Medical and Veterinary Entomology

Medical and Veterinary Entomology (2014) **28** (Suppl. 1), 98–103

Genetic epidemiology and pathology of raccoon-derived *Sarcoptes* mites from urban areas of Germany

Z. RENTERÍA-SOLÍS[1], A. M. MIN[2], S. ALASAAD[3,4], K. MÜLLER[5], F.-U. MICHLER[6], R. SCHMÄSCHKE[7], U. WITTSTATT[8], L. ROSSI[2] and G. WIBBELT[1]

[1]Department of Wildlife Diseases, Leibniz Institute for Zoo and Wildlife Research, Berlin, Germany, [2]Department of Animal Production, Epidemiology and Ecology, University of Turin, Grugliasco, Italy, [3]Doñana Biological Station, Spanish National Research Council (Consejo Superior de Investigaciones Científicas), Seville, Spain, [4]Institute of Evolutionary Biology and Environmental Studies, University of Zurich, Zurich, Switzerland, [5]Clinic for Small Animals, Faculty of Veterinary Medicine, Free University of Berlin, Berlin, Germany, [6]Group for Wildlife Research, Institute of Forest Botany and Forest Zoology, Technical University of Dresden, Tharandt, Germany, [7]Institute of Parasitology, Faculty of Veterinary Medicine, University of Leipzig, Leipzig, Germany and [8]Department of Animal Diseases, Zoonoses and Infection Diagnostics, Landeslabor Berlin–Brandenburg, Berlin, Germany

Abstract. The raccoon, *Procyon lotor* (Carnivora: Procyonidae), is an invasive species that is spreading throughout Europe, in which Germany represents its core area. Here, raccoons mostly live in rural regions, but some urban populations are already established, such as in the city of Kassel, or are starting to build up, such as in Berlin. The objective of this study was to investigate *Sarcoptes* (Sarcoptiformes: Sarcoptidae) infections in racoons in these two urban areas and to identify the putative origin of the parasite. Parasite morphology, and gross and histopathological examinations of diseased skin tissue were consistent with *Sarcoptes scabiei* infection. Using nine microsatellite markers, we genotyped individual mites from five raccoons and compared them with *Sarcoptes* mites derived from fox, wild boar and Northern chamois, originating from Italy and Switzerland. The raccoon-derived mites clustered together with the fox samples and were clearly differentiated from those of the wild boar and chamois samples, which suggests a fox origin for the raccoon mange infection. These results are evidence of the cross-transmission of *S. scabiei* among wild carnivores. Although our results cannot elucidate whether raccoons became infected by frequent interaction with endemically or epidemically infected foxes or whether these cases resulted from occasional contacts among these animal species, they do nevertheless show that pathogens can be shared among urban populations of native and invasive carnivores.

Key words. *Sarcoptes scabiei*, microsatellites, pathology, raccoon, wildlife.

Introduction

Sarcoptic mange is a contagious skin infection of domestic and wild animals caused by the mite *Sarcoptes scabiei* (Fain, 1968; Pence & Ueckermann, 2002). The ectoparasite is considered as a single species divided into several varieties which show a certain degree of host specificity (Fain, 1978; Zahler *et al.*, 1999; Bornstein *et al.*, 2001; Pence & Ueckermann, 2002). However, the extent of host specificity is still widely discussed. Sarcoptic mange is reported in more than 100 mammal species worldwide

Correspondence: Zaida Rentería-Solís, Department of Wildlife Diseases, Leibniz Institute for Zoo and Wildlife Research, Alfred-Kowalke Strasse 17, 10315 Berlin, Germany. Tel.: + 49 30 5168 227; Fax: + 49 30 5126 104; E-mail: renteria@izw-berlin.de

(Bornstein *et al.*, 2001) and occasionally the infection can reach epizootic proportions in some wildlife species such as Barbary sheep (*Ammotragus lervia*), Spanish ibex (*Capra pyrenaica*), Northern and Southern chamois (*Rupicapra* spp.) and red fox (*Vulpes vulpes*) in Europe (Pence & Ueckermann, 2002; Nimmervoll *et al.*, 2013). *Sarcoptes* transmission may occur by direct or indirect contact because the mite can survive for a few days in dislodged skin crust under certain conditions (Arlian *et al.*, 1988). Similarly to cross-transmission events between wild and domestic animal species (Arlian, 1989), zoonotic infections of *S. scabiei* in humans have been reported (Arlian, 1989). Human infection from an animal source is typically less severe and more short-lived (Arlian, 1989). Pet owners, personnel working with animals, veterinarians and wildlife biologists are most likely to become infected with animal-derived mites (Alasaad *et al.*, 2013).

The disease is characterized by a severe dermatitis, usually accompanied by intense pruritus. Erythematous eruptions, papule formation, seborrhoea and alopecia are often present (Bornstein *et al.*, 2001). Histologically, orthokeratotic and parakeratotic hyperkeratosis, and epidermal hyperplasia with the formation of rete ridges are commonly described and are associated with dermal inflammatory cell infiltration by eosinophils, lymphocytes, mast cells and plasma cells (Nimmervoll *et al.*, 2013).

Descriptive studies of *S. scabiei* epidemiology are regularly conducted in wild populations (Rossi *et al.*, 2007), but the limitations of these classical epidemiological studies in revealing the host origin of an outbreak indicate the need for a population genetics-based approach (Alasaad *et al.*, 2014).

The use of biomolecular investigations in epidemiology studies has contributed towards the gaining of new insights into transmission dynamics (Zahler *et al.*, 1999; Walton *et al.*, 2004; Rasero *et al.*, 2010; Alasaad *et al.*, 2011, 2014; Gakuya *et al.*, 2011). Further, such investigations can improve surveillance studies and help to properly distinguish 'genuine' emerging and re-emerging cases of *Sarcoptes* mite infestation from pre-existing but neglected cases in the population (Oleaga *et al.*, 2008).

The raccoon, *Procyon lotor*, was introduced into Germany from 1934. Currently, two main populations are identified in the country: one is located in central Germany in the surroundings of the city of Kassel, and the other is situated in the northeast in the federal states of Brandenburg and Mecklenburg-Western Pomerania. Additionally to these two main stable populations, small groups of raccoons can be found throughout Germany. First reports on causes of raccoon mortality in sylvatic and rural populations have been published (Michler *et al.*, 2009) and it was concluded that raccoons play no significant role in maintaining and spreading infections of major concern to humans or domestic animals in Germany. However, no similar information is available for urban raccoons.

To date, only one report has been published on sarcoptic mange in raccoons, specifically in three animals from North America (Fitzgerald *et al.*, 2004). Using microsatellite analyses, we traced the origin of raccoon-derived *Sarcoptes* mites collected in two urban areas in Germany.

Materials and methods

Sample collection

Between 2009 and 2013 a larger study on raccoons in Germany was conducted in 139 animals from urban areas and 97 raccoons from a national park. Skin lesions consistent with mange were detected in five of these raccoons. Two of these five were male raccoons collected in Kassel in the federal state of Hesse, central Germany (51°19′00″ N, 09°30′00″ E) during spring 2009. Both animals were killed by a hunter because of their poor condition and were sent to the Leibniz Institute for Zoo and Wildlife Disease for post-mortem investigations. Three additional raccoons were collected in the Berlin metropolitan area in northeast Germany (52°30′02″ N, 13°23′56″ E) between 2011 and 2013. One adult male and one juvenile female raccoon collected in Berlin were presented to the Small Animal Clinic of the Faculty of Veterinary Medicine, Free University Berlin, because of their striking skin lesions. After clinical examination, both animals were killed and submitted to the Landeslabor Berlin-Brandenburg (LLBB) for post-mortem examination. One further female raccoon was killed in a traffic accident and sent to the LLBB for post-mortem evaluation. Further details on the individual raccoons are listed in Table 1.

Necropsy and histology

Macroscopic examination and a routine necropsy were performed on each carcass. Samples of affected skin, brain, heart, intestines, kidney, liver, lung, lymph nodes, reproductive organs, spleen and tongue were extracted for further examination. All tissues were fixed in 4% formalin, processed routinely and embedded in paraffin. Tissue slides were sectioned at 4 µm, stained with haematoxylin and eosin, and examined by light microscopy.

Mite isolation and morphological identification

The affected skin areas of each raccoon were scraped with a scalpel blade to obtain hairs and crusts for mite isolation. The skin scrapings were separately placed on glass slides with distilled water and examined under a light microscope. Mites detected on each animal were manually collected and stored in an Eppendorf tube filled with 70% ethanol for scanning electron microscopy examination. Species identification was based on typical morphological characters as described by Fain (1968).

Molecular analyses

Six representative adult mites were isolated from each of the five raccoons and stored at −80 °C. Mites collected from red foxes in Italy and Switzerland (10 mites each), from Northern chamois (*Rupicapra rupicapra*, 10 mites) in northeast Italy, and from wild boar (*Sus scrofa*, nine mites) in northwest Italy, all gathered during previous studies, were jointly analysed. The DNA of each mite was extracted using the HotSHOT

Table 1. Collection date, weight, sex and age of the raccoons examined.

Raccoon ID	Collection date	Origin	Weight, kg	Age group	Gender
ID1	August 2011	Berlin	2.50	Juvenile	Female
ID2	February 2012	Berlin	3.99	Adult	Female
ID3	March 2013	Berlin	3.65	Adult	Male
ID4	March 2009	Kassel	4.11	Juvenile	Male
ID5	March 2009	Kassel	2.88	Juvenile	Male

Plus Thermal SHOCK technique as previously described in Alasaad *et al.* (2008). Ten specific *Sarcoptes* mite microsatellites (Sarms 33–38, 40, 41, 44 and 45) were used in a 10× multiplex polymerase chain reaction as previously described by Alasaad *et al.* (2011) and Gakuya *et al.* (2011). Expected (H_E) and observed (H_o) heterozygosity, linkage disequilibria (LD) and Hardy–Weinberg equilibrium (HWE) were calculated using GENEPOP Version 3.4 (Raymond & Rousset, 1995). Deviations of HWE and LD tests were calculated using Fisher's exact test and sequential Bonferroni corrections. The heterogeneity of genetic diversity among the different mite populations was estimated by the partition of variance components [analysis of molecular variance (AMOVA)] applying F_{ST} statistics using ARLEQUIN Version 3.11 (Excoffier, 2006). The relationship between mites was calculated using the Bayesian assignment test in the software STRUCTURE 2.3.4 (Pritchard *et al.*, 2000). Twenty independent runs were run for $K = 1–10$. The Markov value for both burn-in and run lengths was 100 000. The most likely number of clusters was first determined by estimating the posterior probability for each K as recommended by the method of Evanno *et al.* (2005) using the website STRUCTURE HARVESTER (http://taylor0.biology.ucla.edu/structureHarvester) (Earl & vonHoldt, 2012). The degree of genetic relationship among populations was calculated using factorial component analysis (FCA) in GENETIX Version 4.05.2 (Belkhir, 1999).

Results

Macroscopic findings

During necropsy, raccoons ID1 and ID4 were found to be in good condition, whereas all other animals were in poor body condition. One of the Berlin raccoons (ID1) also had a fractured femur and a subcutaneous abscess at the left side of the neck. All animals had generally poor fur and displayed skin lesions of increasing severity. Small areas of alopecia and mild crusting starting from the dorsal neck and extending to the scapular region as far as the antebrachial regions of both front limbs were found in animal ID1. Raccoon ID2 exhibited patchy alopecia with mild crusting distributed along the thoracic dorsum and front limbs. Raccoon ID3 presented severe massive crusting and presumed subcutaneous abscesses along the dorsum and flanks. Alopecic areas were present at the front paws, caudal dorsum and tail. One male from Kassel (ID4) presented extensive areas of severe skin alterations with crusting and thickened skin extending along the caudal region of the dorsum towards both hind limbs and the base of the tail. Additionally, the skin of the ear pinna was severely thickened. Raccoon ID5, also from Kassel, showed severe proliferative dermatitis and alopecia over the entire abdominal and gluteal region, along the thighs and down to the knees.

Microscopic skin lesions

Histologically, the two males from Kassel presented severe (ID4) and moderate (ID5) parakeratotic hyperkeratosis of the skin, as well as generalized mild to moderate infiltration of mainly granulocytic inflammatory cells (ID4) or multiple small foci of inflammatory cells (ID5) associated with marked epidermal proliferation. The formation of rete ridges was observed in both male raccoons. There was moderate (ID5) or severe (ID4) distribution of intracorneal mites. Additionally, the pinna of one animal (ID4) had focal ulcerations of the epidermis with severe infiltration of mixed inflammatory cells. The raccoons from Berlin showed mild (ID1) and severe (ID3) parakeratotic and mild orthokeratotic hyperkeratosis (ID2) of the skin with mild intracorneal infestation of mites, mild (ID1, ID2) and severe (ID3) epidermal hyperplasia with rete ridges and mild multifocal superficial bacterial colonization; additionally, small subcorneal pustules filled with granulocytic cell debris and mild lichenoid infiltration of mixed inflammatory cells were found.

Additional unrelated findings consisted of small chronic parasite granuloma in the lymph nodes of one juvenile male (ID4), severe suppurative necrotic nephritis in the adult female (ID2) and mild interstitial pneumonia in the juvenile female (ID1).

Mite isolation and morphological examination

All skin scrapings revealed numerous eggs and roundish tortoise-shaped adult mites, nymphs and larvae. The adult female mites ($n = 7$) measured 203.1×309.9 µm, and the adult males ($n = 2$) measured 149.5×166.6 µm. Characteristically, thin ventral striations covered their idiosoma, triangular cuticular spines were present on the dorsal idiosoma and the anus was terminally located. Both anterior leg pairs carried bell-shaped suckers at the tip of unsegmented pedicels. The two posterior leg pairs did not extend beyond the margin of the body. Morphological features identified the mite species as *S. scabiei*.

Molecular analyses

Of 30 processed mites, only one from a Kassel raccoon and six from all Berlin raccoons were suitable for microsatellite

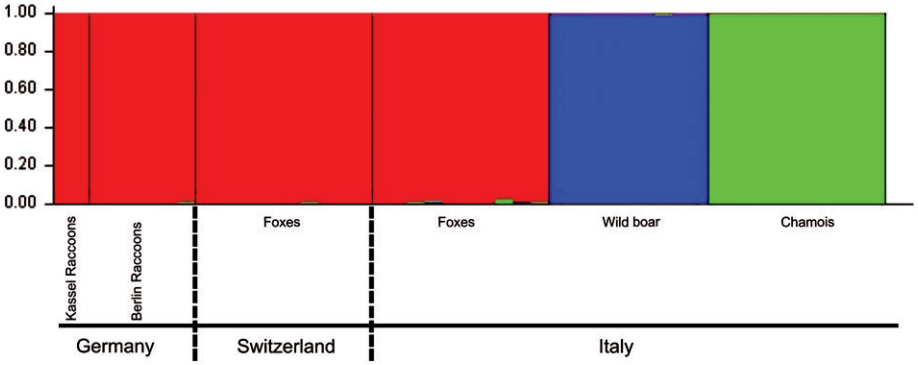

Fig. 1. Bar plot generated with STRUCTURE showing the degree of individual variation among 47 *Sarcoptes scabiei* mites arranged in six host-derived populations. $K = 4$.

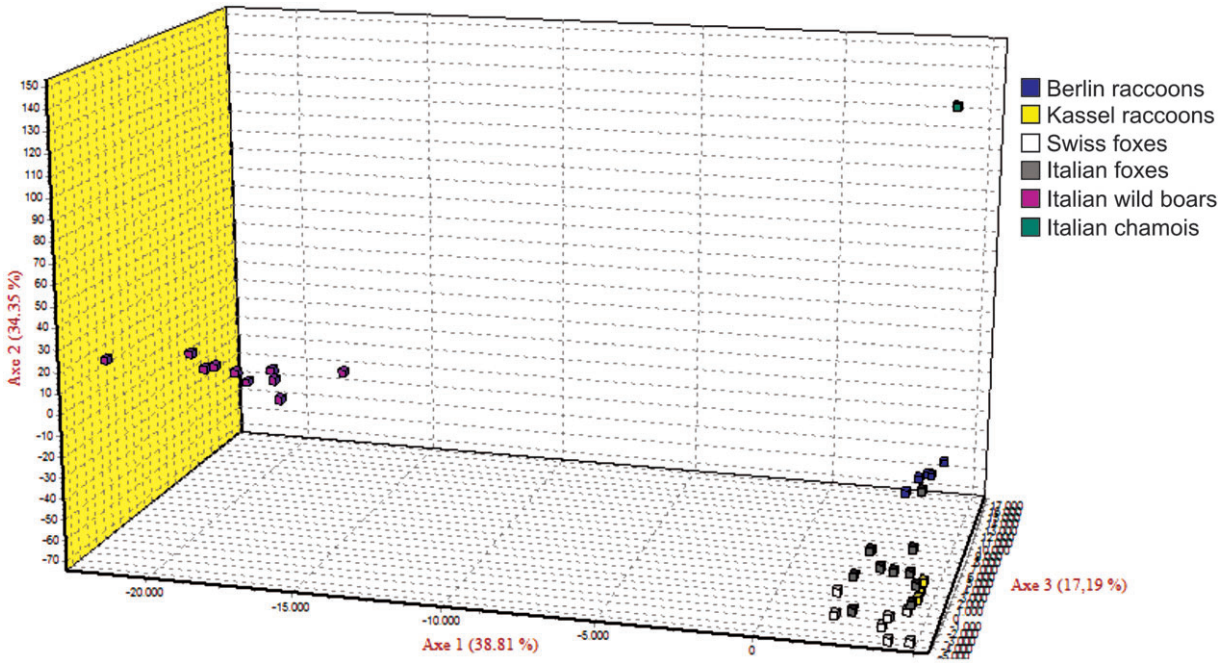

Fig. 2. Factorial component analysis of the proportion of variation in six *Sarcoptes scabiei* populations arranged in clusters calculated with GENETIX.

analyses. Sixteen alleles were detected from nine microsatellite loci. Another microsatellite locus (Sarms 35) was not considered in the study because the data retrieved were insufficient. The Bayesian assignment of the software STRUCTURE, ln Pr (X/K) for the likely number of populations, indicated $K = 3$. For this $K = 3$ comparison, raccoon-derived mites clustered together with fox-derived mites from Italy and Switzerland, whereas mites originating from Northern chamois and wild boar hosts clustered separately (Fig. 1). These results were confirmed in the scatterplot of the FCA for populations (Fig. 2).

Discussion

Sarcoptes infections were investigated in urban raccoons from Germany. The epidemiology and transmission dynamics of *S. scabiei* are still not well understood. Humans have been proposed to represent the original primary host of the mite (Fain, 1978) and it is thought that the mite subsequently spread to domestic animals and further to wildlife species. In free-ranging raccoons, sarcoptic mange has been reported only in Michigan, U.S.A. (Fitzgerald *et al.*, 2004), whereas in Germany there are anecdotal reports of sightings of raccoons bearing overt skin lesions (Michler *et al.*, 2009). In Europe, outbreaks of sarcoptic mange are commonly noted in sylvatic (Nimmervoll *et al.*, 2013) and urban (Börner *et al.*, 2009) foxes and the disease is endemically present in many regions in this species (Börner *et al.*, 2009; Rasero *et al.*, 2010; Nimmervoll *et al.*, 2013).

Traditional epidemiological studies must be enhanced with biomolecular approaches in order to increase current understanding of *Sarcoptes* epidemiology (Alasaad *et al.*, 2014). Genetic studies to differentiate *S. scabiei* are scarce and attempts

using internal transcribed spacer 2 (ITS2) rDNA have proved unsuccessful (Alasaad *et al.*, 2009).

The present study used microsatellite markers, which in the past have efficiently differentiated mite populations from different (non-raccoon) host species and geographical regions, eventually showing a lack of genetic flow among populations and a low degree of heterozygosity among individuals within populations (Walton *et al.*, 2004; Rasero *et al.*, 2010; Alasaad *et al.*, 2011; Gakuya *et al.*, 2011). Using microsatellite markers, Walton *et al.* (2004) showed genetic differentiation among mites from humans and dogs in Australia. Similarly, Rasero *et al.* (2010) and Alasaad *et al.* (2011) described genetic differentiation between *Sarcoptes* mite populations from different host taxa among wild animals in Europe. These authors reported a lack of genetic flow among populations from carnivorous, omnivorous and herbivorous hosts, a phenomenon known as the 'host taxon law' (Rasero *et al.*, 2010), which explains why mange-free populations of herbivores such as the Alpine ibex (*Capra ibex*) and Northern chamois in the Western Alps can share habitat with endemically or epidemically infected foxes (Rasero *et al.*, 2010).

In the current study, the Bayesian assignment test and FCA results showed an obvious differentiation between raccoon-derived and wild boar- and chamois-derived mites, all of which clustered separately. By contrast, mites from raccoons clustered together with Swiss and Italian fox-derived mites, suggesting that they belong to the carnivorous host taxon and that sarcoptic mange infection in raccoons is likely to have originated from infected foxes. However, additional biomolecular studies on mites derived from sympatric foxes and other carnivores, including dogs, would be desirable to rule out other putative sources of raccoon infection within the carnivorous taxon. Cross-transmission of *S. scabiei* mites belonging to the same host taxon (e.g. var. *vulpes* or var. *canis*) can occur between foxes and domestic canids (Rasero *et al.*, 2010), and transmission from dogs or foxes to humans has been reported (Folz, 1984).

With respect to the German raccoon population, rare anecdotal sightings of rural raccoons with mange-like skin lesions have been reported, but the aetiology of these lesions has not been established. Moreover, raccoons in rural or sylvatic populations possibly interact mainly amongst themselves and do not interact much with other carnivore species. Therefore, interspecies transmission of infectious pathogens such as *Sarcoptes* seems to occur only rarely in this ample environment.

By contrast, cityscapes are characterized by habitat defragmentation and the reduction of green spaces. These restrictions force wildlife species to share most of their habitats and result in increasing rates of contact. Thus, the transmission of infectious pathogens such as *Sarcoptes* between raccoons and foxes may be more easily facilitated. As they also share areas such as parks with domestic pets, raccoons may represent a potential source of infectious disease for domestic animals and possibly humans.

However, it could be speculated that despite sharing the same habitat, direct or indirect contact between foxes and raccoons may occur at a low rate so that cross-infection is infrequent and overt clinical disease in raccoons is relatively rare. Moreover, as affected raccoons can be detected more easily in cities than in rural areas, we cannot rule out the possibility that mange cases in raccoons may be subject to reporting bias. To ameliorate this lack of epidemiological knowledge, wildlife professionals and veterinary authorities should be encouraged to record mange-infected raccoons, foxes and other carnivores, despite the fact that such findings are often felt to be of minor significance.

In conclusion, the current study describes the cross-transmission of *S. scabiei* mites among urban wild carnivores, as well as the presence of sarcoptic mange within raccoon populations outside their native range. However, it remains uncertain whether transmission was derived through continuous interaction or sporadic direct or indirect contact. The results of this study increase current knowledge about *S. scabiei* epidemiology in free-ranging hosts, showing that the host taxon phenomenon also applies in the urban wildlife context. Additional studies to elucidate whether *Sarcoptes* infection in raccoons in Germany represents an emerging disease or a neglected and unreported condition are necessary.

Acknowledgements

The authors thank F. Becker, Alt Wahlershausen 4, Kasse, Germany, for providing raccoon samples from Kassel, A. Aue of the Landeslabor Berlin-Brandenburg for facilitating the sample collection of the Berlin raccoons, and Z. Mezoe, M. Biering, D. Krumnow and D. Viertel, Department of Wildlife Diseases, Leibniz Institute for Zoo and Wildlife Research, for their excellent technical assistance. We also thank J. Fickel, Department of Wildlife Diseases, Leibniz Institute for Zoo and Wildlife Research, for his support and advice regarding data analyses and B. A. Michler, Group for Wildlife Research, Institute of Forest Botany and Forest Zoology, Technical University of Dresden, for her helpful comments during the preparation of this manuscript. We are grateful to the German Academic Exchange Service (DAAD) and the Mexican Council of Science and Technology (CONACyT) for the financial support (fellowship of ZR-S) of this study. The funding institutions had no role in this study.

Author's declaration of interests

No competing interests have been declared.

References

Alasaad, S., Rossi, L., Maione, S. *et al.* (2008) HotSHOT Plus Thermal SHOCK, a new and efficient technique for preparation of PCR-quality mite genomic DNA. *Parasitology Research*, **103**, 1455–1457.

Alasaad, S., Soglia, D., Spalenza, V. *et al.* (2009) Is ITS-2 rDNA a suitable marker for genetic characterization of *Sarcoptes* mites from different wild animals in different geographic areas? *Veterinary Parasitology*, **159**, 181–185.

Alasaad, S., Oleaga, A., Casais, R., Rossi, L., Molinar Min, A., Soriguer, R.C. & Gortázar, C. (2011) Temporal stability in the genetic structure of *Sarcoptes scabiei* under the host-taxon law: empirical evidences from wildlife-derived *Sarcoptes* mite in Asturias, Spain. *Parasites & Vectors*, **4**, 151.

Alasaad, S., Rossi, L., Heuckelbach, J., Pérez, J.M., Hamarsheh, O., Otiende, M. & Zhu, X.-Q. (2013) The neglected navigating web of the incomprehensibly emerging and re-emerging *Sarcoptes* mite. *Infection, Genetics and Evolution*, **17**, 253–259.

Alasaad, S., Sarasa, M., Heukelbach, J., Mijele, D., Soriguer, R., Zhu, X.-Q. & Rossi, L. (2014) Advances in studies of disease-navigating webs: *Sarcoptes scabiei* as a case study. *Parasites & Vectors*, **7**, 16.

Arlian, L.G. (1989) Biology, host relations, and epidemiology of *Sarcoptes scabiei*. *Annual Review of Entomology*, **34**, 139–161.

Arlian, L.G., Ahmed, M. & Vyszenski-Moher, D.L. (1988) Effects of *Sarcoptes scabiei* var. *canis* (Acari: Sarcoptidae) in blood indices of parasitized rabbits. *Journal of Medical Entomology*, **25**, 360–369.

Belkhir, K. (1999) GENETIX, Logiciel sous Windows™ pour la Génetique des Populations. Laboratoire Génome et Populations, Université de Montpellier II http://www.genetix.univ-montp2.fr/genetix/intro.htm [accessed on 15 November 2013].

Börner, K., Schneider, R. & Wittstatt, U. (2009) Zum Krankheitsgeschehen des Rotfuchses (*Vulpes vulpes* L.) in Berlin. *Beiträge zur Jagd- und Wildforschung*, **34**, 299–305.

Bornstein, S., Mörner, T. & Samuel, W. (2001) *Sarcoptes scabiei* and sarcoptic mange. *Parasitic Diseases of Wild Mammals* (ed. by W.M. Samuel, M.J. Pybus & A.A Kocan), pp. 107–119. Iowa State University Press, Iowa, IA.

Earl, D.A. & vonHoldt, B.M. (2012) STRUCTURE HARVESTER: a website and program for visualizing STRUCTURE output and implementing the Evanno method. *Conservation Genetics Resources*, **4**, 359–361.

Evanno, G., Regnaut, S. & Goudet, J. (2005) Detecting the number of clusters of individuals using the software STRUCTURE: a simulation study. *Molecular Ecology*, **14**, 2611–2620.

Excoffier, L. (2006) arlequin 3.11. http://cmpg.unibe.ch/software/arlequin3 [accessed 16 October 2013].

Fain, A. (1968) Étude de la variabilité de *Sarcoptes scabiei* avec une revision des Sarcoptidae. *Acta Zoologica et Pathologica Antverpiensia*, **47**, 1–196.

Fain, A. (1978) Epidemiological problems of scabies. *International Journal of Dermatology*, **17**, 20–30.

Fitzgerald, S.D., Cooley, T.M., Murphy, A., Cosgrove, M.K. & King, B.A. (2004) Sarcoptic mange in raccoons in Michigan. *Journal of Wildlife Diseases*, **4**, 347–350.

Folz, S.D. (1984) Canine scabies (*Sarcoptes scabiei*) infestation. *Compendium on Continuing Education for the Practising Veterinarian*, **6**, 176–180.

Gakuya, F., Rossi, L., Ombui, J. *et al.* (2011) The curse of the prey: *Sarcoptes* mite molecular analysis reveals potential prey-to-predator parasitic infestation in wild animals from Masai Mara, Kenya. *Parasites & Vectors*, **4**, 193.

Michler, F.-U., Köhnemann, B.A., Roth, M., Speck, S., Fickel, J. & Wibbelt, G. (2009) Todesursachen sendermarkierter Waschbären (*Procyon lotor*, L. 1758) in Müritz-Nationalpark (Mecklenburg-Vorpommern). *Beiträge zur Jagd- und Wildforschung*, **34**, 339–355.

Nimmervoll, H., Hoby, S., Robert, N., Lommano, E., Welle, M. & Ryser-Degiorgis, M.-P. (2013) Pathology of sarcoptic mange in red foxes (*Vulpes vulpes*): macroscopic and histologic characterization of three disease stages. *Journal of Wildlife Diseases*, **49**, 91–102.

Oleaga, A., Casais, R., González-Quirós, P., Prieto, M. & Gortázar, C. (2008) Sarcoptic mange in red deer from Spain: improved surveillance or disease emergence? *Veterinary Parasitology*, **154**, 103–113.

Pence, D.B. & Ueckermann, E. (2002) Sarcoptic mange in wildlife. *Revue Scientifique et Technique de l'Office International des Épizooties*, **21**, 382–398.

Pritchard, J.K., Stephens, M. & Donnelly, P. (2000) Inference of population structure using multilocus genotype data. *Genetics*, **155**, 945–959.

Rasero, R., Rossi, L., Soglia, D. *et al.* (2010) Host taxon-derived *Sarcoptes* mite in European wild animals revealed by microsatellite markers. *Biological Conservation*, **143**, 1269–1277.

Raymond, M. & Rousset, F. (1995) GENEPOP Version 1.2: a population genetics software for exact test and ecumenicism. *Journal of Heredity*, **86**, 248–249.

Rossi, L., Fraquelli, C., Vesco, U., *et al.* (2007) Descriptive epidemiology of a scabies in chamois in the Dolomite Alps, Italy. *European Journal of Wildlife Research* **53**, 131–141.

Walton, S.F., Dougall, A., Pizzutto, S. *et al.* (2004) Genetic epidemiology of *Sarcoptes scabiei* (Acari: Sarcoptidae) in northern Australia. *International Journal of Parasitology*, **34**, 839–849.

Zahler, M., Essig, A., Gothe, R. & Rinder, H. (1999) Molecular analyses suggest monospecificity of the genus *Sarcoptes* (Acari: Sarcoptidae). *International Journal of Parasitology*, **29**, 759–766.

Accepted 23 April 2014