton, Alberta T6H 4P2, Canada; and D. K.ONDERKA, Alberta Agriculture, Food, and Rural Development, 6909-116 Street, Edmonton, Alberta T6H 4P2 Canada.

In September 1988, health risks associated with importation of game farm cervids were deemed unacceptable to the Government of Alberta and a moratorium on importations was established. Since that time, Alberta has expended considerable energy and resources in assessing importation of cervids in light of potential risks to the health of wildlife, farmed cervids, domestic livestock, and the public. We now have a draft import protocol that was developed on the basis of thorough scientific review of the potential hazards, consultation with our cervid industry, and input from other stakeholders. This presentation will outline the components of the risk assessment, risk management, and risk communication.

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