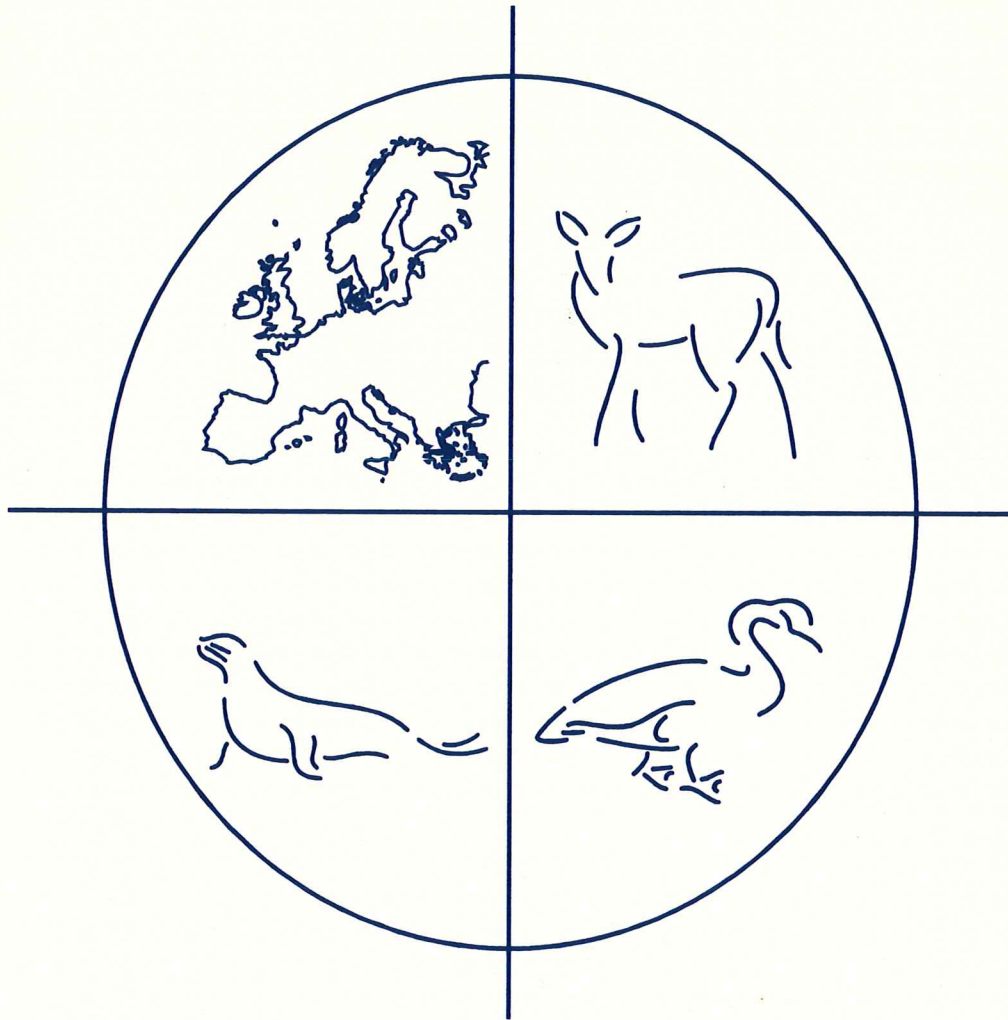


European Wildlife Diseases Association



**Abstracts of the
3rd International Conference
Edinburgh,
16-20 September 1998**



WELCOME TO MOREDUN PENTLANDS SCIENCE PARK

The Organising Committee thank you for supporting your Conference and we hope you have a scientifically stimulating and enjoyable time. We gratefully acknowledge the help of our scientific colleagues and the EWDA Committee in the organisation of the Conference. We especially thank the following organisations without whose most generous support it would not have been possible to arrange the Congress.

SPONSORS

BAYER
(Conference bags)

IDEXX
(Student registrations)

LOTHIAN & EDINBURGH ENTERPRISE LTD (LEEL)

MOREDUN FOUNDATION FOR ANIMAL HEALTH & WELFARE
(Welcome Reception)

EXHIBITORS

IDEXX
VETAID

ORGANISING COMMITTEE

Hugh W Reid
Peter Nettleton
Ranald Munro
David Buxton
Christine Curran

GENERAL INFORMATION

Arrangements for Scientific Presentations

Sessions:

All Sessions will be in the Lecture Theatre.

Posters:

Boards for posters (NUMBERED 1 - 20) will be available in the **ATRIUM**. Please ensure that you use the poster board with the number coinciding with your poster abstract number and ensure that your poster is in position by the morning of Thursday 17 September and removed at the end of the Conference.

Speakers:

The speaker's preparation centre (**LEEL ROOM**) will be open daily from 0815 to allow delegates to view and arrange their transparencies in advance of their talk.

It is the responsibility of the speaker to ensure that the transparencies/slides are in the correct order and position.

It is recommended that you place your name on each transparency/slide and that your transparencies/slides are in plastic frames.

Transparencies/slides should be loaded into the carousel provided and given to the project staff well in advance of your talk.

Carousels will be available in the speaker's preparation centre.

Following your talk please collect your transparencies/slides from the speaker's preparation centre.

Do not exceed the time allowed for your talk as this is discourteous to other speakers.

Chairmen:

Contact the speakers for your Session in advance and ensure that each knows the time allowed for his/her lecture.

Please **briefly** introduce speakers and the topic of the lecture.

Ensure that the speakers keep to the time allowed and that the Session ends promptly.

Allow sufficient time for discussion at the end of each session.

Please remind delegates to view the posters and attend the commercial stands during the breaks.



3RD INTERNATIONAL CONFERENCE OF THE EUROPEAN WILDLIFE DISEASES ASSOCIATION EDINBURGH, 16-20 SEPTEMBER 1998

PROGRAMME

Wednesday 16 September *Registration* 1600-2000 hrs

Thursday 17 September

0900

0900-0930

0930

BUS LEAVES POLLOCK HALLS FOR PENTLANDS SCIENCE PARK

Late registration

Opening: Dr W Donachie
Professor T Morner

SESSION 1
1000-1130

Chairman: Dr Marc Artois (*Lecture theatre*)

1:1 Dr Dolores Gavier-Widen (UK)
The "no visible lesion" presentation of natural bovine tuberculosis in wildlife hosts: a review

1:2 Dr Paddy Sleeman (Ireland)
Oral bait delivery to wild Irish badgers

1:3 Dr Sarah Feore (UK)
The spatial ecology of badgers in a low density area and the implications for the epidemiology of TB

1130-1200 COFFEE (*Atrium*)

1200-1300 1:4 Dr Jacques Godfroid (Belgium)
Isolation and characterisation of *Brucella* spp in a minke whale (*Balaenoptera acutorostrata*)

1:5 Dr Morten Tryland (Norway)
Evidence of *Brucella* infection in marine mammals in the north Atlantic ocean

1300-1400 LUNCH (*Atrium*)

SESSION 2

Chairman: Dr Ranald Munro

1400-1530

2:1 Dr Thomas Mueller (Germany)
A descriptive epidemiological analysis of viral pathogens in migrating bean geese (*A. fabalis*) and white-fronted geese (*A. albifrons*) with special emphasis on Newcastle disease

2:2 Dr Torsten Morner (Sweden)
Winter mortality in waxwings (*Bombycilla garrulus*) caused by ethanol intoxication

2:3 Dr Marc Artois (France)
Wildlife diseases surveillance in France: 10 years of results on diseases shared by cattle and wild ungulates

1530-1600 TEA (*Atrium*)

1600-1730	2:4 Dr Francois Lamarque (France) Surveillance of wildlife diseases in France - causes of mortality of wild ungulates: 10 years of results	
	2:5 Mr Victor Simpson (UK) A study of vitamin A levels in otters (<i>Lutra lutra</i>) in south west England	
	2:6 Mr David Williams (UK) Retinal dysplasia in wild otters: a pathological survey	
1730-1830	BUSINESS MEETING	(Atrium)
1830-2030	RECEPTION	(Atrium)
2030	BUS LEAVES FOR POLLOCK HALLS OF RESIDENCE	
Friday 18 September 0830	BUS LEAVES POLLOCK HALLS FOR PSP	
SESSION 3 0900-1030	Chairman: Dr Hugh Reid 3:1 Dr Emmanuelle Fromont (France) Prevalence and pathogenicity of retroviruses in wildcats <i>Felis silvestris</i> in France 3:2 Dr Sarah Feore (UK) The dynamics of an endemic disease in its wild reservoir host: cowpox and wild rodents 3:3 Dr Malcolm Bennet (UK) The evolution of cowpox virus - a hypothesis	(Lecture theatre)
1030-1100	COFFEE	(Atrium)
1100-1230	3:4 C. Gortazar (Spain) Wildlife diseases in Spain: The SEDIFAS viewpoint 3:5 Dr Martin Cooke (UK) Seychelles magpie-robin (<i>Copsychus sechellarum</i>): veterinary intervention for the conservation of an endangered species 3:6 Dr Peter Nettleton (UK) Current concepts of interactions between red and grey squirrels in the UK	
1230-1330	LUNCH	(Atrium)
SESSION 4	Chairman: Dr Peter Nettleton	
1330-1500	POSTER SESSION	(Atrium)
1500-1530	TEA	(Atrium)
1530-1730	4:1 Mrs Annette Bolte/Dr Walburga Lutz (Germany) Absence of orthomyxovirus and paramyxovirus serotype-1 (aPMV-1) infections among free-living wild geese (<i>Anser anser</i> and <i>Branta canadensis</i>) but detection of aPMV-4, aPMV-6, aPMV-8, Mycobacteria species, Chlamydia species and intestinal parasites 4:2 Mr Thomas Pennycott (UK) Some causes of mortality of the mute swan (<i>Cygnus olor</i>) 4:3 Dr Debra Bourne/Mrs Suzanne Boardman (UK) The London Waterfowl Project: information, communication and expert assistance 4:4 Dr James Kirkwood (UK) Bird feeders and the health and welfare of garden birds	

1730	BUS LEAVES FOR POLLOCK HALLS OF RESIDENCE	
1900	BUS LEAVES POLLOCK HALLS FOR PSP	
1930-2000	PRE DINNER DRINKS	
2000	CONFERENCE DINNER	(Dining Room)
2300	BUS RETURNSTO POLLOCK HALLS OF RESIDENCE	
Saturday 19 September		
0830	BUS LEAVES POLLOCK HALLS FOR PSP	
SESSION 5	Chairman: Dr David Buxton	(Lecture theatre)
0900-1000	5:1 Dr Paul Duff (UK) Rabbit Haemorrhagic Disease in the UK	
	5:2 Dr Martin Cooke (UK) Health threats to endangered free-living wildlife populations	
1000-1030	COFFEE	(Atrium)
1030-1130	5:3 Dr Dominique Gauthier (France) Brucellosis in chamois (<i>Rupicapra rupicapra</i>): relationships with mountain cattle breeding	
	5:4 Dr Jacques Godfroid (Belgium) <i>Brucella suis</i> biotype 2: a cause of positive serological reactions in the brucellosis screening tests in cattle	
1130-1200	CLOSING REMARKS	
1200-1300	LUNCH	(Atrium)
1300-1500	TOUR OF FIFE AND OCEAN WORLD	
1800	DINNER	
1900	RETURN TO EDINBURGH	

TABLE OF CONTENTS

SESSION 1

THE "NO VISIBLE LESION" PRESENTATION OF NATURAL BOVINE TUBERCULOSIS IN WILDLIFE HOSTS: A REVIEW	1
ORAL BAIT DELIVERY TO WILD IRISH BADGERS	2
THE SPATIAL ECOLOGY OF BADGERS IN A LOW DENSITY AREA AND THE IMPLICATIONS FOR THE EPIDEMIOLOGY OF TB	3
ISOLATION AND CHARACTERISATION OF <i>BRUCELLA</i> SPP. IN A MINKE WHALE (<i>BALAENOPTERA ACUTOROSTRATA</i>)	4
EVIDENCE OF <i>BRUCELLA</i> INFECTION IN MARINE MAMMALS IN THE NORTH ATLANTIC OCEAN	5

SESSION 2

A DESCRIPTIVE EPIDEMIOLOGICAL ANALYSIS ON VIRAL PATHOGENS IN MIGRATING BEAN GEESE (<i>Anser fabalis</i>) And White-Fronted Geese (<i>Anser albifrons</i>) WITH SPECIAL EMPHASIS ON NEWCASTLE DISEASE	6
WINTER MORTALITY IN WAXWINGS (<i>BOMBYCILLA GARRULUS</i>) CAUSED BY ETHANOL INTOXICATION	7
WILDLIFE SURVEILLANCE IN FRANCE : 10 YEARS OF RESULTS ON DISEASES SHARED BY CATTLE AND WILD UNGULATES	8
SURVEILLANCE OF WILDLIFE DISEASES IN FRANCE - CAUSES OF MORTALITY OF WILD UNGULATES : 10 YEARS OF RESULTS	9
A STUDY OF VITAMIN A LEVELS IN OTTERS (<i>LUTRA LUTRA</i>) IN SOUTH WEST ENGLAND	10
RETINAL DYSPLASIA IN WILD OTTERS: A PATHOLOGICAL SURVEY	11

SESSION 3

PREVALENCE AND PATHOGENICITY OF RETROVIRUSES IN WILDCATS <i>FELIS SILVESTRIS</i> IN FRANCE	12
THE DYNAMICS OF AN ENDEMIC DISEASE IN ITS WILD RESERVOIR HOST: COWPOX AND WILD RODENTS	13
THE EVOLUTION OF COWPOX VIRUS: A HYPOTHESIS	14
WILDLIFE DISEASES IN SPAIN: THE SEDIFAS VIEWPOINT	15
SEYCHELLES MAGPIE-ROBIN (<i>Copsychus sechellarum</i>): VETERINARY INTERVENTION FOR THE CONSERVATION OF AN ENDANGERED SPECIES	16

SESSION 4

ABSENCE OF ORTHOMYXOVIRUS AND PARAMYXOVIRUS SEROTYPE-1 (aPMV-1) INFECTIONS AMONG FREE LIVING WILD GEESE (<i>Anser anser</i> and <i>Branta canadensis</i>) BUT DETECTION OF aPMV-4, aPMV-6, aPMV-8, MYCOBACTERIA SPECIES, CHLAMYDIA SPECIES AND INTESTINAL PARASITES	18
SOME CAUSES OF MORTALITY OF THE MUTE SWAN (<i>CYGNUS OLOR</i>).....	19
THE LONDON WATERFOWL PROJECT: INFORMATION, COMMUNICATION AND EXPERT ASSISTANCE.....	20
BIRD FEEDERS AND THE HEALTH AND WELFARE OF GARDEN BIRDS	21

SESSION 5

RABBIT HAEMORRHAGIC DISEASE IN THE UK	22
HEALTH THREATS TO ENDANGERED FREE-LIVING WILDLIFE POPULATIONS .	23
BRUCELLOSIS IN FREE RANGING CHAMOIS (<i>Rupicapra rupicapra</i>) RELATIONSHIPS WITH MOUNTAIN CATTLE BREEDING.....	24
<i>BRUCELLA SUI</i> S BIOTYPE 2: A CAUSE OF FALSE POSITIVE SEROLOGICAL REACTIONS IN THE BRUCELLOSIS SCREENING TESTS IN CATTLE?	25

POSTERS

P1: ISOLATION OF <i>E. COLI</i> O86:K61 PRODUCING CYTO-LETHAL DISTENDING TOXIN FROM WILD BIRDS OF THE FAMILY FRINGILLIDAE	26
P2: SEROEPIZOOTIOLOGICAL INVESTIGATIONS OF DIFFERENT INFECTIOUS DISEASES IN BIRDS OF PREY IN PARTS OF EASTERN GERMANY	27
P3: ULTRASTRUCTURE OF EGGSHELLS OF BONELLI'S EAGLES (<i>Hieraaetus fasciatus</i>) WITH REPRODUCTIVE FAILURE IN CENTRAL SPAIN	28
P4: CAUSES OF DEATH IN RHEA AMERICANA CHICKS RAISED IN CAPTIVITY.	29
P5: POISONING OF BIRDS AND MAMMALS BY CARBOFURAN IN AUSTRIA.....	30
P6: PRELIMINARY DATA ON THE INFLUENCE OF SARCOPTIC MANGE ON SERUM PROTEINS AND IMMUNOGLOBULIN G LEVELS IN SPANISH IBEX (<i>Capra pyrenaica</i>) AND CHAMOIS (<i>Rupicapra pyrenaica</i>).....	31
P7: EPIDEMIOLOGY OF SCABIES IN VALANDOVO REGION -REPUBLIC OF MACEDONIA	32
P8: SEROLOGY OF PATHOGENS IN FREE-LIVING BLACK (<i>DICEROS BICORNIS</i>) AND WHITE (<i>CERATOTHERIUM SIMUM</i>) RHINOS IN AFRICA.....	33
P9: FECAL PARASITOLOGY STUDY ON GASTROINTESTINAL NEMATODES OF WHITE RHINOCEROS (<i>Ceratotherium simum</i>) IN LAKE NAKURU NATIONAL PARK (KENYA).....	34

P10: ZOOLOGICAL AND WILDLIFE PATHOLOGY – AN IMPORTANT AND EMERGING DISCIPLINE	35
P11: ON SOME RARE PATHOLOGIC FINDINGS IN CERVIDS.....	36
P12: OUTBREAK OF TULAREMIA IN IBERIAN HARES (LEPUS GRANATENSIS) IN THE WINTER OF 1997-98 IN NORTHWESTERN SPAIN.....	37
P13: A PARALLEL SEROPREVALENCE STUDY OF <i>Neospora caninum</i> AND <i>Toxoplasma gondii</i> IN A SAMPLE OF THE ETHIOPIAN CANID AND BOVID POPULATIONS	38
P14: <i>BRUCELLA MARIS</i> INFECTIONS IN MARINE MAMMALS FROM SCOTTISH WATERS.....	39
P15: ANTIBIOTIC RESISTANCE IN THE NORMAL GUT FLORA OF SMALL WILD RODENTS.....	40
P16: ECTOPARASITES AND MICROPARASITE TRANSMISSION IN WOODLAND RODENT COMMUNITIES.....	41
P17: GRANULOCYTIC EHRLICHIA INFECTION IN RODENTS AND IXODES TRIANGULICEPS TICKS IN A UK WOODLAND.....	42
P18: TRAUMA IS THE MAJOR CAUSE OF MORTALITY IN FINNISH OTTERS	43

THE "NO VISIBLE LESION" PRESENTATION OF NATURAL BOVINE TUBERCULOSIS IN WILDLIFE HOSTS: A REVIEW

D. Gavier-Widen¹ and J. Gallagher²

¹DVM, MSc, PhD, Central Veterinary Laboratory, New Haw, Weybridge KT15 3NB, UK;

²BVetMed, DTVM, MRCVS, Veterinary Investigation Centre, Staplake Mount, Starcross, Exeter EX6 8PE

"No visible lesion" (NVL) tuberculosis refers to the absence of gross tuberculous changes at routine post-mortem examination in animals that are diagnosed as tuberculous by culture or histopathology. It appears to be more frequent in wildlife hosts, particularly in reservoirs, than in domestic species. In the European badger (*Meles meles*) the frequency is subjected to geographical variations suggesting that different strains of *M. bovis* may be involved, up to 80% of the badgers appear as NVL in some areas in England. In New Zealand, it has been shown that 27.8% of feral ferrets (*Mustela furo*) are in the NVL category, and 8.4% of brushtail possums (*Trichosurus vulpecula*) with no gross lesions were diagnosed as tuberculosis by culture of pooled lymph nodes or by histology. The microscopic tuberculosis lesions found in the lungs of NVL badgers are typical early stage granulomas or containment reactions. The former are represented by a nodule of epithelioid cells, with a small centre of necrosis and neutrophils, surrounded by lymphocytes and macrophages. The containment lesions appear as a fibrous lightly mineralised necrotic focus. In the badger, based on the histomorphology, NVL tuberculosis represents early containment or latent infection, a proportion of cases would probably result in resolution while others maintain a potential for reactivation and development of progressive tuberculosis. In feral ferrets, microscopic lesions, predominantly nodular accumulation of macrophages, are more common in the liver than in any other single site, these lesions are most often not detected macroscopically and may be considered pathognomonic of the disease. In possums, the lesions are often minute and generalised and histopathology increases significantly their detection. There is limited information on the pathogenesis, immunological and epidemiological aspects of the NVL form of tuberculosis. Additionally, single site gross tuberculous lesion, usually in a lymph node, is also frequent in wildlife species. The diagnosis of tuberculosis based on post mortem examination only, may largely underestimate the prevalence in the wildlife host populations.

REFERENCES

1. Jackson *et al.* (1995) Naturally occurring tuberculosis caused by *Mycobacterium bovis* in brushtail possums (*Trichosurus vulpecula*): I. An epidemiological analysis of lesion distribution. *New Zealand Veterinary Journal* **43**, 306-314.
2. Gallagher *et al.* (1998) Role of infected, non-diseased badgers in the pathogenesis of tuberculosis in the badger. *Veterinary Record* **142**, 710-714.
3. I.W. Lugton *et al.* (1997) Epidemiology of *Mycobacterium bovis* infection in feral ferrets (*Mustela furo*) in New Zealand: I. Pathology and diagnosis. *New Zealand Veterinary Journal* **45**, 140-150.

ORAL BAIT DELIVERY TO WILD IRISH BADGERS

D.P. Sleeman, J. Prendergast and M.F. Mulcahy

Department of Zoology, University College, Cork, Ireland

There exists a need to develop a vaccine for bovine tuberculosis in badgers (*Meles meles*). To facilitate this a method of vaccine delivery is needed, and a possibility is the oral route. Two biomarkers have been developed to detect oral bait uptake by badgers. This development and subsequent field trials will be described.

THE SPATIAL ECOLOGY OF BADGERS IN A LOW DENSITY AREA AND THE IMPLICATIONS FOR THE EPIDEMIOLOGY OF TB

S. Feore and N.P. French

Centre for Comparative Infectious Diseases, University of Liverpool, UK

The role of the badger in the epidemiology of TB has been a cause for investigation by veterinary and ecological researchers for over 20 years. Efforts to remove low but persistent level of TB in parts of South West England have failed and, in recent years, TB levels have continued to rise and new outbreaks have started to appear in areas that were previously cleared of the disease as far north as Shropshire. Most studies have been carried out in areas of high badger density which are not typical of badger populations elsewhere. The lack of information on the spatial ecology of badgers in lower density badger populations needs to be addressed not only because of the increasing disease incidence in previously unaffected areas, but for the light that such a study might shed on the epidemiology of the disease.

In this present study a survey of badger setts was carried out in a large area of Cheshire where badger densities were reported to be low to medium. The study area incorporated cattle livestock areas to assess the location and activity of badgers in relation to cattle density. The implications of TB appearing in Cheshire would be grave as this is one of the highest cattle density areas in the country. This study presents data which examines badger spatial ecology, environmental factors and land management practices in an area of low to medium badger density and high cattle density and assesses the epidemiological implications.

ISOLATION AND CHARACTERISATION OF *BRUCELLA* SPP. IN A MINKE WHALE
(*BALAENOPTERA ACUTOROSTRATA*)

J. Godfroid¹, C. Clavareau¹, M. Tryland², V. Wellemans¹, P. Michel¹, F. Boelaert¹,
K. Walravens¹ and J.-J. Letesson³

¹Veterinary and Agrochemical Research Center, Brussels, Belgium; ²Norwegian College of Veterinary Medicine, Tromsø, Norway; ³University of Namur, Molecular Biology Unit, Namur, Belgium

We succeeded in isolating a novel, unknown *Brucella* species from a minke whale (*Balaenoptera acutorostrata*) that had been caught during commercial hunting off the Norwegian coast of Finnmark in May, 1995. *Brucella* spp. isolation in seals, dolphins and porpoises along the UK coast have been reported during the past few years. In a preliminary study, we have found serological evidence of *Brucella* spp infections in seals but also in whales caught in the North Atlantic. We have cultured spleen and liver samples from a minke whale classified positive by brucellosis serological tests. Cultures on *Brucella*-specific media yielded positive results. Cultural characteristics, agglutination and biochemical tests are consistent with the diagnosis of a smooth *Brucella* sp. Preliminary DNA work has shown positive PCR results for the specific *Brucella* 16S-23S Spacer and IS6501 sequences. RFLP studies based on the IS6501 sequence have shown a unique profile in comparison to those described for *B. melitensis*, *B. abortus*, *B. suis*, *B. neotomae*, *B. canis* and *B. ovis*.

Moreover, Southern Blot analysis, as well as the sequencing of the *omp2* locus, have shown a unique characteristic: the minke whale *Brucella* appears to have two copies of the *omp2b* gene instead of one copy each of the *omp2a* and the *omp2b* genes as seen in the other *Brucella* species, except for *B. ovis* which has two genes closely related to *omp2a*.

This is the first description of the isolation of *Brucella* spp. in rorquals. The minke whale strain is the first known *Brucella* strain that has 2 copies of the *omp2b* gene. This new *Brucella* strain is pathogenic in the mouse model and immunogenic in cattle. The isolation of a novel agent which is potentially responsible for reproductive disorders in baleen whales, as well as its potential zoonotic importance, is of concern. Finally, the isolation of *Brucella* spp. in whales, seals, dolphins and porpoises raises questions regarding the source(s) of infections and the phylogenetic link between the *Brucella* species found in marine mammals and domestic mammals.

EVIDENCE OF BRUCELLA INFECTION IN MARINE MAMMALS IN THE NORTH ATLANTIC OCEAN

M. Tryland¹, L. Kleivane, A. Alfredsson, M. Kjeld, A. Arnason, S. Stuen and J. Godfroid

¹Department of Arctic Veterinary Medicine, Norwegian College of Veterinary Medicine, N-9005 Tromsø, and Department of Immunoprophylaxis, National Veterinary Institute, P.O.Box 8156, N-0033 Oslo, Norway

The first serological evidence of *Brucella* infections in marine mammals from the Greenland Sea and the Barents Sea and the first evidence of such infections in baleen whales is reported. We investigated 1386 sera, obtained from four seal species and three whale species sampled from 1983 to 1996 in the waters west of Iceland, the pack-ice area north-west of Jan Mayen, and from the northern coast of Norway and the Kola Peninsula, the waters west of Svalbard, as well as additional locations in the Barents Sea, for the presence of anti-*Brucella* antibodies using an indirect ELISA (Protein G conjugate). Positive reacting sera were re-tested with classical *Brucella* serological tests (Serum Agglutination Test, EDTA-modified Serum Agglutination Test, Rose Bengal Test, and Complement Fixation Test) as well as with Anti-Complement ELISA. Anti-*Brucella* antibodies were detected in all marine mammal species investigated, except for the bearded seal (*Erignathus barbatus*), with the following prevalences: hooded seals (*Cystophora cristata*) 35%, harp seals (*Phoca groenlandica*) 2%, ringed seals (*Phoca hispida*) 10%, minke whales (*Balaenoptera acutorostrata*) 8%, fin whales (*Balaenoptera physalus*) 11%, and sei whales (*Balaenoptera borealis*) 14%. An isolate belonging to the genus *Brucella* was obtained from the liver and spleen of one of the seropositive minke whales. Our findings suggest that antibodies directed against the S-LPS of *Brucella* are widely distributed among marine mammals in the North Atlantic Ocean.

A DESCRIPTIVE EPIDEMIOLOGICAL ANALYSIS ON VIRAL PATHOGENS IN MIGRATING BEAN GEESE (*Anser fabalis*) And White-Fronted Geese (*Anser albifrons*) WITH SPECIAL EMPHASIS ON NEWCASTLE DISEASE

T. Müller¹, A. Hlinak², R.U. Mühle³, M. Kramer¹, H. Liebherr⁴, K. Ziedler⁵, D.U. Pfeiffer⁶

¹Institute for Epidemiological Diagnostics and Institute of Epidemiology, Federal Research Centre of Virus Diseases of Animals, D-16868 Wusterhausen/Dosse, Germany, ²Institute of Virology, Veterinary Faculty, Free University Berlin, D-10117 Berlin, Germany, ³Institute for Ecology and Nature Conservation, Department for Ecology of Waterfowl and Wet Lands, University of Potsdam, D-14447 Potsdam, Germany, ⁴Institute for Zoophysiology and Cellbiology, University of Potsdam, D-14447 Potsdam, Germany, ⁵State Veterinary and Food Investigation Centre Frankfurt/Oder, D-15234 Frankfurt/Oder, Germany, ⁶EpiCentre, Institute of Veterinary, Animal and Biomedical Sciences, Massey University, Palmerston North, New Zealand

Migratory waterfowl is considered to be a potential source of viral infections in poultry, especially for Newcastle disease virus. Although year by year high populations of wild geese are resting and wintering in Western Europe there is only limited understanding of their potential role in the epidemiology of avian viral diseases. In a capture - seroscreening - release study conducted in 1991 in the most important resting area for migratory waterfowl in the inland of Germany, the Havel river valley, the infection status of resting bean geese (*Anser fabalis*) and white-fronted geese (*Anser albifrons*) was determined for the first time. Both bird species did show high seroprevalences for Newcastle disease virus (NDV) - 44,6% and goose parvovirus (GPV)- 47,6%. There was also serological evidence of the occurrence of avian reoviruses (REO) - 29,3% and avian adenovirus (EDS 76) - 6,15% out of 130 sera tested. In order to obtain information about migratory behaviour and patterns of the birds in subsequent years, the geese were marked according to international standards prior to release. Between 1991 and 1996, of 65 bean geese and 65 white-fronted geese originally tagged a total of 130 sightings were recorded including 18 (27,6%) and 35 (53,8%) of the birds, respectively. More than 95% of the sightings came from the main resting and wintering areas in The Netherlands and Germany but there were also records from Poland, Russia and Belgium. Nineteen of the 53 birds sighted had serological evidence that they had been exposed to NDV before the time of marking in 1991. Although the origin of these infections in bean geese and white-fronted geese is still unknown the sightings reported in this study indicate that once infected wild geese may be involved in the dissemination and spread of avian viral diseases, specifically Newcastle Disease. Migration patterns of the wild geese provided further evidence that the main resting and wintering areas of migratory waterfowl are likely to be important for the inter- and intraspecies transmission of avian diseases.

WINTER MORTALITY IN WAXWINGS (*BOMBYCILLA GARRULUS*) CAUSED BY ETHANOL INTOXICATION

T. Mörner^{1,3}, A.-L. Barmark², E. Nordkvist², D.S. Jansson¹ and C. Hård af Segerstad¹

¹Department of Wildlife, National Veterinary Institute, PO Box 7073, S-750 07 UPPSALA, Sweden; ²Department of Chemistry, National Veterinary Institute, PO Box 7073, S-750 07 Uppsala, Sweden

Bohemian waxwings (*Bombycilla garrulus*) breeds in the Northern parts of Scandinavia and Russia. The birds migrate during the winter season southwards and are normally found in southern parts of Scandinavia. The birds feed in summertime on insects and in wintertime on fruits and berries, of which rowan berries (*Sorbus aucuparia*) are one of their favourite choices.

During cold periods small groups of waxwings (2-10) can be found dead below their roosting trees in the morning. Post mortem examination of these birds normally show that they are in good body condition and that the primary cause of death is trauma with internal bleedings. The most prominent pathological finding is a severe fattening of the liver, with rupture of the capsule and internal bleedings. No other pathological findings were normally found in investigated birds. The pathological changes are similar to those seen in acute alcoholic liver disease in humans.

Levels of ethanol in the liver were analysed by liquid chromatography using refractive index detection. In four investigated birds were levels of 0.02 - 0.05% ethanol found in the liver. Liver levels of alcohol are lower than in the blood, demonstrating that these birds were affected by ethanol intoxication.

WILDLIFE SURVEILLANCE IN FRANCE : 10 YEARS OF RESULTS ON DISEASES SHARED BY CATTLE AND WILD UNGULATES

M. Artois¹, and F. Lamarque²

¹SAGIR, CNEVA Nancy, BP 9, 54220 Malzeville, France; ²SAGIR, ONC, 78610 Saint Benoît

SAGIR is a network carrying out the epidemiological surveillance of diseases and infections of free ranging wild animals in France. Data on mortality provided by veterinary diagnostic laboratories are gathered and recorded in a computerised data base, at CNEVA Nancy (laboratoire d'études sur la rage et la pathologie des animaux sauvages). Other data on health of wild species are as well recorded on a less comprehensive base, as are serum analysis or results of researches on some pathogens. Data concerning wild ungulates were extracted from the data bases and analysed. This paper will focus on diseases of economical or zoonotical importance in wild species of hoofed mammals. There are 9 species of wild ungulates concerned by this study in France (% of the total number of analysis performed during the concerned period of time): Chamois (and Isard) (5,7%), Fallow deer (0,1%), Ibex (1%), Mouflon (0,6%), Red (2%) and roe deer (71,2%) as well as Wild boar (18%). The SAGIR network mainly provides information on mortality. But as a necropsy is frequently difficult to interpret, results available include as well pathological findings, even if it was not proven they can cause the death. The data presented are limited to list A and B of the OIE, as well as other diseases and infections which are important for domestic species. Among the most significant causes of mortality are rabies, pasteurellosis, in roe-deer, and Classical swine fever and mange in wild-boars. In addition to this list, paratuberculosis and contagious ecthyma are mentioned occasionally in some species as pathological findings. Some pathogens are mentioned as potentially lethal, such are some bacteria (*Corynebacterium pyogenes*, Haemolitical strains of *E. coli*, *Streptococcus bovis* and *Staphylococcus aureus*) as well as parasites (*Dicrocoelium lanceolatum*, *fasciola hepatica*). These results are completed by other types of surveillance: mainly on rabies and on classical swine fever. But local, occasional or more systematically organised sero-surveys are carried out. They are helpful to determine the possible carriage of miscellaneous pathogens from which *Brucella* sp. is the most relevant. The role of wildlife as a reservoir of diseases and infection for livestock is discussed as well as the possible improvements of the SAGIR network to provide better information of the health of free ranging wild ungulates in France.

SURVEILLANCE OF WILDLIFE DISEASES IN FRANCE - CAUSES OF MORTALITY OF WILD UNGULATES : 10 YEARS OF RESULTS

F. Lamarque¹, M. Artois² and C. Hatier²

¹Office National de la Chasse, Saint Benoist - F 78610 Auffargis - France, ²CNEVA-Nancy, Domaine de Pixérécourt, B.P. 9 - F 54220 Malzeville - France.

Created in 1986 by the "Office National de la Chasse" (ONC), a government agency in charge of wildlife, the SAGIR network is the French national surveillance system of wildlife diseases. From its creation up to the end of 1997, 3,996 roe deer, 997 wild boar, 321 chamois and isard, 108 red deer, 59 ibex and 34 mouflon were analyzed by the network.

For the Roe deer, mortality is mainly observed during the fall, from September to November. In this species, the cases of mortality due to only one pathogen are scarce. The principal causes of death are linked to the association of several pathogens, e.g. a parasite and a bacteria (mainly with pulmonary tropism). Traumas of various origin are also a very important cause of mortality.

For the Wild Boar, the highest number of cases of mortality is registered in November and December. No specific infectious cause of mortality is shown in the sample analyzed, excepted maybe, Pasteurellosis. As for the Roe deer, pulmonary infections seem to be one of the commonest lethal affection. Poisoning and trauma are also provoking a high number of deaths in this species.

In the group Chamois/Isard, mortality is mainly observed during September and October. The diseases known as rather specific to these species, like Infectious Kerato-Conjunctivitis or Abscess Disease do not appear as an important cause of mortality in our sample. One may note some cases of mortality due to a contamination by domestic livestock diseases like Brucellosis or contagious Ecthyma. Given the habitat of the species, the traumas are responsible for 36.6 % of the determined causes of mortality.

For the other species of ungulates, an important amount of mortality causes is listed. But, the sample is too small to draw reliable information.

A STUDY OF VITAMIN A LEVELS IN OTTERS (*LUTRA LUTRA*) IN SOUTH WEST ENGLAND

V. Simpson^a, M. Bain^b, B. Brown^c, R. Brown^c, R. Lacey^d

^aVI Centre, Truro, Cornwall, UK, ^bVI Centre, Shrewsbury, Shropshire, UK, ^cEnvironment Agency, Exeter, Devon, UK, ^dWRc Marlow, Buckinghamshire, UK

Britain's otter population went into steep decline in the late 1950's. Over the last 30 years various explanations have been offered, including over-hunting, competition with mink (*Mustela vison*), habitat destruction and pollution. During this period tissue samples from a large number of otters have been analysed for organochlorine pesticides and polychlorinated biphenyls (PCB's) and although there is now general acceptance that either or both of these groups of chemicals were responsible for the decline, this is still an area for debate. A study, started in 1988, sought to systemically examine any otters found dead in south west England. The study protocol included analysis of liver samples for Vitamin A, as well as for polyhalogenated hydrocarbons. Tissues were also examined for gross and histopathological lesions.

Over an eight year period liver samples were analysed from 44 otters for Vitamin A and from 56 for polyhalogenated hydrocarbons. The Vitamin A levels in the early years were mostly very low, often below 7 $\mu\text{mol/kg}$, but this was followed by a steady annual increase. Regression analysis demonstrated a highly significant rate of change from 1988 to 1996 of +94% ($r^2 = 0.28$, standard error 0.064, $p < 0.001$). During the same period there were also highly significant decreases in the levels of certain organochlorine pesticides and PCB congeners. The Vitamin A levels showed a significant inverse correlation with Dieldrin and PCB 153. However, multiple regression analysis showed that the changes in the concentrations of these compounds were more closely related to time than to each other. The possibility that other unidentified pollutants, including coplanar PCB congeners, may have influenced the Vitamin A levels is considered.

Seven otters had hepatic Vitamin A levels of less than 7 $\mu\text{mol/kg}$ and these were considered to be deficient. However, only one of these animals, showed gross lesions consistent with Vitamin A deficiency. This was a stunted cryptorchid male. Histopathological examinations were carried out on various tissues from these deficient otters, including salivary gland from one, urinary bladder from three and kidney and thyroid from five. None of the tissues showed changes suggestive of hypovitaminosis A. Eyes from three of these deficient animals, and from many of the other otters, were also placed in fixative and the results of histopathological examination will be reported elsewhere.

RETINAL DYSPLASIA IN WILD OTTERS: A PATHOLOGICAL SURVEY

D.L. Williams¹, A. Flindall¹, V.R. Simpson²

¹Animal Health Trust, Lanwades Park, Kentford, Newmarket CB8 7UU; ²Polwhele Veterinary Investigation Centre, Truro, Cornwall TR4 9AD

Introduction: Eyes from fifty nine wild otters presented predominantly as carcasses after road traffic accidents, and for which post-mortem findings have previously been reported by Simpson (1997), were investigated both grossly and histopathologically. Gross pathological change and histopathological findings were correlated with liver vitamin A levels and tissue levels of pollutants such as polychlorinated biphenyls.

Materials and methods: Eyes were fixed and stored either in 10% buffered formalin and transferred to 70% alcohol for 24 hours prior to bisection or in 2.5% gluteraldehyde. 10 micrometre sections were cut and stained with haematoxylin and eosin.

Results: Few eyes showed abnormalities on gross examination of the entire or hemitransected globe although some were characterised by obvious gross retinal folds. The particularly striking finding of this study was that of retinal folding and rosetting characteristic of retinal dysplasia. 32% of eyes had apparently artefactual posterior segment change predominantly involving post-mortem retinal detachment. 25% were normal without significant retinal abnormality. 12% had unmistakable dysplastic changes not complicated by any artefactual changes. A further 25% were considered likely to be demonstrating dysplastic change although interpretation of ocular pathology in these eyes was complicated by some concurrent artefactual change. Regression analysis showed the incidence of retinal dysplasia to fall both with time and with liver levels of vitamin A although neither correlation was significant, r^2 in a multivariate regression analysis being 0.035 and 0.004 respectively. 11% of eyes had other non-dysplastic ocular pathology of the ocular surface, the anterior or the posterior segments. Lesions ranged from lymphoid aggregates in the cornea and periocular multinucleate giant cell granulomas with protozoal cysts in periocular fat through to signs of lentivular and vitreal liquefaction, neurectodermal proliferation and choroidal thickening.

Discussion: The finding of retinal folding and rosetting in this study is complicated by the concurrent artefactual changes of retinal detachment occurring when eyes are fixed in formalin or fixed some time after death as occurred in several cases here. Nevertheless although 13% of these eyes had changes which could be entirely artefactual in origin around 30% have retinal folds or rosettes which could not have occurred artefactually. 11% had other non-dysplastic ocular pathology. It is thought most likely that the retinal dysplasia was caused by low maternal vitamin A levels, although this can clearly not be directly substantiated. Low levels of vitamin A level have been documented wild otters, occurring at a time when levels of polychlorinated hydrocarbons (Simpson et al 1998). Thus an association between the retinal lesions and low vitamin A levels during ocular development would seem a plausible hypothesis. The regression analysis performed, demonstrated the incidence of retinal dysplasia to decrease over time with an inverse relationship to liver vitamin A levels although correlation with time or vitamin A concentration was very poor. .

References

1. Simpson, V.R. (1997) Health status of otters (*Lutra lutra*) in south-west England based on post-mortem findings. *Vet Rec* 141, 191-197
2. Simpson, V.R., Bain, M.S., Brown, R., Brown, B., Lacey, R.F. (1998) A longterm study of vitamin A and polychlorinated hydrocarbon levels in otters (*Lutra lutra*) in south west England. In preparation.

PREVALENCE AND PATHOGENICITY OF RETROVIRUSES IN WILDCATS *FELIS SILVESTRIS* IN FRANCE

E. Fromont¹, A. Sager², F. Léger³, F. Bourguemestre⁴, D. Pontier¹, P. Stahl³, E. Cain², M. Artois²

¹UMR CNRS 5558, Université Lyon 1, 43 bd du 11 novembre 1918, 69622 Villeurbanne Cedex, France; ²Centre National d'Etudes Vétérinaires et Alimentaires, Domaine de Pixérécourt, BP 9, 54220 Malzéville, France; ³Office National de la Chasse, Montfort, 01330 Villars les Dombes, France; ⁴Fédération Départementale des Chasseurs de l'Indre, 46, Bd du Moulin Neuf, BP12, 36001 Chateauroux, France

Feline Leukemia Virus (FeLV) and Feline Immunodeficiency Virus (FIV) are major pathogens of domestic cats *Felis catus*. Nevertheless their prevalence and pathogenicity in wildcats *Felis silvestris* were mostly unknown.

We studied 38 wildcats from Eastern and Central France, either captured or found dead following road accident. Nine individuals (23.68%) carried FeLV p27 antigen, and 3 (7.89%) had FIV antibodies. The FeLV prevalence is more important than found previously, and FIV-positive results constitute the first sign of FIV-related virus in wildcats.

We recorded body condition of 25 cats, either as the residual of the regression of body mass on tarsus length, or as an index describing the amount of renal and cutaneous fat. With both measures, there was a significant relationship between body condition and FeLV status, FeLV-positive cats being in poorer condition than negative individuals.

Our sample does not show evidence of selection bias, nevertheless the representativeness of the results remains to be investigated. In any case, this study suggests that FeLV and FIV are more frequent than found previously, and it constitutes an evidence that FeLV can be pathogenic for wildcats. Further data are needed to search for the origin of the high level of infection in wildcats.

THE DYNAMICS OF AN ENDEMIC DISEASE IN ITS WILD RESERVOIR HOST: COWPOX AND WILD RODENTS

S.M. Feore, M. Bennett, J.C. Chantrey, K. Bown, D. Baxby and M. Begon

Centre for Comparative Infectious Diseases, University of Liverpool

The study of host-microparasite dynamics in natural populations has been largely confined to the study of epidemics following the outbreak of a disease to which the population had not previously exposed. Few studies have focused on endemic infections that circulate within a natural reservoir species. Cowpox virus is an orthopoxvirus, found only in Eurasia and serological evidence suggests that wood mice (*Apodemus sylvaticus*), bank voles (*Clethrionomys glareolus*) and field voles (*Microtus agrestis*) are the reservoir hosts for cowpox in Great Britain and most of Western Europe. We have carried out a study into the dynamics of cowpox in two populations of wild rodents dominated by bank voles and wood mice. The study involves detailed longitudinal studies in two woodland areas where each individual is identified and blood sampled every four weeks.

Examination of the data obtained over the last three years has shown that while population and infection dynamics differ for the two species, the prevalence of infection is highest in both in the autumn. Furthermore, while infection causes little overt disease in its natural reservoir hosts, experiments have revealed that infection with cowpox affects fecundity which could in turn affect host population dynamics. A preliminary survey has provided evidence of a threshold population size for maintenance of infection in rodents, and the possibility that arthropods might be involved in transmission is also being investigated. This cowpox and wild rodent study therefore provides a readily-accessible model to determine the general features of endemic disease in a wildlife reservoir.

THE EVOLUTION OF COWPOX VIRUS: A HYPOTHESIS

M. Bennett¹, D. Baxby¹, M. Begon¹, J. Chantrey¹, S. Feore¹ and H. Meyer²

¹Centre for Comparative Infectious Diseases, University of Liverpool, UK; ²Institute of Microbiology, Federal Armed Forces Academy, Munich, Germany

Cowpox virus is an orthopoxvirus which circulates mainly in wild rodents throughout much of Europe and parts of Western. Molecular and phenotypic studies demonstrate a degree of variation amongst cowpox virus isolates which has not, so far, been observed among other orthopoxviruses. Although not enough isolates have been studied in sufficient detail to determine the nature of correlations between local reservoir host, geographic source and isolate properties, spatial variation in cowpox virus isolates, which may reflect local differences in host range, is marked. For example, the degree of difference between isolates within geographically discrete groups of isolates varies, with most variety being seen in Central Europe but little difference being seen between isolates from, for example, Great Britain. Furthermore, the largest differences between isolates, or groups of isolates, are between those found at the extremes of the geographic range.

The modern distribution of both virus and viral variation suggests a Central European origin for cowpox virus, with subsequent radiation. Comparison of the molecular phylogeny and current geographic and host range of cowpox virus, together with the fossil record of putative hosts, enables a hypothesis of co-evolution with rodents, particularly voles, to be developed. Moreover some approximate dates can be ascribed to these events.

The present day host range appears to include both reservoir hosts in which the virus can be maintained, and endemic hosts in which the virus circulates but probably cannot be maintained, in addition to a variety of accidental hosts (including man) with varying susceptibility to infection and disease. Indeed, it is interesting to speculate on the function and origins of cowpox virus virulence genes given that little disease is seen in the reservoir hosts in which, we hypothesise, the virus evolved.

Cowpox virus has the largest genome of the Old World orthopoxviruses, and some have suggested it is evolutionarily the most ancient of the existing orthopoxviruses. Comparison of the known phylogenetic, geographic and ecological features of cowpox virus with the other old world orthopoxviruses, may provide some clues as to evolution of orthopoxviruses overall.

WILDLIFE DISEASES IN SPAIN: THE SEDIFAS VIEWPOINT

C. Gortazar, and D. Fernandez-de-Luco

SEDIFAS, Wildlife Diagnostic Service. Facultad de Veterinaria, Universidad de Zaragoza, c/Miguel Servet 177, 50013 Zaragoza, Spain

SEDIFAS is a Wildlife Diagnostic Service made up of the University of Zaragoza and the private consulting Ebronatura S.L. This team offers laboratory services, field research and information to anyone interested in wildlife diseases in Spain. Its main clients are official fish and game agencies, biologists, game preserves and gamebird farms. From January 1995 to June 1998, 741 cases including about 1,300 necropsies were submitted to the Faculty. These cases belong to carnivores in 177 cases (24%), ungulates, 158 cases (21%), lagomorphs, 157 cases (21%), gamebirds, 144 cases (19%), raptors and other birds, 82 cases (11%), and to other vertebrates, 23 cases (3%). The geographical range covered included 36 out of the 52 Spanish provinces.

The presentation summarizes the most interesting findings and makes a revision of previous papers dealing with wildlife diseases in Spain. The objective is to inform about the current status of wildlife diseases in the study area.

SEYCHELLES MAGPIE-ROBIN (*Copsychus sechellarum*): VETERINARY INTERVENTION FOR THE CONSERVATION OF AN ENDANGERED SPECIES

M. Cooke*

Institute of Zoology, Zoological Society of London, Regent's Park, London NW1 4RY, UK,

*Correspondence: 7 Cheveney Walk, Bromley BR2 0XZ, UK

The Seychelles magpie-robin (*Copsychus sechellarum*) is endemic to the granitic islands of the Seychelles archipelago. The destruction of its mature closed-canopy forest habitat and the introduction of predatory and competitor species have been blamed for decline. By 1965 the entire world population of Seychelles magpie-robins had fallen to no more than 12 birds on Fregate island. There are 68 birds on four islands at the time of writing.

Recovery initiatives for the Seychelles magpie-robin have included habitat restoration, the provision of nest boxes and supplementary feeding. Translocations of magpie-robins to other islands have had mixed success. The deaths of several birds, apparently from *Erysipelothrix rhusiopathiae* infection has alerted conservation managers to the need for veterinary involvement in the project. In addition, the recent arrival of rats (*Rattus norvegicus*) on Fregate island poses an additional threat to the main population of Seychelles magpie-robins.

Following an investigative visit to the Seychelles other disease threats to magpie-robins were identified and a health investigation and management plan formulated. The case of the Seychelles magpie-robin illustrates the need for veterinary involvement in the conservation management of endangered species. It also demonstrates the importance of an ecosystem-based approach to population health planning.

CURRENT CONCEPTS OF INTERACTIONS BETWEEN RED AND GREY SQUIRRELS IN THE BRITISH ISLES

P. Nettleton¹, J.A. Gilray¹, T. Sainsbury², J. Gurnell³

¹Moredun Research Institute, International Research Centre, Pentlands Science Park, Bush Loan, Penicuik, Midlothian EH26 0PZ, ²Veterinary Science Group, Institute of Zoology, Regent's Park, London NW1 4R7, ³School of Biological Science, Queen Mary College, Mile End Road, London E1 4NS

Throughout Europe the indigeneous red squirrel (*Sciurus vulgaris*) is abundant. Only in Britain and Italy is it in decline as a result of displacement by the larger grey squirrel (*Sciurus carolensis*) introduced from North America. In Britain, grey squirrels were established in central England by 1876 and have been spreading from there ever since. In Ireland, grey squirrels were introduced in 1913 and have since become widespread. The current distribution of red squirrels in the British Isles is well known. In England and Wales only a few isolated populations exist except in the northern counties of England where reds are more numerous. In Scotland and Ireland there are still areas the greys have not reached where the red populations are good; there are also regions where reds and greys appear to co-exist. These regions have both conifer forests, favoured by the reds, and broadleaf forests favoured by greys.

The role of infectious disease in the demise of red squirrels has been considered of importance since 1930 when outbreaks of unidentified epidemic disease were first described. Subsequent investigations have implicated coccidiosis and parapoxvirus infections as important diseases. There is evidence that parapoxvirus disease may have a significant impact on red squirrel populations. The virus causes skin lesions and apparently the majority of affected red squirrels die. A high percentage of grey squirrels have antibody to the virus and they may represent a reservoir host. Where both species interact there is evidence for reds, but not greys, being killed by the virus.

ABSENCE OF ORTHOMYXOVIRUS AND PARAMYXOVIRUS SEROTYPE-1 (aPMV-1) INFECTIONS AMONG FREE LIVING WILD GEESE (*Anser anser* and *Branta canadensis*) BUT DETECTION OF aPMV-4, aPMV-6, aPMV-8, MYCOBACTERIA SPECIES, CHLAMYDIA SPECIES AND INTESTINAL PARASITES

A.L. Bolte¹, E.F. Kaleta¹ and W. Lutz²

¹Institut für Geflügelkrankheiten, Justus-Liebig-Universität, Frankfurter Straße 91, 35398 Gießen, Germany, ²Landesanstalt für Ökologie, Bodenordnung und Forsten / Landesamt für Agrarordnung, NRW, Dezernat Forschungsstelle für Jagdkunde und Wildschadenverhütung, Pützchens Chaussee 228, 53229 Bonn, Germany

More than 920 tissue samples from about 150 wild geese (*Anser anser* and *Branta canadensis*) which were collected between 1994 and 1998 were examined for the presence of viruses. No orthomyxo- and paramyxoviruses or other embryo-lethal or cytopathogenic viruses were isolated. In addition sera and egg yolk samples were tested for antibodies against several viruses which may cause disease in wild and domestic geese. Haemagglutination inhibition and virus neutralisation tests were used. High antibody titres against avian paramyxovirus serotype 6 (aPMV-6) and also antibody titres against aPMV-4 and aPMV-8 were detected.

One goose (*A. anser*) showed granulomas with necrotic areas in the spleen which contained acid fast rods (Mycobacteria species). Salmonella species or Riemerella species were never isolated from different organ samples of these geese.

Attempts to detect Chlamydia species were made by using an immunofluorescence test. Chlamydia species were proved in 14 out of 46 geese, mostly from the conjunctival swabs.

In most of the investigated geese Amidostomum species were obvious by necropsy of the intestinal tract. In some cases coccidia oozysts were detected by microscopical examination of gut contents.

The epidemiological aspects and the significance of these findings are discussed.

This study is ongoing and additional results are expected, especially as far as serological attempts are concerned.

SOME CAUSES OF MORTALITY OF THE MUTE SWAN (*CYGNUS OLOR*)

T.W. Pennycott

SAC Veterinary Science Division, Auchincruive Ayr KA6 5AE

As part of a survey into the causes of mortality in wild birds, between January 1995 and December 1996 the carcasses of 41 mute swans (*Cygnus olor*) were examined at SAC Veterinary Science Division, Auchincruive, Ayr, Scotland.

Birds were submitted for post mortem examination directly by members of the general public, by a local wildlife rehabilitation centre, or by other organisations with an interest in wild birds. Fourteen of the birds were received from one site during a five month period and were representative of a greater number of dead swans found on that site. The remaining 27 swans were received over the two years from several different sites as sporadic deaths.

The most significant findings in the birds from the site with heavy mortality were lead poisoning, heavy parasitic burdens, bacterial diseases and aspergillosis. In contrast, in birds submitted as apparent sporadic deaths, the commonest causes of death were trauma, heavy parasitic burdens, starvation, and aspergillosis.

This paper will describe and illustrate the standard post mortem approach and the lesions encountered at post mortem examination, and will discuss the significance of the findings and compare them with other mortality surveys conducted in swans.

THE LONDON WATERFOWL PROJECT: INFORMATION, COMMUNICATION AND EXPERT ASSISTANCE

D. Bourne and S. Boardman

Wildlife Information Network, The Royal Veterinary College, Royal College Street, London NW1 0TU

Greater London contains a wide variety of environments suitable for waterfowl - rivers, canals, lakes, reservoirs and their surroundings, which together provide habitats for more than 15,000 breeding waterfowl in summer and perhaps as many as 60,000 at any one time in winter. For some species this area is of national or even international importance. The habitats used by these birds are managed by many different organisations, including voluntary organisations, Borough Councils, the Corporation of London, The Royal Parks, the Wildfowl & Wetlands Trust, the Environment Agency, Thames Water and British Waterways.

Most of the waterfowl in Greater London are wild or feral, but there are also collections of exotic species maintained in parks and which interact freely with wild birds using the same habitat. Waterfowl are mobile and disease outbreaks may easily spread from one waterbody to another; in such cases managers of affected waterbodies need to be able to inform other local managers of the disease outbreak. Other problems might involve particular sites or individual birds; however the same or similar concerns have to be addressed by different managers. Time and resources are wasted if each manager individually has to learn solutions (by trial and error) which may have been proven for many years at other sites.

The London Waterfowl Project was developed by the Wildlife Information Network to assist waterfowl managers in the Greater London area. The project is designed to address two complementary needs. These are the need for increased ease of communication between managers and the requirement for a readily-accessible source of information relating to waterfowl and their management.

General communication between managers is being assisted by producing and distributing a contacts list, while an Early Warning System is being developed to ensure rapid notification of disease outbreaks. At the same time, a WILDPro™ database module on waterfowl is being created, containing information on all aspects of waterfowl management from species identification through general management to disease investigation, treatment and control.

BIRD FEEDERS AND THE HEALTH AND WELFARE OF GARDEN BIRDS

J.K. Kirkwood¹ and S.K. Macgregor²

¹ Universities Federation for Animal Welfare, The Old School, Brewhouse Hill, Wheathampstead, Hertfordshire AL4 8AN, UK, ² Institute of Zoology, Regent's Park, London NW1 4RY, UK

Feeding garden birds has become a popular activity. It has been estimated that 15,000 tonnes of peanuts are provided for wild birds each year in the UK and that in the USA the industry has a 2 billion dollar annual turnover (Moss & Cottridge, 1998). Presumably, provisioning can compensate, to some extent, for reductions in the availability of natural foods as a result of land use changes in recent times. However, there is some evidence that, under some circumstances, provisioning may increase the risk to wild birds of infectious disease (Kirkwood, 1998; Hartup et al, 1998; Pennycott et al, 1998). *Salmonella typhimurium* has been recognised as one of the most common causes of mortality incidents in garden birds in Europe and the USA. In the UK, *S. typhimurium* phage type 40 is known to affect a variety of species including: greenfinch *Carduelis chloris*, chaffinch *Fringilla coelebs*, bullfinch *Pyrrhula pyrrhula*, house sparrow *Passer domesticus* and it has also been diagnosed as the cause of mortality incidents in pine siskin *Carduelis pinus* and other species in the USA (Kirkwood & Macgregor, 1998; Prescott et al, 1998). Another agent that has recently been found to be a common cause of mortality incidents, especially of finches, in the UK is *E. coli* O86 (Pennycott, 1998). It seems likely that faecal contamination of food is an important route for transmission for both these diseases and thus that transmission is facilitated by high population densities at feeding stations. This hypothesis has been supported by the results of a preliminary study comparing causes of mortality in relation to level of feeding in gardens (Kirkwood, 1998). Steps to avoid high bird densities at feeding stations and to maintain good hygiene are likely to reduce the risks of infectious disease outbreaks (Kirkwood & Macgregor, 1997).

References

1. Moss, S. & Cottridge, D. (1998) Attracting birds to your garden. New Holland, London.
2. Kirkwood, J.K. & Macgregor, S.K. (1997) Infectious diseases of garden birds: minimising the risks. Universities Federation for Animal Welfare, Wheathampstead, Herts, UK.
3. Kirkwood, J.K. & Macgregor, S.K. (1998) Salmonellosis in provisioned free-living greenfinches (*Carduelis chloris*) and other garden birds. Proceedings of the Second Scientific Meeting of the European Association of Zoo and Wildlife Veterinarians. Chester, 21-24 May 1998. Pp 229-233.
4. Kirkwood, J.K. (1998) Population density and infectious diseases at bird tables. Veterinary Record 142, 468.
5. Hartup, B.K., Mohammed, H.O., Kollias, G.V. & Dhondt, A.A. (1998) Risk factors associated with mycoplasmal conjunctivitis in house finches. Journal of Wildlife Diseases 34, 281-288.
6. Pennycott, T.W. (1998) Population density and infectious disease at bird tables. Veterinary Record 142, 523.
7. Prescott, J.F., Poppe, C., Goltz, J. & Campbell, G.D. (1998) *Salmonella typhimurium* phage type 40 in feeder birds. Veterinary Record 142, 732.

RABBIT HAEMORRHAGIC DISEASE IN THE UK

J.P. Duff¹, R.C. Trout², D. Westcott¹, D Chasey¹, G Sharp¹

¹VLA, Penrith VI Centre, Merrythought, Calthwaite, Penrith CA11 9RR, ²Rabbit Wise Consultancy

Rabbit Haemorrhagic Disease (RHD), also referred to as Rabbit Calicivirus Disease (RCD) was first diagnosed in the UK, in domesticated rabbits (*Oryctolagus cuniculus*), in 1992. The disease rapidly became established in southern England. However, it was not until 1994 that the first cases were confirmed in wild (free-living) rabbits. Undoubtedly, large numbers of wild rabbits have succumbed to RHD since 1994, although accurate mortality data is not available for several reasons, not least, because many animals died undetected in their burrows. It is only now that workers can begin to assess the impact of RHD on affected wild populations.

Data from diagnosed cases has been collected by the authors throughout the period in which clinical RHD occurred in the UK up to the present time. From the time of onset in 1992 until 1996, the disease in both domesticated and wild animals was declared notifiable by MAFF and consequently all cases were recorded. Following de-notification, perhaps not surprisingly, the number of diagnosed cases has fallen precipitously. To this MAFF/VLA database, we can add information on wild rabbits from collaborative research. Finally, a small amount of further data is provided from a variety of other sources (eg regional laboratories).

The paper seeks to review, from this data, the history of the disease in the UK. Primary consideration is given to the disease in wild rabbits, but reference will be made to the domesticated animal, where relevant. Sero-positive wild rabbits probably existed in the UK prior to emergence of clinical disease and the bearing that this has had on the expression of subsequent disease is considered significant. The possible origins of disease in the UK are explored, together with the subsequent pattern of geographical spread, which was closely monitored as the epidemic developed. The role of fomites, as vectors of virus transmission as indicated by circumstantial findings, is discussed. We assess, as far as is possible, the current prevalence of RHD and speculate on the future occurrence of the disease in wild populations, touching on the possible consequences for other wildlife and fauna.

HEALTH THREATS TO ENDANGERED FREE-LIVING WILDLIFE POPULATIONS

M. Cooke*

Institute of Zoology, Zoological Society of London, Regent's Park, London NW1 4RY, UK,

*Correspondence: 7 Cheveney Walk, Bromley BR2 0XZ, UK

The presence of disease in wildlife populations may only become apparent after it has reached extreme levels and its effects are obvious. The importance of disease is therefore often underestimated and it remains relatively neglected by conservation biologists. Furthermore, it is well known to veterinarians dealing with production animals that diseases, which do not necessarily themselves cause substantial mortality, may nevertheless have significant effects on parameters such as physical fitness and reproductive performance. Most infectious agents do not result in obvious disease, but the host must nevertheless pay a price in terms of fitness for harbouring them. The effects of genotypic and environmental variation may be reflected in differences in host-parasite interactions.

Current views of wildlife disease place susceptible hosts, disease agents and environmental factors in a three-cornered relationship; disease occurs when the three come together. All else is said to be a state of health. The concept of health is a human construct; however it could be described as a state of balance in which individuals or populations may coexist with agents capable of causing harm, but without suffering impaired function as a result. Newly developing ideas of ecosystem health incorporate the concept of co-adapted groups of organisms with partially overlapping fitness landscapes. Such systems are in a state of punctuated equilibrium, where health breakdowns are often manifested as threshold effects, with no warning of impending crisis. In the case of endangered species the effects of genetic, demographic and environmental stochasticity may reduce the capacity of a population to recover from a disease challenge.

The threat of both manifest and cryptic disease to endangered free-living wildlife populations is reviewed.

BRUCELLOSIS IN FREE RANGING CHAMOIS (*RUPICAPRA RUPICAPRA*) RELATIONSHIPS WITH MOUNTAIN CATTLE BREEDING

D. Gauthier

Laboratoire Départemental d'Analyses Vétérinaires de la Savoie - 321 chemin des Moulins -
BP 1113 - F 73011 CHAMBERY CEDEX

Systemic brucellosis caused either by *Brucella melitensis* biovar 3 or by *Brucella abortus* biovar 1 occurred in Chamois within three focus in French Alps during the last 15 years. This disease brings a risk to both human and animal health. A clinical description based on 21 necropsies shows that Chamois may be a very sensitive species, for which infection generally evolves to letal issue in few monthes. However, some animals are able to survive over one year.

Serological surveys (n = 872, Card Test, Complement Fixation Test) were led in the three focus, from samples of blood and of lungs analyzed through a derivated serological method. It showed that there was no wild reservoir : the infection source was strictly located, and the only recognized intraspecific contamination occurred inside the mother-kid pair. Epidemiological enquiry revealed a domestic source for each focus. The route of transmission might be oro-nasal, through seasonal succession of domestic stock and Chamois in the same pastures.

There is a contradiction between the drastic decrease of domestic prevalence and the recent rise of brucellosis focus in Chamois : the explanation may be in connection with the wild population expansion and the evolution of pastoral traditions towards free-wandering domestic herds. Until now, wild Brucellosis has spontaneously extinguished, but there might be a threshold of sympatry between domestic source and wild victims, beyond which wild focus will become endemic.

BRUCELLA SUIIS BIOTYPE 2: A CAUSE OF FALSE POSITIVE SEROLOGICAL REACTIONS IN THE BRUCELLOSIS SCREENING TESTS IN CATTLE?

J. Godfroid¹, F. Boelaert¹, K. Walravens¹, V. Weynants², J.-J. Letesson²

¹Veterinary and Agrochemical Research Center, Brussels, Belgium; ²University of Namur, Molecular Biology Unit, Namur, Belgium

We have described an enzootic brucellosis (*Brucella suis* biotype 2, BS2) in wild boars (*Sus scrofa*) in the south-eastern part of Belgium. Contacts between cattle and wild boars are regularly reported. In this area false positive serological reactions (FPSR) occur in bovine brucellosis screening tests. All of the causes for the FPSR in cattle are not well known although the cross-reactivity of *Yersinia enterocolitica* O:9 is suspected. In the USA, in Australia and in New Zealand, *B. suis* biotype 1 or 3, which are isolated regularly from feral pigs, have also been isolated from cattle and have been reported to induce positive serological results in brucellosis screening tests. Infections from either of these bacteria may eventually cause a mild disease in cattle; however, no abortions have been reported.

In order to assess if BS2 can cause FPSR in cattle, BS2 experimental infections in cattle were performed. We infected two 12 month-old heifers with 4×10^6 BS2 and two 12 month-old heifers with 4×10^9 BS2 by the conjunctival route. Transient positive serological results were detected by classical brucellosis serology and iELISA in both groups of animals, which is the same phenomenon that is seen in the field in FPSR cases. At slaughter 14 weeks post infection, 14 pairs of organs were sampled. No BS2 could be isolated by classical bacteriological methods.

These results suggest that BS2 can be considered as a potential source of FPSR in bovine brucellosis screening tests.

P1: ISOLATION OF *E. COLI* O86:K61 PRODUCING CYTO-LETHAL DISTENDING TOXIN FROM WILD BIRDS OF THE FAMILY FRINGILLIDAE

G. Foster¹, H.M. Ross¹, T.W. Pennycott², G.F. Hopkins³, I.M. McLaren⁴

¹SAC Veterinary Services, Inverness, ²SAC Veterinary Services, Auchincruive, Ayr, ³SAC Veterinary Services, Thurso, ⁴Central Veterinary Laboratory, Addlestone, Surrey

A non-lactose or late-lactose fermenting *E.coli* was recovered from tissues of forty-three out of a total of forty-six finches found dead in the Highlands of Scotland during April-May of 1994 and 1995 and in subsequent years from similar birds at various locations throughout Scotland and also England. The isolates belonged to serogroup O86:K61, possessed the *eae* gene and produced cytolethal distending toxin. Finches involved were siskins (*Carduelis spinus*), greenfinches (*Carduelis chloris*) and chaffinches (*Fringilla coelebs*).

Although mortality associated with *E.coli* O86:K61 has been a frequent finding in finches, how this might be caused remains unclear.

Strains of *E. coli* within serogroup O86 are recognised as enteropathogenic (EPEC) in man. Strains within this O group have also been associated with diarrhoeal disease in calves and pigs. Other species of bird from which the organism has been recovered include 3 pheasants, 2 crossbills and a seagull,

Wild birds have previously been implicated as a source of various enteric pathogens for humans. Whilst there is no evidence to suggest a role for wild birds in incidents of O86 *E. coli* infection in humans or animals, given that this strain possesses both a toxin and an adhesin and that so many of the birds examined were infected, the potential surely exists.

P2: SEROEPIZOOTIOLOGICAL INVESTIGATIONS OF DIFFERENT INFECTIOUS DISEASES IN BIRDS OF PREY IN PARTS OF EASTERN GERMANY

E. Schettler, K. Frölich, T. Langgemach, P. Sömmer

During a period of three years (November 1994 to December 1997) 448 blood plasma samples from free-living birds of prey from the Berlin and Brandenburg area were collected in cooperation with various rehabilitation centres of birds of prey. The samples were tested for antibodies against Newcastle disease virus (NDV) by haemagglutination-inhibition test, *Chlamydia psittaci* by using a commercial competitive enzyme linked immunosorbent assay and against falcon herpesvirus (FHV) and owl herpesvirus (OHV) by virus neutralisation test, respectively. Of 355 samples from diurnal birds of prey seven (2 %) showed antibodies against NDV with titres ranging from 1:8 to 1:64, whereas none of the owl samples (n=93) tested were seropositive. Thus, a potential threat from ND to endangered species of diurnal birds of prey can not be excluded. Of 442 samples 277 (63%) were seropositive for *Chlamydia psittaci*. Titres varied from 1:5 to 1:256. The seroprevalence in owls was higher (75%) than in diurnal birds of prey (61%). The percentage of seropositive reactors varied between different species of birds of prey. Ninetyfour (61%) of 155 adult, 11 (61%) of 18 subadult, 35 (48%) of 73 juvenile and 28 (38%) of 73 nestling birds showed antibodies against *Chlamydia psittaci*. This reflects the ubiquitous occurrence of *Chlamydia psittaci* in birds of prey in Berlin and Brandenburg. Moreover, preliminary results of the exposure to FHV- and OHV are presented.

P3: ULTRASTRUCTURE OF EGGSHELLS OF BONELLI'S EAGLES (*Hieraaetus fasciatus*) WITH REPRODUCTIVE FAILURE IN CENTRAL SPAIN

J.M. Blanco¹, U. Höfle¹, M.J. Blaquez²

¹Centro de Estudios de Rapaces Ibéricas (C.E.R.I.), 45671 Sevilleja de la Jara, Spain;

²Departamento de Anatomía y Anatomía Patológica Comparada, Fac. de Veterinaria, Universidad de Complutense de Madrid, Madrid, Spain

The Bonelli's Eagle is a highly endangered species of bird of prey. The subspecies *Hieraaetus fasciatus fasciatus* has the widest distribution of the three existing subspecies, including southern Europe, the northern coasts of Africa and reaching to the south of China in the east. Its distribution in the Iberian peninsula is uneven, more than half of the population is found in southern and central Spain and Portugal. Especially in the 1980s this population has experienced a marked decline, due mainly to persecution and loss of habitat.

A number of breeding pairs of a population in the province Ciudad Real in central Spain had been observed to abandon their nest during the incubation period in several consecutive years. In spring 1997 a study on the possible causes of reproductive failure in these pairs was initiated.

Egg shells and addled eggs were collected from nests after confirmed abandon of incubation by the eagles, and during health checks of nestlings from successful breeding pairs. On entire eggs (n=1) full post-mortem examination including microbiology, virology and toxicology were carried out and fragments of all shells and shell remains were processed for scanning electron microscopy.

In this presentation we summarise the ultrastructural abnormalities observed during scanning electron microscopy of the eggshell surfaces and fracture, and the inner and outer eggshell membranes.

Although the breeding territory of the most affected pairs is known to have been an area of mercury mining the lesions encountered are not consistent with previously described lesions in relation to this heavy metal. When comparing shells of eggs that hatched normally and shells of addled eggs or eggs presumably broken during incubation, no significant differences in eggshell thickness are detected. Contents of organochlorine compounds and mercury in the analysed egg contents were low.

The lesions observed in affected eggshells resemble the abnormalities observed by some authors due to diets including osteolathyrogens or semicarbacides. Our observations include lack of separation of fibres of eggshell-membranes, thus uneven nucleation sites and multinucleation, uneven pore size and distribution, lack of establishment between shell and membranes, and large interstitial spaces. Spherulites were observed on the external surface of one eggshell. The described lesions may be the cause of reduced resistance to fracture and probably also increased permeability. Thus the cause of abandon in the affected pairs may have been the breakage of the eggs during incubation. Nevertheless further studies are needed to confirm the lesions and detect the possible cause.

P4: CAUSES OF DEATH IN RHEA AMERICANA CHICKS RAISED IN CAPTIVITY

M. Sanchez Bastos¹, H. Louvandini, E. Reiko Matushima, K. Werther

¹Almirante Barroso 290 apto, 233 CEP 13418-250, Piracicaba- São Paulo, Brasil

In recent years Rhea farming has been gaining in popularity as an alternative farming enterprise. When raised in captivity, the young, between 0 the 3 months of age, are highly susceptible to illnesses that can lead to death. Among these are included: leg abnormalities, stress, impaction, gastroenteritis and respiratory problems, as well as various infectious diseases. In this trial, 23 cases of death of Rhea young (ages, between 1 and 16 weeks), raised in one captivity, were studied with the objective to identify the possible "causes of death". The animals were lodged in one group on concrete floor in an area in cages of 25m². The diet was a mixture of initial concentrate for chicks (Crude Protein 21%, Crude Fibre 6%, Metabolizable Energy 2235 Kcal/kg, Ca 1,50%, P 0,50%, Ether Extract 2,50%, Ash 8,50%) with added cabbage, chicory and minced meat to increase the palatability. Water and the concentrate were supplied daily "ad libitum". The autopsies of the animals were carried out on the day of death, with the collection of different organs for histopatological examination. Of the 23 deaths that occurred, 74% (17 cases) were due to different infectious processes. In the majority of these cases, the animal stopped feeding due to the rapid clinical evolution of the diseases which led to death. The histopatological examination verified septicemia, with presence, in all cases, of enteritis suggesting that the illness, entered by the intestines. In 3 cases of respiratory problems such as pneumonia were verified. One animal death occurred around the 16^o week of life. This chick presented purulent ocular discharge for a period of 15 days, with a reduction in the consumption of food followed by death. At the autopsy, the presence of a focal infection in the liver was verified and the histopatological examination, showed chronic, granulomatosis focal hepatitis with central necrosis and giant cells. Specific Ziehl-Neelsen coloration was carried out, to discard the possibility of tuberculosis. Bone injuries represented about 13% of the deaths (3 cases), 2 for breaking the right femur and one case of leg sprain (type valgus). Bone illnesses in the Ratitas have been reported in captivity, as being due to nutritional problems or lack of physical space. A further three birds (13%) had died due to feeding management. They had been transferred from the adaptation place to a larger area with earthen floor and grass. The following day they were found dead due to stomach impaction, because of the uncontrolled ingestion of earth and grass. It can be concluded from this study that infectious processes were the main reasons for death of the chicks in this age range, followed by bone injuries and inadequate handling. This reiterates that sanitary and nutritional control is essential for the success of preservation or commercial exploration of this species.

P5: POISONING OF BIRDS AND MAMMALS BY CARBOFURAN IN AUSTRIA

F. Tataruch and T. Steineck

Research Institute of Wildlife Ecology, Vienna Veterinary University, Vienna, Austria

We report on incidents of poisoning with the carbamate carbofuran in different wildlife species, like buzzards (*Buteo buteo*), harriers (*Circus spec.*), white-tailed eagles (*Haliaeetus albicilla*), crows (*Corvus spec.*), magpies (*Pica pica*), red foxes (*Vulpes vulpes*) and pole cats (*Mustela putorius*) in Austria.

The first case was proven in December 1993, from this time on in each winter, predominantly between December and April, animals poisoned by carbofuran were sent to our institute for necropsy. Additionally many baits, consisting of parts of carcasses (hares, roe deer, buzzards etc.) treated with great amounts of granular carbofuran, were found, which endangered dogs and cats too. These baits, usually prepared with a great amount of Furadan.

P6: PRELIMINARY DATA ON THE INFLUENCE OF SARCOPTIC MANGE ON SERUM PROTEINS AND IMMUNOGLOBULIN G LEVELS IN SPANISH IBEX (*Capra pyrenaica*) AND CHAMOIS (*Rupicapra pyrenaica*)

M. E. Lastras, L. Viñas, I. Marco, S. Lavín

Patologia General i Mèdica, Facultat de Veterinària, Universitat Autònoma de Barcelona, 08193 Bellaterra, Spain

It is an important aspect to develop an immunologic approach to control scabies in wildlife populations. Arlian (1989) reported in different species significant differences in mean serum proteins and immunoglobulins G (IgG) levels in infested hosts. Sarcoptic mange was detected for the first time in Southern Spain (1988) affecting the Spanish ibex (*Capra pyrenaica*) and in Western Spain (1993) affecting the chamois (*Rupicapra pyrenaica*) populations. A research has been conducted on the influence of sarcoptic mange on serum proteins and IgG levels for Spanish ibex and chamois.

Two groups of Spanish ibex were investigated; the first group was 24 healthy animals and the second group consisted of 16 animals with natural scabies infestation. Two groups of chamois were established as well; a first group of 11 healthy animals and a second group of 14 animals naturally infested with scabies. Total serum proteins were measured by the Biuret method and the serum proteins were separated by cellulose acetate electrophoresis. IgG concentrations were measured by radial immunodiffusion using an antisera to caprine IgG. Student's t-test for unequal groups was used for the statistical data evaluation.

No statistically significant differences were found in albumin, alpha globulin, beta globulin and gamma globulin values, albumin:globulin ratio, total proteins and IgG concentrations from the two groups of Spanish ibex. Gamma globulin values, total proteins and IgG levels were significantly higher ($p < 0.01$) in chamois infested with scabies. Similarly, infested chamois had significantly ($p < 0.05$) larger values for alpha 1 globulin than did healthy animals. Results for the variables are presented.

Such information could hold the key to a better understanding of pathological condition of scabies in wildlife.

Bibliography

Arlian, L. G. (1989). Biology, host relations, and epidemiology of *Sarcoptes scabiei*. *Ann. Rev. Entomol.*, 34, 139-61.

P7: EPIDEMIOLOGY OF SCABIES IN VALANDOVO REGION -REPUBLIC OF MACEDONIA

M. Hristovski¹, B. Janevski², T. Tenekidjiev³, S. Jovana¹

¹Veterinary Institute, Skopje, Str. Lazar Pop Trajkov 5-7, Republic of Macedonia; ²National foundation for animal protection on Macedonia; ³Veterinary Inspection, Valandovo, Republic of Macedonia

Scabies, one of the oldest anthroponoses, occurred in Valandovo, region in the southern part of the Republic of Macedonia, near Macedonian-Greek border. The epidemic of scabies occurred in the period between April and May 1998, during which all endemic species were affected.

The first case was registered in wildlife. The red fox (*Vulpes vulpes* L.) was the most frequently affected species, with 90% of observed cases. Wolves were infected with 50% of the population in the region.

In the next period, scabies occurred in domestic animals. Stray dogs were the connection between infected wildlife and domestic animals. In Valandovo, a big group of stray dogs was present, which was uncontrolled in the movement. In the epidemic 290 sheep, 20 cows and 25 pigs were infected. All of these animals had a contact with the wild living animals or stray dogs. Appearance of scabies in 1998 was also present among human population with 31 cases, mostly among young population. It was estimated on the base of epidemiological survey that the epidemic occurred as a result of migration of wild animals, primarily foxes, wild boars and wolves from the northern part of Greece, where some human activity happened recently. Epidemiological process was characterized with the spread of scabies among different species, from wildlife to domestic animal and human population.

With parasitological investigation *Sarcoptes sarcoptes* var. *vulpes* was determined. In the previous period, in Macedonia scabies in wildlife was registered only sporadically and on small group of wild animals.

Key words: Scabies, red fox, *Vulpes vulpes*, wildlife, *Sarcoptes sarcoptes*, Republic of Macedonia.

P8: SEROLOGY OF PATHOGENS IN FREE-LIVING BLACK (*DICEROS BICORNIS*) AND WHITE (*CERATOTHERIUM SIMUM*) RHINOS IN AFRICA

C. Fischer, C. Hamblin, S. Quandt, K. Frölich

In Africa populations of both black and white rhinos have suffered dramatic declines in the last few decades. They are now subject of intensive and expensive conservation programs. Surprisingly little however is known on distribution and prevalence of pathogens and infectious diseases in free-ranging rhinos.

287 serum samples from free-ranging black and white rhinoceroses were tested for the exposure to several infectious organisms. The samples were obtained from conservation and monitoring programs in the Republic of South Africa, Namibia, Kenya and Botswana. Fourteen pathogens were selected because they are endemic in the study sites or they have been detected in other African wildlife species. Viral, bacterial and protozoal agents were included. A battery of serological methods was applied for the detection of antibodies.

We found antibodies against African horsesickness virus (77 of 278; 27,7 %), bluetongue virus (157 of 277; 56,7%), bovine herpesvirus 1 (7 of 232; 3,0%), equine herpesvirus 1 (25 of 273; 9,1%), bovine viral diarrhoea virus (3 of 258; 1,2%), parainfluenza type 3 (58 of 231; 25,1%), and four *Leptospira interrogans* serovars (60 of 278, 21,5%). No positive reactors were detected for encephalomyocarditis virus, Rift Valley fever virus, *Brucella* spp., and *Trypanosoma equiperdum*.

The exposure to five infectious organisms was significantly different in various study sites. White rhinos were significantly more often exposed to African horsesickness than black rhinos. Conservation programmes for rhinoceroses include translocation of free-ranging individuals or reintroduction of captive animals into the wild. To minimise the risk of introducing exotic diseases to non-enzootic areas, quarantine and screening for diseases and pathogens of animals prior to translocation should become a standard practice. In addition, before introducing a rhinoceros to the wild, disease screening is important in order to clarify which pathogens may be encountered by the reintroduced animal once it is released.

P9: FECAL PARASITOLOGY STUDY ON GASTROINTESTINAL NEMATODES OF WHITE RHINOCEROS (*Ceratotherium simum*) IN LAKE NAKURU NATIONAL PARK (KENYA)

M.E. Lastras¹, D. Mudakha², A. Sayyid² and T. Oloo²

¹Patologia General i Mèdica, Facultat de Veterinària, Universitat Autònoma de Barcelona, 08193 Bellaterra, Spain; ²Kenya Wildlife Service, PO Box 40241 Nairobi, Kenya

The white rhinoceros (*Ceratotherium simum*) has been extinct for several hundred years in Kenya. Lake Nakuru National Park was selected as the first manageable rhino sanctuary to reintroduce the species. An initial group of 19 white rhinoceros was established there since 1994, principally from South Africa, and the population has been increased up to 26 animals.

In free-living wild animals a balance usually exists between hosts and parasites. Under stressful conditions the host's immune system could be awkward. Although Penzhorn *et al.* (1994) documented all parasites that have been recovered from black (*Diceros bicornis*) and white rhinoceros, little is known on the gastrointestinal nematodes in white rhinoceros in Africa.

Lake Nakuru National Park covers an area of some 200 sq. Km. It is a soda lake but it has areas of grassland, bush, forest and rocky cliffs. Rainfall varies from a minimum of 20 mm in July to 200 mm in April and falls essentially in two seasons. During the short rain season of 1997, 19 animals from 7 groups were examined in the park, two herds of six animals, two groups of female and calf and three solitary males. Animals and territories were previously identified and fresh fecal samples were collected from each group. Counts of strongylid eggs per gram of feces were high (minimum: 500 eggs per g feces; maximum: 4400 eggs per feces). Average measures (n=49) for strongylid eggs were 79-97 (m (length) and 47-63 (m (width). Fresh fecal samples were cultured for 5 to 7 days and third stage infective larvae were recovered and fixed in 10% buffered saline. Two kinds of third-larvae were found: Short tail larvae with sheath and large number of intestinal cells, and long tail larvae without sheath and with large number of intestinal cells. Measures of total length, width and tail length were performed in both short tail (n= 61) and long tail (n=44) larvae. Average measures were 358-444 (m (total length), 20-24 (m (width) and 34-64 (m (tail length) from the short tail larvae and 673-873 (m (total length), 25-33 (m (width) and 217-325 (m (tail length) from the long tail larvae.

To our knowledge this is one of the first studies on gastrointestinal nematodes of white rhinoceros. Further studies on intensity of parasitism and characterization of eggs and larvae are needed.

Bibliography

Penzhorn, B.L., Krecek, R.C., Horak, I.C., Verster, A.J.M., Walker, J.B., Boomker, J.D.F., Knapp, S.E. and Quandt, S.K.F. Parasites of African rhinos: a documentation. Proceedings of a symposium on "Rhinos as Game Ranch Animals", Onderstepoort, 9 and 10 September 1994.

P10: ZOOLOGICAL AND WILDLIFE PATHOLOGY – AN IMPORTANT AND EMERGING DISCIPLINE

S.W. Cooke, J.A. MacDonald, J.E. Cooper

Greendale Laboratories, Woking, Surrey GU21 2EW, UK

Zoological and wildlife pathology (hereafter called “zoological pathology”) is concerned with the diagnosis of disease and the monitoring of health of both captive and free-living “exotic” animals.

As the global environmental threat increases, scientists and the public alike recognise the urgent need to conserve species and to promote bio-diversity. The distinction between free-living and captive animals blurs as the former become subject to more rigorous management and as the latter play an increasingly important role in species recovery by captive-breeding and return to the wild. Expertise in zoological pathology contributes to this work by:

1. determining the cause and circumstances of death of animals;
2. assisting in the clinical investigation of ill-health through examination of biopsies, blood, faeces and other samples;
3. providing baseline data on normal values;
4. detecting and interpreting subtle changes in tissues that may adversely affect function, productivity and survival;
5. establishing reference collections of tissues, samples and data.

Zoological pathology is a rapidly evolving, multidisciplinary subject that involves a range of different scientists as well as veterinarians. It necessitates high standards of laboratory investigation and excellent quality control.

P11: ON SOME RARE PATHOLOGIC FINDINGS IN CERVIDS

T. Steineck and P. Kanzler

Research Institute for Wildlife Ecology, Vienna Veterinary University, Vienna, Austria

A report is given on three pathological findings in deer, well known in domestic animals but similar cases in wildlife missing in literature. 1st case: In a small fallow deer farm each year calves died only few days old, all showing similar alterations of the skin in the sacral region. According to the gross findings *epitheliogenesis imperfecta* was diagnosed, microscopic findings were not expressiv due to the poor conservation of the carcass. This congenital malformation is transmitted by the male and diagnosis was confirmed by the fact that no cases occurred after removal of the suspected stag. 2nd case: In a half year old free ranging roe deer, shot because of obvious weakness and difficulties to follow the herd, a *hernia pericardiaca* probably congenital, too, could be diagnosed. 3rd case: The head of a shot female red deer showed a marked swelling of both cheeks giving the appearance like a hamster. 924,94 g respectively 587,02 g of compressed plants, partly rotted, partly fresh could be isolated from the cheek pouches. According to anamnesis the animal was in poor body condition, the year before swellings of only tennis ball size had been observed. After removal of soft tissues paradentitis in the upper and lower jaw became evident which might have been either the reason for or the consequence of this *stagnation of food in the oral cavity*.

P12: OUTBREAK OF TULAREMIA IN IBERIAN HARES (*LEPUS GRANATENSIS*)
IN THE WINTER OF 1997-98 IN NORTHWESTERN SPAIN

D. Fernandez-de-Luco, C. Gortazar, R. Costillas, M. Saco and I. Badiola

SEDIFAS, Wildlife Diagnostic Service. Facultad de Veterinaria, Universidad de Zaragoza,
c/Miguel Servet 177, 50013 Zaragoza, Spain

At the end of 1997 a high mortality among Iberian hares (*Lepus granatensis*) was observed in the northern part of Castilla-Leon Region. At the same time a plague of common voles (*Microtus arvalis*) occurred in the same area of hare mortality. A common finding among these hares was that all dead hares found in the field showed epistaxis. An anticoagulant rodenticide (chlorophacinone) was used to control the rodent plague. Dead hares from this area and from other parts of Spain, where neither abnormal mortality nor diseased hares were detected, were submitted for its study.

A total of 27 hares (23 from the mortality area) and 4 wild rabbits found dead inside the affected zone were necropsied and samples from liver, lung, spleen, intestine, subcutaneous and mesenteric lymph nodes were taken and preserved in 10% buffered formalin for histopathological examination. Liver samples were stored at -20°C for bacteriological studies on cysteine agar plates.

The main lesions observed were small foci of necrosis in the liver, spleen, lymphoid tissue of cecal appendix and sacculus rotundus, and in mesenteric, cervical and popliteal lymph nodes. *Francisella tularensis* was isolated in 18 hares that had died in 1994, 95, 97 and 98. Seventeen out of 26 hares from Castilla-Leon region and one hare from La Rioja region were positive. *F. tularensis* was also isolated from one out of four wild rabbits dead due to RHD in Castilla-Leon. In 1997/98 the disease has also been diagnosed in people after handling with hunted hares.

We can state that tularemia was present in Iberian hares before this outbreak. Moreover, this disease is present in areas far away from the core area affected by the most noticeable outbreak.

P13: A PARALLEL SEROPREVALENCE STUDY OF *Neospora caninum* AND *Toxoplasma gondii* IN A SAMPLE OF THE ETHIOPIAN CANID AND BOVID POPULATIONS

A. O'Keeffe¹, K. Laurenson², S. Wright¹, I. Esteban-Redondo¹, E. Innes¹, I. McKendrick¹

¹Moredun Research Institute, International Research Centre, Pentlands Science Park, Bush Loan, Penicuik, EH26 0PZ, Scotland; ²Centre for Tropical Veterinary Medicine, Easter Bush Veterinary Centre, Roslin, Midlothian, EH25 9RG, Scotland

The recently recognised parasite *N. caninum* is a major cause of bovine abortion and neuromuscular disorders in dogs in many countries. Only limited epidemiological information is available on the seroprevalence of *Neospora caninum* and *Toxoplasma gondii* in canid and bovid populations in East Africa and none relates specifically to Ethiopia. By establishing the seroprevalence of these pathogens it may be possible to further elucidate the biology of *N. caninum* and could lead to the identification of the definitive host. Dog serum samples were obtained from a rabies prevention programme to protect the critically endangered Ethiopian wolf. A total of 211 dog serum samples collected from two provinces of Ethiopia were analysed for the presence of *T. gondii* and *N. caninum* using the Indirect Fluorescent Antibody test (IFAT). Forty-one dogs had specific *N. caninum* antibody titres (19.4%, SE= +/- 3.31 %) and 126 dogs had specific *T. gondii* antibody titres (59.6%, SE= +/- 2.52 %). A strong relationship between increasing age and rising seroprevalence was shown for both *T. gondii* and *N. caninum*. Of the two provinces sampled, *N. caninum* was more abundant in the Bale region where stocking densities are higher than in the predominantly arable Shoa region. *T. gondii* prevalence was uniformly distributed throughout both regions. Studies on the seroprevalence of these parasites in bovid populations sampled from the Bale region of Ethiopia will also be described.

P14: *BRUCELLA MARIS* INFECTIONS IN MARINE MAMMALS FROM SCOTTISH WATERS

I. A. P. Patterson¹, F. E. Howie², R. J. Reid¹, H. M. Ross¹, A. MacMillan³, S. Brew³ and G. Foster¹

¹SAC Veterinary Science Division, "Drummondhill", Stratherrick Road, Inverness IV2 4JZ

²SAC Veterinary Science Division, Bush Estate, Penicuik, Midlothian EH26 0QE

³Brucella Reference Laboratory, Central Veterinary Laboratory, New Haw, Addlestone, Surrey, KT15 3NB

As part of a programme of investigation into strandings of marine mammals, post mortem and bacteriological examinations of stranded and by-caught animals from around the Scottish coast were undertaken by SAC Veterinary Science Division. This led to the isolation of *Brucella* from a porpoise in 1991 and, to date, *Brucella* has been isolated from twenty eight animals. These isolates have come from five species of cetacean, three species of seals and an otter.

Bacteriological investigation of the sea mammal isolates demonstrated that they were significantly different and a new species, *Brucella maris*, has been proposed. Seal and cetacean strains of *B. maris* are readily differentiated however, and it is likely that at least two distinct species exist.

Serological studies have shown evidence of widespread infection in marine mammals and seropositivity has been found in ten species of cetacean and four species of seal.

However, pathological changes associated with *B. maris* isolations have only been found in a total of nine cetaceans and two seals. The most common pathological condition in cetaceans was sub-blubber abscessation. Both of the seals had pneumonia.

There was evidence of a possible abortion in one dolphin and a spinal lesion in a porpoise. These conditions would be more typical of those expected with *Brucellae* infection in terrestrial mammals.

In most of these animals there were intercurrent conditions which may have predisposed to the *Brucella* pathologies. This suggests that *Brucellae* may be well adapted to marine mammals and they are not acting as significant pathogens. However, alternatively, the main problems caused may not be fatal e.g. abortion and/or sterility and these would not be seen in mortality studies.

Transmission studies have demonstrated that the pathogenicity of *B. maris* for sheep appears to be low. However, a laboratory worker was infected indicating that it is a potential zoonotic agent.

P15: ANTIBIOTIC RESISTANCE IN THE NORMAL GUT FLORA OF SMALL WILD RODENTS

M. Gilliver, M. Begon, M. Bennett, S. Feore and T. Hart

Centre for Comparative Infectious Diseases, University of Liverpool, UK

Antibiotic resistance is recognised to be an increasingly common problem in both veterinary and human medicine. Although the high prevalence of resistance in human and animal pathogens is generally agreed to be due to selection through use of those antibiotics, the original sources of the resistance genes are often not known. In a few cases, it may be that both mutation and selection of resistance genes has occurred within the treated host species, but more usually the flora of other species is believed to be the proximate source. However, as a rule, the original sources of resistance genes in animal bacteria are not known. Rather, like the genes encoding the antibiotics themselves, they are generally assumed to have evolved in environmental bacteria as a consequence of millions of years of inter- and intra-specific competition. Current theory further suggests that the expression of antibiotic resistance by bacteria involves an energy cost which is disadvantageous unless antibiotics are present in the environment. In other words, it is predicted that if antibiotics were not used in animals (or man) then resistant bacteria would be at a selective disadvantage compared with non-resistant bacteria, and resistance would gradually disappear from the untreated bacterial population. This scenario is supported by some laboratory experiments and by the apparently low prevalence of resistance in human and animal populations in some parts of the world where antibiotics have not been used.

To test further the hypothesis that the prevalence of antibiotic resistance should be low in the absence of antibiotics - and its apparent corollary that this prevalence should be manipulable by the control of antibiotic usage - we undertook a small survey of resistance in enteric aerobic Gram negative (AGN) bacteria isolated from two wild populations of small rodents. Faeces from 38 bank voles (*Clethrionomys glareolus*) and 70 wood mice (*Apodemus sylvaticus*) were collected from two woodland sites. Overall, up to 86% of coliforms and 100% of oxidase +ve AGNs were found to be ampicillin resistant, in over 50% of cases due to (-lactamase expression. Depending on the species and site sampled, 22-86% of coliforms were tetracycline resistant, 48-77% sulphonamide resistant, and 64-83% trimethoprim resistant. Similarly high prevalences of resistance were found to other antibiotics tested, although only 6-11% of coliforms were apramycin resistant.

Although only a relatively small number of animals were tested, these results demonstrate that the prevalence of antibiotic resistance in some wild rodent populations is much higher than would be expected. These findings raise several questions of both clinical and ecological interest. For example, just how widespread is antibiotic resistance in wild rodents? What is the potential for these wild rodents to act as a source of resistance in domestic animals (and man)? What are the origins of this resistance? And what are the selection mechanisms within wild rodents which maintain it at such high prevalences?

P16: ECTOPARASITES AND MICROPARASITE TRANSMISSION IN WOODLAND
RODENT COMMUNITIES

K. Bown, M. Bennett, S. Feore and M. Begon

Centre for Comparative Infectious Diseases, University of Liverpool, UK

As part of an ongoing study investigating the effects of endemic infections on wild rodent populations, the role of ticks and fleas in their transmission is being studied. Information on the host-tick population dynamics are gathered from two woodland sites one week in every four for the small mammal tick *Ixodes trianguliceps* and its rodent hosts, wood mice (*Apodemus sylvaticus*) and bank voles (*Clethrionomys glareolus*). Fleas are also collected from hosts and identified in the laboratory to determine the species and relative abundance of fleas present.

Using serological (indirect immunofluorescence assay), isolation and molecular (PCR) techniques, hosts, ticks and fleas are screened for the presence of several infectious agents, including cowpox virus and *Bartonella* spp. to determine prevalence of infection in each. This information can then be compared with both host and vector population data to investigate the role of each in infection dynamics, and any relationship between infection and vector dynamics.

To date, the results suggest that neither ticks nor fleas are infected with cowpox virus, but that both are infected with a variety of *Bartonella* spp, and that at least four different species of *Bartonella* circulate in the rodent populations. Sequencing of *Bartonella* amplicons obtained from arthropods is underway to identify the species of *Bartonella*, correlate infection in individual vectors and their hosts, and to determine whether any host or vector specificity exists.

P17: GRANULOCYTIC EHRLICHIA INFECTION IN RODENTS AND IXODES TRIANGULICEPS TICKS IN A UK WOODLAND

N.H. Ogden^{1,2}, K. Bown^{1,3}, B.K. Horrocks^{1,4}, Z. Woldehiwet^{1,4} and M. Bennett^{1,4}

¹Centre for Comparative Infectious Diseases, University of Liverpool L69 3BX, UK; ²Department of Veterinary Clinical Science, Faculty of Veterinary Science, University of Liverpool L69 3BX, UK; ³School of Biological Sciences, University of Liverpool L69 3BX, UK; ⁴Department of Veterinary Pathology, Faculty of Veterinary Science, University of Liverpool L69 3BX, UK

The detection, by PCR, of Ehrlichiae of the *Ehrlichia phagocytophila* genogroup (the granulocytic ehrlichiae), in engorged *Ixodes trianguliceps* ticks and blood collected from wild rodents, indicates that European wild rodents are competent reservoirs. The prevalence of granulocytic *Ehrlichia* (GE) infection in engorged *I. trianguliceps* ticks and rodent blood provides preliminary observations of the dynamics of infection in a woodland in the North of England. GE infection was also detected in questing and engorged *I. ricinus* ticks, collected from herbage and vertebrates of a woodland in the South of England. The prevalence of infection in the three feeding stages of the ticks indicated that granulocytic Ehrlichiae are transmitted transstadially with no, or inefficient, transovarial transmission. Infection prevalence in *I. trianguliceps* adults which engorged on rodents was significantly greater than infection prevalence in questing *I. ricinus* ticks of any stage, and fed *I. ricinus* ticks collected from any of rodents, pheasants and deer. This is consistent with most *I. trianguliceps* ticks feeding on reservoir competent rodents in the woodland in the North of England. In contrast, in the woodland of the South of England, considerable numbers of nymphs of the exophilic *I. ricinus* ticks feed on pheasants which are incompetent or inefficient reservoirs, so reducing GE infection prevalence in *I. ricinus* ticks in this habitat. 16S rRNA sequences of GE from ticks of these woodlands showed a high degree of homology with those of granulocytic ehrlichiae isolated from humans, but also showed some evidence of genetic diversity of granulocytic ehrlichiae in the UK.

P18: TRAUMA IS THE MAJOR CAUSE OF MORTALITY IN FINNISH OTTERS

E. Rudbäck¹, T. Stjernberg²

¹National Veterinary and Food Research Institute, PO Box 368, SF-00231 Helsinki, Finland,

²Zoological Museum, Helsinki University, P.O. Box 17, SF-00014 Helsinki University, Finland

During 1990-1997, 111 otters (*Lutra lutra*) were necropsied in the National Veterinary and Food Research Institute. The physical condition of the majority of animals was good. The most common cause of death was trauma (57 animals, 51 %), of which road traffic accidents were responsible for 58 % (33 animals) and killing traps for 14 % (8 animals). Thirty five otters (32 %), mainly males (26 animals, 74 %), had drowned due to fish traps. Starvation was the primary cause of death in 19 cases (17 %), most frequently these animals were found in winter time. Adiaspiromycosis was diagnosed in the lungs of 45 otters (41 %). This fungal infection did not seem to effect the general health status of the animals. No other infectious agent or evidence of infectious disease was found in these animals.

LIST OF DELEGATES AS AT 10 SEPTEMBER 1998

Dr Marc Artois
CNEVA Nancy
BP 9
54220 Nalzeville
France

Dr John Vincent Beer
127 Church Road
Salisbury
Wilts

Dr Malcolm Bennett
University of Liverpool
Leahurst
Chester High Road
Neston
South Wirral L64 7TE

Dr Juan Manuel Blanco
Centro de Estudios de Rapaces
Ibericas (CERI)
45671 Sevilleja de la Jara
Spain

Mrs Suzanne Boardman
Wildlife Information Network
RVC
Royal College Street
London NW1 0TU

Mrs Annette L Bolte
Institut fur
Geflugelkrankheiten
Justus-Liebig-Universitat
Frankfurter Strabe 91
35398 Gießen
Germany

Dr Debra Bourne
Wildlife Information Network
RVC
Royal College Street
London NW1 0TU

Dr Giusy Cardeti
Via Apfia Nudva 1411
Rome
Italy

Dr Martin Cooke
7 Cheveney Walk
Bromley BR2 0XZ

Professor John Cooper
Wildlife Health Services
PO Box 153
Wellingboro NN8 2ZA

Dr Richard Delahay
CSL Research Unit
Woodchestr Park
Nympsfield
Glos. GL10 3UJ

Dr Paul Duff
Penrith VI Centre
Merrythought
Calthwaite
Penrith CA11 9RR

Dr Sarah Feore
University of Liverpool
Leahurst
Chester High Road
Neston
South Wirral L64 7TE

Dr Daniel Fernandez-de-Luco
Sedifas-Fac Veterinaria
C/Miguel Servet 177
E-50013 Zaragoza
Spain

Dr Carola Fischer
Institute for Zoo Biology and
Wildlife Research
Alfred-Kowalke Str 17
10315 Berlin
Germany

Dr Kai Froelich
Institute for Zoo Biology and
Wildlife Research
Alfred-Kowalke Str 17
10315 Berlin
Germany

Dr Emmanuelle Fromont
UMR 5558
UCB Lyon 1
43 Bd du 11 novembre 1918
69622 Villeurbanne Cedex
France

Dr Dominique Gauthier
Laboratoire Veterinaire de la
Savoie
BP 1113
73 011 Chambéry Cedex
France

Dr Dolores Gavier-Widen
Central Veterinary Laboratory
New Haw
Weybridge KT15 3NB

Dr Marco Giacometti
Centre for Fish and Wildlife
Health
Wild and Zoo Animal Group
Laenggasstrasse 122
CH-3012 Bern
Switzerland

Ms Moira Gilliver
Department of Medical
Microbiology
University of Liverpool
PO Box 147
Liverpool L69 3BX

Dr Jacques Godfroid
Veterinary and Agrochemical
Research Center
Brussels
Belgium

Dr Christian Gortazar
Sedifas-Fac Veterinaria
C/Miguel Servet 177
E-50013 Zaragoza
Spain

Ms Hilde Hansen
Department of Virology
Institute of Medical Biology
Univeristy of Tromso
N-9037 Tromso
Norway

Dr Carl Hard af Segerstad
Department of Wildlife
National Veterinary Institute
Uppsala
Sweden

Dr Ursula Hofle
Centro de Estudios de Rapaces
Ibericas (CERI)
45671 Sevilleja de la Jara
Spain

Mr Paul Holmes
Veterinary Investigation
Centre
Kendal Road
Harlescott
Shrewsbury SY1 4HD

Dr Miso Hristovski
Veterinary Institute
Skopje
Str. Lazr Pop Trajkov 5-7
Republic of Macedonia

Dr Elizabeth Innes
Moredun Research Institute
International Research Centre
Bush Loan
Penicuik
Midlothian EH26 0PZ

Dr James Kirkwood
Scientific Director
Universities Federation for
Animal Welfare
The Old School
Brewhouse Hill
Wheathampstead
Hertfordshire AL4 8AN

Mr Francois Lamarque
Office National de la Chasse
Saint Benoist
78610 Auefargis
France

Dr Eugenia Lastras
Patologia General I Medica
Fac. Veterinaria UAB
Bellaterra 08193
Spain

Dr Karen Laurenson
CTVM
Easter Bush
Penicuik
Midlothian

Dr Antonio Lavazza
Istituto Zooprofilattico
Sperimentale Della Lombardia
e dell emilia
Via Bianchi
7 25126 Brescia

Ms Becky Lawson
Flat 1
5 Middle Street
Taunton
Somerset

Ms Joanna Lellow
University of Liverpool
PO Box 147
Liverpool L69 3BX

Professor Luis Leon-Vizcaino
Facultad de Veterinaria
Universidad de Madrid
Spain

Dr Walburga Lutz
Putzchens Chaussee 228
D-53229
Bonn
Germany

Dr Alex Markovics
Kimron Veterinary Institute
PO Box 12
Beit Dagan 50250
Israel

Dr Torsten Morner
Department of Wildlife
National Veterinary Institute
Uppsala
Sweden

Dr Thomas Mueller
Institute for Epidemiological
Diagnostics
Federal Research Centre for
Virus Diseases of Animals
Seestr 55
D-16868 Wusterhausen
Germany

Dr Ralf-Udo Muhle
Institute for Ecology and
Nature Conservation
Department for Ecology of
Waterowl and Wet lands
University of Potsdam
D-14447 Potsdam
Germany

Dr Plaxidia Muvavarirwa
University of Zimbabwe
PO Box MP167
Harare
Zimbabwe

Dr Tony Patterson
VIC Inverness
Drummondhill
Sratherick Road
Inverness IV2 4JZ

Mr Thomas Pennycott
SAC Veterinary Science
Division
Ayr

Ms Eve Pleydell
East Leigh Cottage
Harberton
Totnes
Devon TQ9 7SS

Dr Eeva Rudback
National Veterinary and Food
Reserch Institute
Finland

Dr Marisa Sanchez-Bastos
Almirante Barroso
290 apto 233
CEP 13418-250
Piracicaba-São Paulo
Brazil

Dr Nuno Santos
PN Peneda-Geres
4845 Geres
Portugal

Ms Elvira Schettler
IZW Alfred Kowalke Str 17
10315 Berlin
Germany

Mr Victor Simpson
VI Centre
Polwhele
Truro
Cornwall

Dr Paddy Sleeman
Department of Zoology
University College
Lee Maltings
Prospect Row
Cork
Ireland

Dr Theodora Steineck
Savoyenstr 1
1160 Vienna
Austria

Dr Frieda Tataruch
Savoyenstr 1
A-1160 Wien
Austria

Dr Paul Tavernier
Polbroek 17
9520 Sint-Lievens-Houtem
Belgium

Dr Morten Tryland
Department of
Immunoprophylaxis
National Veterinary Institute
PO Box 8156 Dep
0033 Oslo
Norway

Mr David Williams
Unit for Comparative
Ophthalmology
Centre for Small Animal
Studies
Animal Health Trust
Lanwades Park
Kentford
Newmarket CB8 7UU

LIST OF AUTHORS

A

Alfredsson, A · 5
Arnason, A · 5
Artois, M · 8, 9, 12

B

Badiola, I · 37
Bain, M · 10
Barmark, A-L · 7
Baxby, D · 13, 14
Begon, M · 13, 14, 40, 41
Bennett, M · 13, 14, 40, 41, 42
Blanco, J M · 28
Blanquez, M J · 28
Boardman, S · 20
Boelaert, F · 4, 25
Bolte, A L · 18
Bourguemestre, F · 12
Bourne, D · 20
Bown, K · 13, 41, 42
Brew, S · 39
Brown, B · 10
Brown, R · 10

C

Cain, E · 12
Chantrey, J C · 13, 14
Chasey, D · 22
Clavareau, C · 4
Cooke, M · 16, 23
Cooke, S W · 35
Cooper, J E · 35
Costillas, R · 37

D

Duff, J P · 22

E

Esteban-Redondo, I · 38

F

Feore, S · 3, 13, 14, 40, 41
Fernandez-de-Luco, D · 15, 37
Fischer, C · 33

Flindall, A · 11
Foster, G · 26, 39
French, N P · 3
Frölich, K · 27, 33
Fromont, E · 12

G

Gallagher, J · 1
Gauthier, D · 24
Gavier-Widen, D · 1
Gilliver, M · 40
Gilray, J A · 17
Godfroid, J · 4, 5, 25
Gortazar, C · 15, 37
Gurnell, T · 17

H

Hamblin, C · 33
Hård af Segerstad, C · 7
Hart, T · 40
Hatier, C · 9
Höfle, U · 28
Hopkins, G F · 26
Horrocks, B K · 42
Howie, F E · 39
Hristovski, M · 32

I

Innes, E · 38

J

Janevski, B · 32
Jansson, D S · 7
Jovana, S · 32

K

Kaleta, E F · 18
Kanzler, P · 36
Kirkwood, J K · 21
Kjeld, M · 5
Kleivane, L · 5

L

Lacey, R · 10
Lamarque, F · 8, 9

Langgemach, T · 27
Lastras, M E · 31, 34
Laurenson, K · 38
Lavin, S · 31
Léger, F · 12
Letesson, J-J · 4, 25
Louvandini, H · 29
Lutz, W · 18

M

MacDonald, J A · 35
Macgregor, S K · 21
MacMillan, A · 39
Marco, I · 31
McKendrick, I · 38
McLaren, I M · 26
Meyer, H · 14
Michel, P · 4
Mörner, T · 7
Mudakha, D · 34
Mulcahy, M F · 2

N

Nettleton, P · 17
Nordkvist, E · 7

O

Ogden, N H · 42
O'Keeffe, A · 38
Oloo, T · 34

P

Patterson, I A P · 39
Pennycott, T W · 19, 26
Pontier, D · 12
Prendergast, J · 2

Q

Quandt, S · 33

R

Reid, R J · 39
Reiko Matushima, E · 29
Ross, H M · 26, 39
Rudbäck, E · 43

S

Saco, M · 37
Sager, A · 12
Sainsbury, T · 17
Sanchez Bastos, M · 29
Sayyid, A · 34
Schettler, E · 27
Sharp, G · 22
Simpson, V · 10
Simpson, V R · 11
Sleeman, D P · 2
Sömmer, P · 27
Stahl, P · 12
Steineck, T · 30, 36
Stjernberg, T · 43
Stuen, S · 5

T

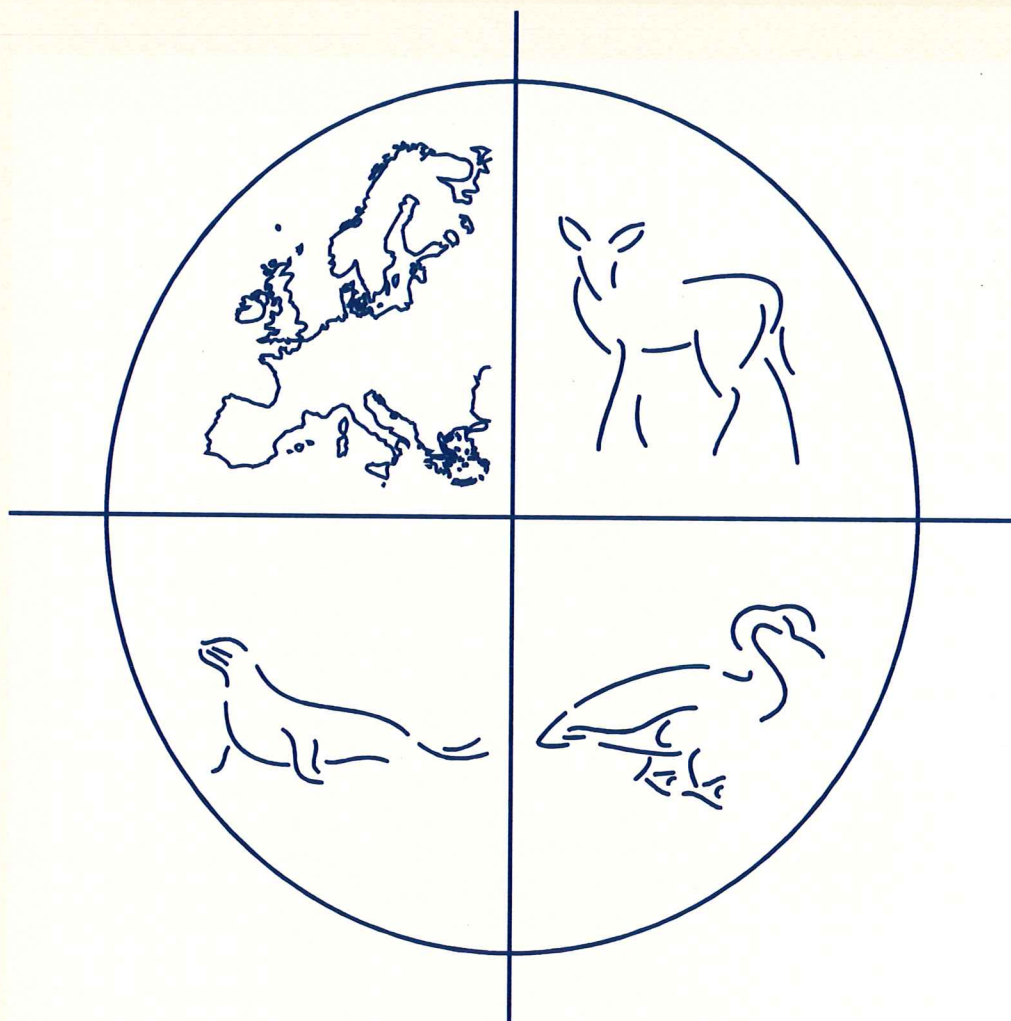
Tataruch, F · 30
Tenekidjiev, T · 32
Trout, R C · 22
Tryland, M · 4, 5

V

Viñas, L · 31

W

Walravens, K · 4, 25
Wellemans, V · 4
Werther, K · 29
Westcott, D · 22
Weynants, V · 25
Williams, D L · 11
Woldehiwet, Z · 42
Wright, S · 38



Moredun

Pentlands Science Park

Edinburgh, EH26 0PZ

Scotland, UK

Telephone (+44) 131 445 5111

Fax (+44) 131 445 6111

