Clinical, histopathological and epidemiological study of canine straelensiosis in the Iberian Peninsula (2003–2007)

Gustavo A. Ramírez*, Jaume Altimira*, Beatriz García*, Marcos Fernández† and Miquel Vilafranca*

*HISTOVET Laboratorio de Diagnóstico Histopatológico Veterinario, Montserrat 9, 08192 Sant Quirze del Vallés, Barcelona, Spain
†Clinica Veterinaria BEADE, Ctra. da Coutada 4, 36312 Beade, Vigo, Spain

Correspondence: Gustavo A. Ramírez, HISTOVET Laboratorio de Diagnóstico Histopatológico Veterinario, Montserrat 9, 08192 Sant Quirze del Vallés, Barcelona, Spain. E-mail: gramirez@histoweb.com

Sources of funding
This study is self-funded.

Conflict of interest
No conflict of interest has been declared.

Abstract
Straelensiosis is a relatively new disease described in dogs and produced by a trombiculid larva known as Straelensia cynotis. Few cases have been reported in the English literature. Straelensiosis has been observed in Southern France and Northern Portugal causing a distinctive nodular dermatitis. The present study describes the clinical, histopathological and epidemiological features of 19 cases diagnosed as straelensiosis in the north of Spain and Portugal (2003–2007). A follow-up of the animals after diagnosis was carried out. Differences with previously described cases were observed, especially concerning epidemiology and histopathology. The grade of response to different treatments and new microscopic features are discussed. The study concludes that S. cynotis can cause a nodular dermatitis in rural and hunting dogs in Spain and Portugal and is associated with particular seasonal and geographical factors.

Accepted 7 October 2008

Introduction
Straelensiosis is a distinctive nodular dermatitis affecting hunting or outdoor dogs which has been recently reported in France1,2 and Portugal.3,4 The causal agent was identified as Straelensia cynotis, a parasitic larva of the family Leeuwenhoekiidae, superfamily Trombidioid.1 Trombiculidosis is an infestation by harvest mites, usually found in soil, litter and other terrestrial habitat. The nymphs and adults of the trombiculid mites are free living or parasitic on plants and other arthropods. Adult harvest mites lay eggs in the soil and the larvae hatch shortly after, being transferred to the host by contact. The parasitic larvae (chiggers) induce a papular highly pruritic dermatitis.5–9 Wild vertebrates are the usual hosts for the trombiculid mite larvae but food-producing domestic animals, pets and humans may be accidentally infested. Factors such as season or soil type influence the prevalence of trombiculidosis.2,5,6,10 Neotrombicula autumnalis, the European harvest mite, Euschoengastia latchmani and Walchia americana are the most common species involved in trombiculidosis in cats, dogs and horses.2,5,10 Straelensia cynotis shows some clear differences with other trombiculid mites, especially in clinical presentation, histopathological features and response to treatment.2,4,10

The aim of this study was to describe the clinical, histopathological and epidemiological characteristics of 19 cases of nodular dermatitis caused by S. cynotis in Spain and Portugal. New aspects of the disease and previously reported cases are also discussed.

Materials and methods
Histopathological diagnosis of straelensiosis was carried out in 19 dogs between 2003 and 2007 at the Histovet Veterinary Anatomopathology Laboratory, Barcelona, Spain. Skin samples were fixed in 10% neutral buffered formalin, processed routinely, embedded in paraffin wax, sectioned at 3 μm, and stained with haematoxylin and eosin or Alcian blue (pH 2.5 for acid mucopolysaccharides). Clinical histories and follow-up information were obtained through e-mail and telephone interviews with the referring clinicians.

The diagnosis was also confirmed by isolation and identification of the mites in two dogs (Table 1: cases 10 and 13). Larvae were isolated from skin nodules, cleared in 10% potassium hydroxide solution for 12 h, fixed in 10% neutral buffered formalin, and mounted in mineral oil for microscopic examination.11

Results
Clinical history
Female dogs (n = 13) overrepresented males (n = 6) in the study (Table 1). The mean age of the animals was 2.1 years (range 3 months to 8 years). Breeds affected were numerous, and no clear predisposition was observed. The animals

This study was presented as a free communication at the 18th Annual Meeting of the Spanish Society of Veterinary Pathology (Rabat, Marocco, 28–30 June 2006).
<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age</th>
<th>Breed</th>
<th>Date*</th>
<th>Lesions</th>
<th>Distribution</th>
<th>Location†</th>
<th>Lifestyle conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>2 y</td>
<td>Cocker spaniel</td>
<td>September 2003</td>
<td>Papules</td>
<td>Back</td>
<td>Braga (P)</td>
<td>Rural</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>1 y</td>
<td>Mixed</td>
<td>October 2003</td>
<td>Papules, crusts</td>
<td>Muzzle, forehead, occipital, neck</td>
<td>Pontevedra (S)</td>
<td>Hunting</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>3 y</td>
<td>Poodle</td>
<td>October 2003</td>
<td>Papules, crusts</td>
<td>Back</td>
<td>Barcelona (S)</td>
<td>Visited woodland</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>1 y</td>
<td>Golden retriever</td>
<td>November 2003</td>
<td>Papules</td>
<td>Muzzle, forehead and back</td>
<td>Barcelona (S)</td>
<td>Rural</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>1 y</td>
<td>Mixed</td>
<td>June 2004</td>
<td>Erythematous papules</td>
<td>Forehead, ears, back and tail</td>
<td>Barcelona (S)</td>
<td>Rural</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>2 y</td>
<td>Mixed</td>
<td>July 2004</td>
<td>Erythematous papules, crusts, purulent exudate, bleeding</td>
<td>Muzzle, cheeks, back and forelimbs</td>
<td>A Coruña (S)</td>
<td>Rural/Outdoor</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>2 y 6 m</td>
<td>Mixed</td>
<td>October 2004</td>
<td>Nodules, purulent exudate</td>
<td>Back and hindlimbs</td>
<td>Barcelona (S)</td>
<td>Visited woodland</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>6 m</td>
<td>Bulldog</td>
<td>October 2004</td>
<td>Nodules, purulent exudate</td>
<td>Back</td>
<td>Madrid (S)</td>
<td>Rural</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>1 y 7 m</td>
<td>Portuguese hound</td>
<td>October 2004</td>
<td>Pustules, pyoderma</td>
<td>Forehead, back and limbs</td>
<td>Vigo (S)</td>
<td>Hunting</td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>1 y 2 m</td>
<td>Bassat Fauve de Bretagne</td>
<td>December 2004</td>
<td>Papules, pustules, purulent exudate, crusts</td>
<td>Forehead, back and tail</td>
<td>Pontevedra (S)</td>
<td>Hunting</td>
</tr>
<tr>
<td>11</td>
<td>F</td>
<td>2 y</td>
<td>Mixed</td>
<td>March 2005</td>
<td>Papules, pustules, crusts</td>
<td>Back and flanks</td>
<td>Pontevedra (S)</td>
<td>Rural</td>
</tr>
<tr>
<td>12</td>
<td>F</td>
<td>2 y</td>
<td>Hound</td>
<td>September 2005</td>
<td>Papules, crusts</td>
<td>Forehead, back and forelimbs</td>
<td>Braga (P)</td>
<td>Hunting</td>
</tr>
<tr>
<td>13</td>
<td>F</td>
<td>3 m</td>
<td>Cocker spaniel</td>
<td>September 2005</td>
<td>Erythematous papules</td>
<td>Muzzle, forehead and back</td>
<td>Vigo (S)</td>
<td>Rural</td>
</tr>
<tr>
<td>14</td>
<td>F</td>
<td>2 y 6 m</td>
<td>Hound</td>
<td>November 2005</td>
<td>Papules</td>
<td>Muzzle, forehead and back</td>
<td>Pontevedra (S)</td>
<td>Hunting</td>
</tr>
<tr>
<td>15</td>
<td>F</td>
<td>3 m</td>
<td>Mixed</td>
<td>March 2006</td>
<td>Papules</td>
<td>Forehead and back</td>
<td>Vila Real (P)</td>
<td>Rural</td>
</tr>
<tr>
<td>16</td>
<td>M</td>
<td>2 y</td>
<td>Hound</td>
<td>September 2006</td>
<td>Nodules, crusts</td>
<td>Forehead, back and flanks</td>
<td>Pontevedra (S)</td>
<td>Hunting</td>
</tr>
<tr>
<td>17</td>
<td>M</td>
<td>8 y</td>
<td>Mixed</td>
<td>December 2006</td>
<td>Erythematous papules, crusts</td>
<td>Muzzle, forehead and back</td>
<td>Coimbra (P)</td>
<td>Hunting</td>
</tr>
<tr>
<td>18</td>
<td>M</td>
<td>6 y</td>
<td>Hound</td>
<td>September 2007</td>
<td>Papules, crusts</td>
<td>Forehead, back and axilla</td>
<td>Pontevedra (S)</td>
<td>Hunting</td>
</tr>
<tr>
<td>19</td>
<td>FN</td>
<td>1 y</td>
<td>Mixed</td>
<td>September 2007</td>
<td>Nodules</td>
<td>Muzzle, forehead and forelimbs</td>
<td>Vigo (S)</td>
<td>Rural</td>
</tr>
</tbody>
</table>

*Date of onset of clinical signs; †Province or city; (S) = Spain; (P) = Portugal.
F, female; M, male; FN, female neutered; y, years; m, months.
lived in northwest Spain (n = 10), northeast Spain (n = 4) and north Portugal (n = 4) with only one dog living in central Spain (case 8; Fig. 1). A seasonal tendency for clinical presentation was also observed. The disease occurred predominantly in the period between September and March (17 of 19, 89.5%), with the largest number of cases diagnosed between September and November (13 of 19, 68.4%). Only two cases (10.5%) did not develop the first lesions in this period (dogs 5 and 6 in June and July, respectively).

The origin of the infestation could not be clearly established, but all dogs lived in rural areas, were outdoors having regular contact with woodlands or natural open-spaces, or were used for hunting.

Clinical signs and distribution of lesions
The lesions presented as multiple, alopecic, small (approximately 1 to 6 mm in diameter), erythematous nodules and papules scattered over the head, neck, dorsum, extremities and lumbar regions, whereas the abdomen and chest were not involved. Lesions first appeared as erythematous macules that rapidly developed into nodules or papules. The lesions were not pruritic. The most common locations on the head included the forehead-temporal region (n = 13) and muzzle (n = 7) (Fig. 2), scattered nodules were also observed in the ears (n = 1), occipital region (n = 1) or cheeks (n = 1). Occasionally, lesions extended to the proximal aspect of the forelimbs (n = 4), hindlimbs (n = 2), flanks (n = 2), tail (n = 2) and axilla (n = 1). The nodules were round-shaped, firm, well circumscribed and protruded from the skin surface. Pustules, purulent exudate, crusts and ulceration, sometimes very extensive (Fig. 3), were common secondary findings (12 of 19, 63.2%). Bleeding was also observed in dog 6. Pruritus or pain was only noted when secondary problem were present. The dogs were otherwise healthy except for case 6 which was anorectic.

The disease duration was variable and the infection persisted for 5 to 12 months. The disease worsened in three females at the time of oestrus (cases 6 and 10) and parturition (case 14).

Skin scrapings and direct impressions were performed in an attempt to find the parasites and were consistently unsuccessful. On one occasion microscopic examination of a crust showed ‘atypical degenerated mites’. The dermatitis was reported to be contagious in three cases. Two puppies of bitch 14 and another dog living with cases 12 and 16 developed the disease without having access to the outdoor environment. Diagnosis was not pursued in these cases but the clinical signs and distribution of lesions were identical to those of the affected dogs. Owners or other humans in contact with the affected animals were not affected.

Treatment response and evolution of the disease
Fifteen of 19 dogs (78.9%) received treatment. Unfortunately, complete details concerning therapy were not obtained.
for each case. Different combinations of several commercial drugs were used (Table 2), including systemic or subcutaneous avermectins, antibiotics, immunosuppressants, glucocorticoids, topical acaricides, bathing with topical disinfectants and hypoallergenic diets. Although a thorough follow-up could not be carried out in all cases, most dogs (12 of 15, 80%) were reported to have only partial reduction in the size/number of nodules or no response. The best results (complete remission in dogs 3, 9 and 14; 20%) were obtained when the following combination of drugs was adopted: ivermectin subcutaneously every 15 days + cephalosporins twice or three times daily + fipronil spray every 15 days. Three of 19 cases (dogs 2, 7, 12; 15.8%) had spontaneous remission.

### Table 2. Therapeutic regimes and responses observed in the 19 dogs included in the study

<table>
<thead>
<tr>
<th>Case</th>
<th>Treatment</th>
<th>Response/Remission of the disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No</td>
<td>ND</td>
</tr>
<tr>
<td>2</td>
<td>Cephalexin (3 weeks)</td>
<td>Negative (remission after 8 months)</td>
</tr>
<tr>
<td>3</td>
<td>Antibiotics (ns) + benzoyl peroxide + ivermectin (x 3/10 days) + hypoallergenic diet</td>
<td>Complete remission after three ivermectin applications</td>
</tr>
<tr>
<td>4</td>
<td>Antibiotics (ns) + chlorhexidine</td>
<td>Negative*</td>
</tr>
<tr>
<td>5</td>
<td>Ivermectin + antibiotics (ns)</td>
<td>Negative*</td>
</tr>
<tr>
<td>6</td>
<td>Amoxicillin/clavulanic + fipronil + chlorhexidine + ivermectin</td>
<td>Partial</td>
</tr>
<tr>
<td>7</td>
<td>No</td>
<td>Remission after 12 months</td>
</tr>
<tr>
<td>8</td>
<td>No</td>
<td>ND</td>
</tr>
<tr>
<td>9</td>
<td>Ivermectin (x 2/14 days) + fipronil spray (every 15 days)</td>
<td>Complete remission after two ivermectin applications</td>
</tr>
<tr>
<td>10</td>
<td>Antibiotics (ns)</td>
<td>Negative (euthanized)</td>
</tr>
<tr>
<td>11</td>
<td>Cephalexin + azathioprine + benzoyl peroxide (2 weeks)</td>
<td>Partial after 2 months (recurrence)</td>
</tr>
<tr>
<td>12</td>
<td>Ivermectin + cephalosporin (2 weeks)</td>
<td>Negative (remission after 5 months)</td>
</tr>
<tr>
<td>13</td>
<td>Selamectin + fipronil spray (every 15 days) + cefadroxil + benzoyl peroxide</td>
<td>Negative after 1 month (euthanized)</td>
</tr>
<tr>
<td>14</td>
<td>Ivermectin (every 15 days) + fipronil</td>
<td>Complete remission</td>
</tr>
<tr>
<td>15</td>
<td>Amoxicillin/clavulanic + methylprednisolone</td>
<td>Negative*</td>
</tr>
<tr>
<td>16</td>
<td>Cephalosporin + chlorhexidine</td>
<td>Partial after 2 months</td>
</tr>
<tr>
<td>17</td>
<td>Ivermectin + antibiotics (ns)</td>
<td>Partial after two ivermectin applications</td>
</tr>
<tr>
<td>18</td>
<td>No</td>
<td>Negative</td>
</tr>
<tr>
<td>19</td>
<td>Antibiotics (ns) + prednisolone</td>
<td>Negative</td>
</tr>
</tbody>
</table>

ND, not determined; ns, not specified; (*), complete follow-up could not be carried out.

### Histopathological features

Histological examination of the nodules revealed the presence of dilated hair follicles containing most of the time a well-preserved larval mite (Fig. 4a). In some cases, no larvae were seen even when the whole follicle was completely sectioned. An eosinophilic, sometimes calcified, dense and amorphous material surrounded the larvae and showed poorly defined limits with the keratinized follicular epithelium (Fig. 4b). The larval mites presented an oval morphology, a gnathostome with typical chelicerae pointing towards the dermis, and a clear striated cuticle (Fig. 4c). The overlying epidermis was acanthotic, with hyperkeratosis, focal spongiosis and serocellular crusts. Affected follicles revealed a marked pseudoeotheilmomatous hyperplasia and abundant perifollicular mucusosis (Figs 4b and 5). Vascular proliferation and ectasia were also common around the follicles (Fig. 4b). The inflammatory component was usually scarce and composed of mononuclear cells (lymphocytes, plasma cells and scattered mast cells) in the absence of eosinophils. Only those cases with clinical evidence of pyoderma and pustules exhibited a more severe

![Figure 4. Photomicrographs of canine skin. Haematoxylin and eosin stain. (a) Case 4. Histopathology of a skin nodule showing marked follicular wall hyperplasia and dilated follicular ostium. Note a larva section occupying the hair follicle infundibulum (arrow). Bar = 250 μm; (b) Case 16. Detail of two hair follicles containing parasitic larvae surrounded by an eosinophilic, calcified amorphous material (arrow), pseudoepitheliomatous follicular wall hyperplasia and an abundant vascular component. Bar = 100 μm; (c) Case 4. Detail of the striated cuticle of the parasite. Bar = 10 μm.]
suppurative mural folliculitis and furunculosis. In two dogs (cases 14 and 19), a particular histopathological feature was that several affected follicles were completely replaced by large blood-filled cysts lined by an endothelium and surrounded by abundant vascular proliferation, mild dermal fibrosis and scarce amounts of mucin (Fig. 6).

**Discussion**

Straelensiosis is a form of trombiculidosis caused by *S. cynotis*, a newly described harvest mite of dogs in France and Portugal.²⁻⁴ Le Net et al.² presented 12 dogs affected by this disease. Those authors did not find breed, age or gender predisposition. However, in our study, female dogs about 2 years old were overrepresented. Most of the affected animals were mixed breed dogs, thus breed predilection could not be determined as in previous reports. Contrary to LeNet et al.² report, seasonal influence was marked in our case series. The vast majority of animals developed lesions in the period between September and November. This feature is common to other trombiculidosis: chiggers cause disease in late summer and autumn.⁵⁻¹⁰ The median duration of the disease was very similar to that described in previous reports.²⁻⁴

Geographical distribution of affected dogs was well defined. Eighteen of 19 animals (94.7%) lived in the northeast and northwest of the Iberian Peninsula. Previous studies reported animals in Southern France, one case in Northern Portugal and three cases in northwest of Spain.²⁻⁴,¹¹ These findings indicate a highly defined geographical distribution of the disease, including Northern Portugal and Spain, and Southern France (Fig. 1). To our knowledge, no cases of straelensiosis have been diagnosed in other areas of Spain. However, considering that straelensiosis has been reported only recently, the disease might not have been recognized or may have been misdiagnosed. The case identified in central Spain (Madrid, dog 8) supports this hypothesis. The incidence and development of trombiculidosis are intimately related to climate and soil type,⁵⁻¹⁰ and may explain the presence of the disease in the Iberian Peninsula (northeast/northwest). In our series, clay and siliceous soils predominate in the geographical regions where dogs lived. The mites of trombiculids are most often seen in fruit-growing areas on chalky soil or have certain predilection for neutral or lightly acidic soils.⁵,¹²

Trombiculid infection is acquired in rural and periurban environments.⁵,⁶ Straelensiosis occurs in hunting dogs or in dogs that have contact with woodlands, a common

---

**Figure 5.** Photomicrographs of canine skin. Haematoxylin and eosin. (a) Case 1. Abundant mucinosis was a common finding surrounding affected follicles. Bar = 250 μm. (b) Alcian blue-stained (pH = 2.5) section demonstrating the mucinous nature of the perifollicular material. Bar = 250 μm.

**Figure 6.** Photomicrographs of canine skin. Haematoxylin and eosin stain. Case 14 (a) and 19 (b). Some lesions were completely replaced by large blood-filled spaces lined by an endothelium and surrounded by abundant vascular proliferation. Bar = 50 μm.
Our cases were hunting dogs (n = 8), outdoor or rural-living dogs (n = 9), or animals that regularly go to woodlands (n = 2). Some authors have suggested that foxes’ dens may be a natural habitat for *S. cynotis*. Foxes are widely distributed in the Iberian Peninsula and their habitats are traditionally woodland areas. However, due to the increase of human activities they are also frequently seen in open spaces or woodland areas. However, due to the increase of human presence and periurban rubbish dumps, they are also frequently seen in open spaces or woodland areas. However, due to the increase of human presence and periurban rubbish dumps, they are also frequently seen in open spaces. Given the fact that wild mammals and dogs very rarely come into close contact, it is plausible that the disease spreads from bushes, undergrowth or dens rubbed by both natural hosts and dogs.

Clinical features of the disease were identical to those previously reported. Lesions began as macules that progressed to erythematous, alopecic nodules and papules. Dorsal areas including the head were the most severely affected, whereas limbs, ventral abdomen and chest were less affected or not involved. Pruritus was not initially observed. Many dogs developed purulent dermatitis and crusts. Pruritus has occasionally been observed. Le Net et al., described a pruritic reaction in one of 12 dogs which the authors attributed to flea and lice infestation, and Seixas et al. described one dog with intense pruritus, marked purulent exudation and crusts. In our group, six dogs presented with pruritus associated with pustules, extensive crusting, purulent exudation, folliculitis and furunculosis. Therefore, straelensiosis appears to be typically a non-pruritic dermatitis with pruritus only appearing when secondary infection develops. This is in contrast with other chigger infestations, where variable degrees of pruritus are typically present.

Three cases in our series apparently developed the disease after being in contact with other affected dogs. Cases 12 and 16 shared the same household with another dog that was also used for hunting. However, dog-to-dog transmission in these cases is questionable because all of the dogs were exposed to similar conditions in woodlands. However, puppies of case 14 developed identical papular lesions in the dorsal aspect of the body despite being exclusively indoors. The dogs could have acquired the infection due to direct contact with insects, plants or soil carrying the infective parasite. It is possible that the bitch mechanically carried the parasites on her coat and paws and contaminated the environment where the puppies lived. Unfortunately, the owners of these dogs declined further evaluation to confirm the disease.

Histologically, pseudoepitheliomatous follicular hyperplasia and abundant perifollicular mucinosis were consistently present. These findings can be considered pathognomonic of this condition because they are also present in all cases previously reported. We observed a striking proliferation of vascular channels surrounding some lesions. Also, cases 14 and 19 showed replacement of the hyperplastic follicular epithelium by a blood-filled endothelial-lined lacuna. This change has not been reported before. These dogs had received systemic ivermectin as trial treatment for presumed mange. Seixas et al. described loss of parasite structures and persistence of the pseudoepitheliomatous follicular hyperplasia following treatment with systemic ivermectin. The vascular lacunae found in our cases may be the result of resolution or evolution of the lesions following therapy. Inflammatory infiltrates in our series were usually mild. Mast cells or eosinophils were rarely encountered. This scarce reaction consistently differs from other chigger infestations which cause intense pruritus and eosinophilic inflammation. These differences are probably due to the amorphous intensely eosinophilic material covering the larva that minimizes contact with the host tissues. The parasites in our case series presented the typical characteristics previously described and occupied the follicular ostium causing a marked enlargement. This feature is distinctive of *S. cynotis* as other trombiculid parasites excavate tunnels within the stratum spinosum or in the stratum corneum.

The response to therapy was variable and depended on the treatment regimen applied. Fifteen of 19 dogs (78.9%) received medication. Total (three of 15, 20%), partial (four of 15, 26.7%) or no remission (eight of 15, 53.3%) was observed. The best response was obtained when ivermectin was used, especially in conjunction with other drugs for secondary complications with five of seven dogs (71.4%) showing improvement in clinical signs. Therefore, these results suggest that ivermectin may be effective but other factors such as grade of infestation or secondary infections may influence the response to treatment. On the other hand, spontaneous remission was also suspected in dogs 2, 7 and 12. Dog 7 did not receive treatment, while dogs 2 and 12 received some form of therapy but only initially for a short period of time (three and two weeks, respectively) without any response. The disease resolved between five and eight months after the last treatment, when the effect of the drugs had already disappeared. Alternatively, this apparently spontaneous remission could be explained by the life cycle of *S. cynotis*. The larvae of *Straelensia spp.*, as other trombiculids, must feed on a vertebrate host. Upon completion of feeding, the larvae detach from the host and return to the environment to complete their life cycle. The dog is an incidental host; larvae do not progress to the next phase of the cycle (epidemiological impasse). The duration of the feeding period of *S. cynotis* is not known, but a previous description estimated at least 3 months in dogs kept indoors. The disease duration of the dogs suspected of spontaneous remission was five, eight and 12 months (cases 12, 2 and 7, respectively); however, they were not confined indoors. It could not be determined whether the duration of the disease corresponded to persistent infestation or to continual re-infestation. It is possible that the larvae could have detached from the dogs returning to the environment or died probably due to an incomplete life-cycle. After remission, none of the dogs had recurrence of clinical signs. The intensity of the infestation and secondary infections can produce an adverse clinical course. Similarly to the previously reported case in northern Portugal, two dogs in our series (cases 10 and 13) were euthanized due to generalized cutaneous lesions.

In conclusion, straelensiosis in the Iberian Peninsula shows a well-defined geographical location (north) and appears linked to climatic conditions (cold seasons) and lifestyle (rural and hunting dogs). Treatment based on a combination of macrocyclic lactones and antibiotics may result in complete cure or prevent possible complications.
until remission occurs. Additional studies including better treatment follow-up will be desirable for further knowledge of this parasitic disease.

Acknowledgements
The authors are grateful to the referring clinicians for their cooperation in this study and Dr Estevez-Tenorio for help in providing clinical photographic material (Fig. 3). We acknowledge technicians of the HISTOVET Pathology Diagnostic Laboratory (Barcelona, Spain) for their excellent technical assistance.

References