transplants where fluid exchange with the host was not impeded either by the surface epithelium or by the deep cutaneous musculature of the skin grafts. This dermal calcification was still more pronounced when skin flaps of similarly treated donors were transplanted into DHT-sensitized recipients (group 4). Here the calcification was almost as severe as in similarly treated rats in which the skin was left *in situ* (group 5). It is especially noteworthy that in groups 3, 4, and 5 the calcium deposition occurred mainly throughout the thickness of the dermal collagen (as is characteristic for this type of calciphylactic response); even the adjacent donor tissue was often calcified, while in groups 1 and 2 only occasional damaged muscle fibers underwent mineralization.

Presumably, once calciphylaxis is initiated by a challenging substance (Fe-Dex) in an animal suitably treated with a sensitizer (DHT), mineralization continues even if the challenged region is transplanted into an untreated recipient. Apparently, we are dealing with a trigger reaction which, once actively acquired, can proceed passively in the absence of such blood chemical changes (hypercalcemia, hyperphosphatemia, liberation of calcifiable matrix from the bones) as are induced by calciphylactic sensitizers.

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## Microsporum nanum: First Recorded Isolation from Animals in the United States

Abstract. *The first cases in the United States of swine infections caused by the fungus* Microsporum nanum *are reported. The infections occurred in a herd of Yorkshire swine in Pennsylvania. The dermatophyte, which readily produced experimental infections, was identified by isolation and studies in pure cultures.*

*Microsporum nanum* is a rare dermatophyte first reported from Cuba by Fuentes (*1*). The fungus, isolated from a human case of tinea capitis, was first considered to be a dwarf form of *M. gypseum*. Subsequent study of new isolates from humans led to a redescription of the fungus as a new species (*2*). Dawson and Gentles (*3*) noted that they had received cultures of *M. nanum* isolated from a group of pigs in Kenya. Recently Brock (*4*) reported the isolation of this dermatophyte from a case of tinea capitis in man in Louisiana.

Since there is only one published record of the occurrence of *M. nanum* in the United States and the African pig infections were not described, it was considered worthwhile to report on an outbreak of *M. nanum* in a herd of swine in Centre County, Pennsylvania.

A ringworm-like lesion was first noted on a 3-year-old Yorkshire sow. The lesion, measuring 13 cm in diameter, was located just caudal to the left shoulder. It had a red cast and was covered with many superficial brown crusts. The crusts were especially prominent at the periphery of the lesion, and they formed a prominent band 2.5 cm in width. The skin was somewhat rough in the infected area. There was no apparent alopecia or pruritus. Examination of other swine in the herd has revealed a number of similar lesions.

On 16 August 1963, skin scrapings were taken for mycological examination from the periphery of the lesion on the 3-year-old sow. A small portion of the scrapings was treated with 15 percent KOH and examined microscopically. Pectinate, highly branched, septate hyphae were present. These hyphae were approximately 2.5 $\mu$ wide and of various lengths (Fig. 1). A tentative diagnosis of a dermatophyte infection was made. To confirm this diagnosis, studies in culture media were initiated.

To inhibit bacterial contaminants, scrapings were incubated overnight in 1 ml of distilled water with 1000 units of penicillin and 0.1 g of streptomycin. The specimen treated with antibiotic was then placed on veal-infusion agar and incubated at room temperature for 3 days.

Ten white, rapidly growing floccose colonies appeared. Each colony was 2 mm in diameter. The undersides of the colonies were tannish-orange. During the 1st week of growth, each colony retained its floccose appearance. As the colonies aged, they became granular. The centers were cream-colored, with a buff colored periphery. The reverse side was rose-colored.

Specimens were taken from the colonies and examined microscopically. Numerous macroconidia of *M. nanum* were found. They were all thin-walled,

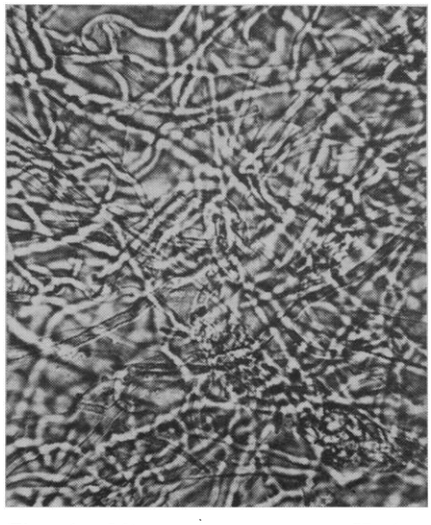

Fig. 1. *Microsporum nanum.* Hyphae present in skin scrapings. (× 400)

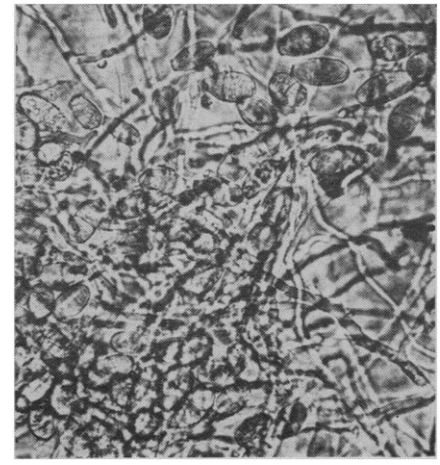

Fig. 2. *Microsporum nanum.* Specimens from veal-infusion agar showing macroconidia. (× 350)

echinulate, elliptical, truncated, two-celled structures that ranged in size from 8.4 to 12.0 $\mu$ in width, and 14.6 to 24 $\mu$ in length (Fig. 2). A few elliptical microconidia 5 by 2 $\mu$ were also noted.

The pathogenicity of the isolates was determined as follows. A shaven skin area (10 by 20 mm) on a rabbit was scratched with the point of a needle and a few drops of the fungus in a physiological saline suspension was deposited on the injured area. In 8 days, at the site of the scratched area, a lesion was noticed (27 by 15 mm). This lesion was generally red, with a zone of deeper red at the periphery approximately 4 mm wide. The lesion was rough and covered with light tan-colored flakes. The overall lesion was raised about 1 mm above the surface area of the skin. The fungus was recovered from the lesion 2 weeks later.

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28 October 1963

## Transferrins in Venezuelan Indians: High Frequency of a Slow-Moving Variant

Abstract. *In 58 percent of the Yupa Indians of Venezuela there is a slow-moving transferrin electrophoretically indistinguishable from Tf $D_{Chi}$, which to date, has only been found in Chinese. This finding is additional evidence for the existence of a racial link between South American Indians and Chinese.*

The electrophoretic study of the distribution of transferrin phenotypes in the native population of the American continent has demonstrated that the common transferrin C is the only one yet found in the Eskimos and Alaskan Indians (*1, 2*). Among the Navajos of the United States (*3*) and Lacandon of Mexico (*4*), the transferrin $B_{0-1}$ has an incidence of 7 and 17 percent, respectively. In other tribes (Itza, Lenca, Kekchi, Jicaque, Chiapaneca, and Rama of Mexico and Quiche of Guatemala) a slow-moving transferrin in heterozygous form (*2*), which has not been completely identified till now, has been found to have an incidence of 1 to 6 percent. Arends and Gallango (*5*) identified in the Irapa, Paraujano, and Macoita of Venezuela, a slow-moving transferrin that behaved as $D_1$. Fast-moving transferrins have also been found in the Quiche Indians of Guatemala, in the Tzotzil, Chinanteca, and Zapoteca of Mexico (*4*), and in the Quechua of Peru (*6*).

Transferrin $B_{0-1}$ is apparently a mutation peculiar to Indians that live in the northern part of the American continent (*7*). In some instances, admixture with neighboring populations might explain the existence of aberrant variants (*8*), but this is not the case for other tribes (*5*). Therefore, these differences in populations, that presumably belong to the same racial stock, indicate the importance of furthering the study of transferrins in other native populations of the American continent.

We have studied the occurrence of transferrins in 91 Yupa Indians, 69 of whom belong to the Pariri tribe and 22 to the Shaparu tribe. They inhabit the foothills of the Sierra de Perijá (latitude 9° to 11°N, longitude 72°40′ to 73°30′W) and linguistically are considered Carib. Serum samples were collected from subjects located in dwellings near the Mission of Los Angeles del Tukuku (*9*). Special care was taken not to include serums from related persons, but since these two tribes represent primitive populations on the verge of extinction, endogamy probably plays an important role in gene distribution.

Serum samples were obtained by venous puncture and tested by means of horizontal starch-gel electrophoresis, the technique of Smithies (*10*) being used with minor modifications as described previously (*11*). To identify the transferrins, $Fe^{59}$ in sulfate form was added to the serum in the proportion of 5 $\mu$c/ml. Autoradiography was performed on Ansco nonscreen x-ray film according to the method of Giblett *et al.* (*12*). The protein fractions were stained with amido black 10B.

Since the two tribes belong to the same linguistical and ethnological group, and because the difference between the frequencies obtained for each individual group was not statistically significant, the results obtained were pooled (Table 1). The difference in frequencies in the two tribes is 2.11 times the combined standard error, which is significant only at the 5-percent level; the $\chi^2$ computation for the two sets of observations gives a value of 5.71, $p > .01$. That we found a slow-moving transferrin of high frequency was remarkable;

Table 1. Transferrin frequencies in two Yupa Indian tribes.

| Tribe | No. | Phenotype | | | $p^{Tf D_{Chi}}$* | S.E. | $\chi^2$† | $P$ (d.f. = 1) |
|---|---|---|---|---|---|---|---|---|
| | | $C$ | $CD_{Chi}$ | $D_{Chi}$ | | | | |
| Pariri | 69 | 0.391 | 0.391 | 0.218 | 0.4135 | 0.042 | 2.57 | >.10 |
| Shaparu | 22 | .500 | .500 | | .2500 | .065 | | |
| Totals | 91 | .418 | .418 | .164 | .3730 | .036 | 1.06 | >.30 |

* $p^{Tf}$ refers to the observed gene frequency. † $\chi^2$ refers to departure from Hardy-Weinberg equilibrium.

Table 2. Populations with unusual frequency of aberrant transferrins.

| Transferrin | Population | Frequency (%) | Reference |
|---|---|---|---|
| $B_{0-1}$ | Navajo Indians (U.S.) | 7 | Parker and Bearn (*3*) |
| | Lacandon Indians (Mexico) | 17 | Sutton *et al.* (*4*) |
| $D_{Chi}$ | Chinese | 6 | Parker and Bearn (*13*) |
| | Yupa Indians (Venezuela) | 58 | This report |
| $D_1$ | Aborigines (New Guinea) | 18 | Barnicot and Kariks (*16*) |
| | Habe (Nigeria) | 15 | Barnicot *et al.* (*17*) |
| | Fulani (Nigeria) | 16 | Blumberg and Gentile (*18*) |
| | Aborigines (New Guinea) | 19 | Bennett *et al.* (*19*) |
| | Aborigines (Australia) | 44 | Kirk and Lai (*2*) |
| D* | Rama Indians (Mexico) | 43 | Sutton *et al.* (*4*) |

* No further subtyping.