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The epizootiology of *Dermatophilus congolensis* infection (A discussion article)

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RÉSUMÉ

Epidémiologie de la dermatophilose *(Dermatophilus congolensis)*

L'épidémiologie de la dermatophilose (*Dermatophilus congolensis*) est discutée à la lumière des acquisitions récentes concernant tant la maladie naturelle qu'expérimentale chez les animaux domestiques et de laboratoire, ainsi que chez les volailles.

Ces investigations suggèrent que les réponses des animaux d'expérience à cet organisme diffèrent de celles des animaux élevés dans les conditions naturelles. L'idée est avancée que cet organisme est un *Dermatophilus congolensis* qui se développe, en les sensibilisant, chez les animaux dont la peau présente le micro-climat qui lui est favorable.

Les animaux qui sont exposés de façon répétée à de faibles doses de cet organisme font une maladie à évolution progressive caractérisée par une hypersensibilité différée du type tuberculinique. Des suggestions sont faites en vue de futures recherches pour préciser le mécanisme exact de la réaction des animaux lors d'infection naturelle ou expérimentale.

Animals that are repeatedly exposed to small doses of the organism develop a progressive type of disease, typified by a delayed hypersensitive response of the tuberculin type.

Suggestions are made for future research to determine the exact mechanism involved in the reaction of animals to experimental and natural infection.

The most important aspect of *Dermatophilus congolensis*, a single species belonging to the family *Dermatophilaceae*, of the order *Actinomycetales*, is the epizootiology of the disease it produces. Previous attempts (9, 37, 50, 70, 98) to explain the epizootiology of *D. congolensis* infection in domestic animals were inconclusive. This necessitates future investigations into the environment, modes of transmission, animal response and the severity of the lesions produced.

In this article, the epizootiology of *D. congolensis* infection is considered and the results of studies on the natural disease together with those obtained from experimental infection of laboratory, domestic animals and the fowl over a period of 8 years are presented. The results are evaluated and discussed in relation to those recorded by previous investigators.

Survival and invasiveness of *D. congolensis*

Dermatophilus congolensis infection occurs when the organism overcomes the three skin barriers protecting the uncornified epidermis, hair or fleece, sebaceous wax and stratum corneum (81). The skin basement membrane was also reported to be an important barrier against dermal invasion by *D. congolensis* (11). The protective role of the sebaceous film is said to be related to its mechanical properties rather than to its bacteriostatic action (81). However, ROBERTS (88) recorded that the experimental

infection of sheep with *D. congolensis* without removing the sebaceous wax film can produce scattered lesions. The hyphae of *D. congolensis* are invasive and exert mechanical force that enable them to penetrate the epidermal cells (84). No relationship was attributed to the seasonal incidence of *D. congolensis* in cattle (10). The experimental infection of rabbits and guinea-pigs in addition to mice with *D. congolensis* was successful on both scarified and non-scarified areas of the skin in which minimum damage had been produced (2, 3).

Zoospores have been found to survive under relatively unfavourable conditions, resist drying and withstand heating at 100 °C (80, 88).

The organism has been found to survive for long periods in fluid and on slope cultures (30, 54), dry soil (16, 82) and under humid conditions (54). Those views were supported by ABU-SAMRA (2) who reported that the organism survived for variable periods in liquid and solid media and resisted a wide range of incubation temperatures (23, 42 °C) and pH (5.4-9.4). He also reported that the organism could survive for longer periods in dry than humid soils. The capsule that encloses the organism was found to be resilient (84). The capsule enclosing the coccoid form of the organism is thick (fig. 1), which suggests that it may have a protective role in enabling the organism to resist unfavourable conditions (4).

It has been suggested that *D. congolensis* exists as a saprophyte (19, 77, 99). The organism can exist in the soil (28). Trials to isolate it from soil samples supporting large numbers of infected animals were unsuccessful (36, 82). However, the success of BIDA and DENNIS in demonstrating the presence of *D. congolensis* in soil samples collected during the dry season is in accordance with the findings of ROBERTS (82).

Numerous investigators have suggested that organism lives as a skin commensal and that infection does not occur until the skin is damaged (29, 31, 99). However, MACADAM (52, 53) believes that the organism can not exist as a skin commensal.

Susceptibility

The susceptibility of animals to infection with *D. congolensis* has received the attention of many workers. Hereditary susceptibility was suggested by KELLEY *et al.* (38). Individual susceptibility was described by MORNET *et al.* (59). Different opinions have been expressed regarding breed, age and sex susceptibility. White Fulani cattle (22), zebu breeds (16) and exotic breeds (16, 45) were found to be susceptible to infection with *D. congolensis*. N'DAMA and MUTURA breeds were reported to be resistant (11, 16, 23, 42). Adult cattle are more susceptible than young ones (45). However, MACADAM (53), OPPONG (70), STEWART (98) were of the opinion that there is no age susceptibility and that adult and young animals are equally susceptible. Both male and

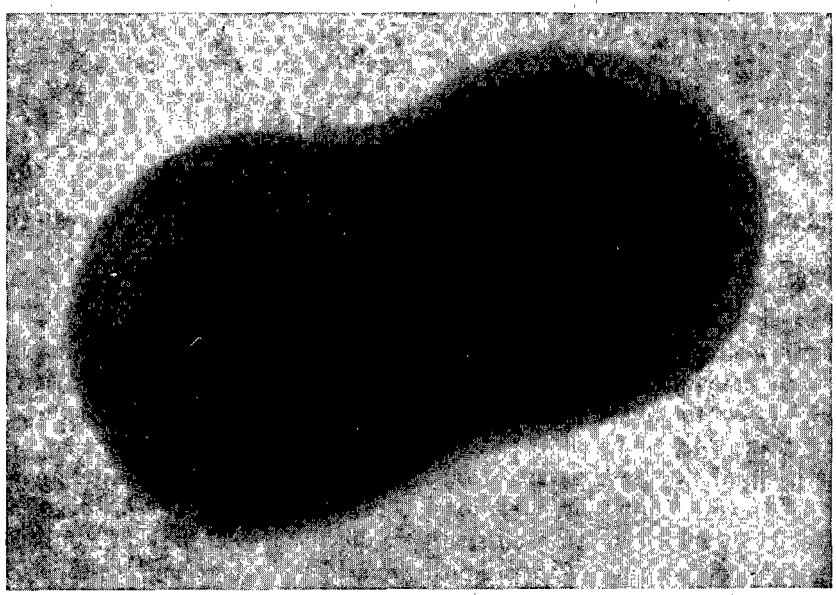


Fig. 1. — Non-flagellate coccus with a thick capsule showing division into two daughter cells in a smooth mucoid colony of *D. congolensis* on brain heart infusion agar. TEM × 13 670.

female animals have been found to be equally susceptible (1, 53, 61), but LLOYD (45) believed that males are more susceptible than females.

Some workers have suggested that the organism is an opportunist and only produces clinical infection when there is increased susceptibility of the host resulting from nutritional deficiency (26, 44, 64). The susceptibility of animals to infection has been found to increase by the presence of concurrent infections, such as rinderpest (33), lumpy skin disease (93, 98), trypanosomiasis, bensonitis (98), strongylosis, babesiosis and histoplasmosis (44) and splenectomized goats and sheep that have Orf infection (62). In the Sudan, the cattle were under the stress of continuous nomadic migration and suffered from generalized or localized debilitation due to blood and internal and external parasitic diseases (5, 13), cutaneous farcy (60) and Demodex infestation (6). In an outbreak of mycotic dermatitis in sheep in Britain, it was interesting to note that all the animals affected were ewes in late pregnancy and lambs that had simultaneous Orf and strawberry foot rot. The majority of the affected horses were also suffering from other ailments (5).

The premedication of mice with steroids increased the severity of the lesions when *D. congolensis* was inoculated intravenously, intraperitoneally and subcutaneously and when the organism was applied on the scarified and non-scarified skin (3). This was due to the anti-inflammatory action of the steroid and the suppression of white cells resulting in an increased susceptibility of mice to infection and allowing the invasiveness of *D. congolensis* to proceed with less check by the host. This was in contrast with the finding of MERKAL *et al.* (58) who reported that the premedication of rabbits with a steroid did not alter the lesions caused by *D. congolensis* and to the findings of SANSI (91) who recorded that steroid therapy did not appear to potentiate the infectivity of *D. congolensis* in the domestic fowl. However, ABU-SAMRA *et al.* (7) reported that the domestic fowl is refractory to infection with *D. congolensis* when applied on the skin.

Carrier animals

Animals with chronic infections are probably responsible for the survival of the organism within a flock or herd during dormant periods and act as a reservoir for clinical infection at the beginning of an outbreak (15, 17, 88, 89, 98). This probability was supported by ABU-SAMRA (5) who reported that cattle with chronic infection suffered from extensive lesions for more than one year. Infection was confirmed during the dry season which is considered by previous workers to be a dormant period.

Factors involved in the transmission of *D. congolensis*

Rain and humidity

The marked association between skin wetting and the clinical infection with *D. congolensis* may have several explanations. Wetting of the scab causes the release of zoospores (82); their subsequent emergence from within the scab is said to be accelerated by a negative chematactic response to their own endogenous carbon dioxide (83, 88). Water may serve as a medium for the transmission of zoospores from one part of the body to another, leaching out the wax, macerating the stratum corneum and facilitating the anchorage of the zoospores to the skin (88).

The possible role of atmospheric humidity and moisture as an important factor in the epizootiology of the disease has received the attention of many workers. It was suggested that humidity has no role in the pathogenesis and epizootiology of *D. congolensis* infection (47). He found that the lesions could heal under humid conditions more rapidly than under dry conditions. However, OPPONG (69) and VANDEMAELE (99) reported that humidity aids in the production of infection. The effect of humidity and moisture as important factors in the skin microclimate of the host and its relationship to the organism, was reported (14, 34, 96). An increased incidence of the disease was reported among sheep with fine rather than coarse fleece (96).

Similarly, many workers have reported an increased incidence of *D. congolensis* infection in the rainy season (9, 19, 22, 25, 27, 28, 43, 46, 57, 92, 95, 97, 98).

Transmission of infection can occur through dipping fluids (41). Wetting of the skin of rabbits helped in the transmission of *D. congolensis* infection with flies (78). Only a small percentage of animals was found clinically infected during the dry season (Summer) in the Sudan and in Britain the outbreaks tend to occur after rainy weather (5). In an earlier study covering

the same areas in the Sudan a high incidence of the disease was recorded in the rainy season (97).

Contact

Some workers have reported that the disease can not be transmitted by contact (19, 56, 97), whereas others such as AUSTWICK (14) and LERICHE (42) have reported that transmission might occur between animals particularly when their coat is wet.

Ticks

Ticks have been incriminated to play an important role in the epizootiology of the disease, either by breaking the skin barriers and/or transmitting the organism (9, 27, 39, 43, 45, 46, 49, 52, 56, 68, 69, 71, 74, 88, 97, 98, 99). *Dermatophilus congolensis* infection was transmitted from cattle to rabbits with *Amblyomma* ticks (48). The organism was isolated from the tick, *Hyalomma asticum* (40) but BIDA *et al.* (16) failed to isolate it from ticks. In the Sudan, both clinically infected and healthy cattle had variable degrees of infestation with ticks of the genera *Amblyomma*, *Hyalomma* and *Boophilus* (5). The role of ticks especially *Amblyomma* which flare up during the rainy season may be instrumental in spreading the disease by providing portals for entery to *D. congolensis* and probably by exciting granulomatous sores through allergy to tick proteins (1).

Flies

Flies and biting insects such as *Stomoxys calcitrans*, *Glossina morsitans*, *Musca domestica* and mosquitoes have also been reported to be involved in the transmission of the disease by breaking the skin barriers during feeding and releasing variable amounts of serum and blood which provide moisture, nutrition and a suitable microclimate for the multiplication of *D. congolensis* (2). Rabbits were successfully infected with contaminated flies (78). Many other workers have also stressed the role of flies in the epizootiology of the disease (9, 28, 43, 51, 52, 53, 66, 68, 88, 95, 98).

Lice and mange mites

Lice and mange mites such as *Demodex* and *chorioptes* have been suspected to help in the establishment, transmission or spread of *D. congolensis* (52, 59, 68, 70, 90, 94).

In the Sudan, various genera of the flies *stomoxys*, *Tabanids* and *Glossina* as well as lice inhabited the areas where *D. congolensis* infection was prevalent (13).

Wounds

The skin barriers can be damaged by various agents which help in the establishment of the disease. These agents are exemplified by the Ox-pecker bird (16, 35, 52, 66, 68), shearing injuries (88) ; truma associated with concrete floors and sharp stones (5, 64, 65). Branches of trees and spines of thorny bushes (2, 32, 66, 68, 97, 100). In all cases, the serum or blood released by such injuries attracts flies and provides a suitable microclimate and nutrients for *D. congolensis*. Such injuries might provide a portal for entery of the organism from the surroundings.

Fig. 2. — Confluent lesions of cutaneous streptothricosis in a cow.

Natural infection

The disease observed under natural conditions was reported to be quite severe and extensive (5) and is characterized by confluent (fig. 2) thick, multistrata scab spreading to involve a large area of the skin of cattle, horses and sheep with mycotic dermatitis and strawberry foot rot (fig. 3).

Thick whorling layers of scab composed of alternating strata of keratin, shed necrotic epithelial debris and leucocytes with many mycelia of *D. congolensis* (fig. 4) were seen in skin sections prepared from the natural disease in cattle. Under the scab the stratum corneum had undergone degenerative and necrotic changes and was infiltrated with neutrophils and lymphocytes. A granulomatous reaction characterized by lymphocyte, macrophage, epithelioid and giant cell infiltration was noticed in deeper layers in addition to proliferation of fibrous tissue (8).

Experimental infection

In spite of the numerous factors that were suspected to be instrumental in the spread of the disease, the author is not aware of any

Fig. 3. — Lamb with strawberry foot rot showing severe proliferative lesions at the coronet.

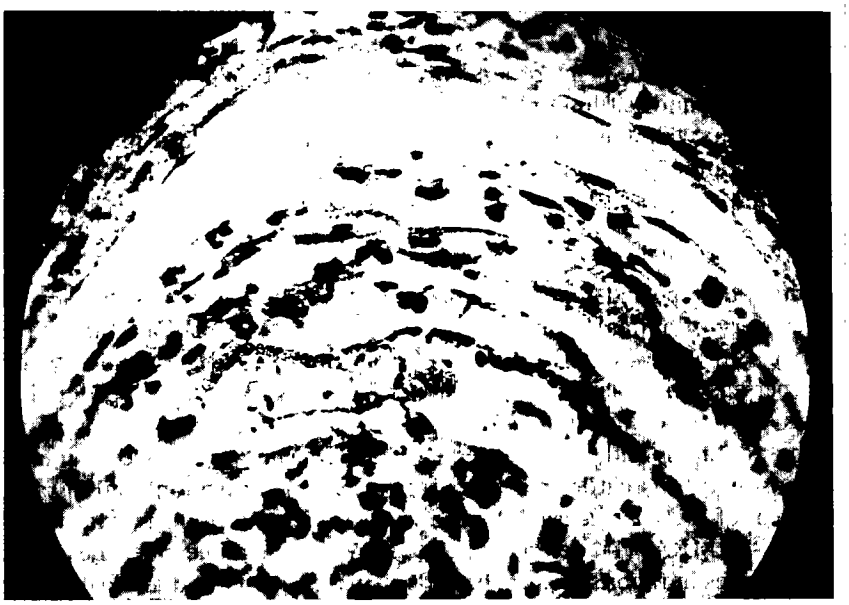


Fig. 4. — Thick whorling layers of scab from a field case of bovine streptothricosis showing *D. congolensis* mycelia dividing longitudinally and transversely. Giemsa × 1 400.

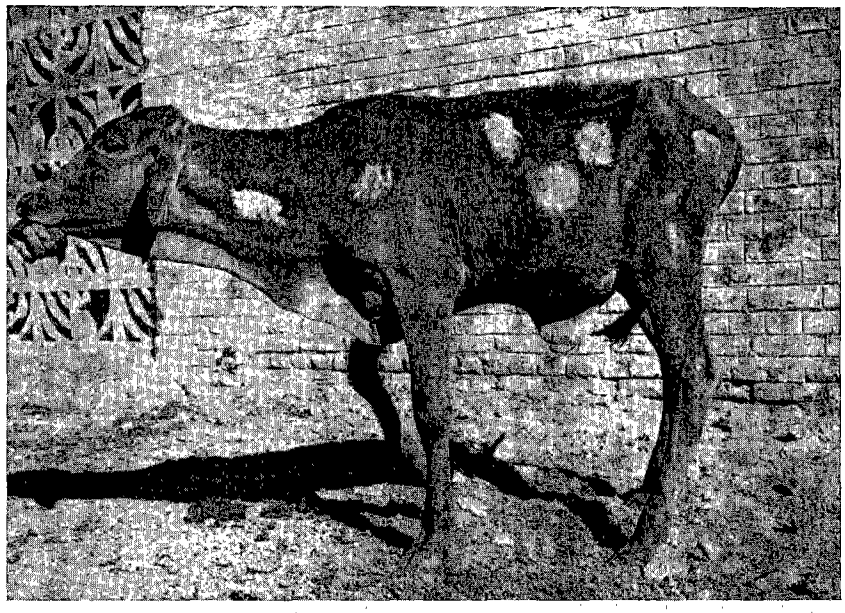


Fig. 5. — Calf 10 days after infection by scarification showing localized non-spreading lesions covered with thick scab on different areas of the skin.

adequate explanation for the complex problem involved in the disease.

Many workers have attempted experimental infection of various species of animals with *D. congolensis* by skin scarification and application of the organism (7, 17, 19, 38, 50, 52, 55, 67, 73, 85) ; on the defatted skin after being clipped or shaved (67, 81, 85) ; by the intradermal inoculation of *D. congolensis* and complete Freund's adjuvant (7) ; by the application of the organism along skin incisions or on minute lesions made by needle jabbing (17, 47), by contaminated ticks and flies (48, 78).

All these workers succeeded in producing localized, non-spreading and self-limiting lesions (fig. 5). It appears that all their attempts to produce generalized and spreading lesions as encountered in natural field cases have failed (7).

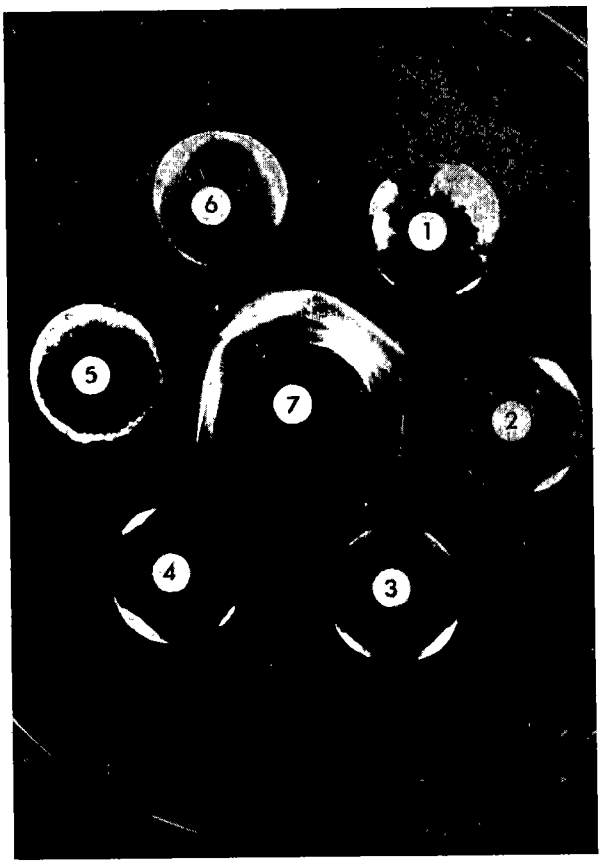
Hypersensitivity and immunity

Hypersensitivity was reported to play a role in the speedy recovery of animals after reinfection (18, 20, 86, 88). However, BIDA (16) believed that hypersensitivity played no role in the pathogenesis of bovine infection with *D. congolensis*. The authors did not observe delayed hypersensitivity in cattle inoculated with killed antigen of *D. congolensis*. On the other hand, ABU-SAMRA (1) demonstrated hypersensitive responses in rabbits, goats, sheep, donkeys, calves and camels. When those experimental animals were challenged three weeks after infection, mild or no lesions developed (1, 7).

Many workers advocated successful immunization of cattle against *D. congolensis* infection by using whole-cell antigen (20, 21, 75, 76). However, other workers reported that neither infection nor vaccination protects animals against reinfection (17, 18, 52, 58, 63, 66, 72, 78, 79, 86, 87, 88).

More severe lesions were produced in lambs by a combination of Orf virus and *D. congolensis* than by either agent alone (2). This suggested that *D. congolensis* may exist as an opportunist. When the lambs were challenged, circulating antibodies were demonstrated serologically by gell diffusion (fig. 6) and by immunoelectrophoresis. When the animals were skin tested an immediate as well as delayed-cell mediated hypersensitivity was noticed.

Rabbits and guinea-pigs that were previously infected after skin scarification and the application of *D. congolensis* as well as others that were immunized with killed whole cell antigen of the organism developed quick lesions characterized by severe inflammatory changes and rapid healing (2). Deep inoculation of *D. congolensis* in the foot pad of rabbits resulted in severe lesions. When skin tested the rabbits showed an immediate, delayed-cell mediated hypersensitivity and an Arthus immunocomplex reaction (2). Circulating antibodies were also present in the sera of those animals (fig. 6).



Immunodiffusion

Fig. 6. — 1. Serum, guinea pig, immunized with coccoid form isolate 1 and challenged with *Dermatophilus*. 2. Serum, field case, mycotic dermatitis. 3. Serum, field case, mycotic dermatitis. 4. Serum, rabbit, infected twice cutaneously with *Dermatophilus*. 5. Serum, rabbit, injected deeply with *Dermatophilus* into the foot pad and challenged cutaneously. 6. Serum, lamb, infected with Orf and *Dermatophilus* and challenged with *Dermatophilus*. 7. Soluble antigen, coccoid form, isolate 3.

However, in the sera from naturally infected animals (2) only faint or no precipitin lines were demonstrated (fig. 6). High levels of γ-globulins were demonstrated in the sera of naturally infected cattle but no evidence for their protection against the disease was found by AMAKIRI (12). He also reported that the natural resistance of N'Dama cattle is probably due to the high percentage of lymphocytes and neutrophils in the healthy skin of those breeds.

The establishment of various allergic responses and the demonstration of circulating antibodies probably accounted for the change in reaction after reinfection of experimental animals with *D. congolensis*. One probable explanation for the difference in the reaction between field and experimental animals may have been caused by the large size of the inoculum (2).

Alikely hypothetical mediation for the spread of the organism to cause progressive lesions is the presence of an extrinsic or intrinsic allergen introduced with the organism from the soil or surroundings. Evidence obtained from experimental work was not in support of intrinsic factors because the lesions produced were localized, non-spreading and self-limiting (7). Conversely such intrinsic factors, if present, may occur as incomplete haptens which need to be supplemented before they could induce allergy. As many saprophytes are found in the soil and on the skin of animals, such complementary allergens may be in the form of saprophytic bacteria, fungi or any other form of protein (1). This hypothesis was tested in a limited experiment using complete Freund's adjuvant and although the cutaneous lesions regressed eventually an extensive tuberculin — type reaction was detected on histological examination (7).

The clinical manifestation of the disease under natural conditions is probably due to the existence of *D. congolensis* in the environment of

the animal, the availability of the right skin microclimate and the introduction of the organism in small doses through damaged skin barriers. The response of the sensitized animal to such repeated small doses of *D. congolensis* is by the release of the mediators of delayed hypersensitivity such as macrophage inhibition, chemotactic and lymphotoxic factors. Such a reaction mimics Type IV allergic reaction, the classical example of which is tuberculosis as described by COOMBS *et al.* (24). In support to this view ABU-SAMRA *et al.* (8) described histopathological changes in natural field cases of *D. congolensis* infection in cattle indicative of a progressive disease of the delayed tuberculin — type hypersensitivity.

A possible diagramatic illustration of the epizootiology of the disease is shown in (fig. 7).

In conclusion, past and present investigations highlight the need for further research, which is in progress, into the immunology and allergic hypersensitive responses under experimental and natural conditions. This includes immunological studies, as well as skin testing at different stages in the disease process, with parallel investigations on passive and active (humoral and cellular) immunization of experimental animals in order to clarify the exact nature of the responses in different animal species, at different stages of *D. congolensis* infection.

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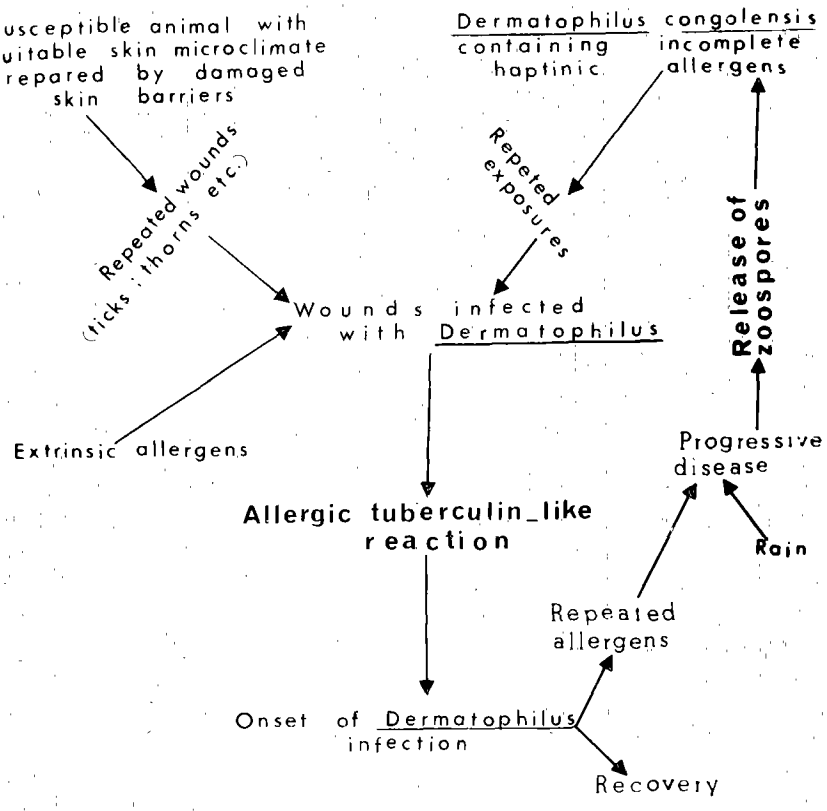

Fig. 7. — A possible diagramatic illustration of the epizootiology of *Dermatophilus congolensis* infection.

SUMMARY

The epizootiology of *Dermatophilus congolensis* infection is discussed, in the light of close observations on the natural disease and the experiments performed in domestic, laboratory animals and the fowl. These investigations suggested that the responses of experimental animals to the organism differed from

those under natural field conditions. The view is put forward that the organism is an apportunist to which animals with the right skin microclimate become sensitized.

RESUMEN

Epidemiologia de la dermatofilosis *(Dermatophilus congolensis)*

Se discute la epidemiologia de la dermatofilosis (*Dermatophilus congolensis*) a la vista de las adquisiciones recientes concernientes a la enfermedad natural tan bien como experimental en los animales domésticos y de laboratorio, y en las aves de corral.

Estas investigaciones sugieren que las reacciones de los animales de experiencia para con dicho organismo difieren de las de animales criados en las condiciones naturales. Se emite la idea que este organismo es *Dermatophilus congolensis*.

Los animales frecuentemente expuestos a pequeñas dosis de este organismo desarrollan un tipo progresivo de enfermedad.

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