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MICROSPORUM CANIS SCALP RINGWORM:
ITS PRIMARY OR SECONDARY ECTOTHRIX CHARACTER

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Abstract

This study supports the view that, in cases of tinea capitis due to a Microsporum canis infection, ectothrix arthroconidium formation is extrapilary and arises from intrapilary hyphae. The hyphae of M. canis perforate and digest the hair cuticle to alter its appearance from a normally identifiable structure of imbricated cells with a distal free border, to a grossly altered and pathological layer. Conidium production mainly takes place outside the hair shaft and forms thick clusters between the cuticular tiles. Finally, a shaft of conidia is formed around the hair. The cuticular covering of such a conidium sheath belongs to the root sheath of the hair follicle, and not to the hair structure proper.

Key Words: Microsporum canis, tinea capitis, scalp ringworm, hair cuticle, ectothrix, endothrix, scanning electron microscopy.

Introduction

Shelley et al. (1987) recently departed from tradition by claiming that all arthroconidia in Microsporum canis infection of the scalp are initially of an endothrix type. These conidia are said to arise exclusively in the hair shaft in a subcuticular or cortical position. They then achieve a secondary ectothrix character after traumatic rupture of the encapsulating hair cuticle. Their studies and interpretations were based mainly on scanning electron microscopy (SEM) with freeze-fracture. Two of their statements were puzzling to us, namely to speak of subcuticular ectothrix (a contradictory assertion), and to refer to the hair cuticle as a thick wrapping of multiple layers when it is normally only one cell thick, but layered purely through overlapping (Montagna and Van Scott, 1958; Rook and Dawber, 1982).

Arthroconidium production as a sheath in the follicle depths was regarded in Sabouraud’s laboratory (Rivalier, 1953) as having two origins: a peripilar, mosaic-like fragmentation of a fungal mantle, extending down between the cuticular layers of the hair and the inner root sheath; and production of conidia in a similar position derived from intrapilary hyphae lying in a transverse feltwork above Adamson’s fringe.

Both sources of conidia were formed from mycelium which rapidly lost its identity. Kligman (1955) could only find evidence for the second of these two sources of conidium sheath production, and clearly stated that conidium-formation was extrapilary, arising from intrapilary hyphae.

In our studies of M. canis infection of the follicle, examined with the SEM we offer a reinterpretation of Shelley’s findings, and describe the pathology of the hair cuticle in this infection. Of the views presented above, we found evidence supporting Kligman’s view alone.

Materials and Methods

Several patients infected with M. canis were previously examined (Findlay, 1974; Vismer and Findlay, 1988). Direct microscopy using Azo black (Chlorazol Black E, Gurr), for fungi present in skin (Burke and Jones, 1984), is successfully applied to hairs to detect
Figures are light micrographs (Figures 1 and 2) and scanning electron micrographs (Figures 3 to 10).

**Figure 1.** Final stages of a *Microsporum canis* infection of a hair. A sheath of conidia are present around the hair shaft.

**Figure 2.** Developmental stages of a *Microsporum canis* infection. Thalloconidia are being produced from intrapilar hyphae (arrow). hs: hair shaft.

**Figure 4.** Broken hair strand revealing the position of the peripilar layer of spheroid conidia in a hair infected by *Microsporum canis*. irs: inner root sheath.

**Figure 5.** Conidia held together by thready connections. It may represent the remains of the plasmodesma between the conidia, before final segmentation (arrows).

**Figure 6.** An infected hair with the inner root sheath (irs) partially intact showing the non-imbricated multilayered cellular arrangement, i.e., puff-pastry. Part of the hair cuticle (hc) is uncovered. The marked area is enlarged in Fig. 7, and the arrow indicates the direction of hair growth.

Fungal hyphae and ectothrix arthroconidia produced by *M. canis* and other similar fungi. Normally only the final stages of the infection are seen. During the routine examination of such cases, some were in the developmental (early) stages of the infection. This prompted us to examine infected hairs by means of SEM, with particular interest in the role the cuticle play in the infective process. After selecting such a case, plentiful material was derived from a 9-year-old white girl by manually plucking infected hair from active scalp lesions with a pair of tweezers. Apart from her tinea capitis, the girl was in good health.

Using a stereomicroscope, infected hairs were selected from the hair sample of the patient for SEM. Hairs were placed in a glass petri-dish in a desiccator, pre-fixed at room temperature for 12 hours in 1% osmium tetroxide (OsO₄) fumes, followed by an 8-hour fixation in the fumes of 25% glutaraldehyde, without vacuum. Some of the hairs were either broken, twisted or bent sharply before mounting on copper stubs and sputter-coating with gold. A JEOL JSM35 SEM was used.

**Results**

In the final stages of hair infected with *M. canis* examined by light microscopy, a sheath of conidia is formed around the hair shaft (Figure 1). In the early stages of the infection, intrapilar hyphae appear to be the origin of the conidia and their formation disrupt the normal structure of the hair (Figure 2).

On SEM, several features of the infective process can be demonstrated in more detail.

**Conidium sheath**

The spheroid arthroconidia form a distinctly peripilar layer. These lines of conidia may settle into smooth concavities or troughs in the disrupted cuticle bed (Figures 3 and 4). The conidia are seemingly held
Figure 7. Partially intact hair cuticle (hc), imbricated cuticular cells with free distal borders. irs: inner root sheath.
Figure 8. Disrupted, swollen and retracted hair cuticle cells are depicted. Cuticular imbrications as well as the roughened cell surfaces in places, are evident (large arrows). Subcuticular hyphae (h) are seen to produce conidia in places (small arrow); co: cuticular hole with a thickened collar around it; c = conidia.

Figure 9. Intact, smooth, undigested cuticular cells; roughened, partially digested cuticular cells; and cuticular cells in an advanced stage of disruption and digestion. Droplets of possible enzymatic origin as well as small depressions in the newly attacked cuticular cells (arrows), are noted.

Figure 10. An advanced stage of infection. Broad collapsed hyphae-like burrows are present and the hair cuticle is becoming unidentifiable. Cuticular cells appear retracted, with tilted, curled, pleated or lumpy surfaces. Numerous cuticular holes and cavities as well as conidia are present.

M. canis scalp ringworm

Discussion

The intrapilary hyphae of an infected hair can be differentiated into two sets: straight hyphae, descending directly, branching, and ending at Adamson's fringe, and contorted hyphae making a feltwork above the fringe, giving rise to the outer cuticular conidium sheath. This arrangement has been frequently observed by us. It has been proposed by Rivalier (1953) that the straight hyphae nourish the conidium-forming contorted branches, at the expense of which the conidium layer is formed.

Considering the classical work of Kligman (1955), describing the dynamics of the host-parasite relationship of tinea capitis due to M. canis and M. audouini by means of light microscopy, our material strongly suggests that the conidium sheath is mainly of intrapilary origin. It is not possible to place the conidium sheath other than outside the hair cuticle when seeing the pathological changes that the conidigenous hyphae cause to this layer. The subcuticular conidia as noted by Shelley et al. (1987) do in fact lie beneath a cuticle, but this cuticle belongs to the inner root sheath.

The hyphae which generate the conidia in M. canis infections are able to disrupt the hair cuticle cells, making them thicker and detaching them. Conidia apparently form in abundance through these defects. The
droplets as well as small depressions in the newly attacked cuticular cell may be due to exo-enzymatic digestion by the fungus, resulting in the eventual destruction of the cuticular layer. In addition, a large number of characteristic perforations occur through the cuticle cells, seemingly made by the conidiogenous hyphae. The production of the thalloconidia leave the hyphal structures unidentifiable.

Furthermore, compared to the scanning electron microscopy of an endothrix infection, e.g., as caused by *Trichophyton violaceum*, it clearly indicates an intact hair cuticle, and in transverse and longitudinal sections, the position of the subcuticular conidium sheath is unmistakable (Tosti et al., 1970). The conidium sheath is also held together by the plasmodesma connections between them (Tosti et al., 1970), quite similar to what is described here.

**Acknowledgements**

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**References**


**Discussion with Reviewers**

**B. Forslid**: The infected patient is a 9 year-old girl. It is well-known that *Trichophyton mentagrophytes* can only infect hair fibers from pre-adolescents but not grown-ups. Is there any difference in the capacity to infect hair fibers with your organism with respect to the age of the infected person and are the patterns of the infection the same?

**Author**: It is our experience that all children of pre-adolescent age can be infected by *M. canis*. Apart from the atypical clinical appearance that is seen in some cases of *M. canis* infections (Findlay, 1974), no obvious differences in the infective process have been noted in this age group (Vismier and Findlay, 1988). Among the anthropophilic dermatophytes, for example *T. violaceum* and *T. tonsurans*, new clinical trends are being described with regard to post-pubertal tinea capitis infections. Such infections are not only becoming more frequent, but should the infection be acquired before puberty, it shows a lesser tendency to clear up spontaneously after puberty (Herbert, 1988). On the other hand, *in vitro* differentiation between *T. mentagrophytes* and *T. rubrum* is done by inoculating sterilized hair fibers from children under the age of 10 years, where *T. mentagrophytes* shows the ability to penetrate hair while *T. rubrum* does not (Rippon, 1988). In my experience this test works equally well, irrespective of the age of the child (up to 10 years) from which the hair is collected.

**B. Forslid**: You do not mention any effects on the cortex in your paper. Is the cortex really not influenced by the infection?

**Author**: Where cortical keratin was visible through hair fracture, the horny cells appeared dissociated, and cavities or troughs were in places filled with conidia (Fig. 4). The cortex of the hair is therefore severely damaged by the fungus.

**B. Forslid**: How deep into the follicle do the conidia of the organism reach. Will they penetrate into the root area below the zone of keratinization?

**Author**: It appears that the hyphal tips of Adamson’s fringe do not penetrate into the nucleated cells at the upper limit of the keratogenous zone. Conidium formation commences in the hyphae just above Adamson’s fringe and the root of the hair remains free from infection (Kligman, 1955).

**Additional References**
