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Review of animal mycoses in Australia

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Abstract

This review covers the available literature on the mycoses of animals in Australia since the last review published in 1967. Of the cutaneous infections, dermatophytoses have been recorded in a wide range of animals: cattle, horses, goats, pigs, sheep, cats, dogs, mice, guinea-pigs, rabbits, a lion, kangaroos, a camel, koalas and wallabies. These infections were caused by several species and varieties of the genera, *Microsporum* and *Trichophyton*. Eight agents of ringworms have been recorded in the horse. Two subcutaneous mycoses, phaeohyphomycosis and sporotrichosis have been reported. Phaeohyphomycosis is becoming more common but sporotrichosis is rare having been recorded only once in a cat.

The following systemic mycoses have been recorded: adiaspiromycosis, aspergillosis, candidiasis, cryptococcosis, dactylariosis, fusariomycosis, histoplasmosis, miscellaneous mycoses, mycotic abortion and related conditions, zygomycosis, pythiosis, protothecosis and green algal infections. Cryptococcosis has affected 11 different animal species. Mycotic abortion is a serious disease in Victoria. Pythiosis of horses has been extensively studied in northern Australia.

Abbreviations used: NSW, New South Wales; NT, Northern Territory; SA, South Australia; WA, Western Australia; NZ, New Zealand; CDC, Centers for Disease Control, Atlanta, USA; CMI, Commonwealth Mycological Institute, Kew, England; CNS, central nervous system

Index

Introduction

The mycoses of animals in Australia were reviewed by Connole & Johnston (1967) [10] who included the available literature and reports. The present review will attempt to cover the subsequent literature. For completeness some unpublished reports are included. In the case of mycoses not reviewed previously, for example, zygomycosis and pythiosis all the literature will be reviewed. In general, the topics will be treated in chronological order.

The mycoses will be considered under three sections:

I. *Cutaneous infections* which include dermatophytosis, caused by imperfect states (anamorphs) of the three genera, *Epidermophyton*, *Microsporum* and *Trichophyton*. There is no record of animal infection in Australia caused by species of *Epidermophyton* although rare infections have been described in other countries.

II. *Subcutaneous mycoses* include infections in which a lesion develops at the inoculation site. In animals in Australia, two of these mycoses have occurred, phaeohyphomycosis and sporotrichosis.
Phaeohyphomycosis is the name given to those subcutaneous and systemic diseases caused by various black moulds that develop in tissue in the form of dark-walled, septate mycelium (Ajello *et al.*, 1974) [2].
Sporotrichosis which is caused by *Sporothrix schenckii*, is usually a chronic pyogranulomatous infection originating by a wound, where a lesion develops. Local lymphatics may become involved.

III. *Systemic mycoses*. These mycoses will be treated in alphabetical order as adiaspiromycosis, aspergillosis, candidiasis, cryptococcosis, dactylariosis, fusariomycosis, histoplasmosis, miscellaneous mycoses, mycotic abortion and related conditions, zygomycosis, pythiosis, and protothecosis and green algal infections.

References will be listed after each of the sections.

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SECTION ONE: CUTANEOUS MYCOSES

Dermatophytoses

Previous isolations of dermatophytes [11] include: cattle, *Trichophyton verrucosum*; horses, *T. equinum* var. *autotrophicum*, *T. mentagrophytes* var. *quinckeanum*; sheep and rabbits, *Microsporum canis*; goats, *T. mentagrophytes* var. *mentagrophytes*; pigs, *M. nanum*; camel and rat, *M. gypseum*; cats and dogs, *M. canis*, *M. gypseum*; kangaroo, guinea pig, rat, mouse, *T. mentagrophytes* var. *mentagrophytes*; and mouse, *T. mentagrophytes* var. *quinckeanum*.

Cattle

The common cause of bovine ringworm in Australia is *T. verrucosum* [11]. Although cattle have often been cited as the source of human infection with *T. mentagrophytes* [17, 35] this dermatophyte has apparently not been isolated from cattle in Australia. However McAleer (1980) [33] isolated *T. mentagrophytes* from at least 13 dairy farmers with lesions on their hands and wrists, and from several patients who were sure they had contracted the infection from cattle. On two occasions, men who skinned cattle at abattoirs contracted infections, possibly from the animal hides. *T. mentagrophytes* has occasionally been recorded from cattle in the United Kingdom [1] but Dawson [15] stated that in general, it was a rare cause of bovine ringworm.

An unusual cause of bovine ringworm due to *M. canis* was recorded in Tasmania (B. Peel, personal communication 1988).

In WA, bovine ringworm is not common in either animals or humans. This is probably because cattle are not housed indoors in the mild winters and there is less close contact between cattle in the open fields than indoors. Handling of cattle is also minimal and therefore any ringworm present is not readily transmitted to humans [30]. These conditions would apply equally well to cattle in Queensland.

Horses

Connole (1967) [7] reported the first case of *M. gypseum* ringworm in a horse in Queensland. The usual ringworm agent affecting horses in Queensland is a variety of *T. equinum*. Smith *et al.* (1968) [54] named this variety *T. equinum* var. *autotrophicum* because it lacked a complete requirement for nicotinic acid which is characteristic of *T. equinum* var. *equinum*, the usual cause of ringworm of horses in the Northern Hemisphere. At that time the variety *autotrophicum* had been recorded only from Australasia [54].

In SA, *M. equinum* was isolated from two horses and *T. equinum* var. *autotrophicum* from horses on several properties [51].

Pascoe & Connole (1974) [45] described *M. gypseum* infections involving 39 horses in Queensland. Moist atmospheric conditions and the presence of biting insects appeared to be factors in the spread and degree of infection in the two major outbreaks described. Lesions were generally much smaller than those associated with *T. equinum* var. *autotrophicum* and showed less scab formation and depilation.

Pascoe (1976) [42] described a survey of ringworm among horses in racing and breeding stables in the Darling Downs area of Queensland. Of 568 horses in training, 32% were clinically affected, while only 1.1% of 2535 horses on breeding farms were clinically affected with dermatophytes. Most lesions on racing horses were on the girth areas. Untreated lesions of *T. equinum* var. *autotrophicum* ringworm measured up to 35 mm. Lesions due to *M. gypseum*, *M. equinum* and *M. canis* were all smaller, usually less than 10 mm. *T. equinum* var. *autotrophicum*, *M. equinum* and *M. canis* were restricted to racing horses. *M. gypseum* occurred in racing, riding and breeding horses.

Perfect state (teleomorph) studies of 27 *M. gypseum* strains from horses in Queensland were carried out by Dr P. Stockdale of CMI and reported by Connole (1977) [9]. The results were that 24 were *Arthroderma gypsea* (then called *Nannizzia gypsea*, 1 was *A. incurvata* and 2 were *A. fulva*. *A. gypsea* occurred in Queensland soils and appeared to be the usual type affecting horses. Epidemiological studies of equine ringworm caused by *M. canis* and *M. equinum* were reported [9].

Pascoe (1979) [43] reported the epidemiology of ringworm due to *T. equinum* var. *autotrophicum* in several thoroughbred stables in southeast Queensland. The infection was readily transmitted, particularly by infected saddle-girths on which the fungus survived for 12 months. Mild abrasion from the saddle during work favoured the development of lesions and prolonged recovery. Young horses under 3 years were most susceptible and the majority of cases occurred in periods of high humidity.

McAleer (1980) [33] in a study of zoophilic dermatophytes and their natural hosts in WA, reported the first Australian case of *T. mentagrophytes* infection in a horse, and two cases of *T. equinum* var. *autotrophicum*.

In 1981 Connole & Pascoe [12] recognised *T. equinum* var. *equinum* ringworm in three different outbreaks in horses in southeast Queensland. One outbreak was in a thoroughbred stable that had a recent intake of NZ yearlings. This variety had been detected for the first time in the Southern Hemisphere in 1977 in horses in NZ [6]. In an attempt to determine its initial presence in Australia, a retrospective nutritional study was done on 38 strains collected in Queensland between 1970 and 1982, and on five strains from horses in Tasmania.

Thirty-six Queensland strains and the five Tasmanian strains were confirmed as *T. equinum* var. *autotrophicum*. The other two strains isolated in 1980 in Queensland were identified as *T. equinum* var. *equinum*. These were from horses bred in southeast Queensland. So this study did not show when or how the variety *equinum* was introduced into Australia but it was present in Queensland in 1980. Although one of the first outbreaks of *T. equinum* var. *equinum* infection was a clinically severe form, experimental infection of two horses with subcultures of the two varieties of *T. equinum* developed identical clinical responses to infection [12]. Subsequently, isolations of *T. equinum* var. *equinum* were made

from two horses in Victoria (M. Maslen, personal communication 1984) and from two horses in SA (G.W. Kaminski, personal communication 1984).

Pascoe (1984) [44] tested 12 medicaments for their efficacy in the treatment of experimental *T. equinum* var. *autotrophicum* infections in horses. Only four preparations – povidone iodine, thiabendazole ointment, captan ointment and Wellcome ringworm ointment, were satisfactory. He considered that their usefulness might be limited for treatment of large numbers of horses.

T. verrucosum was isolated from a horse in Queensland in 1984 (T. Smeltzer, personal communication).

Tasmania has a variety of causes of equine ringworm. The usual cause is *T. equinum* var. *autotrophicum* but there have been sporadic cases due to *M. equinum*, *M. canis*, *T. verrucosum* and *T. mentagrophytes* var. *mentagrophytes* (B. Peel, personal communication 1988).

In Victoria, Maslen and Thompson (1984) [38] recorded two cases of human ringworm caused by *T. equinum* var. *autotrophicum*. In one case the fungus was also isolated from the patient's horse. Treatment with econazole nitrate was successful.

Muir *et al.* (1984) [39] isolated four strains of *T. equinum* var. *autotrophicum* from lesions in humans in NSW.

Eight agents of equine ringworm have been recorded in Australia. In order of frequency of isolation they are:- *T. equinum* var. *autotrophicum*, *M. gypseum*, *T. equinum* var. *equinum*, *M. equinum*, *M. canis*, *T. mentagrophytes* var. *mentagrophytes*, *T. verrucosum* and *T. mentagrophytes* var. *quinckeanum*. The latter has been recorded only once, from SA [3].

Equine ringworm is of some economic importance because thoroughbred and standardbred horses with detectable infections are not allowed to race until the condition has cleared. Also, all eight dermatophytes are potentially transmissible to man.

Goats

References to ringworm of goats are few. Wirth [57] stated that fungal skin disease was commonly seen in Victoria where goats were kept on moist litter. Affected areas were the escutcheon and the udder. The sores had normal appearance for fungal skin lesions and the condition responded readily to soluble iodine baths. King [27] stated that ringworm appeared regularly in milking herds but also had been seen in Angoras and feral goats. The usual cause was *Trichophyton* sp. (probably *T. mentagrophytes* var. *mentagrophytes*).

In Queensland, *T. mentagrophytes* var. *mentagrophytes* is the usual cause but recently a case of ringworm due to *M. gypseum* has been recorded at the Animal Research Institute (ARI) (T. Smeltzer, personal communication 1988).

In Tasmania there have been cases of *M. canis* and an outbreak of *T. verrucosum* ringworm (B. Peel, personal communication 1988).

Pigs

The usual form of porcine ringworm in Australia is due to *M. nanum* [11]. In 1968 a Large White sow at Townsville, Queensland developed several flank lesions up to 55 mm diameter. Scabs were positive microscopically and the geophilic fungus *M. gypseum* was grown. A similar strain of *M. gypseum* grew from soil in the pig's yard. Both strains were typed by Dr P. Stockdale of CMI as *Arthroderma gypsea* (Connole 1977) [9].

In SA, *M. canis* was found in humans, cats and pigs that were closely associated [51].

In NSW, O'Keeffe [41] reported three human infections due to *M. nanum*. Two of these cases had contact with pigs. As only these three cultures had been received for identification at the School of Public Health & Tropical Medicine, University of Sydney, Sydney, over a period of 12 years, it seemed that *M. nanum* was as rare a cause of *tinea corporis* in Australia as it was in other parts of the world. O'Keeffe considered that reasons for the reported low incidence of *M. nanum* infections could be the occurrence of mild infections which healed spontaneously, or treatment topically

without attempting to isolate the causative organism.

Turner & Kaminski [55] reported two cases of human ringworm caused by *M. nanum* from SA. Pigs or soil were considered to be the source of infection in both affected children. An *M. nanum* culture from one case was used to produce lesions on a guinea-pig. Retro-culture grew *M. nanum* similar in all respects to the strain inoculated. The authors stressed the importance of collecting a specimen for culture from all patients with ringworm so that the causative fungus can be specifically identified. Usually a parent blames the family kitten or dog whenever a child gets ringworm. Exact mycological diagnosis can save a family pet, since *M. nanum* has not been reported as causing infection in domestic pets.

Kelly & Searls [26] in Victoria reported a case of *M. nanum* ringworm on the wrist of a slaughterman who handled pigs.

McAleer [33] recorded three outbreaks of *M. nanum* in pigs in WA. A suspension of soil from an area housing infected pigs contained macroconidia typical of *M. nanum*. In 1978 McAleer [33] first recorded *M. nanum* in WA from an abdominal lesion of a 12-month-old child. Efforts to trace the source of the infection failed and there was no family association with pigs.

Subsequent to the outbreak previously recorded by Munday (1964) [40], a further outbreak of ringworm in pigs occurred in Tasmania. *M. canis* was isolated from the pigs and cats sleeping in the heated farrowing shed which was confirmed as the source. All 96 piglets in 12 litters were infected, lesions appeared at 2–3 weeks of age (Buddle, 1985) [5].

In 1986 *M. canis* was isolated from a skin scraping from a pig in Queensland (M. Connole, unpublished).

In 1982 *T. mentagrophytes* var. *mentagrophytes* was isolated from skin lesions on a sow at an agricultural college in Dalby, Queensland. The herd was a minimal disease herd of 35 sows. The two lesions were dry, scaly about 3 cm diameter, and were not like lesions caused by *M. nanum*. The field officer suspected mites. The scraping was microscopically positive for ringworm and *T. mentagrophytes* var. *mentagrophytes* was cultured. Although rats were present in the adjacent feed shed, there were no signs of possums, goats or rabbits which may have acted as reservoir hosts. No other pigs were affected and there have been no further cases (M. Connole, unpublished data). *T. mentagrophytes* var. *mentagrophytes* is a rare cause of porcine ringworm, other cases having been recorded in Scotland [36], in England and in USA [18].

Muir *et al.* (1984) [39] in NSW isolated *M. nanum* twice from human specimens.

Sheep

M. canis has been reported in sheep in Tasmania (B. Peel, personal communication 1988).

Cats and dogs

In 1968 Connole [8] recorded the first *T. mentagrophytes* var. *granulare* (*T. mentagrophytes* var. *mentagrophytes*) infection in a young dog in Townsville, Queensland. The infection was transmitted experimentally to another dog. Skin scrapings of hair samples were collected from this dog at regular intervals and were positive by culture until 125 days after inoculation. Microscopical examinations were positive up to 90 days after inoculation.

Green & Kaminski (1973) [21] and Kaminski & Green (1977) [25] in surveys of ringworms in Aborigines living in the Northern Territory, found a high incidence of *tinea capitis* in children at Maningrida. The cause was a variant of *M. canis* which the authors referred to as the 'Maningrida' type. It differed from characteristic strains of *M. canis* in both the appearance of the colonies and the lack of golden-yellow pigment. Brush samples were collected from 12 cats and 10 dogs which belonged to the Aborigines. *M. canis* was isolated from both of two kittens with very scaly skin and almost complete loss of hair and from two of ten cats showing no abnormality. *M. canis* was isolated from two of eight dogs showing skin lesions but not from two dogs showing no

abnormality. All *M. canis* isolates were the 'Maningrida' variant.

Kamien (1976) [23] in a study of the health of Aboriginal children in Bourke, NSW, found that 29 of 346 children under 15 years of age had ringworm due to *M. canis*. This ringworm was ubiquitous in the large population of dogs associated with many Aboriginal dwellings. Presumably the *M. canis* strains were of the characteristic type as no variant among the isolates was described.

Wilkinson (1979) [56] in Brisbane, Queensland, reported a rare case of multiple infections of the skin of a dog with *M. gypseum*, *T. mentagrophytes*, *Candida albicans* and *Staphylococcus aureus*. The author considered that a possible sequence of infection might have been: infection of the head with *M. gypseum* from soil contact, followed by a *T. mentagrophytes* colonisation of the damaged skin with *C. albicans* and *Staph. aureus* following in turn.

In four surveys on different animal groups in the metropolitan area of Perth, WA, McAleer [32] found that *M. canis* was the only fungus isolated from cats and was prevalent in these animals. In dogs, dermatophyte infections were less common and were caused by *M. canis*, *M. gypseum* and *T. mentagrophytes*. McAleer [29] observed that *M. canis* was the most common dermatophyte species causing *tinea corporis* of humans in WA. The main predisposing factor was contact with other infected humans or animals. Zoophilic infections caused by *M. canis* were very common in children and kittens in both city and country areas of WA (McAleer [30]).

The major factor in the spread of *tinea capitis* in WA was opportunity of contact with infected animals [35]. The initial infection was almost invariably acquired by a child through direct contact with an infected animal, usually a kitten, occasionally a dog. There was also evidence of transfer among humans within small groups such as families and neighbours. Stray kittens in the community were the major factor contributing to the occurrence of *tinea capitis*. Abandoned kittens infected with *M. canis* were a frequent source of human infection.

Rowbottom & Goldsmid (1986) [53] in a survey of domestic animals in Tasmania found *M. canis* was the most common zoophilic isolate from animals, followed by *T. mentagrophytes*. One strain of *M. nanum* was isolated from a farm dog. Also, *T. verrucosum* was isolated from a dog in Tasmania (B. Peel, personal communication 1988).

Rodents and laboratory animals

Brown & Suter (1969) [4], while studying mouse plagues which produced favus in humans in SA, isolated *T. quinckeanum* from eight mice. These authors concluded that mouse favus was probably endemic in Australia amongst wild mice.

McAleer [31] reported a widespread epizootic due to a granular variety of *T. mentagrophytes* which occurred in a new stock of guinea pigs imported to Perth, WA from an eastern Australian state. The infection spread quickly amongst these animals and infection occurred in rabbits and mice and in four people at the breeding station. The human infections were contracted either directly through handling animals or indirectly by means of fomites. The strain of *T. mentagrophytes* causing the epizootic was distinctive from strains previously isolated in WA. *T. verrucosum* was isolated by McAleer [33] from a mouse with bald crusted lesions on its back.

Wild and native animals

Hyne *et al.* (1969) [22] isolated *M. canis* from a young lion in NSW. A small group of lions was affected and a decision was made not to treat the animals but to allow effective natural immunity to develop.

In SA, *T. mentagrophytes* var. *mentagrophytes* was recorded from two cases in which humans and kangaroos were closely associated. In one case it seemed that the disease spread from man to animal, and in the other, that the reverse occurred [51].

The strain of *M. gypseum* isolated from a camel in Queensland by Connole in 1964 was typed as *Arthroderma gypsea* by Dr P. Stockdale of CMI

[9]. Kuttin *et al.* (1986) [28] in a survey of camels in Israel showed that over 25% of young animals had *T. verrucosum* infection and less than 0.5% had *T. mentagrophytes* infection. In Australia ringworm in camels remains a rare condition as there have been no cases recorded since 1964.

Connole (1983) [10] isolated *M. canis* and *M. gypseum* from lesions of koalas (*Phascolarctos cinereus*) in an animal sanctuary in Brisbane, Queensland. One koala was infected with both species and *M. gypseum* was isolated from four koalas. As Alsatian dog on which koalas rode, was a carrier of *M. canis*. All *M. canis* isolates from the koalas and the dog were a variant of the characteristic *M. canis* and were confirmed by Dr P. Stockdale, CMI, and Dr G. Kaminski, Adelaide, SA as the 'Maningrida' type.

No koala colonies exist in the harsh tropical climate of the NT. It is interesting that the same variant of *M. canis* was found in koalas in the sub-tropical area of south east Queensland at Brisbane, some 2500 km south-east of Maningrida. However, colony variants of *M. canis* are not uncommon (Rippon, 1988) [52].

Dr P. Stockdale tested five of the koala strains for production of the perfect state (of *M. canis*), *Arthroderma otae*, using Japanese tester mating strains. Although the controls were positive the koala strains failed to react (P. Stockdale, personal communication 1982).

Kaminski (1983) [24] in SA isolated *T. mentagrophytes* var. *mentagrophytes* from 43 humans and from 16 young kangaroos. In three instances *T. mentagrophytes* var. *mentagrophytes* was isolated from the humans that had contact with kangaroo. These strains were different from the usual strain of *T. mentagrophytes* var. *mentagrophytes* isolated from guinea pigs, mice or dogs and were called the 'kangaroo variant'. The variant strains usually produce a large number of clavate, multiseptate macroconidia, often with terminal appendages, whereas the normal strains produce fewer macroconidia and without appendages. This work confirmed the association of ringworm of humans due to contact with kangaroos first reported by Donald (1960) [16]. At that time specimens from animals were not available for culture.

McAleer [32] in Perth, WA conducted a survey of wild animals by taking brush cultures from 90 native animals. These consisted of quokkas, Tammar wallabies, Western grey kangaroos, red kangaroos, Western euros, agile wallabies, Western native cat, rufous rat kangaroo, hairy-nosed wombat, Northern brushtailed possum, bushtailed rat kangaroo and short-nosed bandicoot. Several strains of *M. cookei* and *T. ajelloi* were isolated but not *T. mentagrophytes*. Soils from the yards in which these animals were held were cultured for keratinophilic fungi. *M. cookei* was isolated from most of the soil samples. *T. ajelloi* and *M. gypseum* were also isolated from soil in some yards.

McAleer [33] in WA isolated *T. mentagrophytes* from a young kangaroo with no clinical sign of infection.

In Queensland, *T. mentagrophytes* var. *mentagrophytes* 'kangaroo variant', has been isolated from kangaroos and *M. gypseum* (*A. gypsea*) isolated from a kangaroo and a purple neck rock wallaby (M. Connole, unpublished).

McAleer [34] sampled 299 soils in WA to determine which species of geophilic dermatophytes were present. Most samples were collected from areas frequented by people and animals such as home gardens, parks and animals yards. Of the total, 271 (90.6%) yielded keratinophilic fungi and a total of 181 dermatophytes. *M. gypseum* (30.7%) was the most prevalent dermatophyte recovered, followed by *M. cookei* (21.7%) and *T. ajelloi* (8.0%). There was no isolates of *T. mentagrophytes* recovered from WA soils, despite its presence in domestic and wild animals.

Glazebrook (1980) [20] isolated *Paecilomyces* sp. from shell lesion of a green turtle (*Chelonia mydas*) in Townsville, Queensland.

Maslen (1981) [37] in Victoria reported two human infections with *T. mentagrophytes* var. *erinacei*. This variety affects the hedgehog (*Erinaceus europaeus*) in NZ but does not occur in Australia. One infection occurred on a woman who handled a hedgehog during a visit to NZ, the

other on a woman who handled an unsealed culture plate of *T. mentagrophytes* var. *erinacei* imported from NZ.

Muir *et al.* (1984) [39] also reported the isolation of *T. mentagrophytes* var. *erinacei* from a patient who had recently handled a hedgehog in NZ. These authors recorded dermatophytes identified at the Australian National Reference Laboratory in Medical Mycology, Sydney, over the period 1966–1982. They emphasised the point that human infections caused by zoophilic dermatophytes, for example, *M. canis*, *T. mentagrophytes* var. *granulare*, *T. verrucosum* and *T. equinum* var. *autotrophicum* are almost invariably acquired through contact with infected animals.

Crozier (1980) [14] surveyed soils in the Illawarra area of NSW for the prevalence of geophilic dermatophytes. *M. gypseum* was isolated from 74.5%, *M. cookei* from 56.4% and *T. ajelloi* from 55.7% of soil samples. He pointed out the potential pathogenicity of such a high content of certain geophilic dermatophytes within soils of the area.

Crozier [13] proposed that the increased colonization of well-composed soils by keratinophilic dermatophytes recorded in his soil survey, could be due in part to the transportation of fungi within these soils by nematodes, as well as by lumbricid worms and by other methods of surface dispersal.

T. mentagrophytes var. *mentagrophytes* has been isolated from a wallaby in Tasmania (B. Peel, personal communication 1988).

Other animals

Rees (1967) [48] described a new species, *Arthroderma flavescens*, which he isolated from feathers of 3 different species of birds in Queensland.

In Queensland, Rees, in a series of studies of keratinophilic fungi isolated the following fungi: *Chrysosporium* spp., *A. curreyi*, *A. cuniculi*, *A. tuberculatum*, *Nannizzia* (*Arthroderma*) *cajetana*, *M. cookei*, *M. gypseum* and *Keratinomyces* (*Trichophyton*) *ajelloi*, from hairs or scales of various mammals and reptiles [46]; *Chrysosporium* spp., *A. curreyi*, *A. cuniculi*, *A. tuberculatum*, *A. flavescens*, *N. cajetana* and *M. gypseum*, from feathers of wild birds [49]; *Chrysosporium* spp., *A. tuberculatum*, *A. ciferrii* and *M. gypseum*, from feathers of domestic fowls [50]. None of the animals studied showed evidence of infection.

Miscellaneous

Frey *et al.*, 1979 [18] prepared a well-illustrated, useful textbook on pathogenic fungi. The text covered dermatophytosis and subcutaneous and systemic mycoses.

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SECTION TWO: SUBCUTANEOUS MYCOSES

Phaeohyphomycosis

Cats and dogs

In Victoria, Bostock *et al.* (1982) [4] described the infection in a domestic shorthair cat which had a granuloma in the subcutaneous tissues of the nose. *Exophiala jeanselmei* was isolated. The lesion was excised widely and the wound healed normally.

In Queensland in 1984 McKenzie *et al.* [13] reported on subcutaneous phaeohyphomycosis caused by *Moniliella suaveolens* in two cats. The first cat had a series of ulcerated black, abdominal lesions which recurred despite repeated surgical removal. The other had a single black subcutaneous lesion near one dew claw. This lesion did not recur following surgery. *M. suaveolens* was isolated in pure culture from both cats. This fungus had not been associated previously with disease in animals or man. More intensive investigation of dark lesions by veterinarians may reveal more phaeohyphomycoses in cats. Certainly the need for differentiation of these lesions from melanomas is indicated.

In Townsville, north Queensland in 1985, Shinwari *et al.* [17] reported a female cat which showed circling and incoordination. The pupils were dilated and pupillary reflex was absent. The cat died, and necropsy revealed a solid black lesion (2 × 2.5 cm) in the left occipital lobe of the cerebrum. A pure culture of *Cladosporium bantianum* (*Xylohypha bantiana*) grew from a sample of the lesion. The gross appearance, histopathology and fungal morphology of the brain lesion resembled those of lesions induced by *C. bantianum* in the brain of a woman in south Queensland [19].

Cattle

Carbonell (1976) [5] reviewed the condition known as 'bovine nasal granuloma'. A variety of diseases come under this name. One, nasal mycetoma, was considered due to fungal infection and could be differentiated from nasal granuloma by histology of the lesions. Carbonell referred to the work of Hore (1966) [6] who consistently isolated fungi of the genus *Fusarium* from the nasal mucus of cattle with nasal granuloma but not from a small number of normal cattle. He found also that fungal spores and hyphae were more numerous in nasal scrapings of cattle with nasal granuloma. Carbonell concluded that nasal granuloma was probably a form of allergic rhinitis.

In 1977 McKenzie & Connole [12] reviewed laboratory diagnoses of mycotic nasal granuloma in cattle in Queensland during 1966–1975. They described seven cases of eosinophilic granulomas containing fungal chlamydospores and short septate hyphae in the submucosa of the anterior nasal cavity from beef cattle. Specimens from four cases were available for mycological examination. *Drechslera rostrata* was isolated from two cases, and unidentified dematiaceous fungi were isolated from the other two. *D. rostrata* has been reclassified as *Exserohilum rostratum*.

McKenzie & Connole considered that the severe tissue eosinophilia and numerous mast cells in the lesions may have indicated a chronic hypersensitivity reaction to fungi in the tissue [7]. Lesions of bovine atopic rhinitis ('nasal granuloma') also contained epithelial hyperplasia, numerous mast cells and tissue eosinophilia but no fungal organisms, macrophages or giant cells (Pemberton & White, 1974) [14]. Pemberton *et al.* (1974) [15] demonstrated that the so-called 'nasal granuloma' of cattle in Victoria could be reproduced experimentally by provoking an allergic response in the nasal mucosa. This disease thus should be known as bovine atopic rhinitis.

Cases of bovine atopic rhinitis occur rarely in Queensland and have been reported only from some coastal areas and the southern Darling Downs. It appears that mycotic nasal granulomas occur sporadically in Queensland cattle and should continue to be considered in the differential diagnosis of space-occupying lesions of the nasal cavity of cattle in Australia [12].

In 1977 Pritchard *et al.* [16] reported in NSW a case of eumycotic mycetoma in a beef cow with lesions in the skin, nasal cavity and lymph nodes. *E. rostratum* was isolated from various tissues. Koch's postulates were satisfied by the reproduction of a liver granuloma in a mouse inoculated with a suspension of a culture of *E. rostratum*. This case could be classified as a phaeohyphomycosis (L. Ajello, personal communication 1977).

In 1985 McGinnis *et al.* [10] identified the two previously unidentified dematiaceous fungi isolated from bovine nasal granulomas by McKenzie & Connole [12]. The isolates were identified as *Phaeosclera dematioides* Sigler, Tsuneda et Carmichael, a species which was first described in 1981 and was isolated from pith of *Pinus contorta* in Alberta, Canada [18].

The diseases classified as phaeohyphomycoses have been caused by a wide variety of opportunistic dematiaceous fungi. Ajello (1986) [1] listed 71 species of such fungi classified in 39 genera as aetiologic agents of the disease in humans and animals. This list included all the currently well-documented agents on a worldwide basis. The Australian cases include nine caused by five different species. The classification of two of these agents has been changed recently. McGinnis *et al.* [9] reclassified *C. bantianum* as *Xylohypha bantianum*; McGinnis *et al.* [11] based upon morphological characteristics of 13 isolates, and the observations of Alcorn (1983) [2] in Queensland, decided that *D. rostrata* should become *Exserohilum rostratum* and confirmed it as an agent of phaeohyphomycosis in humans and animals.

Sporotrichosis

The first case of sporotrichosis in an animal in Australia was in a cat as described by Mackay *et al.* (1986) [8] in Brisbane, Queensland. The Siamese cat had a circular crusty lesion on the dorsum of the nose. Biopsy material indicated multiple encapsulated spores within histiocytes, morphologically suggestive of cryptococci. However, the culture was identified as *Sporothrix schenckii*. Treatment with potassium iodide was unsuccessful. Combined amphotericin and ketaconazole therapy was apparently curative but the original lesion returned after therapy had been discontinued because of toxicity. The cat was killed and at necropsy, severe renal damage due to drug therapy as well as active, localized cutaneous sporotrichosis were found. The source of infection was not determined, although a puncture would on the nose during fighting seemed a likely route of entry.

While sporotrichosis is very rare in animals in Australia, sporotrichosis in humans has been reported more often in Queensland than elsewhere in Australia, possibly because of the prevailing high temperature and high humidity, which favours saprophytic growth of the fungus (Auld & Beardmore, 1979) [3].

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SECTION THREE: SYSTEMIC MYCOSES

Adiaspiromycosis

In 1980 Krivanec & Mason from Tasmania [52] reported finding small spherules in the lungs of two species of wombats, *Lasiorhinus latifrons* (Owen, 1945) and *Phascolomis ursinus* (Shaw, 1800). Adiaspiromycosis was suspected following histology of lung tissue and the aetiological agent was thought to be either *Chrysosporium parvum* or a new fungal species.

In 1982 Mason & Gauhwin [65] recorded the first confirmed occurrence of adiaspiromycosis in wombats. Fresh lung tissue from adult hairy-nosed wombats (*L. latifrons*), fixed lung tissue from 22 pouch young *L. latifrons*, 12 rabbits (*Oryctolagus cuniculus*), a feral cat (*Felis catus*) and a fox (*Vulpes vulpes*), all from SA, were examined. No spherules were detected in the lungs of the rabbits, the feral cat or the fox. Spherules were detected in the lungs of the six adult and eight of 22 pouch young wombats.

Apparently the hairy-nosed wombats acquired infection by the inhalation of spores from dust in their burrows. A prey-predator cycle was considered not to be involved. A similar although smaller organism to the one in *L. latifrons* was observed in Tasmania in the common wombat (*Vombatus ursinus*). It was believed that both organisms were *C. parvum* with different varieties occurring in the two species of wombat.

Aspergillosis

Aspergillus fumigatus is the species usually isolated from animal infections but others, including *A. flavus*, *A. nidulans*, *A. niger* and *A. terreus*, are sometimes identified.

Mycotic abortion and related conditions due to *Aspergillus* spp. will be treated separately.

Previous reports described aspergillosis in domestic poultry, caged birds, black swans and penguins [15]. Here, further reports are given of the disease in cats, dogs, horses and birds (a galah, an albatross, ostriches, penguins, Japanese quail, ducks and turkeys).

Cats and dogs

In Queensland, Stokes (1973) [121] recorded a case of infectious feline enteritis with concomitant fungal enteritis. The fungus was identified tentatively as *Aspergillus* sp. on morphological characteristics.

In WA, Peet and Robertson (1976) [92] described multiple lesions of suspected systemic aspergillosis in a dog.

In Queensland, Wilkinson *et al.* (1982) [132] reported an *Aspergillus* sp. infection associated with orbital cellulitis and sinusitis in a cat.

From WA, Kabay *et al.* (1985) [48] reported a unique, disseminated *A. terreus* infection in 10 previously healthy adult dogs – nine German Shepherds and one Dalmatian. The disease was characterized by the presence of multiple granulomas and infarcts in a wide range of organs. The kidney, spleen, and skeletal system were most commonly and severely affected. Fungal hyphae were demonstrated in large numbers within granulomas and thrombi, and *A. terreus* was readily isolated by culture. In the series of cases there was no apparent predisposing factor, portal of entry, or primary focus for dissemination of the infection.

Day *et al.* (1985) [18] made an immunologic study of systemic aspergillosis in eight of the above German Shepherd dogs. Day *et al.* (1986) [19] reported clinical and pathological findings from the series of 12 cases of disseminated aspergillosis in 11 German Shepherd dogs and one Dalmatian.

Oxenford and Middleton (1986) [86] reported osteomyelitis and arthritis associated with *A. fumigatus* in a dog from NSW.

In WA, Day and Penhale (1988) [20] studied aspects of humoral immunity in 17 dogs with disseminated aspergillosis (16 cases *A. terreus*, 1 case *A. flavipes*).

At the University of Queensland Veterinary School, *A. fumigatus* was isolated from the nasal discharge of a cat (M. Mutimer, personal communication).

At the University of Melbourne Veterinary School, *A. fumigatus* was isolated from nasal and ocular discharges of a dog (K. Hughes, personal communication).

Birds

In a report from SA, *A. fumigatus* infection of the beak of a galah (*Kakatoe roseicapilla*) was described, and from fungal granulomata in the kidneys of a grey-headed albatross (*Diomedea chrystoma*) an aspergillus of the *A. flavus-oryzae* group was isolated (Report 1973) [98].

Obendorf & McColl (1980) [85], in a study of Little penguins (*Eudyptula minor*) along the coast of Victoria, diagnosed pulmonary aspergillosis in two adult penguins. In Victoria, Rousseaux and Dalziel (1981) [110] diagnosed pneumonia due to *A. fumigatus* in an ostrich (*Struthio camelus*).

In Melbourne, *A. fumigatus* caused pericarditis and pneumonia in a penguin (K. Hughes, personal communication). Also in Melbourne, *A. fumigatus* was isolated from the air sac of an ostrich (K. Hughes, personal communication). *A. flavus* was isolated from the air sac of a penguin from Currumbin, Queensland (T. Smeltzer, personal communication).

In 1986 in Victoria, Reece *et al.* [94] reported mycosis of commercial Japanese quail, ducks and turkeys. In the quail there were 22 flocks with an incidence of mycosis from 2.5 to 95%. The mycosis comprised (1) mycotic pneumonia and airsacculitis; (2) kyphosis and paresis associated with mycotic spondylitis of the last cervical or first

thoracic vertebrae; (3) tremors, paddling and incoordination associated with mycotic encephalitis. Four flocks had an incidence of 2.5% mycosis comprising (1) and (2). Two flocks had an incidence of 25% mycosis comprising (1) only. *A. fumigatus* was the causal fungus in all cases.

Four flocks of ducks had an incidence of 5% mycosis of type 1 disease and was caused by *A. fumigatus*. One flock of turkeys had an incidence of 10% mycosis. Both disease types were present. However, *Penicillium* sp. was the fungus attributed as the cause of disease.

Horses

Guttural pouch mycosis is a proliferative and necrotising inflammatory lesion of the equine guttural pouch. Various fungi have been isolated from overseas cases with *A. nidulans* being the most commonly reported.

In NSW, Rawlinson & Jones (1978) [93] reported 2 cases of guttural pouch mycosis. One horse gradually recovered with conservative medical treatment, the second was destroyed. Autopsy failed to establish the initiating cause of the lesion but later stages were clearly associated with an invasive septate fungus morphologically resembling *Aspergillus*.

In WA, Hilbert *et al.* (1981) [36] reported an erosion of the internal carotid artery and cranial nerve damage caused by guttural pouch mycosis in a horse. Histologically, numerous branching septate hyphae were seen in the lesion.

Candidiasis

Candidiasis has been described mainly in poultry [15]. The disease has been seen more recently in native animals.

Obendorf (1980) [84a] in Victoria isolated *Candida albicans* from the upper alimentary tract lesions of four young eastern grey kangaroos (*Macropus giganteus*), which had been hand-reared.

Dixon & Baird (1984) [24] in NSW reported systemic candidiasis in a grey kangaroo (*Macropus giganteus*). *C. tropicalis* was isolated from the bile and hepatic lesions, and the presence of blastoconidia and mycelia in foci of non-inflammatory hepatic necrosis was considered evidence for a systemic infection.

At James Cook University, Townsville, in a study of macropods, candidiasis was one of the more common diseases seen, particularly in hand-reared 'joeys' [104, 105].

Tham *et al.* (1985) [125] in SA reported a case of generalised infection in an adult male musk lorikeet (*Glossopsitta concinna*). *C. albicans* was isolated in pure culture from granulomas in the skeletal muscle, liver, heart, kidney and lung.

Cryptococcosis

Previously [15] in Australia cryptococcosis had been recorded in cats, koalas, a horse, a sheep, a cow and a ring-tailed possum; *Cryptococcus neoformans* had been isolated from soils and pigeon droppings.

Cryptococcosis is recorded here, again in cats, koalas, horses and a cow but also in dogs, a beaver, quokkas, a pig-tailed monkey and a ferret; *C. neoformans* has been isolated from droppings of a swallow.

Cats

In a cat in Queensland the disease was characterised by infection of the cervical lymph nodes and surrounding connective tissues of the neck, meningitis, encephalitis and ophthalmitis (Clark & Roubin, 1970) [10].

In NSW Howlett *et al.* (1973) [39] reported a case of generalised lymphadenopathy, and Wanner & Baird (1974) [127] isolated *C. neoformans* from the cerebrospinal fluid and nasal secretions. Meningitis had followed direct invasion of the cranial cavity from the frontal sinus.

Cryptococcosis was diagnosed in the upper respiratory tract of a cat in Perth, WA [99].

Wilkinson (1979) [129] from the University of Queensland Veterinary School Clinic reviewed feline cryptococcosis and reported seven more cases in Queensland, two of which were treated unsuccessfully with amphotericin B. He stated that any case presenting in poor bodily condition associated with chronic rhinitis, signs of CNS involvement, generalized skin nodules, or chorioretinitis, should raise strong suspicions of cryptococcosis. The review showed that chronic rhinitis is so common in the cat that clinicians should always exclude cryptococcal infection, by examination of a suitably stained smear (India ink), in all cats showing a chronic nasal discharge or snuffling. The use of this simple procedure might reveal that feline cryptococcosis is more widespread, particularly in non-tropical regions than hitherto thought.

Wilkinson stated that the generalized distribution of skin nodules in the three cases showing cutaneous involvement suggestive of haematogenous dissemination, was rare in the cat. Cats appeared to be more susceptible to cryptococcal infection than dogs. During the 30 month period covered by the case reports, no cases of canine cryptococcosis were observed although the clinic population contained a 2 : 1 majority of dogs and cryptococcal contamination of the environment must have been equal for both species. Similarly dogs appear more resistant than cats to cutaneous mycobacterial infections, suggesting that cats have a less effective immune response than dogs. Prognosis in feline infection was unfavourable as treatment was expensive and required careful laboratory monitoring. Successful therapy had been recorded only in cases with small and localised lesions.

Wilkinson *et al.* (1983) [131] reported four more cases in Queensland in Siamese cats. Lesions were mainly confined to the nasal passages and skin and were treated orally with 5-fluorocytosine augmented in two cases by intradermal injections of an autogenous vaccine. Treatment was successful although one cat showed recurrence of skin lesions when 5-fluorocytosine therapy was discontinued. The condition cleared after a further course. No untoward side-effects were seen. Wilkinson (1984) [130] recommended combination therapy using either 5-fluorocytosine or ketoconazole together with an autogenous vaccine.

In Victoria, Emms (1987) [25] successfully treated cryptococcosis in two cats by using ketoconazole. This drug showed promise of being a safer treatment than previously available drugs.

In SA a Siamese cat had large subcutaneous granulomas containing many cryptococci. At necropsy lesions containing the organism were found in the subcutis, pancreas, lung, interstitium and meninges [103a].

Dogs

Woodbury (1974) [133] in NSW reported a fatal cryptococcal granuloma in a dog. Cryptococcosis was diagnosed histologically in brain tissues of dog in WA [100].

Browning & Montgomery (1981) [4] in Victoria found that treatment of a generalised infection in a dog with amphotericin B was successful. Sutton (1981) [122] described 6 cases in large breed dogs seen over a 14-month period at the University of Queensland Veterinary School. The main features were CNS involvement in all cases and diversity of the initial presenting signs. The respiratory tract was affected in one case but the bronchopneumonia observed did not appear to be of cryptococcal origin. Immunosuppressive factors and other diseases, which are believed to increase the susceptibility of man and animals to cryptococcal infection were not implicated in these cases.

In Queensland, a case of osteomyelitis due to *C. neoformans* was diagnosed in a dog at the Veterinary School (A. Frost, personal communication) and at the ARI in 1987, *C. neoformans* was isolated from pharyngeal fluid of a dog (M. Connole, personal communication).

Horses

Among the few cases of equine cryptococcosis reported in Australia, there appear to be three distinct conditions, a pulmonary form [23, 35], a

neurological form [3, 43] and granulomatous involvement of the nasal and paranasal regions [107, 128].

Dickson & Meyer (1970) [23] reported two cases of cryptococcosis in horses in WA. One horse had a large granulomatous mass replacing most of the lung tissue. The other horse died from a ruptured colon. Encapsulated nodules were sparsely distributed in the dorsal area of the diaphragmatic lobes of the lungs. No cultural techniques were attempted in either case. Diagnosis was made on the typical histological appearance of the encapsulated coccoid bodies, considered sufficiently accurate for identification (Jubb & Kennedy, 1963) [47].

Watt (1970) [128] in Queensland reported a large cryptococcal granuloma in the nasal cavity of a horse. The lesion appeared to originate from the anterior portion of the ventral turbinate bone.

In WA cryptococcosis in the miliary form was diagnosed in a horse [96] and a cryptococcal osteomyelitis in a filly [100].

Barton & Knight (1972) [3] in NSW reported a case which presented as meningitis.

Hilbert *et al.* (1980) [35] in WA described cryptococcal pneumonia in a mare.

Roberts *et al.* (1981) [107] in Queensland described protracted cryptococcal nasal granuloma in a Standardbred stallion. *C. neoformans* was identified by direct examination of, as well as culture of, nasal discharge. Treatment with amphotericin B was considered inadvisable, and the stallion's good general health precluded destruction on humane grounds. The horse completed two successful stud seasons. When represented, he had lost weight and the nasal condition had steadily worsened. He was destroyed and autopsied. A large lobulated fleshy mass occupied the left nasal cavity. Also a nodule was found in the wall of the jejunum. This finding appears to be the first report of a cryptococcal lesion in the gastrointestinal tract of a horse.

Koalas

In WA, cryptococcosis was diagnosed in koalas on 3 occasions [95, 99, 100].

Munday (1976) [81] stated that cerebral cryptococcosis was quite common in some colonies of koalas and had been found in other native mammals. Signs were referable to the CNS but respiratory involvement may occur, especially swelling of the sinuses. The disease should be suspected if typical lesions are present in the sinuses. Otherwise a diagnosis can be confirmed only by demonstration of the organisms in sections or body fluids. Munday considered that treatment was neither warranted nor likely to be highly successful. He suggested that as the main reservoir of the organism was the soil, control was difficult but suspect areas could be fenced off and good grass cover encouraged.

Dickens (1976) [22] stated that the disease was suspected in koalas exhibiting neurological signs such as ataxic gait or cycling, but that sudden death may occur without predisposing nervous signs. Cryptococci may be detected in brain, lungs, kidneys or elsewhere throughout the body.

Canfield *et al.* (1986) [8], investigating a disease outbreak involving pneumonia in captive koalas in NSW, isolated both *Bordetella bronchiseptica* and *C. neoformans* from the inflamed lungs and nasopharynx of one koala. Also, *C. neoformans* was seen in the spleen when examined histologically.

Canfield (1987) [7] in a study of 127 free range koalas from the north coast of NSW isolated *C. neoformans* from two of seven koalas with pneumonia.

Other animals

Cryptococcal lymphadenitis was diagnosed in a beaver from the Adelaide zoo, SA [97].

In WA, cryptococcal pneumonia was diagnosed twice in quokkas (*Setonix brachyurus*) [101, 102] and in a pig-tailed monkey [102]. Also it was diagnosed histologically in a bovine lung [100].

In WA, Lewington (1982) [54] reported

pneumonia due to *C. neoformans* in a hob Fichet ferret. The ferret had been used to reduce the number of mice attracted by seeds used to feed birds, mainly finches, in an aviary. It seemed that contact with bird excreta around the aviary was responsible for the infection.

Glasziou & McAleer (1984) [33] in WA, reported a fatal case of cryptococcal meningitis in a 42-year-old male motor mechanic. A search for an environmental source of infection revealed that the patient, four months previously, had been exposed to dust and debris from the nest of a swallow (*Hirundo neoxena*) being removed from the roof of the patient's workshop. Samples of the reconstructed nest and weathered droppings from the workshop floor yielded large numbers of *C. neoformans* Serotype A, the same type isolated from the patient's CSF. The association of *C. neoformans* with roosts of some birds, particularly pigeons, is well documented, but this appears to be the first report of its association with the roost of the common swallow.

Dactylariosis

This mycosis was first described as an encephalitis in turkeys in South Carolina, USA, by Georg *et al.* (1964) [31] who named the fungus *Diplorhinotrichum gallopavum*. In 1968 it was reclassified as *Dactylaria gallopava* by Bhatt and Kendrick [3a].

The second reported isolation of this fungus was by Connole (1967) [13]. In that outbreak, 550 of 1600 five-week-old Australorp chickens maintained near Brisbane, Queensland, died after showing signs of encephalitis. A brown fungus isolated from the brains of two birds was identified by Dr P.K.C. Austwick, Weybridge, England as *D. gallopavum*. Later, cultures and tissue sections of the brains were made available to Dr L.K. Georg, CDC, Atlanta, who confirmed the identification as *D. gallopava* and the presence of characteristic brain lesions. Tissue changes caused by this fungus are somewhat similar to those caused by the *Aspergillus* species and a diagnosis must be based on the morphological and pigmentation differences between these two fungi (Chandler *et al.*, 1980) [9].

Fusariomycosis

Hodgson & Jacobs (1982) [37] in Queensland reported keratomycosis caused by *Fusarium* species in two horses with corneal ulcers. In both cases, topical treatment with natamycin, EDTA plasma and atropine with subconjunctival tetanus antitoxin resulted in resolution of the ulcers.

McAleer (1983) [57] in WA described 'black shell disease' of the western rock lobster (*Panulirus cygnus*) caused by *Fusarium solani*. *F. solani* was isolated from all lesions. The isolate had a deep magenta-purple pigmentation both on the surface of the colony and diffusing into the medium. Superficial injection of *F. solani* into the tail flesh of healthy mature lobsters resulted in death of all animals within eight days. This mycosis was unusual in that the outbreak occurred in the wild and not in cultured lobsters. *F. solani* is not a marine organism but lives and adapts well to salt water conditions.

In Tasmania, *F. solani* was isolated from ear lesion of a cat (B. Peel, personal communication).

Histoplasmosis

Hoffmann *et al.* (1985) [38] reported a case of suspected histoplasmosis capsulati in a dog necropsied at Ingham, Queensland. Only formalin-fixed portions of colon, rectal wall and mesenteric lymph node were available. Replicate sections of colon were examined by direct immunofluorescence staining using conjugates prepared at the CDC, Atlanta, Georgia. Although the diagnosis was a yeast-form mycosis caused by organisms morphologically compatible with *H. capsulatum* var. *capsulatum*, the authors were unable to identify the organism by direct immunofluorescence. They suspected the yeast forms represented a unique serotype of *H. capsulatum* var. *capsulatum* that did not react with the FA conjugate prepared at the CDC. This was the first

report of gastrointestinal histoplasmosis in an animal in Australia.

Fewings *et al.* (1970) [27] in SA isolated *H. capsulatum* from soil samples in and near a fowl yard adjacent to the home of a man who developed disseminated histoplasmosis.

Following the report of 13 clinical human cases of acute pulmonary histoplasmosis associated with the Church Cave at Wee Jasper in NSW in 1977, Hunt *et al.* (1984) [40] began a 7-year investigation with the aim of isolating and identifying the causative microorganism of the disease from the cave environment. Although three clinical cases of acute pulmonary histoplasmosis occurred during the investigation, *H. capsulatum* was isolated on only one visit, in November 1983. The organism was isolated using three different techniques, from mice caged in the cave environment, from the cave soil and respirator filter material by the mouse inoculation technique, and from the sputum of a person who contracted acute pulmonary histoplasmosis after visiting the cave.

Hunt *et al.* strongly supported the assumption that there was a continuing natural reservoir of the fungus *H. capsulatum* in the Australian environment. Bats of the genus *Miniopterus* have been reported to be associated with caves from which the fungus has been isolated (Taylor *et al.*, 1962) [124]. Since the cave in question, Church Cave, was only one of 11 known breeding sites for the bent-wing bat *Miniopterus schreibersii* in eastern Australia and there was some evidence for the interchange of bats between breeding caves, it was possible that *H. capsulatum* existed in the other known bat breeding caves.

Miscellaneous systemic mycoses

Infections which are not classifiable under the separate mycoses include geotrichosis, paecilomycosis, eumycotic and black-grained mycetomas, fungal pneumonias and fungal myocarditis/nephritis. Relevant reports are reviewed here, in chronological order.

In his study of a chronic diarrhoea syndrome in racehorses in Sydney, NSW, Manahan [62] found large numbers of *Geotrichum*-type fungi in faecal samples of two of 32 cases examined. Also in Sydney, Sykes (1978) [123] observed the same syndrome in a champion racehorse; its faeces yielded a pure culture of *Geotrichum* sp.

In Queensland, van den Hoven and McKenzie (1974) [126] tentatively diagnosed a paecilomycosis in a dog. Histologically, granulomatous lesions were found in the forepaw, prescapular lymph node, lung and cerebrum. Attempts to isolate the causal fungus from tissues were unsuccessful.

At the University of Queensland Veterinary School in 1977, *Curvularia senegalensis* was isolated from a black-grained mycetoma in a dog in 1977 (M. Mutimer, personal communication), and *Paecilomyces lilacinus* from a case of epididymitis in a sheep in 1978 (A. Frost, personal communication).

Glazebrook (1980) [34] in Townsville, Queensland isolated a variety of fungi from lung lesions of green turtles (*Chelonia mydas*): *Paecilomyces* spp., *Penicillium* spp., *Alternaria* sp., *Fusarium solani*, *Aspergillus terreus*, *Aureobasidium pullulans*, *Heterocephalum aurantiacum* and *Drechslera hawaiiensis*.

Miller *et al.* (1980) [78] reported black-grained mycetoma in two horses in Queensland. Only preserved material was submitted for examination.

From WA, Peet *et al.* (1981) [91] reported fungal myocarditis and nephritis in a horse. Histopathological examination revealed zygomycete hyphae.

Coyle *et al.* (1984) [17] in Queensland reported a case of canine mycetoma of the fourth tarsal bone of a female dog, caused by *Curvularia geniculata*. Factors in the successful treatment of this case were radical surgery, the absence of secondary bacterial infection, a normal immunological response and early vigorous treatment with sodium iodide.

From Melbourne, Victoria, Maslen *et al.* (1988) [63] reported a systemic mycosis in a captive crocodile hatchling (*Crocodylus porosus*). Granuloma-like lesions were seen in the liver, left lung and spleen. *Paecilomyces lilacinus* was isolated from liver lesions.

Mycotic abortion and related conditions

Munday (1967) [80] listed the mycotic infections of cattle in Tasmania as:
Bovine abortion: *Aspergillus* spp. (including *A. fumigatus*), *Absidia* spp. (including *A. corymbifera*) *Mucor* spp., *Mortierella* spp. and *Acremonium* spp.
Bovine acute, mycotic pneumonia: *Mortierella* spp. and unidentified species.
Bovine chronic, mycotic pneumonia: *Aspergillus* spp. (on histophatology).
Bovine, mycotic rumenitis: causative agent not identified.
Bovine, mycotic meningoencephalitis: causative agent not identified.

In a study of diseases of the CNS of cattle in Tasmania, Munday *et al.* (1973) [82] included mycotic meningoencephalitis. The diagnostic criteria used included microscopic pathology and isolation of *Aspergillus* or *Mucor* spp.

Also from Tasmania, Mason (1971) [64] recorded the first case of porcine mycotic abortion in Australia. The lesions consisted of a suppurative mycotic bronchiolitis and a necrotic placentitis. *A. fumigatus* was recovered in pure culture from both a foetus and the placenta.

In SA, mycotic abortion in two herds of cattle was attributed to *A. fumigatus* [98].

In WA, Dennis (1974) [21] in a study of perinatal lamb mortality, reported a mycotic abortion due to an unidentified fungus which was responsible for the abortion of twins.

In Victoria, Neilan *et al.* (1982) [84] reported that within 22 days after 30 pregnant cows inadvertently received silage *ad libitum*, one had a mycotic abortion and two gave birth to calves with mycotic encephalitis. The latter two cows both developed severe mycotic pneumonia within 3 days of parturition. *Mortierella wolfii* was cultured from the cow's lung, both calf brains and the silage which was the probable source of infection.

Later investigations showed that *M. wolfii* was responsible for about 20% of all mycotic abortions in Gippsland, Victoria, and that 15–20% of affected cows died from *M. wolfii* pneumonia within three days of calving (McCausland & Neilan, 1982) [60].

A survey of Gippsland herds in which fungal abortions occurred, showed that mycotic abortion was almost always associated with a grossly recognisable placentitis but that foetal lesions were rare. Also, the predominant cause of abortion in cows fed hay was *Aspergillus* spp., and in cows fed silage, *M. wolfii* [58, 59].

Mycotic bovine abortion was diagnosed four times over a three-month period in south-east Queensland, an unusually high incidence [103]. One of these abortions was due to *M. wolfii*. Skilbeck (1984) [118] developed a reliable complement fixation test for the serological diagnosis of bovine abortion caused by *M. wolfii*.

Jerrett *et al.* (1984) [44] studied 265 bovine abortions in Victoria. The cause of abortion was identified in 98 (37%) of cases. Of these, 27 (28%) were mycotic infections, the most common cause of abortion. *Aspergillus* spp. were isolated from the placenta in 18 cases and from fetal stomach contents in five cases. Fungal hyphae resembling *Mortierella* spp. were identified in four of the remaining mycotic abortions, but fungi other than *Aspergillus* spp. were not isolated. The reason was that the isolation of *M. wolfii* required additional mycological techniques not performed in this survey.

McCausland *et al.* (1987) [61] in Victoria examined placentas and foetuses from 1107 bovine abortions, and a fungal infection was diagnosed in 131 (11.8%) instances. Fungi seen in silver-impregnated sections of tissues were placed into three categories designated as aspergillus, zygomycete and atypical. Culture indicated that the first two of these categories were due to *Aspergillus* sp. and *M. wolfii* respectively. The infections in the atypical category were probably also due to *Aspergillus* sp. Gross or microscopic examination and culture of the placenta were valuable diagnostically but examination of the foetus was seldom of value as infection in most instances did not involve foetal tissues. Questionnaires indicated that many *M. wolfii* abortions were associated with the feeding of poorly prepared or stored grass silage. As the majority of cattle were

fed hay or silage, no association could be shown between *Aspergillus* sp. abortion and these feeds. Fertility following fungal abortion was apparently unimpaired.

Seviour *et al.* (1987) [112] in Victoria, tested several media for inducing sexual reproduction in confirmed or suspected isolates of *M. wolfii* obtained from cattle in various geographical locations. Only silage extract agar worked consistently and rapidly. Its use should provide a simple reliable culturing procedure to assist with identification of clinical isolates of *M. wolfii*. Substantial variation among isolates with morphology characteristic of *M. wolfii* was revealed by temperature growth response curves and electrophoretic patterns of soluble protein extracts.

In 1988 in Queensland bovine abortion due to *A. fumigatus* was diagnosed (M. Kelly, personal communication).

Zygomycosis and pythiosis

These two diseases are grouped as Phycomycosis in the older literature. They will be considered together in this review.

Horses

These diseases were not included in the review by Connole and Johnston (1967) [15] due to a lack of recent references. Their history will therefore be discussed here in some detail.

In 1912 Gilruth [32] first reported the disease known as 'swamp cancer' (pythiosis) in horses in the NT of Australia. Later Lewis (1914) [55] produced a comprehensive report on equine granuloma (swamp cancer) in the NT. It seems the disease first appeared soon after horses were introduced into tropical Queensland early in the 19th century. It was reported in NT by 1881 and was then confined to the northern tropical parts of Australia corresponding fairly closely with the areas subject to heavy tropical rainfall.

The common position of lesions on the legs and lower surface of the abdomen indicated that the infection was acquired while horses were frequenting swamps and marshes. Lewis did not detect any organisms or other parasites in the foci of lesions by microscopy or by cultural methods. He failed to reproduce the lesion artificially. So the aetiology of the disease remained a mystery.

In 1916 Bull [5] published his first report on cutaneous habronemiasis in temperate South Australia and in 1919 he presented further research suggesting that Lewis' swamp cancer and cutaneous habronemiasis were the same condition [6]. This theory was held for many years (Seddon, 1967) [111].

Johnston (1971) [45] first noted subcutaneous zygomycosis and swamp cancer in horses in NSW. Hutchins and Johnston (1972) [41] described these conditions in detail. *Hyphomyces destruens* was isolated from two cases of swamp cancer, having the pathology of ulcerating lesion. The lesions were on the legs and were characterised by their size, rapid spread, intense pruritus and the presence of distinctive necrotic cores or kunkers in the inflammatory tissue. Subcutaneous zygomycosis was diagnosed in a third horse and *Entomophthora coronata* was recovered from a large ulcerated proliferative and obstructive lesion of the left nasal orifice. The pathology was that of an infectious granuloma. Surgical intervention was successful in the three horses.

Pascoe (1973) [88] in Queensland recorded two cases of swamp cancer. Neither recurred after surgical excision followed by topical iodine therapy.

Connole (1973) [14] in Queensland reported three cases (two zygomycosis; one swamp cancer). No fungus was isolated from the first case of a granuloma over the hind cannon bone. Histopathology confirmed the diagnosis of zygomycosis. In the second case *Basidiobolus haptosporus* was isolated from a granulomatous eye lesion. In the third case *H. destruens* was isolated from a lesion of the right fore carpal joint and from the lower cervical lymph node (as a result of metastasis).

Johnston & Henderson (1974) [46] in the NT examined two cases of pythiosis called by them phycomycotic granuloma. They considered that

H. destruens was responsible because the histopathology of the lesions was the same as that of the ulcerative leg lesions in NSW which Hutchins and Johnston [41] attributed to *H. destruens*. As all these lesions exemplified the 'swamp cancer' described by Lewis in 1914 [55] Johnston and Henderson suggested that the aetiological name of 'cutaneous habronemiasis' applied to the condition for the past 50 years was open to question. Further, the available evidence suggested that swamp cancer of horses in Australia was a fungal granuloma attributable to a 'phycomycete' then called *H. destruens*, now known to be a *Pythium* (Oomycete).

Meanwhile in Papua New Guinea, Austwick and Copland (1974) [2] studied isolates, from 4 cases of swamp cancer, which closely resembled limited description available for *H. destruens*. They tried several methods to encourage these fungi to sporulate and succeeded when undifferentiated filamentous sporangia were seen releasing biflagellate zoospores. This showed that *H. destruens* was an Oömycete belonging to the family Pythiaceae in the order Peronosporales, and could be included in the genus *Pythium* Pringsheim.

Pascoe (1974) [89] and Arundel (1978) [1] discussed aspects of these diseases and habronemiasis.

From Townsville, Murray *et al.* (1978) [83a] reported a case of subcutaneous phycomycosis (pythiosis) of the hindlimb of a horse, with metastasis to the inguinal lymph node. *H. destruens* was isolated from the limb and lymph node.

In north Queensland, Murray *et al.* [83] conducted a clinicopathological study of cutaneous lesions resembling swamp cancer. Of 150 lesions studied 37 cases were 'subcutaneous phycomycosis (Pythiosis) five of which were also infected with *Habronema* sp. larvae, considered as secondary invaders. A fungus having the morphology of *H. destruens* was isolated from three lesions. The incidence of 'phycomycosis' in north Queensland was high compared to more temperate southern areas of Queensland and appeared to be related to the wetter seasons. Young horses were more commonly affected.

In Townsville, Miller and Pott (1980) [79] isolated *B. haptosporus* from a case of subcutaneous zygomycosis of the abdominal wall. Although some clinical signs were similar to the disease caused by *H. destruens*, the gross and microscopic pathology were different in several respects. The main characteristic of *B. haptosporus* infection was the narrow eosinophilic sleeve around wide, frequently septate hyphae scattered throughout the affected tissue and found commonly at the advancing border of the lesion. A retrospective histopathological study of 63 cases of 'phycomycosis' diagnosed at the Department of Tropical Veterinary Science, James Cook University, Townsville, since 1970 indicated a further 11 cases with similar histological features typical of true subcutaneous zygomycosis. Another difference between *H. destruens* lesions (pythiosis) and *B. haptosporus* lesions (subcutaneous zygomycosis) was their location. Granulomas of the lower limbs, which are common in *H. destruens* cases, were not seen in those caused by *Basidiobolus*. All lesions of the latter were found on the trunk or head.

Pascoe & Summers (1981) [90], in a retrospective survey of tumours and tumour-like lesions in horses in south-east Queensland between 1958 and 1978, included five lesions of fungal granuloma in a total of 68 tumour-like lesions. The five cases were diagnosed histologically and *H. destruens* was isolated from one. Lesions occurred on the lower limbs in four cases and on the lower abdomen in one. Surgical removal was successful in all cases.

Miller (1981) [67] in north Queensland used a vaccine derived from ultrasonicated hyphae of *H. destruens* to treat 30 cases of clinical pythiosis under the name hyphomycosis, 10 cases of pythiosis following unsuccessful surgery and five cases of *Basidiobolus* infection. Approximately 53% of animals with clinical pythiosis were cured after vaccination and a further 33% clinically improved. All horses with pythiosis treated within two weeks of unsuccessful surgery were cured. There was no response to vaccination in horses with *Basidiobolus* infection, while surgery alone resulted in a cure of approximately 69%.

Response to vaccination at the site of the lesion was observed within seven to ten days of inoculation. There was progressive reduction of pruritus, drying of the surface of the lesion, expulsion of kunkers, fibrosis of the granuloma and eventually complete epithelialisation.

Previously, in Australia and other countries, treatment of equine 'phycomycosis' had involved surgery and systemically administered and/or topical application of drugs. Surgical excision of the whole lesion was the most common and successful treatment and was widely used. Systemic administration of antifungal drugs had been used with varying success. Amphotericin B and sodium iodide given intravenously were apparently successful [89]. Vaccination appears to be a safe and effective alternative control method.

Shipton *et al.* (1982) [116] described the asexual and cultural characteristics of a number of Australian isolates of a pythiacious organism which caused 'equine phycomycosis'. Zoospores were laterally biflagellate with anterior flagella of the tinsel type and were shorter than the posterior whiplash flagellum. Cell wall analysis agreed with data from other studies of recognized *Pythium* species. Oospores were not found, so precluding positive identification as to the species of *Pythium*. In Japan a similar fungus from characteristic equine lesions was identified as *P. gracile* by Ichitani & Amemiya (1980) [42].

Shipton (1982) [113] used various media to examine the physiology of growth and asexual reproduction of a *Pythium* sp. isolated from a lesion on a horse in Townsville. The strain possessed a restricted ability to utilize carbohydrates. A formulation providing about 10 atoms of carbon for every atom of ammonium nitrogen was close to optimum for both growth and asexual reproduction. All forms of sulphur tested, including sulphate, supported growth. Asexual production was achieved on colonies grown on cystine, thiosulphate and some sulphates.

Miller & Campbell (1982a) [72] reported a clinical study of 'phycomycosis' in northern Australia. They identified three specific forms of 'phycomycosis'. Of 266 cases diagnosed in five different laboratories in Queensland and the NT, hyphomycosis (pythiosis) caused by *H. destruens* was responsible for 76.7%, subcutaneous zygomycosis caused by *B. haptosporus* for 18.0% and subcutaneous zygomycosis caused by *C. coronatus* for 5.3%. Most cases of hyphomycosis were observed between March and July, that is after the monsoonal wet summer, but the initial infections apparently began in the wet season between November and May. Both *Basidiobolus* and *Entomophthora* infections occurred regularly during the year and no seasonal incidence could be determined. All of these conditions occurred in horses in similar tropical regions of Queensland and the NT. No breed, sex or age predisposition was found but significant differences between the three fungal diseases in site of infection and appearance and size of the lesion were observed.

Hyphomycosis (pythiosis) was seen as an ulcerative granuloma containing characteristic coral-like kunkers. Lesions were most commonly found on the legs and ventral parts of the chest and abdomen, areas where contact with swamp water was apparent.

Basidiobolus infection was clinically similar to hyphomycosis but was found mostly on the lateral parts of the trunk and face where contact with contaminated soil might be expected. All lesions of *Entomophthora* infection were located in the nasal region thus suggesting infection by inhalation.

Miller & Campbell (1982b) [73] reported immunological studies. Two *in vitro* and one *in vivo* tests were developed to study immunological aspects of 'phycomycosis' in clinically infected, recovered and normal in-contact horses. Serum from all infected horses gave positive readings in an agar-gel double diffusion test; serum from normal and recovered horses did not react. A complement fixation test detected antibody against *H. destruens* in 82% of clinical cases at an average titre of 20. Serum from recovered and in-contact horses reacted sporadically at positive titre. An intradermal hypersensitivity test (Heaf test) was used to detect evidence of cellular immunity to *H. destruens*. Positive tests were observed in 64% of clinically infected horses,

100% of recovered animals and 31% of normal in-contact horses. Negative tests in the clinically infected group were thought to be due to either anergy in chronic cases or no stimulation in very acute cases. It was concluded that many horses showed evidence of past contact with *H. destruens* and had acquired resistance to infection.

Miller and Campbell (1982c) [74] presented results of a survey of granulomatous and neoplastic diseases of equine skin in north Queensland during the period 1970–1980. Of 338 horses affected, 102 (30.2%) suffered from 'phycomycotic' infections. These comprised hyphomycosis 23.4%, *Basidiobolus* infections 5.9% and *Entomophthora* infections 0.9%. More stockhorses (63%) were infected than Thoroughbreds (25%). This was expected as stockhorses were the predominant breed. 'Phycomycotic ' lesions were significantly larger than all other types.

The results confirmed that 'phycomycosis' in horses, was restricted to tropical and subtropical areas where optimal environmental conditions for fungal growth, that is, higher temperature and seasonal surface water, occur. These conditions exist in north Queensland and almost certainly explained the large number of cases collected in the study.

Miller (1983a) [68] reported his study on the biology of three 'phycomycotic agents' pathogenic for horses in Australia. He described the laboratory methods for diagnosis. In Queensland these revealed 38 cases caused by *Pythium* sp. (*H. destruens*), six cases caused by *B. haptosporus* and two caused by *C. coronatus*. Laboratory studies on the chemotactic behaviour of zoospores of *Pythium* sp. showed that they are strongly attracted to both animal hairs and plant tissue. Based on this principle, human hair was used to trap the fungus from water samples taken from different locations near Townsville. By combining previously published information with data obtained in this study, Miller proposed ecological life-cycles for *Pythium* sp., *B. haptosporus* and *C. coronatus*. The life-cycles included mechanisms whereby horses might become infected.

Miller & Campbell (1983a) [75] described experimental pythiosis in rabbits. Rabbits were injected by subcutaneous, intraperitoneal and intravenous routes with suspensions of motile spores of a *Pythium* sp. isolated from a subcutaneous lesion of a horse in north Queensland. Some animals injected by the subcutaneous route were also given cortisone. Animals in each group were highly susceptible to infection. Injection by the subcutaneous route resulted in progressive granulomatous eosinophilic abscesses in all normal and immunosuppressed animals. Preferential hepatic invasion developing into severe necrotizing hepatitis was the most common lesion in the intraperitoneally injected group. In intravenously injected animals severe embolic mycotic nephritis was the principal lesion. A significant progressive leukocytosis with moderate neutrophilia and mild monocytosis was observed in the subcutaneously and intraperitoneally injected immune competent animals. Cortisone-treated subcutaneously injected rabbits did not develop a leukocytosis. Many of the cortisone-treated control animals showed increased susceptibility to bacterial infections while the cortisone-treated fungal-injected animals did not.

Miller (1983b) [69] discussed equine 'phycomycosis' and the three fungi which have been incriminated, *Pythium* sp. (*H. destruens*), *B. haptosporus* and *C. coronatus*. He reviewed the aetiology, taxonomy, history and distribution, pathology, life cycle and pathogenesis, diagnosis, differential diagnosis and treatment.

Miller & Campbell (1983b) [76] described haematological changes in horses with these diseases. Mean values and standard deviations obtained for 34 horses with 'phycomycosis' and 81 normal horses were presented. The data showed that 'phycomycosis' caused a microcytic, hypochromic anaemia with a moderate leucocytosis and absolute neutrophilia and eosinophilia. In 'phycomycosis' the location of chronic blood loss was the ulcerative granuloma. The amount of serosanguineous fluid observed dripping from the surface of the lesion was usually copious but varied widely between individuals. Surprisingly, mean serum protein levels were not altered. How-

ever, in the few chronic cases examined, a reduction of serum protein was marked. Mean globulin levels were significantly raised and probably occurred as part of the immunological response to a chronic infection. Their term phycomycosis includes pythiosis and subcutaneous zygomycosis of *Basidiobolus* and *Entomophthora* etiology.

Shipton (1983) [114] studied the field behaviour of two strains of *Pythium* sp. isolated from horses in north Queensland, by examining their responses to various environmental parameters under controlled conditions. Studies showed the limits of temperature suitable for mycelial growth were close to 17° and 41 °C, with the optimal range being 30–35 °C. Zoospores were produced in low numbers at 19 °C and 41 °C; the highest zoospore populations were formed at 35 °C. Colonies grown at the optimal temperature gave rise to substantial populations of zoospores when transferred to temperatures near the limits suitable for growth. The optimum pH for growth was about 6.5. The pH during growth exerted a marked influence on subsequent sporulation. Few zoospores were produced on colonies grown at pH 6 and 8; the highest populations formed on colonies grown at pH 6.7–6.8. The pH of the induction medium also exerted effects on sporulation. The occurrence of high temperatures and falling pH values in bodies of fresh water during the wet summer months in north Queensland created conditions favourable for both mycelial growth and sporulation of *Pythium*, and could account for the corresponding high incidence of equine infection during that period.

Miller (1983c) [70] reviewed granulomatous and neoplastic diseases in the horse including diseases caused by bacteria, fungi, nematodes, foreign bodies and trauma, as well as unknown factors. The review includes historical aspects of equine cutaneous granulomas, the fungal granulomas especially the 'phycomycoses', and cultural methods for the most common 'phycomycotic' agents.

Parsons & Ladds (1984) [87] performed an immunohistological study of some equine dermatoses in north Queensland. They used commercial anti-equine IgM, IgG (whole molecule) and IgA antisera and an immunoperoxidase method to determine the class and distribution of immunoglobulins within sections of six selected dermatoses. A total of 31 lesions were examined, including six cases of pythiosis, five cases of basidiobolomycosis, three cases of conidiobolomycosis and seven cases of cutaneous habronemiasis. IgG was the major immunoglobulin class present both extra- and intra-cellularly in all lesions. Other than occasional IgM staining within vessels, no IgM or IgA was detected extracellularly. IgG staining varying in intensity was found inconsistently within eosinophilic precipitates (kunkers) of the 'phycomycotic' and cutaneous habronemiasis lesions, supporting the view that an immune-complex reaction might play a part in the formation of such precipitates. Distinct IgG staining of fungal hyphae was observed. The finding of IgG and/or IgA staining of glandular epithelial cells, nearby lymphoplasmacytic cells and of glandular lumen material of equine nasal, sweat and sebaceous glands, supported the concept of a local immunological activity in these sites.

Miller (1984) [71] reviewed current taxonomy and histologic characterization of 'phycomycoses' in domestic animals. He emphasised his belief that 'phycomycosis' is a valid term and justified its use, discussing how to identify the variety of organisms found in 'phycomycotic' lesions. In the current taxonomy, *Pythium* sp., which was within the class Zygomycetes is no longer a true fungus, is now within the kingdom Protista and is the only mammalian pathogen in the Protista. All other organisms within the 'phycomycosis complex' are fungi. The most common are members of the Mucorales, and include such pathogens as *Absidia*, *Rhizomucor*, *Mucor* and *Mortierella*. The 2 pathogens described in the Entomophthorales are *Basidiobolus* and *Conidiobolus*. In veterinary mycology it is appropriate to name the disease after the causative organism based on its current taxonomic position. In most cases it is extremely difficult to identify the genus of infecting organism from clinical or pathologic specimens. Three basic diseases can be diagnosed histologically: pythiosis, entomophthoromycosis (subcutaneous zygomycosis) and mucormycosis (zygomycosis).

Demographic data such as species, breed, sex and age are evaluated with facts such as seasonality, geographic location of the animal and site of the lesion to try to arrive at the most probable etiologic diagnosis. Miller summarized the comparative histologic findings in 'phycomycosis'. A few diseases need to be differentiated from 'phycomycosis' histologically. These include aspergillosis, phaeohyphomycosis and the uncommon opportunistic infections. The most important non-fungal diseases to rule out, especially in equine pythiosis, are cutaneous mastocytosis and cutaneous habronemiasis.

Miller & Campbell (1984) [77] reported the comparative pathology of the 266 cases of 'cutaneous phycomycosis' recorded by Miller & Campbell [72]. *Pythium* sp. hyphae were 2.6 to 6.4 μm in diameter, had thick walls and occasionally were septate. *B. haptosporus* hyphae were 5.1 to 20.5 μm in diameter, had thin walls and commonly were septate. *C. coronatus* hyphae were 5.1 to 12.8 μm in diameter, had thin walls and commonly were septate. A perihyphal eosinophilic cuff (Splendore-Hoeppli phenomenon) with a radius of up to 20 μm was associated with the latter two fungi. Ultrastructurally, *Pythium* sp. was composed of a thick, single density cell wall while *B. haptosporus* and *C. coronatus* had thin, double-layered cell walls.

Little & Kabay (1984) [56] in WA described 'phycomycosis' in a thoroughbred filly with a granulating mass of the upper right foreleg. The horse had access to a swampy area. The mass was treated initially by surgically excising the bulk of the lesion and freezing the base. The followed treatment with sodium iodide, potassium iodide and topical amphotericin B. In biopsy material, necrotic areas were seen containing clumps of abundant branching, occasionally septate structures resembling fungal hyphae. An unidentified non-sporulating fungus was isolated from biopsy material. Fungal granulomas had not been reported previously in WA. Morphology of the fungus, its failure to sporulate, the gross appearance of kunker formation and the marked eosinophil response, were considered to be typical of *H. destruens*. Thus this was in retrospect a case at pythiosis.

During the period 1983–1985, 6 papers of which R. Miller was author or co-author were published on 'phycomycosis'. As these reports dealt with investigations done while Dr Miller was stationed at Louisiana State University, Baton Rouge, Louisiana, they will not be discussed in this review of mycoses in Australia. However, they are relevant to the unfolding sage of the 'phycomycoses' of animals and are listed below:
Miller RI. Letter to editor: Nomenclature of fungal diseases. Vet Pathol 1983; 20: 251–53.
Miller RI, Wold D, Lindsay WA, Beadle RE, McClure JJ, McClure JR, McCoy DJ. Complications associated with immunotherapy of equine phycomycosis. J Am Vet Med Assoc 1983; 182: 1227–29.
Miller RI, Qualls CW, Turnwald GH. Gastrointestinal phycomycosis in a dog. J Am Vet Med Assoc 1983; 182: 1245–46.
Miller RI. Gastrointestinal phycomycosis in 63 dogs. J Am Vet Med Assoc 1985; 186: 473–78.
Miller RI, Olcott BM, Archer M. Cutaneous pythiosis in beef calves. J Am Vet Med Assoc 1985; 186: 984–86.
French DD, Haynes PF, Miller RI. Surgical and medical management of rhinophycomycosis (conidiobolomycosis) in a horse. J Am Vet Med Assoc 1985; 186: 1105–1107.

Shipton (1987) [115] in Townsville described the equine phycomycotic agent known commonly as *H. destruens* or as *P. gracile* (Ichitani & Amemiya 1980) as a new species: *Pythium destruens*. Separation from other species of *Pythium* was on the basis of morphological features, temperature growth profiles, esterase/lipase activity and carbohydrate utilization ability. *P. diclinum* (synonymous with *P. gracile* sensu Middleton) showed minor differences in vesicle, oospore and oogonium size from *P. destruens*. *P. destruens* grew at 40 °C on corn meal agar and hydrolysed esters of lauric and oleic acids. These abilities were not displayed by *P. diclinum*. Equine isolates from Australia, Japan and New Guinea were similar. Also in 1987 a collaborative paper by DeCock *et al.* [11] described *P. insidiosum* sp. nov. as the etiologic agent of pythiosis, a cosmopolitan disease of horses, cattle, dogs and man.

Later Mendoza *et al.* [66] considered *P. destruens* to be a synonym of *P. insidiosum*. Shipton and Zahari (1987) [117] studied sporulation media for *Basidiobolus* species. The use of medium which incorporated glucosamine hydrochloride and casein hydrolysate was found to overcome partially the problem of loss of sporulation ability by *Basidiobolus* species. The medium also appeared to be suitable for the culture of *Conidiobolus* species.

Other animals

Gardiner (1976) [30] in a survey of pulmonary disease in cattle slaughtered in the Kimberley district of WA, incidental to the monitoring of lungs for contagious bovine pleuropneumonia (CBPP), found a few cases of zygomycosis in 1970, three cases (1.2% of total pulmonary cases) and in 1971, 10 cases (5.1%). Most of these were due to species of non-septate zygomycetes. Material suitable for culture was not submitted from these lesions as it was regarded by the inspectors as actinobacillosis of tuberculosis. The typical feature of these pulmonary lesions was the intense concentration of eosinophils around the mycelia, which were mainly large (6–8 microns wide), much branched, of an irregular conformation and very rarely septate.

Lee *et al.* (1976) [53] in Townsville reported a rare case of zygomycosis in the ileum of a dog. Only fixed tissues were available, so isolation of the fungus was not possible. The histological appearance of lesions consisting of numerous granulomatous foci located superficially and scattered in the deep muscular layers of the intestine, was typical of zygomycosis.

English & Frost (1984) [26] diagnosed pythiosis ('phycomycosis') in a German Shepherd dog referred from the NT to the Small Animal Clinic, University of Queensland, Brisbane. The dog had discharging lesions at the junction of the middle and distal thirds of the tail. *H. destruens* was isolated from aspirate collected from the enlarged left inguinal lymph node. Hyphae characteristic of *H. destruens* were seen in stained sections of a biopsy specimen of affected skin. It is noteworthy that this canine infection occurred in the tropical zone where the prevalence in horses is high.

Miller (1983) [68] isolated *B. haptosporus* by indirect culture from the faeces of five bearded dragon lizards (*Amphibolurus barbatus*) captured near a small creek near Brisbane. This lizard is a common Australian agamid and the result confirmed the opinion of Coremans-Pelseneer (1973) [16] that agamids frequently harbour *B. haptosporus*. Miller postulated that in Australia *A. barbatus* might be a natural reservoir for the fungus when environmental conditions become adverse and might help to concentrate fungal material in areas where lizards congregate. The distribution of this lizard included all areas of Australia from which *Basidiobolus* infections had been reported.

As mammals had not been reported to act as reservoir hosts for *B. haptosporus*, Speare and Thomas (1985) [119] in northern Queensland examined the gastrointestinal tracts and faeces of macropods. They cultured for fungi, 285 specimens from 162 macropods which included 16 different species. Seventeen isolates of *Basidiobolus* sp. were recorded from 14 macropods: 15 from faeces, one from stomach contents and one from small intestinal contents. *Basidiobolus* sp. including *B. haptosporus* was isolated from seven species of macropods including wallabies, wallaroos and kangaroos. *Basidiobolus* sp. was not isolated from the adult macropods but was found in 14/112 (12.5%) orphaned pouch young (joeys) or juvenile macropods. *Basidiobolus* sp. was isolated at autopsy from both stomach and rectal contents of one joey and from two other joeys on two occasions, one three months apart at two different locations in Townsville and the other three weeks apart at the same location. Faecal specimens from 23 possums, seven reptiles (including three lizards) and one native bird were negative for *Basidiobolus* as were six samples from artificial pouches and 16 soil samples from three of the seven locations which has been frequented by joeys from which *Basidiobolus* sp. had been isolated.

Immature wallabies and kangaroos in northern Queensland have significant carriage rates of

Basidiobolus sp. The isolation of *Basidiobolus* sp. from two macropods on two occasions suggested a true carrier state might exist. However, its presence in the gastrointestinal tract might merely reflect its common occurrence in the environment. Retrospective sampling of likely sources of *Basidiobolus*, that is pouches and soil, were negative.

Ketterer *et al.* (1989) [51] reported 5 cases of nasal and rhinocerebral zygomycosis caused by *Conidiobolus incongruus* in sheep in Queensland. The cases were in ewes pastured in a low-lying paddock. They are believed to be the first infections due to *C. incongruus* recorded in animals.

Protothecosis and green algal infections

Protothecosis is an infection caused by members of the genus *Prototheca*. The four valid species are considered to be achlorophyllous microorganisms similar morphologically to the green algae in the genus *Chlorella*. Four species are recognized: *P. moriformis*, *P. stagnora*, *P. wickerhamii* and *P. zopfii*. Although the disease is sporadic, over 90 cases in wild and domestic animals have been reported on a worldwide basis (Rippon, 1988) [106].

In Australia the first report in 1977 was of a disseminated infection in a dog in Melbourne, Victoria. The dog was submitted as having a suspected dermatophytosis with the surface of its feet being hard and necrotic, reflecting its exposure to water in a cesspool. *P. wickerhamii* was isolated from subcutaneous granulomata, regional lymph nodes, spleen and rectal swabs. The culture and histological sections were sent to the CDC, Atlanta, where Dr M.S. Sudman confirmed the identification by fluorescent antibody tests (K.L. Hughes, personal communication).

Also in Victoria, two cases of protothecosis in cats have been reported. In one, *P. wickerhamii* was isolated from the popliteal lymph node and a granulomatous tarsal lesion on the left hindlimb (Coloe & Allison, 1982) [12]. In the other, histological examination of a granuloma on the forehead of a cat revealed large numbers of *Prototheca* sp. As only preserved material was submitted, the organism could not be isolated and classified (Finnie & Coloe, 1981) [28].

In 1974 Rogers [108] reported that a pale green lesion in the medulla of a retropharyngeal lymph node of a steer, was detected at slaughter in north Queensland. Only fixed tissue was examined so cultural identification of the organism was impossible. The causal organism was classified as a *Prototheca* sp. because of morphological similarity with descriptions of protothecosis in the literature. Later Kaplan (1977) [49] examined tissue sections from this case by light and electron microscopy. This revealed the presence of well-defined chloroplasts in the cytoplasm of the cells which morphologically, probably belonged to the algal genus *Chlorella*.

Rogers *et al.* (1980) [109] reported on a series of green lymph nodes detected in eight cattle at slaughter over a period of eight years. Seven cases were in north Queensland and one in the NT. The infection was restricted to retropharyngeal and mandibular lymph nodes in all but one instance, when a mediastinal node was also involved. The affected nodes were usually enlarged up to three or four times in advanced infections. The infection evoked a largely proliferative inflammatory response. Non-progressive or slowly progressive infections were established in the peritoneal cavity of rats inoculated intraperitoneally with cultures of the organism. From three cases only preserved material was submitted but four strains of green algae were isolated by culture from the other five cases. All strains were similar on tinctorial and morphological grounds. Two strains were examined by Dr E. Kessler, Erlangen, Germany. He stated that the strains did not fully fit any of the well-defined *Chlorella* species, so he did not consider they belonged to the genus *Chlorella*. They could just as well be unicellular representatives of the genus *Scenedesmus*. Further, it appeared that these strains belonged to a rather common but still ill-defined group of green algae.

The presence of numerous, strongly periodic acid-Schiff (PAS) and Gomori methenamine silver (GMS) positive granules, well-developed

chloroplasts and the green colour of the organisms, both of individuals and colonies, served to differentiate it from the morphologically similar *Prototheca* species which are probably achloric algae.

Retropharyngeal lymph nodes were affected in all 8 cases reported indicating oral infection, presumably by drinking contaminated water. A break in the oral mucosa may be necessary for the organism to penetrate to the regional lymph node. The fact that only a few cases had been detected from a cattle population of about 11.6 million over an eight year period suggested that very few animals were susceptible to infection rather than that few had been exposed. Since the publication of the paper by Rogers [109] there has been another case of a similar green algal infection in a retropharyngeal lymph node of a steer detected at slaughter in Rockhampton, central Queensland (M.D. Connole, unpublished).

To date, infections due to chlorophyll-containing unicellular green algae have been recognized in cattle (10 in Australia and four in the USA), sheep (two in the USA and two in India), a beaver trapped in Canada and a human in the USA – see summary by Kaplan [50].

The puzzling question concerning the development of green lesions in internal organs under conditions of darkness, a phenomenon that appears to reflect the production of chlorophyll by the aetiological agent, has been elucidated by Kaplan [50]. Many members of the Order Chloroccales produce chlorophyll in the dark (Fritsch, 1935) [29]. Kaplan obtained two of the cultures isolated from infected Australian cattle and grew them for two years in continuous darkness, with only occasional transfer, without their losing their green colour [50].

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