Case 1: A 52-year-old geologist has returned from Borneo. He presents with a 9-day history of migratory serpiginous, intensely pruritic lesions on his left ankle (Figure 1). FBC is normal.

Discussion
Cause: Cutaneous larva migrans due to dog and cat hookworm *Ancylostoma braziliense*, or less commonly other animal hookworms including *A. caninum*, *Uncinaria stenocephala* and *Bunostomum phlebotomum*. Rarely human hookworms, *Strongyloides stercoralis* and *Gnathostoma spinigerum* and insect larvae may be responsible.

Diagnosis: This is usually a clinical diagnosis. Papules develop at the penetration site. Subsequently the larvae migrate a few millimetres up to centimetres per day between the stratum corneum and stratum granulosum of the skin. Eosinophilia is present in only approximately 40% of cases. No serology is available.

Treatment: Without treatment, skin lesions gradually disappear. The larvae can, however, migrate for 2 years. Treatments include topical thiabendazole (crush up a 0.5g tablet and combine with petroleum jelly) for 5-15 days. Albendazole, 400mg/d for 3-7 days is an alternative but Ivermectin 12mg stat is considered most effective, with results usually within 3 days.

Case 2: A 20-year-old male presented with a 1-week history of fever and rash after returning to Australia from Vanuatu. He had received appropriate pre-travel vaccinations, as well as malarone for malaria prophylaxis. Three days after return he developed fatigue, anorexia and fever (temperature 38.6°C), and complained of headaches, rhinorrhea, myalgias and a rash that started on his face and spread to the trunk, extremities, palms, and soles. Physical examination was notable for persistent fever, diffuse erythroderma (Figure 2), scattered petechiae and bilateral conjunctival suffusion. Laboratory studies included: WBC count, 4500 cells/L (with 39% polymorphonuclear neutrophils, 8% bands, 26% lymphocytes, and 21% atypical lymphocytes); platelet count, 105,000 platelets/mm³; aspartate aminotransferase level, 192 IU/L; and alanine aminotransferase level, 180 IU/L.

Discussion
Cause: Dengue fever. The most commonly diagnosed tropical infections that cause fever in the returned traveller are malaria, dengue, typhoid, and viral hepatitis. The epidemiology may change with appropriate pre-travel vaccination and use of malaria prophylaxis. Dengue is transmitted most commonly through a human-mosquito cycle with the *Aedes aegypti* mosquito the most common vector. The incubation period of dengue virus is generally 4-7 days. The short incubation period is an important epidemiological clue.

Diagnosis: Leucopaenia, atypical lymphocytes and thrombocytopaenia in the full blood count, and mild abnormality of liver function, are all characteristic. Serological testing of an acute-phase serum specimen demonstrated a strongly positive dengue specific IgM titre. Convalescent testing demonstrated a seroconversion for dengue IgG.

Treatment: No specific treatment is available. The risk for dengue haemorrhagic syndrome increases with subsequent exposure to different serotypes of dengue virus, which has implications for persons with a history of dengue fever who travel to regions where dengue is endemic, where they may be re-exposed. The greatest risk for dengue hemorrhagic syndrome however is usually in children rather than adults living in endemic areas.

Case 3: A 42-year-old ambulance officer presented to his GP with a boil on his shoulder following a trip to Peru, Bolivia, Brazil, Argentina and Chile, including the basin of the Amazon River in Bolivia. A small punctum was noted and he was advised to apply magnaplasm to the lesion. The following day he returned with this specimen (Figure 3) which popped out of the punctum.

Discussion
Cause: Human botfly larva − *Dermatobia hominis*
DISCUSSION

Cause: Myiasis due to Dermatobia hominis (botfly). Myiasis, from the Greek word myia for ‘fly’, is defined as the infestation of live human and vertebrate animals with dipterous (fly) larvae. A number of fly species in the order Diptera can cause human myiasis. Most reports of furuncular myiasis involve infestation by 
D. hominis in travellers returning from Mexico and Central and South America. There have been recent reports of a clustering of cases infected in the Amazon basin in Bolivia. The African tumbu fly (Cordylobia anthropophaga) causes cutaneous infestation with furuncular lesions in sub-Saharan Africa.

Treatment: Use of an occlusive ointment or mechanical extraction is usually successful in treating cutaneous disease, with no long-term consequences. A unique aspect of the life style of 
D. hominis is the means of host infestation. The female captures a tick or an insect – usually a diurnal mosquito – and attaches her eggs to its underside, a method of egg delivery called ‘phoresy’. When the mosquito takes a blood meal, the eggs hatch and the larvae penetrate the skin creating a furuncular lesion. The African tumbu fly, on the other hand, lays its eggs on clothing that has been spread out to dry on the ground. The larvae hatch and when the clothes are worn, penetrate intact skin, producing cutaneous abscesses. The heat from ironing clothing before wearing is effective in destroying the larvae.

Case 4: A 41-year-old male recently returned from northern Iraq with an 8-week history of 3 sores around his ankles which had been unresponsive to antibiotic therapy (Figures 4 and 5).

Figure 4: Curaneous leishmaniasis sores
Figure 5: Curaneous leishmaniasis sores

Cause: Cutaneous leishmaniasis. This is the most common form of leishmaniasis. Infection is characterised by one or more sores or nodules on the skin, which are often described as being volcano-like in appearance, with raised edges and a central crater. The sores are usually painless (unless they are secondarily infected with bacteria), and may be associated with swelling of lymph nodes proximal to the lesions. Old World leishmaniasis (i.e., infection with Leishmania major, L. tropica, or L. infantum) is endemic in many parts of Iraq and the Middle East. More than 600 cases have been reported in US soldiers returning from Iraq following deployment along the Iraq-Syria border and the Iran-Iraq border. Leishmaniasis is transmitted by the bite of an infected sandfly. Most sores appear within a few weeks of the sandfly bite, but can appear months later.

Diagnosis: The diagnosis was confirmed by histological examination of skin biopsy. In several of the giant cells and a few macrophages, small organisms without capsules (amastigotes) were seen. A fresh biopsy was cultured in Schneiders Drosophila media and yielded promastigotes. The specimen was also referred for PCR species identification.

Case 5: A 35-year-old engineer working intermittently for the past 2 years in Indonesia returned on 6 weeks’ leave and presented with low grade fever, widespread maculopapular rash (Figure 6) which also involved the palms of the hands (Figure 7) and generalised painless lymphadenopathy.

Figure 6: Maculopapular rash lower limbs
Figure 7: Maculopapular rash palms of hands

DISCUSSION

Diagnosis: Secondary syphilis. The diagnosis was made serologically with syphilis screening EIA Positive; RPR 1:256; TPPA Reactive. The incubation period is 9-90 days. Serology in primary syphilis may be negative and should be repeated in 6 weeks; other STDs should be screened for at the same time. Studies of travellers indicate that 5% of travellers will have casual sex during travel. This figure rises to 20% for men travelling alone. Most casual sex is unprotected. Overseas-acquired infection is a growing proportion of new HIV diagnoses in Australia and sexually transmitted diseases such as syphilis, HIV, gonorrhoea and Chlamydia need to be considered.

Treatment: He received intramuscular injections of benzathine penicillin once weekly for 3 weeks. Screening for other STDs yielded a positive Chlamydia PCR and he was treated with azithromycin 1g stat. Follow-up HIV testing was negative.

Case 6: A 30-year-old traveller presented 2 weeks after a holiday to East Africa, visiting wild game parks and bush walking. His illness was characterised by fever, headache, myalgia, and a cutaneous eruption (Figure 8) including the lesion on his leg shown in Figure 9.

Figure 8: Cutaneous eruption
Figure 9: Eschar at the site of tick bite
DISCUSSION

**Cause:** African tick bite fever caused by Rickettsia africae.

Figure 8 shows the eschar (tache noire), the inoculation site of the vector and reservoir, the cattle tick, genus *Amblyomma*. This aggressive biter is common on vegetation in the summer, and readily bites humans. The incidence of African tick bite fever among short-term safari tourists may be as high as 5% and exceeds that for other tropical fevers among short-term visitors to sub-Saharan Africa. As well as an eschar, which may be multiple, vesicular cutaneous rash and mouth blisters are seen in up to 30% of patients. As is not uncommon, his travelling companion presented with a similar illness within one day of his presentation.

**Diagnosis:** Although the acute serum sample had no antibodies to the spotted fever Rickettsial group, a convalescent sample demonstrated seroconversion for IgG to a titre of 512 using an Immunofluorescence assay (IFA). The antigen used for testing cross-reacts with all rickettsiae belonging to the spotted fever group.

**Treatment:** He received the standard regimen of doxycycline 100mg bd for 10 days, with improvement 24 hours after the start of therapy.

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**Nodular lymphangitis – a distinctive syndrome**

A distinctive form of lymphangitis occurs as nodular subcutaneous swellings along the involved lymphatic chains; its presence should suggest infection with a circumscribed group of micro-organisms.

<table>
<thead>
<tr>
<th>Common</th>
<th>Unusual</th>
<th>Rare</th>
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<tbody>
<tr>
<td><em>Nocardia brasiliensis</em></td>
<td><em>Leishmania braziliensis</em></td>
<td><em>Mycobacterium kansasii</em></td>
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<tr>
<td><em>Mycobacterium marinum</em></td>
<td><em>Francisella tularensis</em></td>
<td><em>Blastomyces dermatitidis</em></td>
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<tr>
<td><em>Sporothrix schenckii</em></td>
<td><em>Other Nocardia spp</em></td>
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<td><em>Leishmania major</em></td>
<td><em>Cryptococcus neoformans</em></td>
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<td><em>Streptococcus pyogenes</em></td>
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<td></td>
<td><em>Bacillus anthracis</em></td>
<td><em>Cowpox</em></td>
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Nocardia spp. are Gram positive aerobic actinomycetes with a worldwide distribution in soil and organic matter. Immunocompetent patients develop primary cutaneous nocardiosis, generally manifested as a single tender abscess or ulcerated papule, a few days to 6 weeks after a minor wound contaminated with soil. Infection with *Nocardia brasiliensis* in particular may develop nodular lymphangitis, sometimes associated with regional adenopathy and mild systemic symptoms. The initial wound may drain frank pus, or have already healed except for a dry scab and surrounding erythema. The lymphangitic nodules often ulcerate and may suppurate (Figure 1). Patients usually present having failed to respond to antistaphylococcal antibiotics.

Epidemiological and clinical features may suggest specific entities. The first 3 are the most common aetiological agents in Australia. To differentiate and diagnose these conditions a tissue biopsy for histology and culture is usually necessary. The form should request specialised culture for mycobacteria, fungi and *Nocardia*.
Mycobacterium marinum

*M. marinum* is an environmental atypical mycobacterium associated with fresh and salt water. Human infection usually occurs after injuries in aquariums, swimming pools or other bodies of water, or injuries related to fish spines; hence other names include swimming pool or fish tank granuloma. After a variable incubation period of 2-3 weeks, small violet papules develop which may become nodular, verrucous, or ulcerative. Nodular lesions may develop along draining lymphatic glands (Figure 4), but regional lymphadenitis and systemic complaints are uncommon.

The diagnosis is often delayed for 5 months or more. The acid fast organism (Figure 5) *M. marinum* is cultured after incubation at 32°C on specialised solid (Figure 6) or liquid mycobacterial media. Mycobacterial culture needs to be requested; cultures are held for 6 weeks, although most *M. marinum* grow in 7-10 days.

Treatment includes combination rifampicin and ethambutol for 3-6 months, although doxycycline, minocycline, clarithromycin and trimethoprim/sulfamethoxazole have all been used with success. Routine susceptibility testing is unnecessary unless relapse occurs. At SNP approximately 6-12 *M. marinum* are cultured each year.

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Sporothrix schenckii

*Sporothrix schenckii* is a thermally dimorphic hyphomycete (fungus) found in soil and plant debris such as decaying vegetation and sphagnum moss. Most cases occur sporadically following trauma in an outdoor setting. Occasional outbreaks are described as occurred on the Darling Downs when Wilson et al described an outbreak of 16 cases between January and October 1995 following distribution of contaminated hay. Infrequently sporotrichosis is a zoonotic infection following contact with infected cats. The inoculation site becomes ulcerated, verrucous, nodular, plaque-like, or pustular following an incubation period of 3 days – 12 weeks (mean 3 weeks). Cutaneous nodules develop along draining lymphatics which may suppurate and ulcerate in 30% of cases (Figure 7).

Histology shows a mixed suppurative granulomatous process with occasional micro-abscess formation and characteristic asteroid body seen in the centre of the micro-abscess (Figure 8). Biopsies are cultured at 30°C and incubated for 28 days, although most isolates grow in 3-5 days. Microscopy shows single conidia lining delicate septate hyphae and occasional branching conidophores at 90 degrees (Figure 9). Conversion of the mould to a yeast by subculturing at 35°C demonstrates the dimorphism.

The treatment of choice is itraconazole for 3-6 months. Until recently, potassium iodide was standard treatment. It is inconvenient to take and associated with side effects including metallic taste, salivary gland enlargement and rash. Local hyperthermia may be effective for treating fixed cutaneous disease — used exceptionally, e.g sporotrichosis in pregnancy, where the azoles are contraindicated, as is potassium iodide.

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**Dr Jenny Robson**

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Dr Jenny Robson graduated from The University of Queensland and has worked at Sullivan Nicolaides Pathology since 1989. She is interested in all things infectious, but particularly zoonoses, immunisation, tropical and travel medicine, antibiotic resistance, infection control, and the molecular diagnoses of infectious diseases.

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