

Dermatophytosis due to *Microsporum persicolor*: A retrospective study of 16 cases

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Abstract — A retrospective study of 16 cases of dermatophytosis due to *Microsporum persicolor* in dogs is reported. Hunting dogs were overrepresented (12/16). Skin lesions were observed on the face in all cases, but also on other locations (limbs, neck). The lesions included alopecia (15/16), erythema (13/16), scales (14/16), and crusts (13/16). Histopathology was performed in 10 cases and showed folliculitis and a lichenoid interface dermatitis. Fungal culture was positive in all cases and clinical resolution was achieved with standard antifungal agents (enilconazole, ketoconazole, griseofulvin). Two recurrences were observed (new contacts with rodents).

Résumé — **Dermatophytie à *Microsporum persicolor* : Étude rétrospective de 16 cas.** Une synthèse rétrospective de 16 cas de dermatophytie à *Microsporum persicolor* chez le chien est présentée. Les chiens de chasse sont particulièrement représentés (12/16). L'atteinte faciale est systématique mais d'autres zones corporelles peuvent être concernées (membres, cou). Les lésions observées sont une alopecie (15/16), un érythème (13/16), des squames (14/16) et des croûtes (13/16). L'analyse histopathologique (9 cas) montre une folliculite et une dermatite lichénoïde d'interface. Le diagnostic définitif est fondé sur une culture mycologique positive et le traitement fait appel aux antifongiques classiques (énilconazole, kétoconazole, griséofulvine). Une récurrence est observée chez deux chiens (recontamination par des rongeurs sauvages très probable). Une synthèse bibliographique complète cette étude rétrospective.

(Traduit par les auteurs)

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Introduction

Dermatophytoses are superficial mycoses which are infectious and contagious, and are caused by keratinophilic and keratinolytic epidermal fungi belonging to the genera *Microsporum* and *Trichophyton*. The classification of dermatophytes is based on their characteristics of sexual reproduction but identification relies more upon their asexual reproduction (septate mycelium, microconidia, and macroconidia).

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Most cases of dermatophytosis affecting dogs are due to *Microsporum canis* (85% of cases). In many areas (Africa, America, Australia, Europe), dermatophytosis due to *Microsporum persicolor* has been anecdotally reported since 1945 and most authors agree that this accounts for between 2% and 10% of canine dermatophytosis cases (2.86% in a French study) (1–7). *Microsporum persicolor* is also referred to as *Trichophyton persicolor*, *Sabouraudites persicolor*, *Ctenomyces persicolor*, or *Microïdes persicolor*, and displays a perfect sexual form called *Nannizzia persicolor*.

Materials and methods

Here, we report a series of 16 cases of dermatophytosis due to *Microsporum persicolor* referred between January 1990 and December 2008. Examination of case files allowed the analysis of certain epidemiological and clinical data: breed of animal, age, sex, nature and topography of lesions, presence of pruritus, and hunting activity. During the consultations, standard complementary examinations were performed: scrapings, smears, Wood's light examination, trichogram and fungal culture from hairs, scales and crusts taken from the lesions [Dermatophyte Test Medium (DTM) or Sabouraud chloramphenicol-actidione medium]. The dogs included in this study were those in which a definitive diagnosis of dermatophytosis due to *M. persicolor* was made based on a positive mycological culture and microscopic identification of the fungus. In 9 cases, 6-mm cutaneous Trepan punch biopsies were stained with hematoxylin and eosin

Table 1. Sixteen cases of dermatophytosis due to *Microsporum persicolor* (between 1990 and 2005)

Case	Breed	Age (y)	Sex	Hunting	Lesional topography	Lesions	Pruritus
1	German shorthaired pointer	3	F	hound	face	A, E, Sc, Cr	0
2	Fox terrier (smooth-haired)	5	F	terrier	face, limbs	A, E, Sc, Cr	++
3	Fox terrier (rough-haired)	10	M	—	generalized	A, E, Sc	+++
4	Labrador retriever	3	M	hound	face	E, Cr	+
5	Fox terrier (smooth-haired)	6	F	terrier	generalized	A, E, Sc, Cr	++
6	Belgian shepherd	2	M	—	muzzle	A, Sc, Cr	0
7	Fox terrier (smooth-haired)	4	M	terrier	face	A, E, Sc, Cr	+
8	Beagle	2	M	terrier	muzzle	A, E, Sc	+
9	Fox terrier (rough-haired)	4	M	terrier	face	A, E, Sc	+
10	Pointer	6	M	hound	muzzle	A, Sc, Cr	0
11	Jack Russell terrier	3	F	terrier	muzzle	A, E, Sc, Cr	0
12	Jack Russell terrier	4	F	terrier	muzzle	A, E, Sc, Cr	0
13	Fox terrier (smooth-haired)	2	M	terrier	face	A, E, Cr	0
14	German shepherd	1,5	F	—	lip	A, Sc, Cr	0
15	Fox terrier (smooth-haired)	9	F	terrier	face, limb	A, E, Sc, Cr	+
16	Jagd terrier	5	M	terrier	face	A, E, Sc	+

A — alopecia, E — erythema, Sc — scales, Cr — crusts, face — muzzle + one other area of the face, generalized — at least 3 sites.

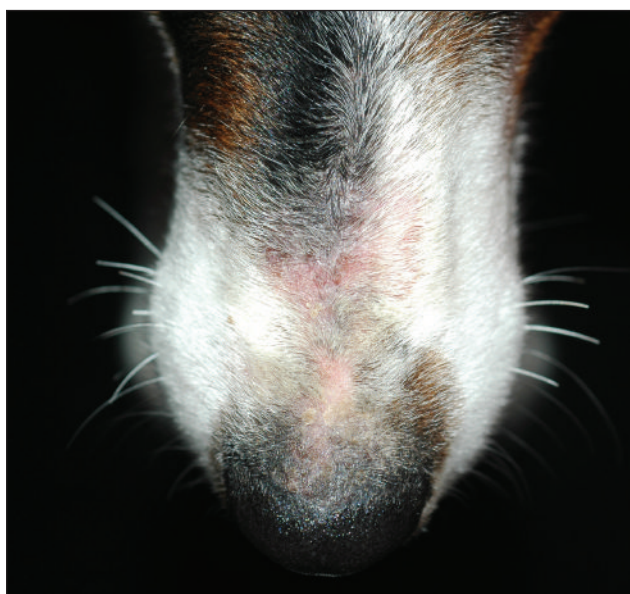


Figure 1. Alopecia, scaling, and crusting on the nose of a fox terrier.

and periodic acid-Schiff stains. Treatment involved standard anti-fungal agents: griseofulvin [50 mg/kg body weight (BW) per day, PO], ketoconazole (10 mg/kg BW, per day, PO) or enilconazole (2% solution, applied locally every 4 d for 3 wk). All cases were followed clinically for 8 to 12 mo.

Results

Eight breeds were represented (Table 1). Consistent with the mode of contamination (contact with wild rodents), the hunting dogs most affected were terriers (fox terrier, Jack Russell terrier). Ten dogs were terriers and 3 were hounds. For these 13 dogs, symptoms appeared during or just after a hunting period. Dog number 3 only had contact with its owner's guinea pig (probably an asymptomatic carrier) and dogs 6 and 13 were frequently walked in the countryside but did not have any known contact with a wild rodent. The age when the symptoms appeared was variable (1.5 to 10 y) and there was no evidence for any sexual predisposition (9 males and 7 females).

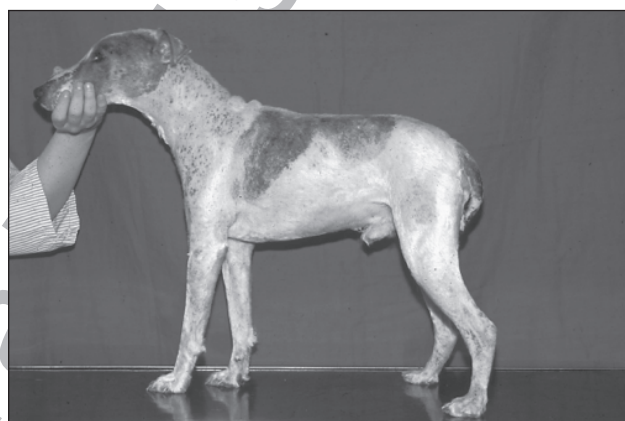


Figure 2. Alopecia and generalized erythema on a smooth-haired fox terrier.

All dogs had lesions on the face (16/16 cases), particularly the nose (15/16), but other sites were also involved: anterior limbs (4/16) and neck (2/16). The lesions showed alopecia (15/16), erythema (13/16), scaling (13/16), and crusting (13/16) (Figures 1, 2). A moderate (8/16) to intense (1/16) pruritus was observed in most cases, although 7 of the dogs had no pruritus. The dog presenting with severe pruritus was affected by a particularly severe and generalized form of the disease.

Complementary examinations

Scrapings, cutaneous smears, Wood's light test, and direct examination of the hair shafts were negative in all cases. Fungal culture was positive for all 16 dogs with growth detected on average 4 d after seeding and a change in color of the DTM medium observed after 7 d. Microscopic examination of colonies showed the characteristics of *M. persicolor*: numerous fusiform macroconidia, with a thin and spiny wall, and wide spirals.

Histopathological examination showed that all 10 dogs examined had a lichenoid interface dermatitis consisting mostly of a lymphoplasmacytic infiltration (Figure 3), associated in some cases with perifolliculitis and folliculitis. Only 6 cases showed evidence of mycelial filaments (without arthrospores) in epidermal and follicular keratin (Figure 4).

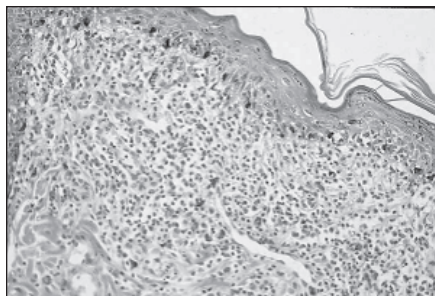


Figure 3. Lichenoid interface dermatitis. Note the lymphoplasmacytic infiltration in groups under the epidermis (H & E, 200×).

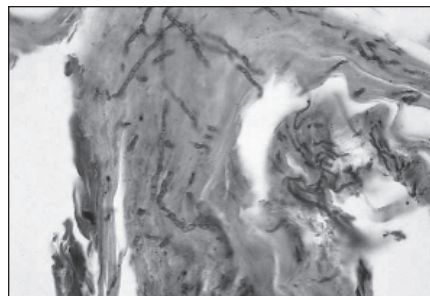


Figure 4. Mycelial filaments without arthrospores in epidermal and follicular keratin. (H & E 800×).

Table 2. Treatment and follow-up in the 16 affected dogs

Number of cases	Treatment	Duration of treatment (d)	Result	Recurrence
4	Griseofulvin	40	Cure	0
4	Ketoconazole	40		0
7	Ketoconazole	60		2
1	Ketoconazole + Enilconazole	60		0

Treatment

Clinical recovery was achieved in all 16 dogs, whichever treatment was used (Table 2). Enilconazole was used in only 1 case (dog 3, in combination with ketoconazole for the dog and used as monotherapy for the guinea pig). Ketoconazole was used on 12 dogs (40 d for 4 dogs and 60 d for 8 dogs), and griseofulvin was used on 4 dogs (40 d). The average duration of treatment was 50 d (40 d for griseofulvin and 53 d for ketoconazole). Among the 10 cases followed for 1 y, 2 recurrences were observed, 3 and 6 mo following recovery. In both cases the dogs had been in renewed contact with rodents and recontamination (rather than a relapse of the first episode) was therefore strongly suspected.

Discussion

Microsporum persicolor is a zoophilic (associated primarily with animals) fungus which may also be geophilic (reservoir in soil and may infect humans or animals) (3,8). It is a natural resident and occasional pathogen in small rodents such as voles, hamsters, and field mice. These rodents represent the main reservoir (up to 50% can be carriers) and are the major source of human and animal infection, contamination occurring essentially through direct contact with their hair (preferential localization is in the hair of the tail) (9,10). The fungus has also been isolated from bats, rabbits, and a diverse range of birds. Dogs and, much more rarely, cats may serve as intermediate hosts between rodents and humans; however, no case of human contamination has ever been reported in descriptions of dermatophytosis due to *M. persicolor* in domesticated carnivores.

These epidemiological findings indicate the importance of considering the medical history for dermatophytosis suspected to be due to *M. persicolor*, and of questioning the owners regarding the possibility of contact between the infected animal

(particularly hunting dogs) and small mammals. There is no predisposition based on age or sex. The breeds exposed to small rodents are particularly well represented among published cases: hunting dogs such as spaniels or Weimaraners, and ratting dogs such as terriers (fox, Jack Russell, jagd) (1,3).

Microsporum persicolor is a strict epidermatophyte, meaning that it only grows on keratin in superficial dead tissue. It therefore never penetrates the hair (and does not cause genuine ringworm) and is limited to the *stratum corneum* or sometimes to follicular keratin. This fungus produces keratinases which break down keratin into easily assimilated metabolites and allow invasion of the *stratum corneum*. Differences in the nature of hair keratins and keratins in the *stratum corneum* are the reason *M. persicolor* is not found in hair (1,2). Lesion formation in the host results from mechanical rearrangements due to the growth of the fungus, and also from the action of fungal metabolites (acting as toxins). These metabolites may be partially responsible for the marked cutaneous inflammation observed in most dogs with dermatophytosis due to *M. persicolor* (11).

Our study is the first to report evidence of a lichenoid interface dermatitis in all of the biopsied cases. The complementary studies were necessary to characterize the infiltration precisely and to better understand the mechanism of this reaction. Elimination of the dermatophyte by the host (spontaneous recovery) is linked to the development of cellular immunity (11). However, *M. persicolor* does not penetrate to the deep layers of the epidermis and remains very superficial, so the host does not necessarily develop sufficient immunity; this may explain why spontaneous recovery is not observed in affected dogs.

The most frequently affected areas are those that are most likely to come in contact with small rodents: principally the face (particularly the muzzle) and the thoracic limbs. In a previous study in France, 10 of 13 cases also presented with lesions on the muzzle, the ears, around the eyes, the neck, axillary zones, and thoracic limbs (1). Although classical dermatophytic lesions can be observed (alopecic, nummular erythematous-squamous lesions), it is not unusual to find more puzzling forms: erythema, papular or pustular lesions, furuncles, cellulitis, crusting dermatitis, and kerato-seborrhoea. Intense variable pruritus is sometimes present and can be very severe. Depigmentation of the nasal planum has also been reported (1,3).

The differential diagnoses are essentially demodicosis, pyoderma, or a dermatophytosis not due to *M. persicolor*, although

the more unusual clinical presentations (crusting dermatitis, presence of pruritus) evoke other possibilities (such as scabies, dermatitis due to zinc deficiency, autoimmune disease).

Examination of hairs under the light microscope is uninformative because *M. persicolor* does not invade hair. In contrast, examination of scales may reveal mycelial filaments on the surface keratin. The transparent adhesive cellophane test is therefore useful. *Microsporium persicolor* does not produce pteridine, which is responsible for fluorescence in Wood's light examination, so this test will always be negative. One of the characteristics of *M. persicolor* is its rapid rate of growth (3 to 5 d) in traditional culture media [Sabouraud's agar or DTM]. In DTM medium, therefore, growth will precede any change in color by a few days.

Microsporium persicolor colonies have a peach-colored front, which becomes shiny and pink with age, and a powdery surface (felt-like at the center), with a yellow to yellow-brown back. Culture on a medium that is poor in sugars, such as Sabouraud's medium, produces a back with a characteristic "wine-red" color (2). Microscopic examination should be performed using Roth's Flag technique: a piece of adhesive tape is applied onto the culture and is then transferred onto a slide upon which a drop of dye (lactophenol cotton blue) has been placed beforehand. A second drop of the dye is then placed onto the tape and the whole fungus is recovered on a coverslip. Microscopic examination shows numerous round or oval microconidia organized as acledia and subsequently clusters (10th day). Macroconidia are also numerous, and are fusiform (cigar-shaped) with a thin wall and 6 or more cells. The macroconidial echinulations which alone allow differentiation of *M. persicolor* from *Trichophyton mentagrophytes* are not always visible, especially in young cultures. Wide spirals are also frequently observed (1–3,6,7).

Fungal infection can be identified by histopathology in 70% to 80% of dermatophytosis cases. However, this examination can be unsatisfactory in cases of *M. persicolor* infection because this fungus is very superficial and often lost during sampling for histology (1,3). Standard staining with hematoxylin and eosin, or even better, Gomori-Grocott or periodic acid-Schiff staining, may in some cases reveal the presence of mycelial filaments. A diffuse epidermal orthokeratotic hyperkeratosis with acanthosis and presence of crusts is generally found, sometimes associated with a superficial perivascular and perifollicular dermatitis. Folliculitis and furunculosis are sometimes present. Note the systematic discovery of an interface dermatitis with hydropic degeneration of the basal cell layer in all the cases described in our study; this finding needs to be confirmed in other studies. This discovery suggests that *M. persicolor* is a possible cause of hydropic interface dermatitis in dogs.

A topical treatment (enilconazole, 0.2%, twice per week) is applied if necessary in combination with a systemic treatment (ketoconazole, 10 mg/kg BW per day or griseofulvin, 10 to 60 mg/kg BW per day or itraconazole, 5 mg/kg BW per day). Treatment is continued until disappearance of the lesions (2 mo on average) and ideally until culture tests are negative (1,3,12–14). The response to treatment is always excellent but

the lifestyle of the affected animals (hunting dogs in particular) leads to possible recurrences due to recontamination from the environment.

Human dermatophytosis due to *M. persicolor* mostly affects young women. It represents 2% to 3% of the zoophilic dermatophytosis cases in humans, and manifests as large-scale herpes circine, vesicular or bullous erythematous-squamous lesions on the face, arms, forearms, hands and fingers, as well as the legs. Contamination occurs following direct contact with animal hosts (asymptomatic or presenting with lesions), but no case of canine dermatophytosis due to *M. persicolor* has been reported as having contaminated humans. Human contamination seems instead to originate from direct contact with wild rodents.

This retrospective study confirms the clinical and epidemiological data that have been previously reported in the veterinary literature. The study also suggests that dermatophytosis due to *M. persicolor* is one of the possible causes of lichenoid interface dermatitis, which, to our knowledge, has never previously been suspected. Although this dermatitis can present with some puzzling and even severe clinical aspects, the prognosis is good because appropriate treatment always leads to recovery within 1 or 2 mo. Finally, it is imperative that owners be made aware of the possibility of recontamination, especially in hunting dogs that are frequently in contact with small wild mammals. CVJ

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