

Mycoses Newsletter

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EDITORIAL

With this issue of the Newsletter readers will find a readership questionnaire. I would be grateful if you could fill in this questionnaire and return it to me at your earliest convenience. This is your opportunity to have some input into the proposed content of future issues. So if there is some area that you feel is not covered, and you think could become a regular feature, let us know. Any changes to the Newsletter will be done in a democratic way. If sufficient readers tell us of some shortcoming, then we will do something about it.

We always welcome suggestions for leading articles, special articles, etc, so please give this some thought when completing the questionnaire. Of course, if you would like to contribute a leading article, this would be most welcome! Readers are reminded that we are keen to include case histories in the Newsletter, so if you have had an interesting case recently, please share it with us, and the rest of the readership.

Following on from the leading article featured in the last issue, "Mycology Resources on the Internet", I would recommend that readers visit David Ellis's website (www.mycology.adelaide.edu.au) and go for a roam through the "Fungal Jungle". There has been a great deal of hard work put into this site and it is well worth a visit. It is special to the readership of the Newsletter as it is the first such site in Australasia and one of which we can be justly proud. Whilst roaming in the "jungle" you will notice that the *Mycoses Newsletter* can now be viewed there. There are some small

differences between the electronic and the hardcopy versions. The main difference is that details of reference sources for the quarterly surveillance have been omitted in the electronic version.

With the availability of the Newsletter on the Internet, readers now have a choice as to whether they wish to continue to receive a hard copy of this publication. There are three options outlined in the questionnaire, so please could you indicate which one you prefer. If you do not have access to the Internet, then option one is the only one open to you! There is no pressure on the readership to move to the electronic form of the Newsletter; we are merely offering you the option.

There is always the perennial problem of delays in the publication of the Newsletter and for that we do apologise. There are no full time editorial staff for the *Mycoses Newsletter* and the preparation of each issue has to be prioritised along with the other activities in the laboratory. This year has been exceptional with the need to attend to other priorities more pressing than the Newsletter, so unfortunately there have been unavoidable delays in publication.

Australian readers will find attached a draft copy of Guidelines for assuring the quality of medical mycological culture media. These have been prepared by a committee of the Mycology SIG chaired by Sue Coloe of Melbourne Pathology.

This is very much a discussion document and it now requires the input from individuals and organisations interested in this important area of medical and veterinary mycology. The future success of these Guidelines depends on consensus being reached by all interested parties.

Having read the draft document, please send amendments, suggestions etc so that these reach Sue Coloe by Monday 4 January 1999.

Sue's address: c/o Microbiology Department,
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Ctd. p25

Acknowledgment

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The *Mycoses Newsletter* can now be seen at www.mycology.adelaide.edu.au.

LEADING ARTICLE

Chromoblastomycosis and Phaeohyphomycosis - two separate entities?

Alan Woodgyer,
Microbiological Diagnostic Unit,
The University of Melbourne

The term chromoblastomycosis was first used by Terra and his colleagues in 1922 to distinguish a cutaneous fungal infection from a skin disease known as dermatitis verrucosa. In 1935 chromoblastomycosis was contracted to chromomycosis as it was considered that the former term implied that the causal agents occurred as yeasts in tissues (1). The term chromomycosis was unfortunately used inappropriately for infections caused by a growing number of diverse dematiaceous fungi from a number of different genera and species. This prompted Ajello to introduce the term phaeohyphomycosis for those infections, which on the basis of clinical, pathological and mycological grounds, could be distinguished from chromoblastomycosis (2).

What follows is an overview of some aspects of these two diseases. Readers will find a wealth of detail in the excellent publications by Ajello (3), McGinnis (1), McGinnis and Fader (4), and Matsumoto and Matsuda (5).

The fungi which produce chromoblastomycosis and phaeohyphomycosis are described as dematiaceous. The term dematiaceous is taken to mean that the fungi in culture produce melanin-like pigment in the walls of the hyphae and/or conidia. A recent publication has suggested that in fact dematiaceous originally referred to the bundles of hyphae formed in culture giving a tufted or floccose appearance. More recent definitions emphasise colour and omit any reference to the original meaning (floccose) (6). The word dematiaceous is deeply entrenched in mycology taxonomy and there may well be some resistance in using an epistemologically more correct term, for example, phaeoid fungi, where phaeoid refers to the melanin-like pigment in the cell walls.

In what follows, the aetiology, plus clinical and pathological aspects of each of these two disease entities will be briefly reviewed.

Chromoblastomycosis

Clinical Features: The infection mainly affects exposed body sites, typically the feet, legs, and arms. The initial lesion is a small, pink, scaly papule which occurs at the site of inoculation of the causal

fungus through trauma (thorns, splinters, wood debris). The papules gradually enlarge producing superficial erythematous plaques. These lesions may be scaly or have a warty appearance. At this stage, the infection may superficially resemble phaeohyphomycosis. The lesions further evolve to produce large papillomatous growths which have a cauliflower-like appearance. Satellite lesions may follow localised dissemination via the superficial lymphatics, or scratching the lesions (autoinoculation) which can be pruritic. Ulceration may follow a secondary bacterial infection or injury to the lesions. Haematogenous dissemination of *F. pedrosoi* to the central nervous system has been documented in rare cases of chromoblastomycosis (1).

Epidemiology: Most cases occur in tropical to subtropical countries and are predominantly in male agricultural and forestry workers.

Pathology: The characteristic tissue forms are called muriform cells (Figure 1). In older literature they have been referred to as sclerotic cells, copper pennies, fumagoid cells, Medlar bodies, chromo bodies and chlamydospores. The muriform cells are brown, round to polyhedral, 5 - 12 µm in diameter, and have septa in two planes. Muriform bodies are considered to be a growth form arrested between hyphal and yeast cells (1).

Aetiology: There are seven recognised causal agents of chromoblastomycosis which are in order of descending frequency: *Fonsecaea pedrosoi*, *Phialophora verrucosa*, *Cladophialophora carrionii*, *F. compacta*, *Rhinochrysiella aquaspersa* (1), *Exophiala jeanselmei* (7) and *E. spinifera* (8, 9). The last two are known from a small number of infections. The majority of cases seen in Australia are caused by *C. carrionii* and frequently involve the upper limbs, the hands especially. The cases seen in New Zealand, have all been caused by *F. pedrosoi* and, apart from one case, have been patients from other countries in the Pacific (10). In a recent issue of the Journal of Medical and Veterinary Mycology, a newly described fungus, *Ascosubramania melanographoides*, is documented from a case of chromoblastomycosis in an Indian patient (11).

Phaeohyphomycosis

Clinical Features: This encompasses a number of different presentations which are summarised in table 1. For the purposes of the present discussion we are only interested in subcutaneous and disseminated phaeohyphomycosis.

Subcutaneous infections occur at the site of traumatic implantation of the causal fungi. The lesions usually remain localised as subcutaneous nodules and in many cases present as abscesses encapsulated in fibrous connective tissue. There is no tendency for the abscesses to rupture through the skin (1). In some cases, the lesions may resemble the cauliflower-like lesions that typify chromoblastomycosis (5).

Disseminated infections - the usual initial focus of infection is in the lungs with dissemination to other organs. The most common aetiological agent in such cases is *Xylohypha bantiana*.

Epidemiology: In many cases, these infections occur in patients who are immunocompromised (5); certainly most of the disseminated infections occur in such patients.

Pathology: Muriform cells are not found. Instead the fungal elements appear as dematiaceous septate

hyphae, toruloid hyphae (like chains of yeast cells), pseudohyphae, isolated yeast cells that divide by a process of septation or budding in one plane only, or a combination of any of these (Figure 2) (1). In some instances the dematiaceous pigment may not be apparent in which case the Fontana-Masson silver stain, which specifically stains hyphae containing melanin, can be used to confirm that the hyphae are dematiaceous (3).

Aetiology: In 1994, Ajello published a list of fungi known to produce phaeohyphomycosis which included 57 genera and 101 species (pers comm, ISHAM Congress, 1993). Since then many more fungi have been added to the list. The twelve most common causal fungi are; *Cladophialophora bantiana*, *Curvularia* spp, *Bipolaris* spp, *Exserohilum* spp, *Exophiala jeanselmei*, *Scedosporium apiospermum*, *Ochroconis gallopava*, *Coniothyrium fuckelii*, *Phialophora parasitica*, *P. repens*, *Wangiella dermatitidis*, *Lasiodiplodia theobromae* (12).



Figure 1. Muriform cells characteristic of chromoblastomycosis.



Figure 2. Phaeohyphomycosis - toruloid hyphae (like chains of yeast cells).

Intermediate Cases

It has been suggested that the two disease entities, chromoblastomycosis and phaeohyphomycosis, represent reference points occurring on a continuum of variation (4). Cases of phaeohyphomycosis caused by *F. pedrosoi* and *P. verrucosa* have been reported in patients in the absence of concurrent

chromoblastomycosis. In these infections, the tissue form of the fungus has been identical to that of the fungi causing phaeohyphomycosis. Conversely, *E. jeanselmei* and *E. spinifera* have been reported as the cause of chromoblastomycosis from a small number of patients, and in tissues, the isolates have produced the muriform bodies that are pathognomonic for this infection (7, 8, 9).

Mycetoma might also be considered to occur along the same continuum but is sufficiently different not to be considered here.

In the small numbers of patients with chromoblastomycosis caused by *F. pedrosoi*, where the organism has disseminated to the central nervous system (where the tissue forms are the same as those seen in phaeohyphomycosis), should the resulting infections be described as disseminated chromoblastomycosis or phaeohyphomycosis?

These cases are not especially helpful to us when we try to categorise types of infections and their aetiologic agents. However, they do illustrate the fact that such infections are the outcome of a complex interaction between the host's immune system and the inciting fungus and that the two disease entities are points on a continuum.

It has been suggested that until more detailed information is available, retaining both diseases under the term chromomycosis might be a sensible option (13). While this argument may have some merits, personally I think that this would be a retrograde step in that inevitably cases of the two disease entities will be combined under the umbrella term without distinguishing between them.

Both diseases have also been recorded in animals other than humans, e.g. cats, dogs, fish, frogs (13).

Conclusion

In the majority of cases, there will be good agreement between the observed lesions, the tissue

form and the identity of the causal fungus. The finding of muriform bodies indicates that an infection is chromoblastomycosis but care should be taken to observe that such structures have septa in two planes and can thus be differentiated from budding forms in phaeohyphomycosis where the septa are in one plane only. In many cases of subcutaneous and systemic phaeohyphomycosis, the patient has some underlying immunological deficiency, whereas in chromoblastomycosis the patients are in good health. Where there is any doubt, sections, together with the clinical history, and of course the isolate, should be referred to a reference centre for further evaluation

Table 1 Clinical Classification of Phaeohyphomycosis (from McGinnis)

Category	Specific Disease	Aetiological Fungi
Superficial Phaeohyphomycosis	Black piedra	<i>Piedraia hortae</i>
	Tinea nigra	<i>Phaeoannellomyces werneckii</i> , <i>Stenella araguata</i>
Cutaneous Phaeohyphomycosis	Dermatomycoses	<i>Scytalidium dimidiatum</i> (pycnidial synanamorph of <i>Nattractia mangiferae</i>)
	Onychomycosis	<i>S. dimidiatum</i> (pycnidial synanamorph of <i>N. mangiferae</i>), <i>Lasiodiplodia theobromae</i>
Corneal Phaeohyphomycosis	Mycotic keratitis	<i>L. theobromae</i> , <i>Curvularia geniculata</i> , <i>C. lunata</i> , <i>Exophiala jeanselmei</i>
Subcutaneous Phaeohyphomycosis		<i>Alternaria alternata</i> , <i>E. jeanselmei</i> , <i>E. spinifera</i> , <i>Phialophora parasitica</i> , <i>P. repens</i> , <i>P. richardsiae</i> , <i>P. verrucosa</i> , <i>Phoma</i> spp, <i>Wangiella dermatitidis</i>
Sinusitis		<i>C. lunata</i> , <i>Bipolaris</i> sp, <i>Exserohilum</i> sp.
Systemic Phaeohyphomycosis (in compromised patients)		<i>C. lunata</i> , <i>E. jeanselmei</i> , <i>Ochroconis gallopava</i> , <i>W. dermatitidis</i> , <i>Xylohypha bantiana</i>

4. Fader, RC, McGinnis, MR. Infections caused by Dematiaceous fungi: Chromoblastomycosis and phaeohyphomycosis. Infect Dis Clin North Am 1988; 2: 925-38.

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8. Barba-Gomez, JF, Mayorga, J, McGinnis, MR, Gonzalez-Mendoza, A. Chromoblastomycosis caused by *Exophiala spinifera*. J Am Acad Dermatol 1992; 26: 367-70.9
9. Padhye, AA, Hampton, AA, Hampton, MT, Hutton, NW, Prevost-Smith, E, Davis, MS. Chromoblastomycosis caused by *Exophiala spinifera*. Clin Infect Dis 1996; 22: 331-5.
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11. Rajendran, C. *Ascocubramania* gen. nov., and its *Fonsecaea*-like anamorph causing chromoblastomycosis in India. J Med Vet Mycol 1997; 35: 335-9.
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13. Smith JMB. Opportunistic mycoses of man and other animals. Wallingford: CAB International, 1989.

QUARTERLY SURVEILLANCE

The following isolates of interest were referred to: 1 the Mycology Reference Laboratory at the Microbiological Diagnostic Unit, Department of Microbiology, The University of Melbourne; 2 the Australian National Reference Laboratory in Medical Mycology, Royal North Shore Hospital, St Leonards; 3 the New Zealand Mycology Reference Laboratory, Auckland and Children's Hospitals. A full listing of referral sources is given at the end of this section.

Under clinical data, the term "immunocompromised" also includes HIV + patients. Please note that not all immunocompromised patients are HIV +.

January - March 1998

Dermatophyte infections

<u>Sex, age</u>	<u>Direct examination</u>	<u>Site/clinical data</u>
<i>Trichophyton mentagrophytes</i> var. <i>mentagrophytes</i>		
F 4y	D +	scalp
<i>T. mentagrophytes</i> var. <i>nodulare</i>		
F 14y	D -	ear swab
		This variety of the species, together with the variety <i>interdigitale</i> , is largely confined to producing infections of the feet and toenails. Both have been documented as causing infections of the groin but such infections are infrequent. The isolation of <i>T. mentagrophytes</i> var. <i>nodulare</i> from the external ear is very unusual.
F 82y	D +	great toenails
<i>T. rubrum</i>		
F 45y	D +	finger nail
		The isolate produced a brown diffusible pigment on peptone and Dermasel agars. On lactritmel agar the isolate produced the usual vinaceous red pigment and the micromorphology resembled that of the downy strains of <i>T. rubrum</i> . These features are characteristic of the melanoid, "M", variants of the species. Such strains are rare.
M 15y	D +	hand
		The isolate was atypical in that it failed to produce any pigmentation on lactritmel despite prolonged incubation. The characteristic vinaceous red pigment of the species was formed on potato glucose agar and on bromocresol purple milk glucose agar after 4 weeks incubation.
F 18y	D +	skin scrapings (site not given)
		The isolate failed to produce any pigmentation on either potato glucose agar or lactritmel agar after 4 weeks incubation. The characteristic vinaceous red pigment was formed on Dermasel agar after 4 weeks incubation.
M 23y	D +	groin

<u>Sex, age</u>	<u>Direct examination</u>	<u>Site/clinical data</u>
The isolate was a yellow, "Y", variant of the species.		
<i>T. soudanense</i>		
M 7y	D ?	scalp, kerion, African immigrant
<i>T. tonsurans</i>		
F 41y	D +	skin scraping (no site given)
F 7y	D +	skin scraping (no site given)
F 25y	D +	wrist, Samoan strain. Patient was a Samoan.
<i>T. violaceum</i>		
M 5y	D +	scalp, "exclamation mark hairs" and scaling, no other details available
F 51y	D +	nose and forehead, immigrant from Eritrea
M 2y	D -	scalp, African immigrant
M 3y	D ?	face, African immigrant
M 5y	D +	knee, no details available

Onychomycosis - Non-dermatophyte

<i>Onychocola canadensis</i>		
F 56y	D +	toenail
<i>Scytalidium dimidiatum</i>		
F 13y	D +	soles of feet
M 18y	D +	toenail
		The isolates resembled the Form 1 type of this fungus as described by Dr M K Moore (J Med & Vet Mycol 1988; 26: 25-39).
F 50y	D +	great toenail
		The isolate resembled the Form 3 type of this fungus as described by Dr M K Moore (J Med & Vet Mycol 1988; 26: 25-39).

Otomycosis

<i>Aspergillus niveus</i>		
M 71y	D +	R & L ear swabs
		This fungus has been documented as a rare cause of otitis externa.

Chromoblastomycosis

<i>Fonsecaea pedrosoi</i>		
M 39y	D +	right hand, 14y history.
		The infection appears to have been acquired in New Zealand. The patient could not recall any trauma to the site. The patient was started on a course of itraconazole but has failed to return for follow up, so the efficacy of the treatment cannot be assessed.

Miscellaneous filamentous fungi

<i>A. fumigatus</i>		
M 48y	D ?	sputum, methicillin resistant <i>Staphylococcus aureus</i> also isolated from same site.
		The isolate was a poorly sporulating albino variant of the species.
<i>Aureobasidium pullulans</i> var. <i>melanogenum</i>		
M 1y	D ?	foreign body in eye
<i>Fusarium episphaeria</i> subspecies <i>merismoides</i>		
M ?	D +	right malleolus

<u>Sex, age</u>	<u>Direct examination</u>	<u>Site/clinical data</u>
<i>Histoplasma capsulatum</i>		
F ?	D ?	aspirate (site unknown), immunocompromised patient, from Philippines, deceased.
<i>Rhizopus microsporus</i> var. <i>rhizopodiformis</i>		
F 90y	D +	sputum
<i>Scedosporium apiospermum</i>		
F 49y	D +	brain tissue, lung transplant patient on immunosuppressive therapy - deceased
F ?	D ?	bronchial wash
<i>S. prolificans</i>		
F ?	D ?	sputum and bronchial lavage
F 68Y	D ?	sputum

Cryptococcosis

<i>Cryptococcus neoformans</i> var. <i>neoformans</i>		
M 28y	D +	CSF, immunocompromised, meningitis
M 57y	D nd	blood, post-transfusion
F 74y	D ?	wound swab of arm, renal transplant patient
M 28y	D +	CSF
F 57y	D ?	lung aspirate
M 66y	D ?	blood, chest wall

Aerobic actinomycete infections

<i>Dermatophilus congolensis</i>		
M 38y	D +	left forearm, patient from a rural area.
<i>Nocardia asteroides</i>		
F 55y	D ?	sputum, loss of weight, recurrent upper respiratory tract infection
M 55y	D +	sputum
M 42y	D +	sputum
<i>N. farcinica</i>		
F 18y	D ?	aspirate lung abscess
F 49y	D ?	sputum, febrile
F 62y	D +	lung
F 66y	D +	lung, post mortem
M 63y	D +	brain abscess
M 21y	D ?	sinus swab, abscess R buttock drained 3 weeks previously with subsequent formation of sinus
<i>N. nova</i>		
M 67y	D ?	pus, abscess L hand; isolated on two occasions
F 49y	D ?	lung tissue (PM), lung transplant patient

Veterinary mycology

Filamentous fungi

<i>Scedosporium apiospermum</i>		
Bovine	D ?	milk, bovine mastitis

<u>Sex, age</u>	<u>Direct examination</u>	<u>Site/clinical data</u>
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Yeast infections

C. neoformans var. *neoformans*

Feline	D ?	nasal swab
Feline	D +	lymph nodes

C. neoformans var. *gattii*

Feline	D ?	nasal swab
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Referral Sources for data used in Quarterly Surveillance for January - March.

Australia: ALFRV Alfred Hospital; AUSTV A & RMC; BALLV Dorevitch Pathology, Ballarat; BENDV Bendigo Base Hospital; CBHCQ Cairns Base Hospital Pathology; DOREV Dorevitch Pathology; GRIBV Gribbles Pathology, South Yarra; GRIFN South West Pathology, Griffin NSW; LIVHN Liverpool Hospital; MLDPV Melbourne Pathology; NCHWN New Childrens Hospital, West Mead Campus, Parramatta; POWHN Prince of Wales Hospital, Randwick NSW; RNSHN Royal North Shore Hospital; STVDN St Vincents Hospital, Darlinghurst; SDVLN Scone Diagnostic Veterinary Laboratory; VETSN Dept Veterinary Pathology, Uni of Sydney.

New Zealand: AKAHZ Auckland & Childrens Hospitals; AKDIZ Auckland Diagnostic Laboratory; AKMLZ Auckland Medical Laboratory; AKMMZ Middlemore Hospital, Auckland; BNWHZ Wairau Hospital, Blenheim; CHCHZ Christchurch Hospital; CHPSZ Medlab South, Christchurch; DGDHZ Dargaville Hospital; HNMLZ Hamilton Medical Laboratory; HNWHZ Waikato Hospital, Hamilton; LHVDZ Valley Diagnostic Laboratory, Lower Hutt; TITHZ Medlab South, Timaru. AKVDZ Auckland Veterinary Diagnostics.

April - June 1998

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Under clinical data, the term "immunocompromised" also includes HIV + patients. Please note that not all immunocompromised patients are HIV +.

Dermatophyte infections

<u>Sex, age</u>	<u>Direct examination</u>	<u>Site/clinical data</u>
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Trichophyton concentricum

F 2y	D +	buttock, Pacific Islander
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T. equinum var. *equinum*

F 19y	D +	right forearm, contact with horses Human infections caused by this dermatophyte are rare.
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T. mentagrophytes var. *mentagrophytes*

F 3y	D +	scalp
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T. mentagrophytes var. *nodulare*

M ?	D +	foot
F 26y	D +	toenails

T. rubrum

M 51y	D +	foot The isolate produced a brown diffusible pigment on peptone and Dermasel agars. On lactrimel agar the isolate produced the usual vinaceous red pigment and the micromorphology of the isolate resembled that of the downy strains of <i>T. rubrum</i> . These features are characteristic of the melanoid, "M", variants of the species. Such strains are rare.
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T. rubrum var. *oceanicum*

F 13y	D ?	legs, Samoan This variety of <i>T. rubrum</i> is endemic in parts of the South Pacific.
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T. soudanense

<u>Sex, age</u>	<u>Direct examination</u>	<u>Site/clinical data</u>
F 7y	D +	scalp, Somali immigrant
M 4y	D ?	scalp, Somali immigrant siblings The isolates from the brother were pleomorphic variants of <i>T. soudanense</i>
<i>T. tonsurans</i>		
M ?	D +	face, Samoan strain Subsequent inquiries revealed that the patient was Samoan.
F 8y	D +	scalp
F 1y	D +	scalp
M 5y	D +	ear
F 1y	D -	scalp
F 1y	D +	? site
F 20y	D -	scalp
M 1y	D +	face
<i>T. verrucosum</i>		
F 56y	D +	leg, no details of contact with cows / cattle
M 46y	D +	leg, no details of contact with cows / cattle, patient from a rural area
M 26y	D +	? site, no details of contact with cows / cattle
M ?	D ?	? site, no details of contact with cows / cattle
<i>T. violaceum</i>		
F 9y	D +	scalp, African immigrant
M 4y	D +	scalp. Patient born in Australia, no other information available. The initial Woods lamp examination of the patient's scalp showed fluorescing hairs. This dermatophyte does not produce fluorescent metabolites when growing on hair. The false positive result for this test was probably caused by some other fluorescent material on the patient's scalp, eg topical medication
F 35y	D +	hand, African immigrant
M 9y	D +	scalp, African immigrant
M 6y	D +	scalp, African immigrant
M 8y	D +	abdomen, African immigrant
M 3y	D +	neck & arm, no details available

Onychomycosis - Non-dermatophyte

Onychocola canadensis

F 64y	D +	great toenail
F 81y	D +	great toenail

Scytalidium dimidiatum

M 56y	D +	soles of feet and interdigital spaces, patient originally from Mauritius
F 47y	D +	toenails
M 63y	D +	toenail, the isolate formed pycnidia characteristic of the pycnidial form of the fungus, <i>Natrassia mangiferae</i> . The isolates resembled the Form 1 type of this fungus as described by Dr M K Moore (J Med & Vet Mycol 1988; 26: 25-39).
M 31y	D +	toenail
F 38y	D +	toenail
F 67y	D +	sole The isolates resembled the Form 3 type of this fungus as described by Dr M K Moore (J Med & Vet Mycol 1988; 26: 25-39).

Otomycosis

<u>Sex, age</u>	<u>Direct examination</u>	<u>Site/clinical data</u>
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Scedosporium apiospermum

F ?	D ?	ear swab
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Miscellaneous filamentous fungi*Absidia corymbifera*

F 4y	D +	bronchial washing
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Bipolaris hawaiiensis

M 16y	D ?	antrum tissue
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B. spicifera

M ?	D ?	fine needle aspirate of lymph node in mass on thigh
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Exophiala jeanselmei

M 77y	D ?	wound swab of foreign body abscess on wrist
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Exserohilum rostratum

F 40y	D ?	tissue, acute myeloid leukaemia (AML) patient
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Fusarium solani

M 77y	D +	nasal septum
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Phaeoannellomyces werneckii

F ?	D ?	tinea nigra, ? site
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S. apiospermum

M ?	D ?	sputum
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Wangiella dermatitidis

M ?	D ?	black dots inside CAPD catheter line
F 9y	D ?	sputum

Yeast infections*Candida guilliermondii*

F 4w	D nd	blood, sepsis, chronic lung disease
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Malassezia pachydermatis

M 4w	D nd	blood, ? sepsis
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Cryptococcosis*Cryptococcus neoformans* var. *neoformans*

M 38y	D ?	CSF & blood, fever, cough, headache
M 42y	D nd	blood

Aerobic actinomycete infections*Nocardia asteroides*

M 83y	D ?	bronchial washing
F 60y	D +	bronchial washing, skin nodules
M 65y	D ?	sputum, immunosuppressed patient with pneumonia. The isolate was a slow-growing type II form of the bacterium.
F 47y	D ?	bronchi-alveolar lavage, necrotic lung. The isolate was a type I form of the bacterium.
M 77y	D ?	sputum, post cardiac surgery

<u>Sex, age</u>	<u>Direct examination</u>	<u>Site/clinical data</u>
<i>N. brasiliensis</i>		
F 84y	D ?	pus, abscess on foot
M 74y	D ?	wound swab, finger - diabetic patient
M 30y	D ?	wound swab, ? site
<i>N. farcinica</i>		
M 50y	D nd	blood
F 50y	D ?	sputum
<i>N. nova</i>		
M 4y	D nd	face
F 49y	D -	sputum
<i>N. nova</i> ctd		
F 73y	D ?	sputum
M 68y	D ?	wound exudate, wound to R hand of oncology patient
F 26y	D ?	sputum, lung transplant patient with pneumonia

Veterinary mycology

Filamentous fungus

Paecilomyces lilacinus

Tortoise D + necrotic dermatitis on extremities

Fusarium dimerum

Snake D + trachea
The identification was confirmed by Dr Brett Summerell, Royal Botanic Gardens, Sydney.

Yeast infections

C. neoformans var. *neoformans*

Feline D - sneezing & upper respiratory signs, swab from R nostril
Feline D + nose

C. neoformans var. *gattii*

Feline D + nose

Referral Sources for data used in Quarterly Surveillance for April - June.

Australia: ALFRV Alfred Hospital; BARON Drs Barratt & Smith, Orange; CBHCQ Cairns Base Hospital Pathology; DOREV Dorevitch Pathology; DUBBN Orana Pathology, Base Hospital, Dubbo; GEELV Pathcare, Geelong; GOSFN Gosford District Hospital; GRIBV Gribbles Pathology, South Yarra; HAMPN Hampson Pathology, Kotara; LIVHN Liverpool Hospital; LHNMN Lower Hunter & Northumberland Pathology Service; LYNRQ Dr T B Lynch, Rockhampton; MLDPV Melbourne Pathology; POWHN Prince Of Wales Hospital, Randwick; RAHCN Royal Alexander Hospital for Children; RNSHN Royal North Shore Hospital; RPAHN Royal Prince Alfred Hospital, Camperdown; RYCHV Royal Childrens Hospital, Parkville; RYHHT Royal Hobart Hospital; SFXCV St Francis Xavier Cabrini Hospital, Malvern; STGKN St George Hospital, Kogarah; STVDN St Vincents Hospital, Darlinghurst; SULLQ Drs Sullivan, Nicolaidis & Partners; WDPSV Wangaratta District Pathology.

DVLRN Diagnostic Veterinary Laboratories, Randwick; SVLSN Scone Diagnostic Veterinary Laboratory; VETSN Dept Veterinary Pathology, Uni of Sydney.

New Zealand: AKAHZ Auckland & Childrens Hospitals; AKDIZ Auckland Diagnostic Laboratory; AKMLZ Auckland Medical Laboratory; CHCHZ Christchurch Hospital; HNMLZ Hamilton Medical Laboratory; HTHHZ Memorial Hospital, Hastings; LHVDZ Valley Diagnostic Laboratory, Lower Hutt; ROMLZ Rotorua Medical Laboratory; TGMLZ Tauranga Medical Laboratory; WNWHZ Wellington Hospital.

BIANNUAL SUMMARY OF OPPORTUNISTIC MYCOSES JULY - DECEMBER 1997

The following data were collated from replies received from twelve out of twelve sentinel laboratories from throughout New Zealand. See list of participating laboratories at the end of this section.
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Sex, age	Direct examination	Site	Clinical data - including treatment and outcome (where known)
A. Filamentous fungi			
<i>A. fumigatus</i>			
F 46y	D +	heart valve	ALL
M 70y	D +	blood & aortic valve	previous valve replacement, endocarditis
M 54y	D nd	renal bed tissue	renal abscess
F 11y	D +	fine needle aspirate from lung	
<i>Cladophialophora bantianum</i>			
M 27y	D +	temporalis muscle	trauma
<i>Exophiala jeanselmei</i>			
M 31y	D nd	ball of foot	
<i>Geosmithia argillacea</i>			
F 57y	D +	pigtail drain from pleural cavity	bronchiectasis
<i>Scedosporium prolificans</i>			
M 76y	D +	R mastoid cavity	discharging R mastoid cavity
B. Yeasts			
<i>Candida albicans</i> and <i>Malassezia furfur</i>			
M 6m	D +	bag urine	
<i>Candida albicans</i>			
M 11y	D +	abdominal pus	
M 83y	D -	paraspinal collection & L 5 disc	
F 39y	D +	CAPD & tenckhoff tip	
F 14y		catheter blood culture	
F 59y		catheter blood culture & catheter tip	
M 77y		catheter blood culture	
M 78y		catheter urine	
F 68y	D +	perinephric pus	
F 23y		catheter blood culture	
F 65y		blood	? mycotic abscess
F 65y		blood & CVL tip	
M 6y		blood	long term TPN
M 75y		blood & CVL tip	
M 1m		blood, knee aspirate	neonatal infection
M 64y	D -	CAPD & tenckhoff tip	renal failure
M 68y	D -	CAPD	renal failure
F 65y	D +	CAPD	renal failure
M 58y		catheter blood culture	oncology patient
M 67y	D +	intra-abdominal abscess	
F 51y		blood & CVL tip	

Sex, age	Direct examination	Site	Clinical data - including treatment and outcome (where known)
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C. glabrata

F 62y	D -	CAPD	
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C. guilliermondii

M 50y	D +	CAPD & tenckhoff tip	
F 69	D +	CAPD & tenckhoff tip	
F 48	D -	CAPD	renal failure

C. guilliermondii & *C. parapsilosis*

F 56y	D +	finger tissue	
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C. parapsilosis

F 71y	D +	corneal graft site	
F 30y		catheter blood culture	
F 92y		blood	
F 66y		blood	
F 2m		blood	pneumonia
F 76y		blood & CVL tip	long term intensive care
M 72y		blood	
M 59y	D -	hip tissue	total hip joint replacement
F 54y	D -	CAPD	renal failure

C. pelliculosa

F 35y		blood	
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C. tropicalis

M 86y		blood	
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C. neoformans var. *neoformans*

M 40y	D +	pleural fluid, sputum, urine, faeces & blood	HIV +
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C. Pneumocystis carinii

No data received. This may be due to prophylaxis in patients at risk.

D. Aerobic actinomycetes

Nocardia asteroides

M 53y	D -	bronchial washing	HIV +, recovered
F 36y	D -	tracheal aspirate	Ca oesophagus
M 80y	D -	sputum	

N. farcinica

M 63y		blood	
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Note: The above data were supplied by the following participating sentinel laboratories: Auckland and Children's Hospital; Canterbury Health Laboratories; Dunedin Hospital; Greenlane Hospital; MedLab Central (serving Palmerston North Hospital); Medlab, Bay of Plenty (serving Tauranga Hospital); Memorial Hospital Hastings; Middlemore Hospital; Rotorua Hospital; Southland Hospital; Waikato Hospital; Wellington Hospital.

Data collected and collated by Dinah Parr, Auckland & Children's Hospitals.

ALL	: acute lymphocytic leukaemia	CVL	: central venous line
CAPD	: continuous ambulatory peritoneal dialysis	TPN	: total parenteral nutrition

NEWS, VIEWS AND REVIEWSASM Meeting, Hobart, 27 September - 2 October 1998

This year the Australian Society for Microbiology held its Annual Scientific Meeting and exhibition at Wrest Point Casino in Hobart, Tasmania.

The mycology program was diverse with symposia entitled "Fungi from Fauna", "Harvest to Home; Fungi in Food", "Clinical Mycology" and "Fungal Genetics I and II". The proffered papers session outlined current mycological research being undertaken at universities, research organisations and hospitals. All sessions were well attended, very enjoyable, with much discussion being evoked. It was also pleasing to see the high quality of posters pertaining to mycology that were exhibited.

The Mycology SIG would like to thank Professor JMB (Sandy) Smith, the Mycology International Guest Speaker, for his participation and his excellent presentations in several of the symposia. We would also like to take this opportunity to thank Pfizer for sponsoring his visit.

Report by Sue Coloe, Mycology SIG Convener.

IUMS Congress of Mycology, Sydney, 16 -20 August, 1999.

The International Union of Microbiological Societies (IUMS) will hold its triennial Congresses in Sydney in August 1999. The Virology Division of IUMS will meet during the week August 9-13 and, in association with the Bacteriology and Applied Microbiology Division, the Mycology Division will meet from August 16-20. An extremely varied and interesting program has been planned for the Mycology Congress and once this has been finalised, details will be publicised in a future issue of the Newsletter.

Because of this meeting, there will not be an Annual Scientific Meeting of the ASM for 1999. It is expected that many members of the ASM will want to avail themselves of the opportunity to attend the IUMS Congresses.

ASM Annual Scientific Meeting, Cairns, 9-14 July 2000.

The Scientific organising committee for the Cairns 2000 meeting are calling for suggestions for overseas speakers to be invited to this meeting. The Mycology SIG would like to have an international mycologist present at this meeting. Please send suggestions to Sue Coloe, Microbiology Department, Melbourne Pathology, 32 Smith Street, Collingwood, VIC 3066, by Friday 18 December.