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Source: Journal of Wildlife Diseases, 21(2) : 186-188

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-21.2.186

Journal of Wildlife Diseases, 21(2), 1985, pp. 186–188

Progressive Digital Necrosis in the Eastern Blue-tongued Skink, *Tiliqua scincoides* (Shaw)

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A private reptile breeder in Sydney, Australia reported that following the introduction of an adult eastern blue-tongued skink with several toes missing, into a colony of eight adult skinks, all eight adults and subsequent generations developed progressive digital necrosis (toe-rot). The condition had not been seen previously in the colony since its establishment 3 yr earlier. The skinks were housed in a moist environment in an enclosure comprising grassed areas, soil and rockeries.

Toe-rot in young skinks first became apparent following first ecdysis, approximately 6 wk after birth and the condition was always associated with failure to shed skin from the terminal digits. Subsequent constriction of the terminal digit resulted in a swollen, gangrenous toe which eventually sloughed and was followed by a progressive necrosis. This dermal necrosis progressed for some months with eventual loss of toes and in many cases loss of feet, resulting in a stump which appeared dessicated and covered with bran-like flakes. Two of three unaffected adult skinks introduced into the colony did not develop the condition. However, subsequent generations were found to be susceptible.

Skin scrapings, toes and swabs from the affected feet of one skink and an additional two live, affected skinks were submitted to the Central Veterinary Laboratory, Glenfield for examination. Progressive digital necrosis was evident in all four feet of the live skinks. From the live skinks submitted, skin scales from affected feet were removed for culture and microscopic examination.

The two toes submitted were fixed in 10% neutral buffered formalin and processed for light microscopic evaluation. Histologic sections were stained with hematoxylin and eosin, by the periodic acid Schiff method (PAS) and by Gram. Skin scrapings, scales and swabs of digital lesions were cultured on blood agar (Blood Agar Base No. 2, Oxoid Ltd. Basingstoke, Harts., United Kingdom) containing 7% citrated sheep blood, on Sabouraud Dextrose Agar (Oxoid Ltd. Basingstoke, Harts., United Kingdom) and Mycosel Agar (Baltimore Biological Laboratories, Cockeysville, Maryland 21030, USA), and incubated to 37 C and 30 C for bacteria and fungi respectively.

On primary bacterial culture of toe swabs, scales and skin scrapings, *Staphylococcus epidermidis* and a *Bacillus* sp. were isolated in sparse growth. Culture on Sabouraud and Mycosel agar resulted in a profuse pure growth of *Trichophyton terrestre* from each affected site in all skinks examined. Wet preparations of skin scales (KOH/blue black ink) revealed abundant fungal hyphae throughout. The unaffected abdominal area of one lizard was cultured to see if *T. terrestre* was a common skin contaminant. This resulted in the isolation of a *Penicillium* sp., but not *T. terrestre*.

Histopathology of affected toes revealed a severe chronic necrotizing dermatitis with necrosis occurring deep into the subcutis. There were numerous sub-

Received for publication 27 January 1984.

acute to chronic inflammatory cell foci and widespread fibrosis. Stained sections (PAS and Gram) revealed abundant fungal hyphae distributed throughout the necrotic tissue as well as in dense accumulations in the more superficial areas (Fig. 1). Occasional superficial clumps of large Gram positive bacilli, consistent with *Bacillus* sp., were also seen.

Toes from a clinically indistinguishable case, geographically isolated from the above, were also submitted for histological examination. Stained sections revealed lesions appearing similar to the earlier submissions, with fungal hyphae demonstrable by PAS throughout the necrotic tissue. Material from this case was not cultured for bacteria or fungi.

The toe-rot condition appears to be common in eastern blue-tongued skinks seen at Taronga Zoological Park, Sydney (Reddacliff, pers. comm.) but its etiology has not been previously investigated. Similar cases have earlier been attributed solely to dysecdysis whereby failure to shed scales around the toes results in constriction as the skink grows, with resultant ischemic necrosis and loss of digits (Reddacliff, 1981, *In* Post Grad. Comm. Vet. Science, University of Sydney, Proc. No. 57 Dermatology, p. 415).

The dermatophyte *T. terrestre* is usually regarded as a contaminant and many "human" isolates are the result of environmental contamination at the time of sampling (Gip, 1968, Acta Dermatovenerol. Jugosl. 48: 19–25). However, it is capable of degrading human hair (Hsu and Volz, 1975, Mycopathologia 55: 179–183) and has been isolated from a human cutaneous mycotic granuloma (Grimmer, 1974, Mykosen 17: 333–338). *T. terrestre* has been described as part of the cutaneous flora of hedgehogs (Marples and Smith, 1962, Sabouraudia 2: 100–107), but in certain circumstances has been considered pathogenic to animals (Gupta et al., 1970, Indian J. Anim. Health 9: 85). Connole (1977, *In* Recent Advances in Medical and Veterinary Mycology, Iwara (ed.), Univ. of Tokyo Press, Tokyo, p. 125) reported an association between *T. terrestre* and ringworm-like lesions in both cattle and horses, while Scott et al. (1980, J. Am. Anim. Hosp. Assoc. 16: 53–59) isolated *T. terrestre* from dermatitis lesions in a dog and a cat, and demonstrated fungal elements in biopsy specimens from the dog. They concluded that *T. terrestre* was the causative agent in both cases.

FIGURE 1. Histological section through epidermis and dermis of affected toe of an eastern blue-tongued skink (*T. scincoides*) showing superficial necrosis and deeper infiltration with numerous hyphae of *Trichophyton terrestre* (arrows). PAS, ×300.

The significance of our isolation of *T. terrestre* is thus equivocal. The presence of fungal hyphae in necrotic tissue and superficial areas does not necessarily indicate a pathogenic role for this organism, as its presence could be the result of saprophytic invasion of a pre-existing lesion. Ischemic tissue following dysecdysis could offer this organism such a site for establishment. It is possible that once established, the fungus may play a role in the further development of progressive digital necrosis in *T. scincoides*. The cases reported here are, to our knowledge, the first reported isolations of *T. terrestre* from a pathological condition in reptiles. If *T. terrestre* is the principal agent in the condition described, then the blue-tongued skink may represent a natural host for this dermatophyte.

The authors wish to thank Dr. D. Frey of the Australian National Reference Laboratory in Medical Mycology, Royal North Shore Hospital, Sydney, for confirming the identification of *T. terrestre*, and Mr. R. Gates for his assistance in providing a detailed history of the skinks and materials for examination.

Journal of Wildlife Diseases, 21(2), 1985, pp. 188–190

Increased Mortality in Gray Wolves Captured with Acepromazine and Etorphine Hydrochloride in Combination

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Free-ranging gray wolves (*Canis lupus*) have been immobilized successfully with phencyclidine HCl (Sernylan®, Bio-ceutic Laboratories, Inc., St. Joseph, Missouri 64502, USA), ketamine HCl (Vetelar®, Parke-Davis Co., Detroit, Michigan 48232, USA), and etorphine HCl (M-99, D-M Pharmaceuticals, Inc., Rockville, Maryland 20850, USA), from a helicopter and after trapping (Seal et al., 1970, Int. Zoo. Yearb. 10: 157–170; Ballard et al., 1982, J. Wildl. Dis. 18: 339–342). Frequently, the use of immobilizing drugs is supplemented with tranquilizers to reduce excitability or convulsions during immobilization. This report examines the deaths of four wolves following immobilization with a combination of etorphine HCl (EH) and acepromazine (Acepromazine, Ayerst Laboratories, Inc., New York, New York 10017, USA) (AP) delivered from a helicopter.

All wolves were immobilized with either 2.5 mg EH or a combination of EH and 5 mg AP administered in 3 ml darts fired with a Cap-Chur gun (Palmer Chemical Equipment Co., Douglasville, Georgia 30134, USA) from a helicopter (Ballard et al., 1982, op. cit.). Mortality rates of wolves immobilized with either EH or EH/AP were compared with those immobilized with phencyclidine HCl and promazine HCl (Sparine®, Wyeth Laboratories Inc., Philadelphia, Pennsylvania 19105, USA) (PP/HCl). All wolves were captured in the Susitna and Copper River Basins, an area which has been thoroughly described elsewhere (Skoog, 1968, Ecology of the caribou (*Rangifer tarandus granti*) in Alaska, Ph.D. Thesis, Univ. California, Berkeley, California, 699 pp.; Bishop and Rausch, 1974, Nat. Can. (Que.) 101: 559–593; Taylor and Ballard, 1979, Proc. 15th N. Am. Moose Conf. Workshop, Kenai, Alaska. pp. 169–186).

During the period 1982–83, 27 wolves were immobilized with the EH/AP combination. Of that total, four (15%) died immediately after immobilization. A juvenile female immobilized within 2 min after an injection of EH/AP with a total chase time of 20 min died in 5 min. A thermometer was not available, so exact body temperature was unknown, but it appeared elevated based on physical examination. Onset of rigor mortis was rapid, also indicating an elevated body temperature. An adult female receiving the same combination of drugs was immobilized within 4 min of drug injection. Chase time was 30 min. During anesthesia, the wolf had severe convulsions and died within 15 min. Rigor mortis also occurred

Received for publication 26 October 1983.