

European Association of Zoo
and Wildlife Veterinarians



EWDA Session

[Scroll down to view documents](#)

European Association of Zoo- and Wildlife Veterinarians (EAZWW)
4th scientific meeting,

joint with the
annual meeting of the European Wildlife Disease Association (EWDA)

May 8-12, 2002, Heidelberg, Germany.

This manuscript is reproduced in the IVIS website with the permission of EAZWW
www.eazww.org

European Association of Zoo- and Wildlife Veterinarians (EAZVW) 4th scientific meeting,
joint with the annual meeting of the European Wildlife Disease Association (EWDA)
May 8-12, 2002, Heidelberg, Germany.

SURVEILLANCE AND CONTROL OF WILDLIFE DISEASES IN EUROPE

*M. ARTOIS*¹, *R. DELAHAY*², *V. GUBERTI*³ and *C. CHEESEMAN*²

Affiliation:

1. ENV Lyon - B.P. 83 - 69280 Marcy l'Etoile, France. Email : m.artois@vet-lyon-fr
2. Central Science Laboratory – Sand Hutton, York, YO4 1LZ, UK. Email: r.delahay@csl.gov.uk
3. Istituto Nazionale per la Fauna Selvatica – Via Ca' Fornacetta, 9 – 40064 Ozzano dell'Emilia, Italy. Email: infs_vete@iperbole.bologna.it

Key words : Infectious diseases – Monitoring – Control – Europe – Wildlife

Abstract

During the last thirty years new epidemiological patterns have emerged as free-ranging wildlife have become progressively more involved in the epidemiology of both common and emerging infectious diseases of humans and domestic animals. This has been seen in rabies, bovine tuberculosis and more recently in wild-boar classical swine fever. Emerging diseases are of interest for veterinarians as well as public health officials. Attempts to control these diseases have not always been successful as in wildlife populations control of either host or pathogen can present particular problems. Lessons should be learnt from previous experiences to help in the management of new emerging diseases in the future.

Résumé

Au cours des trente dernières années l'Europe a connu l'apparition de maladies infectieuses ou parasitaires de la faune sauvage (notamment et chronologiquement : la rage du renard, la tuberculose *bovine* du blaireau et la peste porcine classique du sanglier). Compte tenu de leur importance économique ou sanitaire, les autorités vétérinaires des pays concernés se sont efforcés de lutter contre ces maladies. Les tentatives de contrôler ces maladies n'ont pas nécessairement conduit à des succès. Les problèmes rencontrés doivent être analysés afin d'améliorer les chances de gérer les crises du futur.

European Association of Zoo- and Wildlife Veterinarians (EAZVV) 4th scientific meeting,
joint with the annual meeting of the European Wildlife Disease Association (EWDA)
May 8-12, 2002, Heidelberg, Germany.

WILDLIFE DISEASE IN THE UK: A SHORT HISTORY

J.P. DUFF

Affiliation:

Veterinary Laboratories Agency, VLA Penrith, Merrythought, Penrith, Cumbria CA11 9RR

Key words : history, wildlife diseases, UK

Abstract

This paper first discusses the history of wildlife disease in the UK, why the subject should be examined, sources of information and the possible significance of wildlife disease in the UK down the centuries. The history of wildlife disease is examined in three periods – pre 1800, 1800-1900 and 1900 to the present day. The diseases considered are then discussed in three general categories, zoonotic disease, diseases of importance to domesticated stock and diseases of importance to wildlife, social structures and the environment. A further aspect that will be considered are the institutes, organisations and scientists that have been involved in wildlife disease investigations in more recent years. The development of investigation techniques is followed together with discussion on the current status of wildlife investigation in the United Kingdom.

Zusammenfassung

Dieser Bericht betrachtet zunächst die Geschichte von Erkrankungen unter wilden Tieren im Vereinigten Königreich und untersucht dann die Gründe für eine Erörterung dieses Themas, die Informationsquellen und möglichen Auswirkungen von Erkrankungen unter wilden Tieren im Vereinigten Königreich im Laufe von Jahrhunderten.

Die Geschichte von Erkrankungen unter wilden Tieren wird in drei Zeiträumen untersucht, d. h. vor 1800, 1800-1900 und 1900 bis zur Gegenwart. Die berücksichtigten Erkrankungen werden dann im Rahmen von drei allgemeinen Kategorien, d. h. der Zoonose, Erkrankungen unter domestizierten Tieren und drittens den Auswirkungen von Erkrankungen auf wilde Tiere, soziale Strukturen und die Umwelt untersucht.

Ein weiterer zu berücksichtigender Aspekt betrifft die Institutionen, Organisationen und Wissenschaftler, die in jüngster Zeit Erkrankungen unter wilden Tieren untersucht haben. Die Entwicklung von Untersuchungsverfahren wird verfolgt, und es findet eine Diskussion über den aktuellen Stand von Untersuchungen von wilden Tieren im Vereinigten Königreich statt.

Résumé

Cet article examine d'abord l'histoire des maladies des animaux sauvages au Royaume-Uni, pourquoi ce sujet devrait être étudié, les sources d'information, et l'importance possible des maladies des animaux sauvages au Royaume-Uni au cours des siècles.

L'histoire des maladies des animaux sauvages est étudiée en trois périodes – avant 1800, de 1800 à 1900, et de 1900 à nos jours. Les maladies considérées sont ensuite examinées en trois grandes catégories, à savoir les zoonoses, les maladies qui ont de l'importance pour les animaux domestiques, et les maladies qui ont de l'importance pour la faune, pour les structures sociales et pour l'environnement.

Un autre aspect est considéré, à savoir les établissements, les organisations et les chercheurs qui ont participé aux études sur les maladies des animaux sauvages ces dernières années. Le développement des techniques d'étude est suivi et l'état actuel de l'étude des maladies des animaux sauvages au Royaume-Uni est examiné.

European Association of Zoo- and Wildlife Veterinarians (EAZWV) 4th scientific meeting,
joint with the annual meeting of the European Wildlife Disease Association (EWDA)
May 8-12, 2002, Heidelberg, Germany.

BOVINE TB IN BRITISH WILD MAMMALS

R. J. DELAHAY

Affiliation:

Central Science Laboratory, Sand Hutton, York, YO41 1LZ

Key words: bovine tuberculosis, wildlife disease reservoir, surveillance.

Abstract

Bovine tuberculosis caused by *Mycobacterium bovis* is a zoonotic infection with a wide range of mammalian hosts. In parts of the UK *M. bovis* infection in cattle is a persistent problem. The European badger (*Meles meles*) is implicated in the transmission of *M. bovis* to cattle, and is widely believed to constitute the most important reservoir of infection in British wildlife. TB is endemic in the British badger population. Infected individuals may excrete bacilli in sputum, faeces, urine and/or pus from wounds and burst abscesses. However, infected badgers can survive for relatively long periods and breed successfully, and the impact of the disease on population size and structure appears to be minimal. The foraging habits of badgers may bring them into close contact with pasture and farm buildings, providing opportunities for disease transmission to cattle. However, few studies have been carried out on the status of *M. bovis* infection in other wild mammals in the UK. Infection has been identified in several species of deer, foxes, moles, rats, mink, a feral ferret, farm cats and a field vole. Although the evidence from these studies does not support the existence of a significant self-maintaining reservoir of infection in any wild mammal other than the badger, there is a clear lack of sufficient data to rule out the involvement of other species. This paper describes the methodology and preliminary findings from a contemporary survey for *M. bovis* infection in wild mammals in south-west England, and discusses its limitations and the interpretation of results.

FIRST REPORT OF EUROPEAN BROWN HARE SYNDROME VIRUS (EBHSV) IN FREE-RANGING EUROPEAN BROWN HARES (*Lepus europaeus*) FROM SWITZERLAND AND ARGENTINA

**K. FRÖLICH, G. HAERER, L. N. BACCIARINI, M. JANOVSKY, M. RUDOLPH, S. SPECK, L.
RONSHOLT and M. GIACOMETTI**

Affiliation:

1. Institut for Zoo and Wildlife Research Berlin (Director: Prof. Dr. H. Hofer),
2. Center of Fish and Wildlife Health,
3. Institute of Animal Pathology, University of Berne (Director: Prof. Dr. M. M. Suter),
4. Wildvet Projects, Stampa,
5. Danish Veterinary Institute, Lindholm, Kalvehave

Extended Abstract

In 1989, Lavazza and Vecchi (11) found viral particles in European brown hares (*Lepus europaeus*) which had died from European brown hare syndrome (EBHS) by negative staining immune electron microscopy of the liver. The causative agent of EBHS is a small (30 to 35 nm) icosahedral, non-enveloped and hemagglutinating virus (6) and is classified as a calicivirus (16). European brown hare syndrome has been demonstrated in European brown hares and in mountain hares (*Lepus timidus*), and has been reported in many European countries (2, 3, 5, 7, 8, 9, 13, 14, 15, 17, 19, 21, 22, 23). However, until now neither EBHSV-antigen nor corresponding pathology has been found in Lagomorphs in Switzerland. Only one report exists for antibodies against caliciviruses in European brown hares in this country (1). Our objective was to determine, whether free-ranging European brown hares and mountain hares were naturally infected by EBHSV in Switzerland. From 1997 to 2000, complete necropsy and histopathological investigations were performed on 157 free-ranging European brown hares found dead throughout Switzerland. Organ samples of all these individuals (157 livers and 107 spleens available) were tested for European brown hare syndrome virus (EBHSV)-antigen by enzyme-linked immunosorbent assay (ELISA) test kit (4). Furthermore, 60 blood samples available were tested for antibodies against EBHSV by ELISA. In addition, liver samples of 87 free-ranging mountain hares hunted in 1996 were tested for EBHSV-antigen. In two European brown hares from southern Switzerland lesions suggestive of changes induced by EBHSV were present, and high titres of EBHSV-antigen were detected in both liver and spleen samples of the same animals. The two liver samples were positive up to a dilution of 1: 1,000 and the two spleen samples were positive when diluted 1: 10,000 and 1:1,000, respectively. Major histological lesions were restricted to the liver parenchyma and characterised by midzonal- and perilobular, large extensive to coalescing areas of coagulative and lytic necrosis and partial replacement of the parenchyma by erythrocytes. Additional lesions were characterised by multiple subendocardial haemorrhages and tubular nephrosis. Based on negative staining electron microscopy investigations of liver and spleen homogenates, we observed calicivirus in one antigen-positive hare. These viral particles morphologically resemble those observed by Poli et al. (18) in spleen and liver of hare with acute hepatic necrosis. Low EBHSV-antigen titres were found in three additional European brown hares from central and western Switzerland, but EBHS-lesions were absent. Antibodies against EBHSV were not detected in any of the sera of European brown hares, and EBHSV-antigen was not found in the samples of mountain hares. The two individuals showing EBHS originated from a region south of the Alps. This local subpopulation unit is part of the European brown hare metapopulation of northern Italy, where the disease is known to be widespread (20).

EBHS has been reported in many different European countries (5) but until now it was not known outside Europe (12). In 1888, imported European brown hares were released for hunting in the province of Santa Fé in Argentina. Genetic studies on European brown hares from Argentina might indicate their European origin (Faber, pers. comm.). However, detailed genetic investigations about the origin of European brown hares from Argentina do not exist until now. Due to the presence of optimal habitat the hare populations increased rapidly and in 1907 the European brown hare was officially declared a pest. As a consequence, commercial hunting started in the early 1930's (10). Our objective was to determine whether European brown hares in Argentina were naturally infected with EBHSV. From 1998 to 2000, serum samples of 80 shot European brown hares from Argentina were examined for antibodies against European brown hare syndrome virus (EBHSV) and 80 spleen samples were investigated for EBHSV-antigen by enzyme linked immunosorbent assay (ELISA). Nine hares were positive for EBHSV-antigen and antibodies against EBHSV were detected in only one individual. Based on negative staining electron microscopy investigations of spleen homogenates, we observed calicivirus in one of five EBHSV-antigen-positive hares. However, EBHS has not been reported to cause any mortality. Consequently, a less pathogenic variant of EBHSV may be present in the population, which does not cause clinical symptoms suggestive of EBHS.

In conclusion, this is the first report of EBHS in Switzerland and of specific antibodies to EBHSV, EBHSV-antigen and electron microscopy findings in free-ranging European brown hares from South America.

Zusammenfassung

Im Jahr 1989 gelang es Lavazza und Vecchi (1989) das ätiologische Agens des European Brown Hare Syndrome (EBHS) in der Leber von an EBHS gestorbenen Feldhasen (*Lepus europaeus*) nachzuweisen, das von Ohlinger und Thiel 1991 als Calicivirus klassifiziert wurde. Mit Ausnahme der Schweiz, ist das Auftreten von EBHS seitdem in vielen europäischen Ländern, jedoch nicht über die Grenzen Europas hinaus, beschrieben worden. Im Zeitraum von 1997-2000 standen 157 tot aufgefundene freilebende Feldhasen aus der Schweiz zur Untersuchung auf EBHS zur Verfügung. Diese Tiere wurden einer patho-histologischen Untersuchung unterzogen; Leber (n=157), Milz (n=107) und Serum (n=60) wurde auf das Vorhandensein von EBHSV-Antigen und Antikörper gegen EBHSV mittels ELISA untersucht. Zusätzlich wurden Leberproben von 87 freilebenden Schneehasen (*Lepus timidus*) auf EBHSV-Antigen untersucht. Zwei Feldhasen zeigten Läsionen in der Leber, die typisch für EBHS sind. In beiden Fällen konnte EBHSV-Antigen in der Leber und in der Milz in hohen Titern nachgewiesen werden. Die elektronenmikroskopische Untersuchung der Leber- und Milzhomogenate erbrachte den Nachweis von Calicivirus-Partikeln in einem der beiden Antigen-positiven Tiere. Drei weitere Tiere waren EBHSV-Antigen positiv, jedoch ohne die für EBHS typischen Läsionen. Antikörper gegen EBHSV konnten in keinem der untersuchten Seren nachgewiesen werden. Alle untersuchten Schneehasen waren EBHSV-Antigen negativ.

Im Zeitraum von 1998-2000 wurden Serum- und Milzproben von geschossenen freilebenden Feldhasen aus Argentinien ebenfalls auf das Vorkommen von EBHSV-Antigen und Antikörpern gegen EBHSV untersucht. Neun Tiere waren EBHSV-Antigen positiv. Milzhomogenate von fünf positiven Tieren wurden mittels Elektronenmikroskopie untersucht, und in einem Fall wurden Calicivirus-verdächtige Partikel gefunden. Antikörper gegen EBHSV waren nur in einer von 80 Serumproben nachweisbar.

Dies ist der erste Bericht über den Nachweis von EBHS in der Schweiz und ferner der erste Nachweis von Antikörpern gegen EBHSV, EBHSV-Antigen und die elektronenmikroskopische Darstellung des Erregers in Europäischen Feldhasen aus Süd-Amerika.

References

1. Büttner S. (1996): Gesundheitszustand der Feldhasen (*Lepus europaeus*) in der Schweiz. Thesis, Veterinary Medical University of Berne, Berne, Switzerland, 99 pp.
2. Chasey. D and P. Duff (1990): European brown hare syndrome and associated virus particles in the UK. The Veterinary Record 126, 623-4.
3. Eskens U and Volmer K. 1989. Untersuchungen zur Ätiologie der Leberdystrophie des Feldhasen (*Lepus europaeus*, Pallas). Deutsche Tierärztliche Wochenschrift 96: 464-6.
4. Frölich K, Meyer HHD, Pielowski Z., Ronsholt I., V. Seck-Lanzendorf S and Stolte M. (1996): European brown hare syndrome in free-ranging hares in Poland. Journal of Wildlife Diseases 32, 280-5.
5. Frölich K, Haerer G, Bacciarini I. Janovsky M Rudolph M and Giacometti M. 2001. European brown hare syndrome in free-ranging European brown and Mountain hares from Switzerland. Journal of Wildlife Diseases. 37, 803-7.
6. Gavier-Widen D, and Mörner T. (1991): Epidemiology and diagnosis of the European brown hare syndrome in Scandinavian countries: Revue Scientifique et technique. Office International des Epizooties 10, 453-8.

7. Gavier-Widen D, and Mörner T. (1993): Descriptive epizootiological study of European brown hare syndrome in Sweden. *Journal of Wildlife Diseases* 29, 15-20.
8. Gortazar C, and De Luco F. (1995): La enfermedad hemorrágica de la liebre. *Trofeo* 295, 30-4.
9. Henriksen P, Gavier D and Elling F (1989): Acute necrotising hepatitis in Danish farmed hares. *The Veterinary Record* 125, 486-7.
10. Kujawski O and Graf O. 1998. *Die neue Wild Küche*. 2. Aufl., Leopold Stocker Verlag, Graz, Germany, 72 pp.
11. Lavazza A and Vecchi G. (1989): Osservazioni su alcuni episodi di mortalità nelle lepri. Evidenziazione al microscopio elettronico di una particella virale. Nota preliminare. *Selezione Veterinaria* 30, 461-7.
12. Lenghaus C., Studdert MJ and Gavier-Widen D. 2001. Calicivirus infections. In *Infectious diseases of wild mammals*, 3rd Edition, E. S. Williams, and I. K. Barker (eds.). Iowa State University Press, Ames, Iowa, pp. 280-91.
13. Marcato PS, Benazzi C, Galeotti M and Salda Della I. (1989): Infective necrotic hepatitis of leporids. *Rivista di Coniglicultura* 26, 41-50.
14. Morisse JP, (1988): Hemorrhagic septicemia syndrome in rabbits: First observations in France. *Le Point Vétérinaire* 20, 79-83
15. Nauwynck, H, Callebaut P, Peeters J, Ducatelle R and Uyttebroek E. (1993): Susceptibility of hares and rabbits to Belgian isolate of European brown hare syndrome virus. *Journal of Wildlife Diseases* 29, 203-8.
16. Ohlinger VF and Thiel HJ.(1991): Identification of the viral hemorrhagic disease virus of rabbits as a calicivirus. *Revue Scientifique et technique. Office International des Epizooties* 10, 311-23.
17. Okerman, I., P. Van de kerckhove, s. Osaer, I. Devriese, and e. Uyttebroek (1989): European brown hare syndrome in captive hares (*Lepus capensis*) in Belgium. *Vlaams Diergeneeskundig Tijdschrift* 58, 44-6.
18. Poli A, Nigro M, Gallazzi D, Sironi G, Lavazza A and Gelmetti D. 1991. Acute hepatitis in the European brown hare (*Lepus europaeus*) in Italy. *Journal of Wildlife Diseases* 27, 621-9.
19. Salmela P, Belak K and Gavier-Widen D. (1993): The occurrence of European brown hare syndrome in Finland. *Acta Veterinaria Scandinavia* 34, 215-7.
20. Scicluna M, Lavazza TA and Capucci I. 1994. European brown hare syndrome in northern Italy: results of a virological and serological survey. *Revue Scientifique et Technique, Office International des Epizooties* 13, 893-904.
21. Slamecka J, Hell P and Ratislav J. (1997): Brown hare in the Westslovak bwland. *Acta Scientiarum Naturalium Academiae Scientiarum Bohemicae Brno* 31, 1-115.
22. Sostaric B, Lipej Z and Fuchs R (1991): The disappearance of free living hares in Croatia: 1. European brown hare syndrome. *Veterinarski Arhiv* 61, 133-50.
23. Steineck Th and Nowotny N. (1993): European Brown Hare Syndrome (EBHS) in Österreich: Epizootiologische Untersuchungen. *Tierärztliche Umschau* 48, 225-9.

European Association of Zoo- and Wildlife Veterinarians (EAZVV) 4th scientific meeting,
joint with the annual meeting of the European Wildlife Disease Association (EWDA)
May 8-12, 2002, Heidelberg, Germany.

PUUMALA (HANTAVIRUS) EPIZOOTIOLOGY IN THE ARDENNES REGION (FRANCE)

**F. SAUVAGE¹, C. PENALBA², Ph. VUILLAUME³, F. BOUÉ⁴, D. PONTIER²,
D. COUDRIER⁵ and M. ARTOIS⁶**

Affiliation:

1. UMR-CNRS 5558 "Biométrie et Biologie évolutive", Université C. Bernard Lyon 1, 43 Bd du 11 novembre 1918, 69622 Villeurbanne cedex, France.
2. Centre Hospitalier, 08000 Charleville-Mézières, France
3. Entente Interdépartementale de Lutte contre la Rage, BP 43, 54220 Malzéville, France.
4. AFSSA Nancy, Domaine de Pixercourt, 54220 Malzéville, France.
5. Centre National de Référence des Arbovirus et virus des Fièvres hémorragiques, Institut Pasteur, 25 rue du Dr Roux, 75724 PARIS Cedex 15 France.
6. ENVL Unité Pathologie infectieuse, BP 83, 69280 Marcy l'Etoile, France. **Corresponding author**

Key words: Puumala virus, HFRS, Ardennes (France), *Clethrionomys*, seroprevalence.

Abstract

Human cases of the so-called "haemorrhagic fever with renal syndrome" (HFRS) were routinely recorded at the CHR (regional hospital) of Charleville, (Ardennes) France. This hospital has registered the maximum number of human disease cases in this country. Epidemic outbreaks of acute infection have been recorded every third year, on a regular basis since the year 1990. During the three years preceding the last peak of the disease (1999), Bank vole (*Clethrionomys glareolus*) the rodent reservoir, was monitored by trapping and antibody screening. Voles were box-trapped on standard trap-lines in a randomised selection of wooded habitats near the city of Charleville. This surveillance was aimed to test the hypothesis that the population level increase in the reservoir will amplify the virus prevalence in voles and the infection hazard in exposed human beings afterward.

If the sampled rodents are representatives of the whole population of reservoir to which humans are exposed, the data are in accordance with a synchrony of the infection rate of both species during the three years epidemiological cycle. But the assumption that the vole demographic population peak precedes the epidemic outbreak in humans is not supported by the data.

During the whole study, the demography of the monitored vole population can be described by a global trend of increase (x by four) with seasonal fluctuations (top in September and bottom in spring). But the prevalence rate reached a maximum 8/10 % level already during the autumn of 1998 (year 2) and remained at this level up to the population peak in September 1999. It can be speculated that the amount of available virus in voles was at its highest level in between September 1998 and 1999.

In accordance with previous records, the greatest number of HFRS was registered during this period at Charleville CHR. Provided that the data of this preliminary study are accurate, the temporal correlation between the infection rates in the human victim and the reservoir host strongly suggests a common source of infection. We hypothesise that mechanism of virus excretion is related to the social structure of the reservoir. In combination with an indirect transmission route it can explain the observed infection figure in voles. These hypotheses are to be challenged during ongoing epidemiological studies.

Résumé

Dans les Ardennes Françaises les cas humains de Fièvre hémorragique avec syndrome rénal (FHSR, due à l'infection par l'Hantavirus Puumala) apparaissent sous forme de flambées épidémiques tous les trois ans depuis 1990. Les auteurs ont décrit la cinétique de la séropositivité des campagnols roussâtres (*Clethrionomys glareolus*) qui est le réservoir de virus. Les résultats suggère une synchronie de l'incidence chez l'homme et le réservoir ce qui pourrait indiquer une contamination à une source commune. Les auteurs évoquent de ce fait, l'hypothèse d'une transmission indirecte du virus.

European Association of Zoo- and Wildlife Veterinarians (EAZWV) 4th scientific meeting,
joint with the annual meeting of the European Wildlife Disease Association (EWDA)
May 8-12, 2002, Heidelberg, Germany.

Erysipelothrix rhusiopathiae INFECTION IN STRANDED HARBOUR PORPOISE (*Phocoena phocoena*) AND HARBOUR SEAL (*Phoca vitulina*)

G. BOSERET¹, T. JAUNIAUX² and J. MAINIL¹

Affiliation :

1. Bacteriology and pathology of bacterial diseases , Department of infectious and parasitic diseases, Faculty of Veterinary Medicine, University of Liège, B43a, Bd de Colonster, 20, B-4000 Liège, Belgium.
2. Pathology, Department of morphology, Faculty of Veterinary Medicine, University of Liège, B43, Bd de Colonster, 20, B-4000 Liège, Belgium.

Key words : Wild animals, porpoise, seal, *Erysipelothrix rhusiopathiae*, septicaemia, enteritis.

Abstract

An adult female harbour porpoise (*Phocoena phocoena*) and a juvenile male harbour seal have been found dead on a Belgian beach in autumn 2001. The two bodies were in good condition (CC = 2). Pure and abundant growth of a small rod-shaped, Gram-labile bacterium was obtained aerobically and anaerobically on Columbia blood-agar from the heart blood, the mouth, the pharynx, the lungs, the intestine and the anus of the porpoise, and from the intestine, the pharynx, the mouth, the nose and the anus of the seal. The colonies were surrounded by a narrow zone of α -hemolysis. The catalase- and peroxydase-tests gave negative results.

Rapid ID 32 Strepto (Biomérieux, France) sugar tests applied on porpoise's heart blood, lungs and intestine, and on seal's intestine and pharynx identified this isolate to *Erysipelothrix rhusiopathiae*.

Erysipelothrix rhusiopathiae is not reported as a common cause of infection and death in wild cetaceans and wild pinnipeds in opposite to respectively captive dolphins and sea lions.

Nevertheless, *E. rhusiopathiae* can be considered as the cause of death of the stranded harbour porpoise as it was present in heart blood and internal organs, and the seal was carrying the bacterium with lesions of enteritis which could be associated with *E. rhusiopathiae* infection

Résumé

Un marsouin commun adulte femelle (*Phocoena phocoena*) et un phoque commun mâle juvénile ont été trouvés échoués sur les plages de la côte belge en automne 2001. Les deux cadavres étaient en bonne condition (CC = 2) et ont été autopsiés immédiatement.

Des analyses bactériologiques ont été effectuées. La mise en culture pendant 24 heures en atmosphère aérobie et anaérobie sur gélose au sang Columbia de prélèvements au départ du sang cardiaque, de la bouche, de l'évent (marsouin) ou des narines (phoque), de l'anus, de l'orifice génital, des intestins et des poumons ont révélé la croissance d'une colonie pure et abondante d'un petit bâtonnet régulier, Gram-labile. Les colonies étaient entourées d'une zone étroite d' α -hémolyse. Les tests catalase- and peroxydase-tests ont donné des résultats négatifs.

Une galerie biochimique Rapid ID 32 Strepto (Biomérieux, France) appliquée sur les isolats de sang cardiaque, poumon et intestin du marsouin, et du pharynx et de l'intestin du phoque a identifié cette bactérie comme étant *Erysipelothrix rhusiopathiae*.

Erysipelothrix rhusiopathiae n'est pas reporté comme un agent infectieux fréquent chez les mammifères marins sauvages, contrairement aux cétacés et pinnipèdes maintenus en captivité.

Néanmoins, *E. rhusiopathiae* peut être considéré comme la cause probable de la mort du marsouin, comme il a été isolé du sang cardiaque et de nombreux autres organes. Le phoque présentait des lésions d'entérite qui peuvent être associées à la présence d' *E. rhusiopathiae* dans l'intestin.

Introduction

Erysipelothrix rhusiopathiae is a Gram positive rod-shaped bacterium reported as an important pathogen for pigs, lambs, turkeys and fish, and less commonly in humans, dogs and cattle (5). The humans can be infected by direct contact with fish (10) (“Erysipelas” or “fisherman’s disease”). This bacterium persists in water and soil, in dampness and coldness. Animals can be carriers in their intestine and excrete the bacterium in their faeces.

Erysipelothrix rhusiopathiae infection has also been described in captive cetaceans (7, 12, 14) and to a lesser extent in captive pinnipeds (15).

Two different forms are known: the acute form (“white erysipelas”) characterised by septicaemia and sudden death, and the chronic form characterised by rhomboid diamond-shaped necrotic skin lesions (6, 7, 13, 14). A vaccine against *Erysipelothrix rhusiopathiae* has been tested in Belgian (9), Hawaiian (2) and Californian (4) delphinaria and showed incentive results.

Only a very few cases were reported from wild stranded marine mammals.

This paper describes two cases of *Erysipelothrix rhusiopathiae* infection in an adult female common porpoise (*Phocoena phocoena*) and in a young male harbour seal (*Phoca vitulina*).

Materials and methods

The two animals were found dead on the Belgian North Sea coastlines. They have been necropsied and sampled following the Kuiken and Garcia-Hartmann (small cetaceans) (8) and Geraci (pinnipeds) (3) protocols.

Mouth, pharynx, blowhole (porpoise) or nose (seal), anus and genital hole were sampled with sterile swabs; heart blood, lymph nodes, lungs and intestinal content were sampled after surface heat sterilisation. All the samples were inoculated on Columbia blood agar and Gassner agar plates, and incubated at 37°C overnight aerobically and anaerobically.

Results

Main necropsy results were acute haemorrhagic enteritis and acute haemorrhagic broncho-pneumonia in the porpoise while the seal presented an acute haemorrhagic enteritis only.

Pure and abundant growth of a small rod-shaped, Gram-labile bacterium was obtained aerobically and anaerobically on Columbia blood-agar from porpoise’s heart blood, mouth, pharynx, lungs, intestine and anus, and from seal’s intestine, pharynx, mouth, nose and anus. The colonies were surrounded by a narrow zone of α -hemolysis. The catalase- and peroxydase-tests gave negative results. The oxidative-fermentative test showed a fermentative metabolism.

Rapid ID 32 Strepto (Biomérieux, France) sugar tests on porpoise’s heart blood, lungs and intestine, and on seal’s intestine and pharynx isolates identified this bacterium to *Erysipelothrix rhusiopathiae*.

Discussion

Erysipelothrix rhusiopathiae has not been reported as a common cause of infection and death in wild cetaceans and wild pinnipeds in contrast to captive ones (7, 12, 14, 15).

Nevertheless, *E. rhusiopathiae* can be considered as the probable cause of death of the stranded harbour porpoise since it was isolated in pure and abundant culture from heart blood and many internal organs.

The bacterium was isolated from seal’s intestine and anus and the seal presented lesions of enteritis, which could be associated with *E. rhusiopathiae* infection. However, no evidence of septicaemia could be found in the seal since no growth was obtained from heart blood).

Erysipelas was described in Belgian captive marine mammals by Lacave and Cox (9) but only rare reports of *Erysipelothrix rhusiopathiae* infection in wild stranded cetaceans and pinnipeds were published (6, 13).

More investigations on wild mammals would help to define the bacterium-carrying status of healthy animals and to have a more precise idea of *E. rhusiopathiae* importance in wild animals causes of death.

These investigations should also study the actual virulence factors of *E. rhusiopathiae* in wild sea mammals and compare them to isolates from fish, birds and terrestrial mammals (1, 11).

References

1. Arhné S., Stenström IM, Jensen NE, Petterson B, Uhlen M and Molin G. Classification of *Erysipelothrix* strains on the basis of restriction fragment length polymorphisms. *Int. Journ. Of Syst. Bact.* 1995 ; 45 (2) : 382-5.
2. Colgrove GS. A survey of *Erysipelothrix insidiosa* agglutinating antibody titers in vaccinated porpoises (*Tursiops truncatus*). *Journ. of Wildl. Diseases* 1975 ; 11 : 234-6.
3. Eamens GJ, Turner MJ and Catt R.E. *Australian Vet. Journ.* 1988; 65 (8) : 249-52 .
4. Geraci and Loundsbury, *Marine Mammals ashore : a field guide for stranding.* Texas A& M University Sea Grant College Program , Galveston, Texas, 1993.
5. Gilmartin WG, Allen Jf and Ridgway S.H. Vaccination of porpoises (*Tursiops truncatus*) against *Erysipelothrix rhusiopathiae* infection. *Journ. Of Wildl. Diseases.* 1971 ; 7 :292-5.
6. Griffiths IB, Done SH and Readman S. *Erysipelothrix* pneumonia in sheep. *Vet.Rec.* 1991 ; 128 : 382-3.
7. Higgins R. Bacteria and fungi of marine mammals : a review. *Can Vet.J* 2000 ; 41 : 105-16.
8. Kinsel MJ, Boehm JR, Harris B and Murnane R.D. Fatal *Erysipelothrix rhusiopathiae* septicaemia in a captive pacific white-sided dolphin (*Lagenorhynchus obliquidens*). *Journ. Of Zoo and Wildl. Medicine.* 1997 ; 28 (4) : 494-49,.
9. Kuiken T and Garcia-Hartmann M. Proceedings of the first European Cetacean Society workshop on cetacean pathology : dissection techniques and tissues sampling, ECS newsletters. 1991, special issue,.
10. Lacave G, Cox E, Hermans J, Devriese L and Goddeeris B.M. Induction of cross-protection in mice against dolphin *Erysipelothrix rhusiopathiae* isolates with a swine commercial vaccine. *Vet. Microb.* 2001 ; 80 : 247-53.
11. Lehane L and Rawlin G.T. Topically acquired bacterial zoonoses from fish : a review. *Med J Aust.* 2000 ; 173 (5) : 256-9.
12. Makino S, Okada Y, Maruyama T, Ishikawa K, Takahashi T, Nakamura M, Ekazi T and Morita H. Direct and rapide detection of *Erysipelothrix rhusiopathiae* DNA in animals by PCR. *Journ. Of Clin.microbiol.* 1994 ; 32 (6) : 1526-31.
13. Medway W and Schryver H.F. Respiratory problems in captive small cetaceans. *JAVMA* 1973; 163 (6) : 571-3,.
14. Medway W. Some bacterial and mycotic diseases of marine mammals, *JAVMA* 1980 ; 117 (9) : 831-4.
15. Thurman GD, Downes SJT, Fothergill MB, Goodwin NM and Hegarty MM. Diagnosis and successful treatment of subacute erysipelas in a captive dolphin. *Journ. Of South Africa Ass.* 1983 : 193-200.
16. Thornton SM, Nolan S and Gulland F.M.D. Bacterial isolates from california sea lions (*Zalophus californianus*), harbor seals (*Phoca vitulina*), and northern elephant seals (*Mirounga angustirostris*) admitted to a rehabilitation center along the central california coast, 1994-1995. *Journ. Of Zoo and Wildl. Medicine* 1998 ; 29 (2) : 171-17,.

Address

Dr. Géraldine Boseret: [mailto: geraldineboseret@yahoo.fr](mailto:geraldineboseret@yahoo.fr)

European Association of Zoo- and Wildlife Veterinarians (EAZVV) 4th scientific meeting,
joint with the annual meeting of the European Wildlife Disease Association (EWDA)
May 8-12, 2002, Heidelberg, Germany.

DISEASES AND MORTALITY IN FREE-RANGING LYNX (*Lynx lynx*), BROWN BEAR (*Ursus arctos*), WOLF (*Canis lupus*) AND WOLVERINE (*Gulo gulo*) IN SWEDEN

T. MÖRNER¹, H. ERIKSSON², C. BRÖJER¹, K. NILSSON¹, H. UHLHORN¹, E. ÅGREN³, M.P. RYSER-DEGIORGIS¹, C. HÅRD AF SEGERSTAD³, D. S. JANSSON,⁴ and D. GAVIER-WIDÉN¹

Affiliation:

1. Department of Wildlife, National Veterinary Institute, SE-751 89 Uppsala, SWEDEN
2. Åkervägen 24E, SE-952 62 Kalix, SWEDEN
3. Department of Pathology, National Veterinary Institute, SE-751 89 Uppsala, SWEDEN
4. Department of Poultry, National Veterinary Institute, SE-751 89 Uppsala, SWEDEN

Key words: brown bear, *Canis lupus*, diseases, *Gulo Gulo*, infanticide, lynx, mange, mortality, *Sarcoptes scabiei*, trauma, *Ursus arctos*, wolf, wolverine

Abstract

One hundred and forty-six lynx, 98 brown bears, 20 wolves and 27 wolverines submitted to the National Veterinary Institute, Uppsala, Sweden in the years 1987 to 2001 were investigated for diseases and causes of mortality. The most common cause of death in lynx was sarcoptic mange, most likely acquired from infected red foxes that had been killed by the lynx. The second most common cause of death was traumatic injuries; animals killed in road accidents or by trains, with 34 cases (23%). Lynx moves over large areas and frequently follow roads or railway tracks, especially in winter when the snow cover is deep. Sarcoptic mange was also common in wolves. The most common cause of death in wolves was traumatic injuries; road accidents with cars or lorries, with seven animals (35%) which died of this reason. The most frequent cause of natural death in brown bears was intraspecies fights (infanticide): with 16 (16%) bears killed by other bears. Territory defending male bears are believed to be the animals causing this mortality. Infectious diseases were not frequently observed in any species and only a few cases were observed. Traumatic injuries, originating from road or railway accidents, were also a common cause of death in wolves and in brown bears. Infanticide, i.e. intraspecies killing, was also observed as a cause of mortality in young wolverines. Malformation of the spinal cord was observed in one lynx and one wolf. It is not known if these malformation are hereditary or not and if they are of importance in the future management of these small and endangered populations. Parasites were only found in a few animals and in all cases the parasitic burden was low. A large number of lynx, brown bears, wolves and wolverines were examined as forensic cases since poaching was suspected. The illegal hunting can be a problem for the future management of these large predator populations, since the illegal killing can be a threat to the future status of the population.

Zusammenfassung

Einhundertsechsvierzig Luchse, 98 Bären, 20 Wölfe und 27 Vielfrässe, die in den Jahren 1987 bis 2001 bei der Staatlichen Veterinärmedizinischen Anstalt in Uppsala, Schweden, eingereicht wurden, wurden vor Krankheiten und Todesursachen untersucht. Die häufigste Todesursache unter den Luchsen war Räude (*Sarcoptes scabiei*), was sie sich höchst wahrscheinlich von räudeinfizierten Fuchse, die die Luchse getötet haben, geholt haben. Die zweit häufigste

Todesursache unter den untersuchten Luchsen und Bären war Verkehrs- und Zugunfälle. Unter den Wölfen war Verkehrs- und Zugunfälle die aller häufigste Todesursache.

Das Verhalten unter den Bären und Vielfrassen Individuen der eigenen Tierart zu töten, war unter diesen beiden Tierarten die häufigste Todesursache.

Ansteckende Krankheiten wurden, abgesehen der Räude, unter keiner der vier Tierarten beobachtet.

Fehlstellungen des Rückenmarkes wurden bei einem Luchs und einem Wolf festgestellt. Es ist nicht bekannt, ob diese Fehlstellungen erblich sind oder nicht, und ob sie für Bedeutung für die zukünftige Management dieser bedrohten Tierpopulationen haben.

Abgesehen der Räude wurden Parasiten nur in einzigen Tieren gefunden.

Bei einer grossen Anzahl der Tieren wurden eine forensische Untersuchung unternommen, nach dem Verdacht auf illegale Jagd entstanden wurde. Illegale Jagd könnte in der Zukunft für diesen Populationen grosser Predatoren Probleme bedeuten.

Résumé

De 1987 à 2001, 146 lynx, 98 ours bruns, 20 loups et 27 gloutons ont été envoyés à l'Institut vétérinaire national à Uppsala (Suède) pour une analyse pathologique. La cause de mort la plus commune chez les lynx examinés était la gale sarcoptique, suivie par les traumatismes (collisions avec une voiture ou un train). La gale sarcoptique a également été fréquemment diagnostiquée chez les loups, mais chez cette espèce les traumatismes dus à des accidents de la route étaient plus fréquents que les cas de gale. Chez les ours, la majorité des animaux examinés étaient morts à la suite de combats intraspécifiques (infanticides). Les combats intraspécifiques semblent être également une cause de mort fréquente chez les gloutons. Chez un lynx et un loup, on a observé une malformation de la colonne vertébrale mais on ignore s'il s'agit là d'une tare héréditaire. Très peu d'animaux étaient infestés par des parasites. Un grand nombre de lynx, ours, loups et gloutons ont été examinés dans le cadre d'une enquête juridique car il y avait soupçon de braconnage.

European Association of Zoo- and Wildlife Veterinarians (EAZVV) 4th scientific meeting,
joint with the annual meeting of the European Wildlife Disease Association (EWDA)
May 8-12, 2002, Heidelberg, Germany.

MORTALITY, AGE STRUCTURE AND FERTILITY OF A NON RANDOM SAMPLE OF 206 DEAD WOLVES RETRIEVED IN ITALY DURING 1986-2001

***V. GUBERTI¹, M. BOLOGNIN¹, M.A. DE MARCO¹,
A. DE FAVERI¹ and F. MARZADORI²***

Affiliation:

1. Istituto Nazionale per la Fauna Selvatica, Ozzano E., Italy
2. Istituto Zooprofilattico Sperimentale della Lombardia e dell'Emilia, Lugo, Italy

Key words: wolf, mortality, age structure, fertility

Abstract

During the period 1986-2001, 206 wolf carcasses retrieved in Italy have been examined. For each dead wolf data and locality of retrieval were available. Standard necropsies and specific examinations assessed causes of mortality. Age was obtained by tooth eruption, cementum-annuli count and cranial sutures. Female fertility was assessed by pregnancy or uterine scars count. Yearly and monthly retrieval trends were calculated. Age structure of the sample has been defined, along with the number of reproductive females and their mean number of uterine scars. No apparent trends have been observed by years, whereas the autumn/winter months are more likely in having dead wolves. Male-female sex ratio was 1.04; when age was clumped in three main classes, 38.6% of the animals were younger than one year, 18.4% ranged from one to two years and 43% were older than three years. Five of the females older than three years were pregnant (N=1) or showed uterine scars (N=4), representing 5.1 % of the whole females collected in the sample. The mean number of uterine scars or foetus was 4.4 (SD 1.67). The main mortality causes was poaching (illegal shooting and poisoning). The data are discussed and criticised.

DENTAL LESIONS AND BITE WOUNDS IN EURASIAN OTTERS (*Lutra lutra*)

V. R. SIMPSON

Affiliation:

Wildlife Veterinary Investigation Centre, Jollys Bottom Farm, Station Road, Chacewater, Truro, Cornwall, TR4 8PB, UK.

Abstract

As part of a long-term study to monitor the health status of otters in south west England post mortem examinations were carried out on 242 specimens submitted between December 1988 and January 2002. The great majority (80%) had died in road traffic accidents but bite wounds were the second most common cause of death (1, 2). This report describes observations on bite wounds and dental lesions.

Bite wounds were commonly present, with 23 out of 102 females (22.5 %) and 35 out of 140 males (25 %) showing lesions. Bites to the head usually involved the cheek and/or lip whilst those to the feet were mostly confined to the central pad and/or the digits. In some cases the digits were amputated. Most bites in the perineal area were around the anus but often also involved the vulva or scrotum. The consistent pattern of the bite wounds suggests stylised fighting due to intraspecific aggression (1, 2). In some cases the wounds showed evidence of infection, with extensive cellulitis and necrosis involving the subcutaneous tissues and muscles.

In a few cases the size and spacing of the wounds were smaller and could have been inflicted by another species, possibly American mink, *Mustela vison*. Two cubs were killed by dogs and in these cases the lesions were distinct, with large puncture wounds, extensive bruising and fractured ribs.

All ages were affected, but it was noticeable that although two female cubs had died as a result of bite wounds, none of the male cubs of less than 4 kg were bitten. There was evidence of a higher incidence of wounds in otters in particular weight ranges. This was particularly noticeable in males between 5 and 5.9 kg where 60 % were affected. The incidence then dropped off progressively and was lowest in the heaviest otters. A similar, though less striking, pattern was seen in females with bites present in a round 29% of animals in the 4 to 6 kg. range. However, many of these otters were not sub adults but had low body weight to length ratios, presumably as a result of fighting.

The degree of wear on the teeth was recorded and any lesions noted. The general health of teeth was good, with very little calculus and no cases of caries. However, damaged or missing teeth were common, with 46 animals (19%) affected.

Fractures or loss of incisors were recorded in 15 males and 7 females (9% overall). Lesions were twice as common on the left side of the mouth as on the right. Lesions affecting canine teeth were more slightly common and fractures or loss were recorded in 11.6% of otters (19 males and 9 females). Frequently there was either a slab fracture or the tooth was sheared off near the base, leaving the pulp cavity exposed. In most cases only one canine was broken but in several otters two or more were fractured and in some cases an entire tooth had been lost. Young animals were not affected and no lesions were seen in females of less than 4 kg or in males of less than 5 kg. In both sexes the highest incidence of canine damage was in the weight range 6 to 6.9 g where around 28% of males and 21% of females were affected.

In some otters the canines on one side of the mouth were more worn than on the other, suggesting that they may be 'handed'. It was also observed that attrition of the enamel on the anterior/lateral aspect near the base of the lower canines was very common. It is assumed that this is caused by some aspect of feeding behaviour.

Lesions involving the cheek teeth were uncommon but often important. Fractures involving the fourth upper premolars, or carnassials, were seen in six otters, five males and one female. As with domestic dogs, this is a significant lesion and in two cases it resulted in a root abscess and osteomyelitis. The infection tracked through the bone dorso-laterally and led to severe cellulitis and necrosis of facial tissues, septicaemia and death. The largest otter examined, a male weighing 11.3 kg., also appeared to have died as a result of a tooth abscess. Unfortunately it was too autolysed for detailed examination. It had fractures to several lower incisors and premolars and a fracture involving the root of the lower right first molar. The latter had become infected, resulting in osteomyelitis and thickening of the ramus. This was the only lesion seen in a molar tooth.

It seems likely that that fractures to carnassial teeth are due to otters biting on hard material but fractures and loss of incisors and canines may often be due to fighting. In several cases fresh bite wounds were seen in otters that also had fresh tooth damage, particularly to canines. Ten out of the 28 otters with canine lesions (36%) had bite wounds, mostly to the head, feet and perineum, and 62% of otters with incisor damage had bite wounds.

The prevalence of bite wounds caused by intraspecific aggression appeared to be fairly constant at around 15 - 17 % up until 1999 but in the year 2000 it was 32 %. This was large due to a marked increase in bitten females, four of which died from their injuries. Mortality associated with bite wounds was 12.5% in 2000 whereas in earlier years it had been in the range 4 - 6.5 %. The overall mortality attributable to fighting was 7.4% (18 out of 242). However, this figure is almost certainly an under estimate, as otters dying in road accidents are much more likely to be seen and submitted for examination than otters dying in the countryside. Intraspecific aggression could be as important as road accidents as a cause of mortality in otters.

References

1. Simpson VR. Health status of otters (*Lutra lutra*) in south-west England based on postmortem findings. *Veterinary Record* 1997; 141: 191-97
2. Simpson VR and Oxbon KE. Intraspecific aggression, cannibalism and suspected infanticide in otters. *British Wildlife* 2000; 11: 423-6

SPATIO-TEMPORAL VARIATIONS IN SERO-PREVALENCE OF CHLAMYDIOSIS AND Q FEVER IN MOUNTAIN UNGULATE POPULATIONS: RETROSPECTIVE DETECTION OF EPIDEMICS

E. FROMONT¹, P.H. GIBERT², J. HARS² and D. GAUTHIER³

Affiliation :

1. UMR CNRS 5558, Biométrie et Biologie Evolutive, Université Lyon 1, 43 Bd du 11 novembre 1918, 69622 Villeurbanne Cedex
2. Office National de la Chasse et de la Faune Sauvage, 8 impasse Champ Fila, 38320 Eybens
3. Laboratoire Départemental d'Analyses Vétérinaire de la Savoie, 321 chemin des Moulins, 73024 Chambéry Cedex

Abstract

Serological data are often used as markers of disease in natural populations. However they often lack external validation. We propose to use descriptive multivariate methods to analyse and interpret large sets of serological data, by comparing the distribution of serological response in different host populations. First, it is possible to determine *a posteriori* a positive threshold, by comparing the distribution of seronegative and seropositive responses across populations. Then we compare the serological response among populations, and show that epidemics can be detected retrospectively. Descriptive analysis constitutes a preliminary investigation but useful to orient field research towards populations of interest.

Key words: epidemiology, serological data, mountain ungulate, chlamydiosis, Q Fever

Introduction

In wild-living populations, investigations concerning disease prevalence are limited by the difficulty to observe clinical signs, specific mortality or reduced fecundity. Because they are simple to record, indirect indicators of disease such as serological measures are frequently searched, however these methods need to be validated. To interpret serological data, the ideal information is the serological profile after infection and its possible variations, which can be measured after experimental infections. However, such information is almost always lacking. It is also possible to make cross-validations of imperfect tests (4), albeit only when several tests are available.

The qualitative interpretation of serological tests depends on the components of the test itself: when a serum is reacting with a given antigen, antibodies reacting against the antigen are present. Then it is possible to infer which pathogen, containing the antigen, is detected. But here we focus on the quantitative aspect of serological tests: how to interpret the amount of antibodies that are detected in the sera of wild-living animals? What is a low or high response? And, primarily, what is the threshold above, which an animal can be considered as positive, or clinically affected? This question raises especially when using serological tests that have been developed primarily for domestic species.

When large serological data sets are available, we argue that it is possible to infer positive threshold and to search for spatio-temporal variations in disease prevalence, by comparing the serological response of different populations. First, determining a positive threshold lies on the detection of false-positive results that have to be considered as negative. If false-positive results occur at random, then their spatio-temporal distribution should be identical to the distribution of negative results. Thus comparing the distribution of titres among populations should help us to

detect titres corresponding to false-positive results and choose a positive threshold. On the other hand, descriptive methods should allow us to classify populations according to the distribution of their serological titres. Then it should be possible to interpret the classification by comparing the distribution of serological titres among groups of populations.

Here we studied serological data from a long-term investigation on chamois (*Rupicapra rupicapra* and *R.r. pyrenaica*), alpine ibex (*Capra ibex ibex*) and mouflon (*Ovis gmelini*) in the French Alps. We analysed the serological response against two bacteria that are known to induce abortion in domestic herds, *Chlamydia psittaci*, agent of chlamydiosis, and *Coxiella burnetti* (Q fever). Both infections have been diagnosed with complement fixation methods, thus serological titres were available. We used descriptive statistical methods with two aims in mind: first, is it possible to determine a positive threshold by comparing the distribution of the serological titres in the different populations? And is it possible to analyse the variation of diseases among populations by comparing the distribution of titres among them?

Materials and methods

Serological data have been recorded from the long-term sanitary follow-up of mountain ungulates that has been driven since 1980. Samples have been collected from 29 sites during one to 15 years, representing a total of 2582 sera. Samples were tested using complement fixation, following AFNOR recommendation (1). Data were then gathered in tables including site, date and serological titre for each of the 2582 sera.

We used Factorial Analysis, which is the classical method to describe contingency tables (3, 5). Factorial analysis makes a description of the distribution of one variable as a function of the second one. Here we first wanted to study the distribution of serological titres among populations in order to compare the serological titres and find a possible positive threshold. But we also aimed to study the distribution of populations based on serological titres to draw types of populations regarding the serological titres. Moreover, we considered the absence of antibodies (titre <8) as a reference situation: all titres were analysed by comparison with the null titre. Several methods are available when all columns are not considered equally (6,7). We used Reference Column Factorial Analysis (CRFA, 2) as the only method considering a reference column.

We first checked the detection of false-positive results by creating at random false-positive individuals (titre = 5) among chlamydiosis-seronegative chamois of the Bauges population. We analysed the new table with CRFA.

Then we applied CRFA to the original tables. We created tables with serological titres as columns and spatial or temporal information as rows. In order to analyse spatial differences we built tables including locations as rows. For the temporal evolution we used data from the largest population (i.e. the Bauges population of chamois) and we compared the 15 years of study as rows. We created two series of tables, concerning chlamydiosis and Q fever respectively.

Results

Detection of false-positive cases and positive thresholds

The table including false-positive individuals showed that their distribution was rather close the distribution of negative individuals, and distant from the distribution of other titres. This result clearly validated our use of CRFA to search for false positive at the condition that false positive occur at random. When applied to original tables, CRFA detected false-positive titres only concerning chlamydiosis in the ibex. In all other cases, serological titres were structured as 2 to 3 levels, suggesting that several thresholds may be useful to classify populations.

Spatio-temporal variations

We classified the studied populations into three groups that were more or less well defined according to the table analysed. We qualified these groups as "epidemic", "endemic" or "healthy" profiles according to the observed distribution of titres. The term "epidemic" is used to describe a population where high titres are relatively frequent compared to intermediate or low titres,

"endemic" populations are populations where low titres are most frequent and at high prevalence, and "healthy" populations have low prevalence of both levels of titres.

Concerning the temporal evolution of distribution in the Bauges population of chamois the different years can be classified according to three factorial axes, which suggests that 3 levels of information are necessary to classify the different years. The analysis of the 3 axes clearly shows an important serological burst in 1987, followed by 2 years of high-level endemic situations. This results allowed us to propose the hypothesis of a "serological epidemics" during this period, with probable transmission among chamois. The question of the domestic or wild origin of the accident cannot be solved here. This analysis also shows that the epidemic tended to disappear from the population with no particular intervention, and it also brings information on the persistence of antibodies in chamois.

Discussion

Descriptive methods are only rarely used in epidemiology (3, 7). Here, description of serological tables allowed us to classify populations studied into groups of probable epidemic, endemic or "healthy" conditions. This classification is only based on serology: the terms of "epidemic", "endemic" and "healthy" do not define any clinical status, nor the consequence of possible epidemics on the host populations dynamics. However the classification obtained is in good accordance with field observations on the exposure of wild populations.

Descriptive analysis could be particularly useful when studying the epidemiology of wild populations, which is severely constrained. However our approach does not solve all questions linked to the use of serological data. Positive serological responses can be observed long after infection, thus time since infection is unknown. Also the clinical significance of a "serological epidemic" remains to be investigated. Descriptive analysis does not replace the gold standard, but can help to search whether it is necessary to look further in the question, and where and when to search for detecting an impact of the disease on host population dynamics, if any.

References

1. AFNOR 2000. Norme française U 47-006. Recherche d'anticorps contre la chlamydie et/ou la fièvre Q chez les mammifères par la technique de fixation du complément.
2. Chessel D, Thioulouse J. Programmathèque ADE-4, Analyses multivariées et expression graphique des données environnementales, Université Claude Bernard Lyon I, 1995. <http://pbil.univ-lyon1.fr>
3. Ducrot C., Legay J.M., Grohn Y.T., Enevoldsen C., Calavas D. 1996. Approach to complexity in veterinary epidemiology: example of cattle reproduction. *Natures Sciences Sociétés*, 4(1): 23-34.
4. Enoe C, Georgiadis MP, Johnson WO. Estimation of sensitivity and specificity of diagnostic tests and disease prevalence when the true disease state is unknown. *Prev Vet Med* 2000 ; 45 : 61-81.
5. Escoffier B, Pagès G. Analyses factorielles simples et multiples. Objectifs, méthodes et interprétation. 2ème édition, Bordas, Paris, 1990, 263p.
6. Lebart L. Exemple d'analyse d'un tableau dont l'une des colonnes a un poids prédominant. *Les Cahiers de l'Analyse des Données* 1979 ; 4 : 417-22.
7. Tillard E, Faye B. Analyse d'un tableau de taux - Exploration multidimensionnelle de la circulation des agents infectieux.

THE STUDY OF THE ANORMAL MORTALITY OF THE ROEDEER IN FRANCE

M. E.TERRIER and M.ARTOIS

Affiliation :

1. AFSSA Nancy, Domaine de Pixérécourt, BP9 , 54 220 MALZEVILLE, France
2. Ecole Nationale Vétérinaire de Lyon, 1, avenue de Bourgelat, 69 280 MARCY L'ETOILE

Abstract

Since 1997, some departementals federations of hunters describe an abnormal mortality in roe deer, by the finding of some cadavers in the same area in a short time.

In 1999 the committee of the network SAGIR decided to organise a study of this phenomenon. The work has been going on for two years. The comparison between the roe deer from areas with declared abnormal mortality and from control areas doesn't showed any differences nor in the causes of death neither in the lesions and pathogens associated to the death.

Because of the insufficient number of analyses, we can't come to a conclusion. It was decided to invite bids to study further.

Zusammenfassung

Seit 1997 beklagen sich mehrere departementales Verbänden von Jäger über ungewöhnliche Sterblichkeit der Rehirsch. Sie finden in kurzer Zeit mehrere Leichen in einer gleichen Gegend.

In 1999 hat der Beirat von dem Netz SAGIR entschlossen, diese ungewöhnliche Sterblichkeit („MAC“) zu studieren. Die Arbeit hat zwei Jahre lang gedauert. Das Vergleichen von Tiere aus Gegenden mit MAC und Kontrollgebieten zeigt weder sinnvolle Unterschied in den Todesursache noch in den Schädigungen oder Keime im Verhältnis mit der Tod.

Aber zu wenige Tiere wurden untersucht, um gültige Schlüsse zu ziehen. Eine Ausschreibung soll eine gründliche Arbeit entwickeln.

Resume

Depuis 1997 de nombreuses fédérations de chasseurs déplorent la survenue d'une mortalité perçue comme anormale, par la découverte de plusieurs cadavres de chevreuils sur un même territoire.

En 1999 le comité de pilotage du réseau SAGIR décidait de réaliser une analyse du phénomène qui fut alors désigné sous le nom de MAC (mortalité anormale du chevreuil), le protocole d'étude étant dénommé EMAC (étude de la MAC). Le protocole d'étude a été mis en place pendant deux ans. La comparaison des animaux issus de zones déclarées en MAC et ceux issus de zones témoins ne révèle pas de différences significatives des grandes causes de mortalité ni même des lésions ou agents pathogènes associés à la mort de l'animal.

Mais un trop petit nombre d'analyses a pu être effectué pour permettre de tirer des conclusions précises. Il a alors été décidé de rédiger un appel d'offre pour réaliser une étude plus poussée.

European Association of Zoo- and Wildlife Veterinarians (EAZWV) 4th scientific meeting,
joint with the annual meeting of the European Wildlife Disease Association (EWDA)
May 8-12, 2002, Heidelberg, Germany.

PSEUDORABIES IN WILD BOAR: MORTALLITY AND DEMOGRAPHY IN ITALY

V. GUBERTI¹, G. FERRARÍ, M. FENATÍ, M.A.DE MARCO¹ and T. PASQUALÍ

Affiliation:

1. Istituto Nazionale per Fauna Selvatica, Ozzano E. Italy
2. Istituto Zooprofilattico Sperimentale Lazio e Toscana, Rome, Italy

Abstract

During the period 1997-1999 three Italian wild boar populations have been investigated in order to detect antibodies against Aujeszky Disease using an ELISA test. The number of examined samples (N), their seroprevalencies (P) and standard errors (SE) were:

- 1) Maremma, Tuscany (Grosseto) (N=265; P= 47% SE=3)
- 2) Presidential Estate of Castel Porziano (Rome) (N=136; P=0%)
- 3) Northern Apennines (Bologna) (N=89; P=6%; SE=2.4)

In the study area 2) according to sampling intensity and population number (about 800 animals in an enclosed area of 50 sqkm) the maximum undetected prevalence might be 2%, an unrealistic prevalence for maintaining the infection in a such high-density populations.

No differences were observed according to gender whereas prevalence significantly increase s with age showing (for the maremma population only) and endemic infection. In this population, using age stratified seroprevalence, the force of infection results in 0,00153/wild boar/day. The importance of latency in maintaining the infection in the wild is discussed. The infection in the wild boar appears endemic where population are large and long lasting and when simpatry with free roaming domestic pigs have been reported in past.

European Association of Zoo- and Wildlife Veterinarians (EAZWV) 4th scientific meeting,
joint with the annual meeting of the European Wildlife Disease Association (EWDA)
May 8-12, 2002, Heidelberg, Germany.

***Mycoplasma conjunctivae* IS MAINTAINED IN DOMESTIC SHEEP BUT NOT IN ALPINE CHAMOIS IN THE SWISS ALPS**

**M. JANOVSKY¹, J. FREY², J. NICOLET², L. BELLOY²,
and M. GIACOMETTI³**

Affiliation

1. Amt der Tiroler Landesregierung, Veterinärdirektion, Wilhelm-Greilstr. 25, A-6020 Innsbruck, Austria
2. Institute for Veterinary Bacteriology, University of Berne, Lenggassstrasse 122, CH-3012 Berne, Switzerland
3. Wildvet Projects, CH-7605 Stampa, Switzerland

Key words: Alpine chamois, sheep, ELISA, PCR, culture, epidemiology, infectious keratoconjunctivitis, *Mycoplasma conjunctivae*, *Rupicapra r. rupicapra*, serodiagnosis, Switzerland.

Extended abstract

Infectious keratoconjunctivitis (IKC) caused by *Mycoplasma conjunctivae* is a highly contagious ocular infection, which is common in domestic sheep and goats. In the European Alps, IKC is often observed in Alpine chamois (*Rupicapra r. rupicapra*) and in Alpine ibex (*Capra i. ibex*), but the disease has also been described in other wild Caprinae in the Pyrenees and in New Zealand. The infection is characterised by inflammation of the conjunctiva and cornea, and in the most advanced stages, the cornea is opaque or even perforated. In IKC outbreaks in chamois and ibex, spontaneous recovery is the most prevalent outcome of the disease. However, mortality can occasionally reach 30% (2). On alpine meadows in Switzerland, IKC occurs at the same time in the same regions in several host species. To assess which host species maintains the *M. conjunctivae* infection in Switzerland, we performed bacteriological and serological investigations in both, domestic sheep and Alpine chamois.

Among a sample of 69 sheep showing clinical signs of IKC in 3 Swiss cantons, *M. conjunctivae* was identified 53 times (76.8%). An indirect ELISA based on a membrane fraction of *M. conjunctivae*, prepared by industrial standards (1) was used to detect *M. conjunctivae* antibodies in 674 sera of adult sheep. We analysed a stratified random sample of 123 sheep herds from 25 out of the 26 Swiss cantons. At least one positive animal was detected in 89.4% of the herds. In positive herds (n=110), 57.1% of the individual animals tested positive. To assess the importance of sheep's age in the spread of *M. conjunctivae*, 209 sera of adult sheep and 93 lamb sera among 8 sheep herds were analysed using the indirect ELISA. Seroprevalence in 2-6 month old lambs was 50.5%, indicating that the IKC agent is spread in sheep flocks during raising. Lambs experimentally infected with *M. conjunctivae* carried the agent for 8 and 23 weeks, respectively, depending on the strain used for challenge. We concluded that *M. conjunctivae* are widespread in domestic sheep in Switzerland. In this country, mycoplasmal IKC was found to be endemic and self-maintained in the domestic sheep population (4). In alpine chamois, the occurrence of IKC was assessed in 1950-1999 in Grisons, a canton in eastern Switzerland. First IKC outbreaks were reported in the decade 1950-1959. Since then, the number of affected subpopulations constantly increased and, by the year 1999, IKC outbreaks were reported in 39 out of 51 (77%) chamois subpopulations. In 1992-1999, a total of 243 chamois, which died of the consequences of IKC, were recorded. The number of cases differed between years, and a distinct seasonal trend was observed. IKC was more common during summer and autumn, with 48% of the cases recorded in August-October. Juveniles (< 4 years of age) were mostly represented. To verify the presence of *Mycoplasma conjunctivae* in chamois we have analysed conjunctival swabs taken from animals

affected with IKC. Among a sample of 28 affected chamois, *M. conjunctivae* was identified 14 times (50%). An indirect ELISA was developed to detect specific *M. conjunctivae* antibodies in the sera of alpine chamois with infectious keratoconjunctivitis using serospecific antigens of *M. conjunctivae*. In subpopulations with ongoing IKC outbreaks, seroprevalence was low (8%). Seroprevalence was even lower in subpopulations with recent IKC outbreaks (3%). We concluded that the *M. conjunctivae* infection is not maintained in the chamois population of the eastern Swiss Alps (3), and transmission of the agent from sheep living in proximity during summer may be the source of epidemics in chamois.

Prevention of IKC in wild Caprinae should focus on preventing the spillover of *M. conjunctivae* from livestock. However, studies are required to evaluate the distribution of *M. conjunctivae* infection in domestic sheep in several countries, and molecular markers should be developed to trace spill-over of

M. conjunctivae from domestic animals to wildlife populations. In addition, immunological studies should be performed to develop tools, which could lead to the control of *M. conjunctivae* infection in domestic sheep.

References

1. Belloy L, Giacometti M, Abdo El-M, Nicolet J, Krawinkler M, Janovsky M and Frey J. Detection of specific *Mycoplasma conjunctivae* antibodies in sera of sheep with infectious keratoconjunctivitis. *Vet Res* 2001; 32: 155-64.
2. Giacometti M, Janovsky M, Belloy L and Frey J. Infectious keratoconjunctivitis of ibex, chamois and other Caprinae. *Rev sci tech Off int Epiz*, in press.
3. Giacometti M, Janovsky M, Jenny H, Nicolet J, Belloy L, Goldschmidt-Clermont E and Frey J. *Mycoplasma conjunctivae* infection is not maintained in alpine chamois in eastern Switzerland. *J Wildl Dis*, in press.
4. Janovsky M, Frey J, Nicolet J, Belloy L, Goldschmidt-Clermont E and Giacometti M. *Mycoplasma conjunctivae* infection is self-maintained in the Swiss domestic sheep population. *Vet Microbiol* 2001; 83: 11-22.

MOLECULAR EPIDEMIOLOGY OF *Mycoplasma conjunctivae* IN CAPRINAE: TRANSMISSION ACROSS SPECIES IN NATURAL OUTBREAKS

L. BELLOY, M. JANOVSKY², E. M. VILEÍ, M. GIACOMETTI^{3,4} and J. FREY¹

Affiliation:

1. Institute for Veterinary Bacteriology, University of Berne, Länggassstrasse 122, CH-3012 Berne, Switzerland
2. Amt der Tiroler Landesregierung, Veterinaerdirektion, Wilhelm-Greilstr. 25, A-6020 Innsbruck, Austria
3. Wildvet Projects, CH-7605 Stampa, Switzerland
4. Author presenting the paper

Key words: Alpine chamois, DNA sequence determination, domestic sheep, European Alps, infectious keratoconjunctivitis, molecular epidemiology, *Mycoplasma conjunctivae*, PCR.

Extended abstract

Mycoplasma conjunctivae is the etiological agent of infectious keratoconjunctivitis (IKC), a highly contagious ocular infection that affects both domestic and wild Caprinae species in the European Alps (1). In order to study the transmission and spread of *M. conjunctivae* across domestic and wild Caprinae populations, we developed a molecular method for subtyping and identifying of strains of *M. conjunctivae* (2). This method is based on DNA sequence determination of a variable domain within the gene *lppS*, a gene that encodes an antigenic lipoprotein of *M. conjunctivae*. This domain remains constant upon passaging strains on growth medium. However the *lppS* domain shows variations among different strains and thus allows their identification and also the estimation of their phylogenetic intercorrelations. The variable domain of *lppS* is amplified by PCR using primers that match conserved sequences of *lppS* flanking it. Sequence analysis of the amplified fragment enables fine subtyping of *M. conjunctivae* strains. The method is applicable directly to clinical samples without requesting the cultivation of the strain.

Among the different isolates from sheep, goat, chamois and ibex from European Alpine regions, the typing method was able to distinguish 16 different strains. Of particular interest were four isolates from diseased chamois in the Salzach valley in Austria, which were shown to have the same *lppS* sequence. After diagnosis of *M. conjunctivae* in these chamois, eye swabs from a few sheep that were grazing in these pastures and that showed signs of potential *M. conjunctivae* infections were analysed. Among 3 positive samples, one isolate showed the same *lppS* sequence as the latter four isolates from chamois. A second situation where the same *M. conjunctivae* strain was found in chamois and in sheep grazing in the vicinity was discovered in the San Bernardino region, Switzerland. Thus, our study revealed that the same *M. conjunctivae* strains could be isolated from chamois with IKC and from sheep that were grazing on the same pastures showing that *M. conjunctivae* can be transmitted between domestic small ruminants and free-ranging wild Caprinae. This confirms previous speculations from sero-epidemiological surveillance of *M. conjunctivae* infections in sheep (3) and chamois (4). These studies showed the domestic sheep population to be a reservoir of *M. conjunctivae* from which alpine chamois, which do not maintain the infection themselves, were considered to be infected by mainly neighbouring sheep populations.

Two particular cases showed that sheep can get infected by two different strains simultaneously: two distinct isolates were found in the same sheep, one in the left eye and the other in the right eye, and two other isolates could be separated as two different strains but originated from the

same eye of a particular sheep. Finally the surprising finding that a female chamois and her two months old kid, both affected with IKC, carried two different strains of *M. conjunctivae*. In summary, we have developed a molecular method for subtyping individual strains of *M. conjunctivae* based on the variable segment of the gene *lppS*. This method allowed us to perform a molecular epidemiological study of *M. conjunctivae* in Alpine regions and to demonstrate the possibility of transmission of *M. conjunctivae* between domestic sheep and wild Caprinae.

Zusammenfassung

Zur Erforschung der interspezifischen Übertragung von *Mycoplasma conjunctivae*, dem Erreger der Gembblindheit, wurde eine molekularbiologische Methode zur Subtypisierung und Identifikation des Erregers entwickelt. Die Methode basiert auf der Sequenzierung eines variablen Teils des *lppS* Gens von *M. conjunctivae*. Mit dieser Methode war es möglich, die zwischenartliche Übertragung von *M. conjunctivae* zwischen Schafen und freilebenden Gamsen nachzuweisen.

Résumé

Afin d'étudier la transmission interspécifique de *Mycoplasma conjunctivae*, l'agent étiologique de la kératoconjonctivite infectieuse, nous avons développé une méthode moléculaire d'identification et de sous-typage des souches *M. conjunctivae*. Cette méthode est basée sur les séquences ADN du gène *lppS* propre à chaque sous-type. Cette méthode a permis de mettre en évidence la transmission naturelle de *M. conjunctivae* entre des moutons et des chamois dans l'environnement.

References

1. Giacometti M, Janovsky M, Belloy L and Frey J. Infectious keratoconjunctivitis of ibex, chamois and other Caprinae. Rev sci tech Off int Epiz, 2002, in press.
2. Belloy L, Janovsky M, Vilei E, Giacometti M, and Frey J. Molecular epidemiology of *Mycoplasma conjunctivae* in Caprinae: transmission across species in natural outbreaks. To be submitted to: Appl Environm Microbiol.
3. Janovsky M, Frey J, Nicolet J, Belloy L, Goldschmidt-Clermont E and Giacometti M. *Mycoplasma conjunctivae* infection is self-maintained in the Swiss domestic sheep population. Vet Microbiol 2001; 83: 11-22.
4. Giacometti M, Janovsky M, Jenny H, Nicolet J, Belloy L, Goldschmidt-Clermont E and Frey J. *Mycoplasma conjunctivae* infection is not maintained in alpine chamois in eastern Switzerland. J Wildl Dis, in press.

European Association of Zoo- and Wildlife Veterinarians (EAZWV) 4th scientific meeting,
joint with the annual meeting of the European Wildlife Disease Association (EWDA)
May 8-12, 2002, Heidelberg, Germany.

PATHOLOGY OF COMMON EIDERS FROM THE DUTCH WADDEN SEA IN WINTER 1999-2000

**TH. KUIKEN^{1,2}, TH. BESTEBROER¹, K. CAMPHUYSEN³, H. CREMERS⁴, M. EGGENS⁵,
R. FOUCHIER¹, J. WAGENAAR⁶ and A. OSTERHAUS^{1,2}**

Affiliation:

1. Institute of Virology, Erasmus MC, P.O. Box 1738, 3000 DR Rotterdam, The Netherlands
2. Seal Rehabilitation and Research Centre Pieterburen, Hoofdstraat 94a, 9968 Pieterburen, The Netherlands
3. Netherlands Institute for Sea Research, P.O. Box 59, 1790 AB Den Burg, Texel, The Netherlands
4. Dr H. Th. 's Jacoblaan 62, 3571 BN Utrecht, The Netherlands
5. Ministry of Traffic, Public Works and Water management, Directorate-General for Water Management, National Institute for Coastal and Marine Management/RIKZ, P.O. Box 207, 9750 AE Haren, The Netherlands
6. Veterinary Microbiological Diagnostic Centre, Utrecht University, P.O. Box 80165, 3500 TD Utrecht, The Netherlands

Abstract

In December 1999, there was unexplained high mortality of common eiders (*Somateria mollissima*) in the Dutch Wadden Sea. Gross necropsies, supplemented by histologic, virologic, bacteriologic, parasitologic, and toxicological analyses, were performed on 13 eiders. All birds were severely emaciated and had multifocal enteritis caused by infection with the acanthocephalan parasite *Profilicollis botulus*. The abundance of this parasite varied from 20 to 1833 (average: 865). The primary cause of death was considered to be starvation or *P. botulus* infection, although the latter was considered less likely. There was no evidence of other diseases, including those caused by viral or bacterial infections, or intoxication as the primary cause of mortality in these eiders. Based on analysis of weather and food resources, we hypothesise that the eider mortality was caused by a combination of factors, including overharvesting by the shellfish industry, a series of mild winters, and decreased shellfish quality. This pathological investigation was hampered by lack of historic data of body weights and *P. botulus* burdens of eiders in this part of their range. To rectify this, we recommend the establishment of a national wildlife health surveillance system. The activities of such a system could include (1) detection of disease or disease-causing agents in wildlife, including eiders; (2) diagnosis, or precise identification, of the diseases; (3) collection and analysis of the information gained from detection and diagnosis; and (4) use of surveillance information in making decisions and policies.

SEVERE FEATHER LOSS AND ABNORMALITIES („PINCHING OFF“) IN A JUVENILE FREE-LIVING WHITE-TAILED EAGLE (*Haliaeetus albicilla*) FROM NORTHERN GERMANY

**E. SCETTNER¹, K. MÜLLER², O. KRONE³, K. HATTERMANN⁴, D. SOIKE⁵,
R. JOHNE⁶, H. MÜLLER⁶, R. KOLLMANN⁷ and K. FRÖLICH³**

Affiliation:

1. Wildlife Park Eekholt, 24623 Großenaspe, Germany.
2. Klinik and Poliklinik for small animals, Free University of Berlin, veterinary faculty, Oerzenweg 19b, 14163 Berlin, Germany.
3. Institute for Zoo and Wildlife Research (IZW), P.O. Box 601103, 10252 Berlin, Germany.
4. Robert-Koch Institute, Nordurfer 20, 13353 Berlin, Germany.
5. Federal state institute for the protection of consumers and agriculture, Pappelallee 20, 14469 Potsdam, Germany.
6. Institute of Virology, veterinary faculty, University of Leipzig, An den Tierkliniken 29, 04103 Leipzig, Germany.
7. Project group for the protection of white-tailed eagles, Biology Building, University of Kiel, Olshausenerstr 40, 24118 Kiel, Germany.
8. corresponding author (e-mail: elvira_schettler@freenet.de)

Key words: *avian polyomavirus, biting lice, circovirus, endoscopy, electronmicroscopy, feather abnormalities, feather loss, Haliaeetus albicilla, PCR, pinching off syndrome, quill mites, white-tailed eagle*

Extended abstract

One of 28 free-living white-tailed eagles (*Haliaeetus albicilla*) that hatched in the federal state Schleswig-Holstein (northern Germany) in 2001 suddenly showed a severe symmetrical loss of flight feathers on both wings at the age of 10 weeks. However, loss of body contour feathers was not observed. No obvious preceded incidence could have explained these clinical signs which are described as „pinching off“ (1). The „Pinching off“ syndrome has been observed in several raptors (2), among them two free-living young white-tailed eagles from Schleswig-Holstein (3). However, a scientific explanation for this phenomenon is still missing. Trauma, pathological agents, hormonal disorders or genetical depression are discussed to be involved in the aetiology. In this study we focussed on pathological agents such as parasites and viruses. In the following the clinical signs are documented and preliminary results of several investigations are presented.

Being unable to fly, the white-tailed eagle described in this study was captured at the age of approximately 3 months and brought to the raptor rehabilitation centre at the Wildlife Park Eekholt. A thorough clinical examination including x-ray was performed. Except for the feather loss and abnormalities in both wing- and tail feathers no clinical signs could be diagnosed. However, numerous biting lice of the genus *Colpocephalum* were found especially on the wings. The bird was successfully treated with 0,01% Heptenofos (Ragadan®) solution. A possible correlation between the biting lice and the feather abnormalities was excluded because the clinical signs were still observed after this treatment.

Nearly every second primary feather was missing on both wings. The remaining primaries as well as the secondaries and also the tail feathers showed significant changes in their structure. In most of these feathers not only the calamus and rachis was deformed but also the typical pennaceous character of the vane was missing. Moreover, the feathers were easily bendable and the rachis often showed longitudinal splitting. Lost primaries were permanently replaced by new abnormal

feathers which are also moulted after a while. The abnormal secondaries have not yet been moulted.

Biopsy feather material and blood samples were taken for parasitological and virological investigations.

A small number of quill mites were detected by histological examination of the pulp. Consequently, the bird was treated with 0.2 mg/kg Ivermectin 5 times in two week intervals, and after a 6 weeks break, three times with 0.4 mg/kg Ivermectin in weekly intervals. However, this treatment did not improve the feathering conditions. Therefore, the mites are presumably not the causal agents of the „pinching off“ syndrome.

Blood chemistry showed no abnormalities except for a rise of lactate dehydrogenase (LDH). However, an endoscopic investigation did not give evidence for any pathological process.

Furthermore, no evidence for the presence of viruses like avian polyomavirus (APV) or circovirus was found by preliminary electronmicroscopical examinations. Neither could APV be detected in feather biopsy material or in blood by polymerase chain reaction (PCR). Blood and feather material is currently tested for circovirus using PCR and sequence analysis.

In conclusion, clinical signs of severe feather loss and abnormalities („pinching off“) of yet unknown aetiology in a free-living white-tailed eagle are presented and preliminary results will be discussed.

References

1. Cooper JE. Veterinary aspects of birds of prey. The Stanfast Press, Gloucestershire 1985; 158.
2. Grünhagen H. Federanomalien bei Greifvögeln. In: Deutscher Falknerorden (Ed) Greifvögel und Falknerei. Neumann Neudamm 1989; 73-6.
3. Robitzky U, Artenhilfsprogramm für den Seeadler *Haliaeetus albicilla* in Schleswig-Holstein. In: Møylburg BU and RD Chancellor (Eds.) Eagle studies. World Working Group on Birds of Prey, Berlin, London & Paris 1996; 73-95.

European Association of Zoo- and Wildlife Veterinarians (EAZWV) 4th scientific meeting,
joint with the annual meeting of the European Wildlife Disease Association (EWDA)
May 8-12, 2002, Heidelberg, Germany.

POSTMORTEM FINDINGS IN WILD GREAT BUSTARDS (*Otis tarda*) FROM SPAIN: A CLINICAL APPROACH

**M. GARCÍA-MONTIJANO¹, A. M. TÉBAR², B. BARREIRO³, P. RODRÍGUEZ⁴,
J.C. ALONSO⁵, A. MONTESINOS⁶ and I. LUACES⁷**

Affiliation:

1. Centro de Cría del Águila Imperial Ibérica. Ministerio de Medio Ambiente. Quintos de Mora. Toledo. Spain. (mmontijano@eresmas.com)
2. Centro de Recuperación y Educación Ambiental "Los Hornos". Junta de Extremadura. Sierra de Fuentes, Cáceres. Spain.
3. GIR diagnostics. Madrid. Spain.
4. C/ Abades 18 2º B. (41004). Sevilla. Spain.
5. Museo Nacional de Ciencias Naturales. CSIC. Madrid. Spain.
6. Clínica Los Sauces. C/Murillo, 3. Madrid. Spain.
7. INGENASA. C/Hnos. Gª Noblejas, 41. Madrid. Spain.

Abstract

Causes of death and morbidity are reported for 13 free-living great bustards (*Otis tarda*) from Spain. The main mortality cause for the adult birds and two juveniles was collision with power lines or fences. One wild adult bustard died of *Aspergillus fumigatus* generalised infection. Other causes of death for juveniles were predation, septicaemia, parasitic obstruction of small intestine by cestodes, ventriculus impaction, and trauma with agricultural vehicles. Causes of morbidity were skin injuries, fractures, soft tissue and liver trauma, pneumonia of different aetiology, ectoparasites and haemoparasites.

Résumé

Les causes de mortalité sont rapportées pour 13 outardes barbues (*Otis tarda*) sauvages en Espagne. La principale cause de mortalité pour les adultes et les 2 juvéniles est la collision avec des lignes à haute tension ou des barrières. Un des adultes est mort d'Aspergillose généralisée à *Aspergillus fumigatus*. Les autres causes de mortalité chez les juvéniles sont la prédation, les septicémies, l'obstruction du grêle par des cestodes, impaction du ventricule, et traumatismes dû à des engins agricoles. Les causes de morbidité sont les blessures cutanées, les fractures, les traumatismes des tissus mous et du foie, les pneumonie d'origine variée, les ectoparasites et les hémoparasites.

Key words: Great bustard, *Otis tarda*, mortality, morbidity, post-mortem.

Introduction

There are 22 species of bustards (family Otididae), some of them are endangered species. The great bustard (*Otis tarda*) is a large, highly sexually dimorphic, globally endangered bird. Their numbers have declined considerably during the present century and current populations inhabit cereal steppes of Europe and Asia (2). This decline has been attributed to habitat changes caused by human population growth, farming practices, changes in agricultural practices and hunting pressure (1).

This manuscript is reproduced in the IVIS website with the permission of EAZWV www.eazwv.org

The Iberian population is the largest, being the size of the Spanish population at around 17.000-19.000 birds. Most of the Spanish great bustard nuclei seem to be stable perhaps with a very slight tendency to increase in some particularly well conserved areas (1).

Other bustard species (*Chlamydotis undulata*, *Ardeotis kori*, and *Eupodotis ruficrista*) have attracted much attention from the veterinary profession, especially in the Middle East, where a good amount of information about management and clinical aspects of bustards exists (3-7,9,12,17).

Most of the references regarding great bustards are based on ecological studies, and there are some published papers on captive great bustard diseases (13-15) but the authors could only find three references on veterinary aspects (parasites) of free-living great bustards (10,11,16), none of them being published in an international journal.

Clinical management of great bustards is not an easy task due to the strength, weight, present injuries and stressful nature of these birds. The knowledge of diseases affecting free-living great bustards could help in the daily treatment and follow up of clinical cases. The aim of this retrospective study is to provide an overview of morbidity and mortality causes in the wild Spanish great bustard population.

Material and method

We included for this retrospective study 13 post-mortem cases of free-living great bustards (6 adults and 7 juveniles) that were necropsied in the period 1998-2001. Five of the 6 adults and 3 of the 7 juveniles were males. In one juvenile we could not determine the sex. Included in this paper are only free-living birds that were found dead or died within 24 hours in the rehabilitation centre.

Standard avian post-mortem examination techniques were used (8), including two X-ray projections, samples for histopathology, parasitology, and microbiology when appropriate and biometry. The condition of each bird was recorded as emaciated, poor, fair, good or obese, based on the degree of pectoral muscle wasting. Birds with emaciated and poor pectoral muscle scores were included in the pectoral muscle wasting group (4). For histopathology special stains were used when *Chlamydothila* sp. or tuberculosis were part of the differential diagnosis.

Cause of death was determined from consideration of the clinical history, clinical observations, laboratory findings, and significant post-mortem findings.

Endoparasites were washed in distilled water and preserved in 70% ethanol until processed for identification. When possible a blood smear was prepared to search for haematzoa and faeces were screened by flotation method with zinc sulphate. Arthropods collected for identification were fixed and stored in 70% ethanol.

Results

Causes of death in wild great bustards are summarised in table 1.

Trauma through collisions with electric power lines or fences were responsible for 83.3% of adult and 42.9% of juvenile deaths in wild great bustards. Aspergillosis accounted for 16.7% of adult mortality. Other mortality causes of juvenile birds were predation (14.3%), septicaemia (14.3%), gizzard impaction (14.3%) and obstruction of small intestine with cestodes (14.3%).

Other post-mortem findings in the present study are summarised in table 2.

All the adult bustards (100%) and 57.1% of juveniles presented intestinal parasitism by cestodes. These cestodes were identified as *Otiditaenia conoides* and *Idiogenes otidis*. The nematodes present in the caecum of one adult and one juvenile bird were identified as *Heterakis isolonche*.

Table 1. Causes of death in 7 juvenile and 6 adult free-living great bustards.

Cause of death	Adult	Juvenile	Total
Trauma-electric transmission power lines, fences	5	2	7
Trauma-vehicle	0	1	1
Aspergillosis	1	0	1
Predation	0	1	1
Parasitic obstruction of small intestine by cestodes	0	1	1
Septicaemia	0	1	1
Gizzard impaction	0	1	1
Total	6	7	13

Table 2. Post-mortem findings in 7 juvenile and 6 adult free-living great bustards.

Post-mortem finding	Adult	Juvenile	Total
Intestinal parasitism-cestodes	6	4	10
Trauma-skin	5	4	9
Fractures	4	3	7
humerus	1	1	2
radius	1	0	1
ischium	1	0	1
femur	0	1	1
tibiotarsus	0	1	1
pubic bone	0	1	1
scleral ring	1	0	1
skull	1	0	1
Pneumonia	4	3	7
bacteria	0	2	2
fungus	1	0	1
aspiration	3	1	4
Pectoral muscle wasting	2	4	6
Haemoparasites	2	2	4
Ectoparasites	2	1	3
Trauma-liver	2	1	3
Intestinal parasitism-nematodes	1	1	2
Sternal bone deformity	1	0	1

Also in the faeces of that juvenile *Capillaria* sp. and *Trichostrongylus* sp. eggs were detected, but no adults were recovered.

Skin injuries were a morbidity cause in 83.3% of adult and 57.1% of juvenile great bustards. Bone fractures occurred in 66.7% of adult birds and 42.9% of juveniles. Open fractures of the humerus were the most common fractures.

This manuscript is reproduced in the IVIS website with the permission of EAZWV www.eazwv.org

Pneumonia was observed in 66.7% of adult and 42.9% of juvenile birds. Foreign body inhalation pneumonia was recorded for 50% of adult bustards and 14.3% of juvenile birds. Pneumonia of bacterial origin (*Pasteurella* sp.) was found in 28.6% of juvenile bustards, and *Aspergillus fumigatus* pneumonia and air sacculitis was responsible for the death of one adult (16.7%) great bustard.

Haemoparasites of the specie *Haemoproteus telfordi* and *Haemoproteus tendeiroi* were seen in blood films from 2 adult and 2 juvenile great bustards. However it was not possible to make a blood smear from every bird.

Ectoparasites were found in 33.3% of adults and 14.3% of juveniles. *Qtilipeurus turmalis* (Mallophaga, Insecta) and a tick from the genus *Hyalomma* were identified.

Discussion and conclusions

Mortality causes of free-living juvenile great bustards from Spain have been published previously (2), although it was not the objective of the study. Predation was the main mortality cause in juveniles and collision with power lines was found in two occasions. In this study only one young was found to be predated by a raptor, while other was hit by an agricultural vehicle. The later cause has been reported as common in bustard's chicks as they look after cover in crop fields (18).

In a previous work in Madrid province (18) the authors found that collision with power lines was responsible of the death of 30 wild great bustards in the period 1999-2000. Trauma trough collisions with electric power lines or fences accounted for 63.1% mortality in wild great bustards in the present study. Bone fractures and dislocations, skin and soft tissue injuries, liver rupture, aspiration pneumonia and lung haemorrhage were the most frequent causes of morbidity in such cases. When such cases are admitted to rehabilitation a reasonable valuation and decisions must be done before treatment is accomplished.

A frequent post-mortem finding was a high parasitic burden caused by cestodes (*Otiditaenia conoides* and *Idiogenes otidis*). These species of cestodes have been previously reported in great bustards (wild and captive) in Spain (10,16). They also report the death of one bustard following obstruction of the small intestine by cestodes. This condition has been also described in captive houbara bustards in UAE (4,12). In our study the prevalence of cestode infections in bustards was higher than the previously reported by Reina et al. (11.4%) in Spain and Jones et al. (25.6%) in the UAE. This could be due to many different causes as our small sample size and the different origin of birds. Great bustards admitted to rehabilitation centres are usually weakened, dehydrated, traumatised, and in poor body condition (authors, unpublished data), conditions that may increase the susceptibility to the pathologic effects of cestode infections (12). The clinician must consider these observations when dealing with this specie. Nematodes were seen in low numbers in the caecum of 2 bustards.

Gizzard impaction and foreign-body obstruction have been reported as an important cause of death of captive juvenile rufous-crested bustards and houbara bustard chicks (4,5). One juvenile bird of our study died after a gizzard impaction of unknown aetiology.

Gram-negative bacterial diseases were the most important cause of death over the first 180 days of captive bustard chicks in UAE (5). In our study one week old chick died of septicaemia where *E. coli* was culture from different organs. The isolation of *E. coli* from bustards at necropsy was found on many occasions in one study involving captive houbara, rufous-crested and kori bustards (9).

After collision with fences or power lines most of the birds could live for sometime and also walk away for many metres before die (authors, personal observation). Aspiration pneumonia was found in 50% of the adult birds. This was the result of aspiration of food (normally seeds) from the ventriculus after the collision.

Aspergillosis has been described as a common cause of euthanasia and post-mortem finding in captive and imported adult houbara bustards and also caused mortality in juvenile kori and houbara bustards (4). One adult female died four hours after presentation prostrated and

with obvious signs of dyspnea. At necropsy fungal granulomas were seen in trachea, syringe, lungs, pericardium, air sacs, kidney and pelvic nerve roots. A cream-coloured ovoid plaque (7 cm minimum diameter, 11 cm maximum diameter) was recovered surrounding the abdominal viscera. A pure culture of *Aspergillus fumigatus* was obtained from the granulomas. These birds also presented signs of external parasitism, but only a tick from the genus *Hyalomma* was recovered. A high cestode burden was another incidental finding in this bird. To the authors knowledge this is the first report of aspergillosis in a wild adult great bustard.

Klebsiella sp. pneumonia and pneumonia of unknown aetiology have been described in captive bustards from the UAE (4). Bacterial pneumonia has been also a morbidity cause (28.6%) in free-living juvenile great bustards. In one occasion *Pasteurella* sp. was cultivated from the lungs of a juvenile bird. Other young great bustard had a focal bacterial pneumonia and liver lipidosis based on histopathology. Unfortunately no microbiology results are available for this case. Some authors have stated that aggressive care of bustards during the first 30 days after hatching is clearly important (5).

Haemoparasites from the genus *Haemoproteus* were detected in 4 of the bustards, but it was not possible to obtain a blood film for each bird. Studies of wild great bustards haemoparasites species and prevalence are being carried out by the authors since 1998 in the Spanish population and will be reported in the future.

Management of wild great bustards clinical cases poses a challenge to the veterinarian. The results presented in this paper could help understanding the morbidity causes when attending such patients.

Acknowledgements

Javier Caldera for providing logistic support and Gerry Dorrestein for reviewing some histopathology slides.

References

1. Alonso JC and Alonso JA. The Great Bustard in Spain: Present Status, Recent Trends and Evaluation of Earlier Censuses. *Biological Conservation* 1996; 77: 79-86.
2. Alonso JC, Martín E, Alonso JA and Morales MB. Proximate and ultimate causes of natal dispersal in the great bustard *Otis tarda* *Behavioral Ecology* 1997; 9: 243-52.
3. Bailey TA, Naldo J, Samour JH, and Howlett JC. Bustard Therapeutics. ERWDA External Report No5. 1997.
4. Bailey TA, Naldo J, Samour JH, and Howlett JC. Post-mortem Findings in Bustards in the United Emirates. *Avian Diseases* 1996; 40: 296-305.
5. Bailey TA, Naldo J, Samour JH, Sleight IM, and Howlett JC. Bustard pediatric diseases: a review of clinical and pathologic findings. *Journal of Avian Medicine and Surgery* 1997; 11: 166-74.
6. Bailey TA, Nicholls PK, Wernery U, Samour JH, Cooper JE, Path C, and O'Leary MT. Avian paramyxovirus type 1 infection in Houbara bustards (*Chlamydotis undulata macqueenii*): clinical and pathological findings. *Journal of Zoo and Wildlife Medicine* 1997; 28: 325-30.
7. Bailey TA, Silvanose CD, Naldo J, and Howlett JH. *Pseudomonas aeruginosa* infections in Kori bustards (*Ardeotis kori*). *Avian Pathology* 2000; 29: 41-4.
8. Cooper JE. Disease and Threatened Birds. ICBP Technical Publications 1989.
9. Dáloia MA, Bailey TA, Samour JH, Naldo J, and Howlett JC. Bacterial flora of captive houbara (*Chlamydotis undulata*), Kori (*Ardeotis kori*) and rufous-crested (*Eupodotis ruficrista*) bustards. *Avian Pathology* 1996;25:459-68.
10. Illescas-Gómez MP, and Gómez MP. First citation in Spain of *Schistometra conoides* (Bloch 1782) Skrjabin 1914, parasitizing *Otis tarda* L. *Proceedings of the third European Multicolloquium of Parasitology*, Cambridge UK 1980
11. Illescas-Gómez MP, and Gomez MP. Idiogenous otitis, intestinal parasite of *Otis tarda*. *Revista Ibérica de Parasitología* 1981;41 (4):475-84.
12. Jones A, Bailey TA, Nicholls PK, Samour JH, and Naldo J. Cestode and acanthocephalan infections in captive bustards: New host and location records, with data on pathology, control, and preventive medicine. *Journal of Zoo and Wildlife Medicine* 1996;27:201-8.
13. Körmendy B, Illes J, Glavits R, and Sztojkov V. An outbreak of *Yersinia pseudotuberculosis* infection in a bustard (*Otis tarda*) flock. *Acta Veterinaria Hungarica* 1988;36:173-6.

14. Körmendy P, Sztojkov V, Ivanics E, Illes J, and Csatori G. Pseudotuberculosis in a Great bustard population and comparison with the changes after an experimental tuberculosis infection. *Verh ber Erkr Zootiere* 1982;24:299-302.
15. Kozakiewicz, B. Infection with endoparasites of bustards (*Otis tarda*) under breeding conditions. *Wiadomosci Parazytologiczne* 1984;30:489-92.
16. Reina D, Habela M, Serrano F, Nieto CG, Brena M, Pérez E, Navarrete I, Hernandez-Rodriguez S. Contribución al conocimiento de la parasitofauna de los animales silvestres y de vida libre en la provincia de Cáceres (España). In *Memoriam al Prof. Dr. Francisco de Paula Martínez Gómez, Universidad de Córdoba* 1992;407-28.
17. Sleigh I and Samour JH. *Bird care manual: Management techniques for a collection of bustards*. NARC 1996.
18. Traverso JM. Agricultura y tendidos afectan a las avutardas del este de Madrid. *Quercus* 2001; diciembre:62-3.

European Association of Zoo- and Wildlife Veterinarians (EAZWV) 4th scientific meeting,
joint with the annual meeting of the European Wildlife Disease Association (EWDA)
May 8-12, 2002, Heidelberg, Germany.

THE LIVER FLUKE *Metorchis bilis* – A NEW THREAT FOR THE WHITE-TAILED SEA EAGLE (*Haliaeetus albicilla*) IN MIDDLE EUROPE?

O. KRONE¹ and R. SCHUSTER²

Affiliation:

1. Institute for Zoo- and Wildlife Research, P.O. 601103, 10252 Berlin, Germany
2. Free University Berlin, Institute for Parasitology and International Animal Health, Königsweg 67, 14163 Berlin, Germany

Key words: white-tailed sea eagle, *Haliaeetus albicilla*, causes of death, trematode, liver fluke, *Metorchis bilis*, life cycle

Extended abstract:

The white-tailed sea eagle (*Haliaeetus albicilla*) an endangered species in Germany suffers a lot of threats in the highly civilised landscape. Amongst the main causes of death such as traumata due to interference with human structures, i.e. collisions with trains, wire, electrocution, and poisoning, parasites do also play an important role in the health status of these birds (1). Liver flukes belonging to the genus *Metorchis*, diagnosed at a prevalence of 51% (n=112) in the eagles, were thought to be responsible for frequently detected alterations of the liver bile duct system.

To identify flukes at species level and to understand the biology of this trematode the life cycle was established in the laboratory. As known from the genus *Metorchis* (2) the life cycle includes two intermediate hosts: a water snail as first and a cyprinid fish as second intermediate host. Parallel histological and toxicological examinations were performed to reveal the liver alterations and the cause of death in the eagles.

During routine post mortem examination of a white-tailed sea eagle, which recently died, adult specimen of *Metorchis* sp. was collected from the gall bladder and the bile ducts. Following cleaning the still living trematodes were put into tap water and stored in a refrigerator (5-7°C) for two days where the parasites excreted fully developed eggs. These eggs were fed to *Bithynia tentaculata* (Prosobranchia) in which the miracidium hatches to undergo an asexual multiplication. Within 5 weeks p.i. rediae were found in the hepato-pancreas and two weeks later cercaria started to leave the snail in search for the next intermediate host. The excretion of cercariae could be enhanced by exposing the water basin with the snails under a lamp. The pleurolopho cercariae with a membranous tail reacted positively photo- and chemotactic which helped them to find their next host the fish. Ids (*Leuciscus idus*) were used as second intermediate host. After putting snails into fish basins, cercariae penetrated the skin of the fish to become encysted metacercariae in the fins, muscle or even in the cornea. A reduced visual faculty may result in a higher possibility to fall victim of the definitive host, where the parasite matures. The shape and measurements of the metacercaria revealed the species *Metorchis bilis*. Regardless the high prevalence and intensities (up to 908) only two sea eagles died due to infections with this liver fluke. Histological examinations showed hypertropia of the epithelium cells of the bile duct walls, partial or total obstruction of the bile ducts resulting in thickening of the bile and liver swelling. Very similar organ alterations consisting of stasis of the bile in the gall bladder and the bile ducts and liver swelling could be attributed to high lead levels in the liver (3), which was also often the cause of death (17%, n=112) in these birds. Other eagles which died due to traumata, e.g. territorial fight (n=10) showed liver alterations in four cases due to an infection with *Metorchis bilis* which make them more likely to be the loser.

References

1. Krone O, Langgemach TP, Sömmer P and Kenntner N. Causes of mortality in white-tailed sea eagles from Germany. Proceedings of the Conference Sea eagle 2000 (in press), Björko, Sweden.
2. Raschmaschkin DA. On the species position of metacercariae of the genus *Metorchis* (Trematoda, Opistorchidae) from fishes of west Siberia (in russian). Parasitologija 1978; 12: 68-78.
3. Kenntner N, Tataruch F and Krone O. Heavy metals in soft tissue of white-tailed eagles found dead or moribund in Germany and Austria from 1993 –2000. Environmental Toxicology and Chemistry 2001; 20: 1831-1837.

European Association of Zoo- and Wildlife Veterinarians (EAZVV) 4th scientific meeting,
joint with the annual meeting of the European Wildlife Disease Association (EWDA)
May 8-12, 2002, Heidelberg, Germany.

ENDOPARASITES OF RED DEER (*Cervus elaphus* L.) OF NORTHRHINE-WESTFALIA (GERMANY)

S. REHBEIN¹, M. VISSER¹, R. WINTER¹ and W. LUTZ²

Affiliation:

1. Merial GmbH, Kathrinenhof Research Center, Walchenseestr. 8-12, D-83101 Rohrdorf.
2. LÖBF NRW, Dez. Forschungsstelle für Jagdkunde und Wildschadenverhütung, Pützchens Chaussee 228, D-53229 Bonn.

Key words: Red deer, Germany, Northrhine-Westfalia, endoparasites, helminths

Abstract

The endoparasite fauna of 85 red deer (37 calves <1 year, 18 ~1 year, 30 >1 year) - 76 complete gastrointestinal tracts, 77 livers, 83 lungs - which were shot during the hunting seasons 1998/99, 1999/2000 and 2000/01 was examined. The red deer originated from four areas from the south of Northrhine-Westfalia. Two of the areas are located close to the German-Belgian border, the other two areas are south of Cologne.

In all, 2 cestode species (*Moniezia benedeni* and *M. expansa*) and 24 species of nematodes (21 species of gastrointestinal nematodes, two lungworm species and *Setaria cervi* in the abdominal cavity) were recorded. All deer harboured gastrointestinal nematodes, 80.7% *Dictyocaulus eckerti* (1-162 lungworms) and 12% *Varestrongylus sagittatus*. *Elaphostrongylus* larvae were found in 47% of the faecal samples of the deer.

The prevalence of nematodes in the abomasum, small intestine and large intestine was 100%, 65.8% or 97.4%, respectively. The most prevalent species were *Spiculopteragia böhmi* (100%), *Oesophagostomum venulosum* (86.8%), *Ostertagia leptospicularis* (83.5%), *Oesophagostomum sikae* (81.6%), *Skryabinagia kolchida* (72.2%), *Cooperia pectinata* and *Capillaria bovis* (43.4% each), *Nematodirus roscidus* (35.5%) and *Rinadia mathevossiani* (31.6%). *Apteragia quadrispiculata*, *Hyostromylus rubidus* and *Nematodirus battus* were recorded for the first time as parasites of German red deer. The abomasum (adults + mucosal fourth-stage larvae) harboured the highest worm burden, followed by small intestine and large intestine. The geometric mean gastrointestinal nematode counts of these sections were 863, 14 or 13 nematodes, respectively. Calves harboured 735, approximately one year old animals 2041 and the animals older than 1 year 1032 gastrointestinal nematodes (geometric means). Worm counts of abomasum and large intestine were highest in the approx. one year old animals, small intestinal worm counts and *Dictyocaulus* lungworm counts decreased with higher age. Neither liver flukes nor rumen flukes were seen.

Zusammenfassung

Die Endoparasitenfauna von 85 Stücken Rotwild (37 Kälber <1 Jahr, 18 ~1 Jahr, 30 >1 Jahr) - 76 vollständige Verdauungskanäle, 77 Lebern, 83 Lungen - aus den Jagdjahren 1998/99, 1999/2000 und 2000/01 aus 4 Herkunftsgebieten im südlichen Nordrhein-Westfalen wurde untersucht. Zwei der Herkunftsgebiete sind an der deutsch-belgischen Grenze gelegen, die anderen beiden südlich von Köln.

Dabei wurden 2 Zestodenarten (*Moniezia benedeni* und *M. expansa*) und 24 Nematodenarten nachgewiesen: 21 Arten im Magen-Darm-Kanal, 2 in der Lunge und eine Spezies in der Bauchhöhle (*Setaria cervi*). Alle Tiere waren

mit Magen-Darm-Nematoden befallen, 80,7% mit 1 bis 168 großen Lungenwürmern (*Dictyocaulus eckerti*) und 12% mit kleinen Lungenwürmern, *Varestrongylus sagittatus*. In 47% der Enddarmkotproben wurden *Elaphostrongylus*-Larven festgestellt.

Alle Rothirsche waren mit Labmagen-nematoden infiziert, 65,8% mit im Dünndarm und 97,4% mit im Dickdarm parasitierenden Nematoden. Die am häufigsten gefundenen Nematodenarten im Verdauungskanal waren *Spiculoptera* *böhmi* (100%), *Oesophagostomum venulosum* (86,8%), *Ostertagia leptospicularis* (83,5%), *Oesophagostomum sika* (81,6%), *Skrjabinagia kolchida* (72,2%), *Cooperia pectinata* und *Capillaria bovis* (jeweils 43,4%), *Nematodirus roscidus* (35,5%) und *Rinadia mathevossiani* (31,6%). Erstmals als Parasiten des einheimischen Rotwildes wurden nachgewiesen *Apteragia quadrispiculata*, *Hyostrongylus rubidus* und *Nematodirus battus*. Der Labmagen (adulte Nematoden + histotrope Larven 4) war der am stärksten parasitierte Abschnitt des Verdauungskanals, gefolgt von Dünndarm und Dickdarm, die nahezu gleichstarke Wurmbürden aufwiesen (geometrisches Mittel der Befallsintensität: 863, 14 bzw. 13 Nematoden). Kälber beherbergten durchschnittlich (geometrisches Mittel) 735, die etwa einjährigen Tiere 2041 und die >1 Jahr alten Tiere 1032 Magen-Darm-Nematoden. Die Parasitierung von Labmagen und Dickdarm war am stärksten bei den etwa einjährigen Stücken; der Wurmbefall des Dünndarms sowie der mit großen Lungenwürmern ging mit zunehmendem Alter zurück. Leberegel und Pansenegel waren nicht nachweisbar.

Résumé

La faune endoparasitaire de 85 cerfs (37 <1 an, 18 de ~1 an, 30 >1 an) - 76 tubes digestifs complets, 77 foies, 83 poumons - recueillis à la faveur des saisons de chasse 1998/99, 1999/2000 et 2000/01 et provenant de 4 territoires du Sud de la Rhénanie-Westphalie a été étudiée. Deux du territoires sont à la frontière Allemagne-Belgique, les autres deux sont au Sud du Cologne.

A cette occasion, deux espèces de Cestode (*Moniezia benedeni* et *M. expansa*) et 24 espèces de nématodes ont été identifiées: 21 dans le conduit gastro-intestinaux, 2 dans les poumons et 1 espèce dans la cavité abdominale (*Setaria cervi*). Tous les animaux étaient porteurs de parasites gastro-intestinaux, 80,7% avec 1 à 168 de vers pulmonaires grands (*Dictyocaulus eckerti*) et 12% avec vers pulmonaires petits, *Varestrongylus sagittatus*. Dans 47% des prélèvements de fèces d'intestin terminal *Elaphostrongylus*-larves ont été identifiés.

Tous les cerfs étaient contaminés par des nématodes de la caillette, 65,8% et 97,4% étant respectivement contaminés par des nématodes de l'intestin grêle et du gros intestin. Les espèces de nématodes les plus fréquentes dans le tube digestif étaient *Spiculoptera* *böhmi* (100%), *Oesophagostomum venulosum* (86,8%), *Ostertagia leptospicularis* (83,5%), *Oesophagostomum sika* (81,6%), *Skrjabinagia kolchida* (72,2%), *Cooperia pectinata* et *Capillaria bovis* (chaque fois 43,4%), *Nematodirus roscidus* (35,5%) et *Rinadia mathevossiani* (31,6%). Pour la première fois les parasites suivants ont été identifiés chez le cerf indigène: *Apteragia quadrispiculata*, *Hyostrongylus rubidus* et *Nematodirus battus*. La caillette (nématodes adultes + larves muqueux 4) était la section la plus parasitée du système digestif, suivie de l'intestin grêle et du gros intestin, que présentes la charge parasitaire presque similaire (moyenne géométrique de la contamination de respectivement 863, 14 et 13 nématodes). Les faons hébergeaient en moyenne (moyenne géométrique) 735, les sujets d'un an 2041 et les sujets de plus d'un an 1032 nématodes gastro-intestinaux. La parasitose de la caillette et du gros intestin était la plus forte chez les pièces autour d'un an, la parasitose de l'intestin grêle aussi qu'avec les vers pulmonaires grands diminue avec l'âge des porteurs. Des sujets de douves hépatiques et de sangsues stomacales n'ont pu être vérifiées.

European Association of Zoo- and Wildlife Veterinarians (EAZWV) 4th scientific meeting,
joint with the annual meeting of the European Wildlife Disease Association (EWDA)
May 8-12, 2002, Heidelberg, Germany.

THE ANNUAL SEROLOGICAL SURVEY IN WILD BOAR POPULATIONS IN FRANCE 2000-2001 REPORT

**J. HARS¹, F. BOUE², P. BOIREAU³, B. GARIN-BASTUJI⁴, M.F. LE POTIER⁵,
A. MESPLEDE⁵, X. PACHOLEK⁶ and B. TOMA⁷**

Affiliation :

1. Office national de la chasse et de la faune sauvage. Unité suivi sanitaire de la faune. 8 Impasse Champ Fila F-38320 Eybens.
2. AFSSA Nancy, Unité SGFS, Domaine de Pixérécourt, BP 9, F-54220 Malzéville
3. AFSSA Alfort, Unité BIPAR, BP 67, F-94703, Maisons-Alfort Cedex
4. AFSSA Alfort, Unité zoonoses bactériennes, OIE/FAO Laboratoire de référence pour la brucellose, BP 67, F-94703 Maisons-Alfort Cedex.
5. AFSSA Ploufragan, Unité Virologie Immunologie Porcines , BP 53, F-22440 Ploufragan
6. MAP – DGAI. Bureau santé animale. 251 rue de Vaugirard 75732 Paris cedex 15
7. ENVA, Laboratoire national de référence pour la maladie d'Aujeszky, 7, Avenue du Général de Gaulle, F-94704 Maisons -Alfort Cedex

Key words: aujeszky's disease, classical swine fever, brucellosis, trichinellosis, wild boar, *Sus scrofa*, epidemiology, France

Abstract

The French Ministry of Agriculture (MAP) and the National Hunting and Wildlife Agency (ONCFS) have been concerned increasingly with the risk that wildlife could constitute to the health of reared animals and man in France.

For three reasons, the wild boar has become a subject of special attention:

- ◆ the population figures have been highly increasing for the past 25 years, particularly for the last 10 years (x 350 %).
- ◆ wild boars are or could be a reservoir of several infectious or parasitic diseases, which are very important from an economic or public-health point of view because they are transmissible to the pig (which belongs to the same *Sus scrofa* species), to other domestic species and man.
- ◆ the development of open-air pig breeding increases the chances of contact with wild boars, and consequently the risks of transmission of pathogenic agents.

The MAP launched a national programme for the serological surveillance of the wild boar population in 1991. It was based on the blood sampling of hunter-killed boars, supervised in each French *département* (sub-regional division, 95 of them in France) by the official Veterinary Services in collaboration with the local hunters associations. The respective national reference laboratories in the French Agency for Food Safety (AFSSA) and the National Veterinary School in Alfort carried out the analyses. .

Until 1999, sera were only analysed for Classical Swine Fever and Aujeszky Disease antibody detection. An average of 1000 sera per year has been collected in 60 *départements* (i.e. 10 to 20 analysed sera per *département*). Since the 2000-2001 hunting season, the programme's protocol has been revised by the ONCFS and the MAP so that it be better adapted to the epidemiological situation, but also to increase the reliability and precision of the results. On one hand, the surveillance part was extended to include two other diseases: brucellosis and trichinellosis. On the other hand, the sampling procedure was thoroughly changed (objective: 100 sera per *département* in the 22 *départements* selected according to epidemiological criteria related to the 4 diseases, as

observed in previous surveys in wild or domestic swine populations). In 2000-2001, the objective had exceeded our expectations since 2,548 sera were collected.

The programme's results are the following:

- ◆ for classical swine fever, no spread of the virus has been revealed among the wild boar population in France, except for the Northern Vosges former outbreak (in the Moselle and Bas-Rhin *départements*) which, in 2001, is in the process of natural extinction;
- ◆ for Aujeszky's disease, the virus is carried by wild boars in several *départements*, with variable infection rates;
- ◆ for brucellosis, the *Brucella suis* 2 infection appears extremely widespread, with very high prevalence rates (mean seroprevalence rate = 29 %);
- ◆ for trichinellosis, several seroprevalences which may amount to 16% have been detected in certain *départements*. This, undoubtedly, reveals an underestimation of the presence of *Trichinella*, which, according to the official trichinoscopic verifications, are only exceptionally shown in wild boar

In order to follow the evolution of the incidence and the geographical distribution of the four diseases in France, it is planned to repeat the sero-surveys in the course of next hunting seasons.