Bacterial infections in travellers

The rise in global tourism has resulted in an increase in travel-related infections.

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The world's airlines carry approximately 2 billion passengers per year, and at any one moment close on half a million people are flying in a commercial aircraft.[1] Moreover, no city on earth is now more than 24 hours away from any other. More destinations are available to travellers than ever before, and in recent years the rise of 'adventure tourism' and extreme sporting events have brought travellers into contact with more 'exotic' as well as 'mundane' infections. The rise in global tourism dictates that physicians and nurses faced with illness in a returning traveller not only should have an approach to assessing and managing such patients, but also should understand the common infections that travellers present with. At no time in South Africa's history is this more relevant than the present, as the 2010 FIFA World Cup is the largest mass gathering for a single sporting event and the first on the African continent. In excess of 350 000 international visitors are expected to enter South Africa.

As vaccines for bacterial infections are limited, advice on preventing risk exposure should form the focus of any pre-travel consultation in terms of bacterial diseases.

Bacterial infections in travellers inflict a heavy toll in terms of morbidity. This review will concentrate on common infections, namely bacterial causes of travellers' diarrhoea and skin infections, as well as rickettsioses, in addition to typhoid/paratyphoid fever and leptospirosis, which are equally important diseases to recognise due to their high mortality if incorrectly treated. Important features of rare causes of bacterial infections in travellers are discussed in Table I. As vaccines for bacterial infections are limited, advice on preventing risk exposure should form the focus of any pre-travel consultation in terms of bacterial diseases (Table II). Even when available, vaccine uptake is often poor in travellers, airport studies showing rates of uptake of between 5% and 45%.[2-4]

Interestingly, travellers from developing countries have a very low incidence rate of TD when visiting other developing countries, as do travellers who have recently visited the tropics.

Common bacterial causes of illness in travellers

Travellers' diarrhoea

Travellers' diarrhoea (TD) is the most frequently reported infectious disease health risk in travellers, with an incidence rate of up to 60% of health problems per month of stay in a developing country.[5] Between 80% and 90% of cases of TD are caused by bacterial infections, the most common pathogens being enterotoxigenic *Escherichia coli, Campylobacter jejuni, Shigella* and *Salmonella* species. TD occurs equally in men and women, generally occurs in younger travellers and is more likely in certain destinations: Asia, Africa, Mexico, south and central America and the Middle East. Risk factors include poor water supply and sanitation and poor food hygiene in hotels and restaurants. Interestingly, travellers from developing countries have a very low incidence rate of TD when visiting other developing countries,[6] as do travellers who have recently visited the tropics.[7] This suggests a role for developed immunity in preventing TD.

Table I. Rare bacterial infections in travellers

Infection	Characteristics	Prevention
Brucellosis	Commonest route of infection is via ingestion of unpasteurised dairy products. Nonspecific features and poor attention to travel history commonly lead to presentation as pyrexia of unknown origin. Endocarditis and unilateral sacroilitis are recognised complications. Diagnosis relies on culture. High rate of false-positive diagnoses using brucella IgM. Treatment requires 6-week course of combination antibiotics, rifampicin and streptomycin or doxycycline	Avoid ingestion of unpasteurised dairy products, undercooked meats and contact with animals giving birth
Diphtheria	Extremely rare in travellers due to high background levels of childhood and booster vaccination. Hallmark of respiratory diphtheria is a pseudomembrane covering tonsils and upper airways. Diagnosis is presumptive and treatment is with macrolide or penicillin to stop exotoxin production; diphtheria antitoxin should be administered if available. Supportive ICU care is critical	All travellers should be up-to-date with diphtheria vaccination
Legionellosis	Community-acquired pneumonic illness or less commonly a cause of fever without lung involvement (Pontiac fever). Gram-negative bacteria transmitted via droplet aerosol spread from water cooling towers, air-conditioning systems and found in potable water. Elderly and immunocompromised travellers are at higher risk. Diagnosis relies on urinary antigen test, which diminishes in sensitivity after the first 5 - 7 days, or serology. Macrolide or fluoroquinolones are the treatment of choice	Elderly and immunosuppressed should avoid high-risk activities such as immersion in whirlpool spas No vaccine available Chemoprophylaxis not advised
Lyme disease	A tick-borne infection with *Borrelia burgdorferi,* acquired in forested areas of Europe and northern Asia. Also common in the northeastern coastal areas of the USA. Travellers of all ages are at risk, and as the ticks are often very small, travellers may not know they have been bitten. Presentation after an incubation period of 3 - 32 days is with a classic migrating rash termed erythema chronicum migrans (ECM). Days to weeks later, neuroborreliosis may manifest with cranial nerve palsies, meningitis or radiculopathy. Carditis with atrioventricular block is also recognised. Late sequelae include a debilitating arthritis. Diagnosis is largely clinical, although culture from the ECM, PCR from joint fluid and serology are often used. Doxycycline is the drug of choice. Ceftriaxone should be used for neuroborreliosis	Protective clothing, sprayed with insect repellant Tuck trousers into socks Frequent use of DEET-based insect repellants Treat clothing and shoes with 0.5% permethrin if long periods of exposure to tick habitat are anticipated
Melioidosis	Endemic infection of south-east Asia and northern Australia. *Burkholderia pseudomallei* is found in soil and water, with >85% of infections occuring in the rainy season. Inhalation leads to respiratory or disseminated infection (including CNS) with severe sepsis and shock. Morbidity and mortality increased in diabetics, the immunocompromised and those with chronic renal or pulmonary disease. Cutaneous melioidiosis may result from inoculation through broken skin and is commoner in travellers than pulmonary melioidosis. Diagnosis relies on culture from blood or affected site. Induction phase treatment with a carbapenem or ceftazidime is followed by 6 months of oral co-trimoxazole ± doxycycline	Adequate cleaning of wounds in endemic areas No vaccine available
Q fever	Zoonotic infection with *Coxiella burnetti* acquired from livestock. Travellers visiting rural areas or farms may be exposed. Transmission may occur via inhalation of the organism in droplets, via unpasteurised milk or bites of ticks that feed on the livestock. Commonly asymptomatic, clinically apparent disease presents with mild influenza-like illness, pneumonia, endocarditis, myocarditis, hepatitis, encephalitis or pyrexia of unknown origin. Diagnosis is serological and treatment is with doxycycline	Restrict travel to farms where livestock are known to be infected Avoid unpasteurised dairy products Prevent tick bites and remove ticks, once attached

Table II. Prevention of commonly acquired bacterial infections in travellers

Infection	Preventive measures
Travellers' diarrhoea	• Avoidance of high-risk food and drinks is often difficult and does not take into account food preparation, where hygiene may be poor even with low-risk foods. General measures include avoiding non-potable water including drinks mixed with water or ice from a non-potable source and avoiding high-risk foods such as raw or undercooked meat, unpeeled raw fruit or vegetables and salads • Bismuth subsalicylate (BSS) 2 tablets qds has been shown to reduce the incidence of TD from 40% to 14%.[26] Common side-effects include blackening of stools and tongue, constipation and tinnitus. BSS is contraindicated in children <3 years and should be used with caution in children with viral infections due to the risk of Reye's syndrome • Chemoprophylaxis with fluoroquinolones is commonly employed, yet risks increasing the resistance rates of TD pathogens in the long run and is no longer effective against *Campylobacter* spp. that are quinolone-resistant. They also lack efficacy against non-bacterial causes of TD. A pre-emptive strategy using fluoroquinolones or macrolide antibiotics is preferable to a prophylactic strategy. A non-absorbable antibiotic, rifaximin, has shown promise in reducing TD in Mexico[27] and further trials are awaited
Rickettsioses	• Protective clothing, sprayed with insect repellant • Tuck trousers into socks if possible • Frequent use of DEET-based insect repellants (containing at least 30% active ingredient), especially on the legs, as these repellants have a relatively short duration of action against *Amblyomma* spp. ticks[28] • Travellers anticipating long periods of exposure to tick habitat (safari, bush walks) should also consider treating clothing and shoes with 0.5% permethrin prior to the activity
Leptospirosis	• Protective waterproof clothes and boots • Avoid submersion in and consumption of river water • Cover cuts and abrasions with waterproof dressings • Purification of drinking water (filtration may not protect) • Avoid travel to recently flooded areas if possible • No firm evidence for chemoprophylaxis, but some studies show benefit in short-term travellers with high-risk exposure such as adventure travellers, athletes and military personnel. Doxycycline 200 mg weekly should be used pre- and during exposure • Vaccination – only available in a few countries with safety issues of concern and current vaccines have poor efficacy
Typhoid fever	• Strict observation of hygiene rules to avoid contaminated food and water • Vaccination: protective efficacy against typhoid may be overcome by ingesting a high bacterial load. Vaccine efficacy for either the oral, live-attenuated vaccine (Ty21) or the Vi capsular polysaccharide vaccine (ViCPS) is between 50% and 80% • Travellers to endemic areas for typhoid fever are advised to be vaccinated, although the limitations of the vaccine should be stressed

Unlike protozoal causes of TD, bacterial causes have a short incubation period of 6 - 48 hours followed by mild to severe abdominal pain, diarrhoea with or without blood, fever and sometimes vomiting. Long-term complications are not typical, but persistent diarrhoea lasting ≥4 months occurs in 1% of patients[8] and up to 10% may exhibit symptoms of irritable bowel syndrome.[9] As bacterial infections are such a dominant cause of TD and morbidity is high, empiric treatment should aim to cover the most likely organism(s) and alleviate symptoms. Single-dose or short-course fluoroquinolone or macrolide treatment has proven efficacy in the treatment of TD. Single-dose oral azithromycin 1 g or a 2-day course (500 mg bd, which causes less nausea) has the advantage of covering quinolone-resistant *Campylobacter* infection. Anti-motility agents such as loperamide and rehydration are key adjuncts to therapy. Children generally require more aggressive oral rehydration than adults. Prevention measures for TD are outlined in Table II.

Impetigo is common in children who travel, particularly those visiting the tropics.

Bacterial skin infections

Skin problems represent a very common medical presentation in returning travellers.[10,11] In the largest retrospective study of skin problems in travellers to date, 18% of all visits in ill travellers at GeoSentinel Network clinics were dermatological in nature.[12] In that study, pyodermas (skin abscess, cellulitis, erysipelas) comprised 13% of all conditions. Bacterial skin infections are more common in the tropics, where super-infected insect bites are common and optimal levels of hygiene may be absent. The common causes of cellulitis and erysipelas include β-haemolytic streptococci and *S. aureus*, although Gram-negative bacteria may cause infection. Impetigo is common in

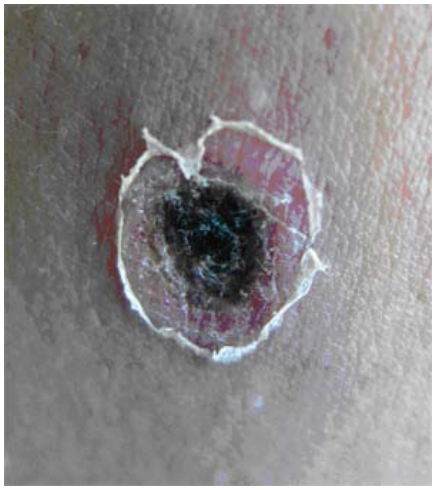

Fig. 1. Eschar with characteristic black, necrotic centre, from a patient with Mediterranean spotted fever caused by R. conorii.

children who travel, particularly those visiting the tropics. Many travel kits will include a topical antibiotic such as mupirocin to treat early bacterial skin infections. An oral antibiotic that covers *S. aureus* and streptococci for treatment of more extensive pyodermas would be beneficial for travellers to remote locations who need to self-treat. Impetigo will usually respond to mupirocin.

Rickettsioses

Rickettsioses refer to a group of zoonotic infections caused by intracellular Gram-negative bacteria. Rickettsial diseases include spotted fever group (SFG) rickettsioses (Mediterranean spotted fever, African tick-bite fever and the typhus group of rickettsiae), scrub typhus caused by *Orientia tsutsugamushi,* human monocytic ehrlichiosis and human granulocytic anaplasmosis. Of these, SFG rickettsioses are by far the commonest reported rickettsial disease in travellers[13] and African tick-bite fever (ATBF) caused by *Rickettsia africae* comprises 99% of SFG rickettsioses in travellers. Moreover, after malaria, it is the commonest diagnosis in ill travellers returning from sub-Saharan Africa.[14]

The vector and reservoir for ATBF is the *Amblyomma* tick, a game and cattle tick found on the vegetation in the bush; it is known to be aggressive and readily feeds on humans. Visitors to game reserves, hunters and ecotourists travelling during late summer months appear to be at heightened risk. Mediterranean spotted fever (MSF), in contrast, is caused by *R. conorii,* transmitted by the brown dog tick and not often associated with travel. ATBF and MSF are characterised by acute febrile illness with the organisms targeting vascular endothelial cells, resulting in vasculitis. After a short incubation period of 5 - 7 days, the classic triad of eschar (Fig. 1), fever and rash, commonly accompanied by headache and myalgia, is characteristic of ATBF and MSF. ATBF is more commonly associated with multiple inoculation eschars, associated regional adenopathy and the rash may be vesicular in nature, rather than the usual maculopapular rash.[15] Diagnosis is largely clinical, and not particularly challenging if the classic triad is present. Routine laboratory tests commonly reveal lymphopenia, raised CRP, moderately elevated liver enzymes, and thrombocytopenia. Specific diagnostic tests include convalescent serology (micro-immunofluorescence) and more recently introduced PCR of blood or eschar biopsy, which is able to differentiate between *R. conorii* and *R. africae.* The Weil-Felix agglutination test, which is neither specific nor sensitive, should no longer be used. Oral doxycycline 100 mg bd for 7 - 10 days is the drug of choice, although if unable to take oral therapy and intravenous doxycycline is unavailable, intravenous chloramphenicol, a macrolide or ciprofloxacin are alternatives. In contrast to MSF, fatalities from ATBF are extremely rare.[15]

Recreational travellers who undertake white water rafting or swim in rivers are at increased risk of acquiring leptospirosis.

Uncommon bacterial causes of illness in travellers

Leptospirosis

The rise of adventure travel and mass participation in water-related sporting events has brought leptospirosis to prominence in the travelling population. A number of high-profile outbreaks have occurred, including an outbreak in Springfield, Illinois in 1998 during a triathlon, which affected 12% of the 834 atheletes,[16] and the 2000 Eco-Challenge multisport race in Malaysia, which saw an attack rate of 42% among 189 atheletes.[17] Similarly, recreational travellers who undertake white water rafting or swim in rivers are at increased risk of acquiring leptospirosis.[18] A number of outbreaks have been related to increase in rainfall, a known factor favouring the survival and transmission of the leptospira spirochete which is shed in animal urine (most commonly rodents), contaminating water and soil. Transmission occurs either through ingestion of contaminated food or water, or through direct contact with breaks in the skin.[19] Leptospirosis is endemic in the tropics but also occurs in temperate climes.

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Infection with leptospira causes a bacteraemia with widespread dissemination including to the central nervous system. Endothelial damage is the primary pathology causing renal tubular necrosis, hepatocellular injury, meningitis, pulmonary haemorrhage and myositis. Following an incubation period of 7 - 14 days (range 1 - 30), one of two recognisable syndromes occur: a self-limiting febrile illness which occurs in ~90% of infections or a severe and potentially fatal form of the disease which is immune-mediated and known as Weil's disease or Weil's syndrome.

Clinical features of the acute bacteraemic phase usually last 5 - 7 days and include fever, chills, rigors, headache, conjunctival suffusion, myalgia, photophobia, abdominal pain, anorexia, nausea and vomiting. The immune phase that follows may last up to 30 days and is characterised by jaundice, hepatomegaly, splenomegaly, renal failure, aseptic meningitis and pulmonary involvement (haemoptysis, haemorrhage and adult respiratory distress syndrome). Weil's disease is associated with a 5 - 15% case fatality rate. The jaundice associated with Weil's disease occurs in the absence of hepatocellular necrosis, hence a normal or low-level increase in liver enzymes is characteristic. This helps differentiate Weil's disease from other causes of acute jaundice.

Sensitivity of laboratory tests for leptospirosis will vary depending on the stage of sampling. During the acute bacteraemic phase, leptospira may be cultured from blood or be detected by PCR. Leptospira IgM may be present in the first 5 - 7 days following onset of illness. The microscopic agglutination test (MAT) has high specificity, but is not available in all laboratories. During the immune phase, sensitivity of culture and PCR diminish greatly.

The drug of choice for treating mild leptospirosis is doxycycline 100 mg bd by mouth, although amoxicillin or ampicillin are alternatives. Weil's disease is commonly treated with intravenous beta-lactams or a third-generation cephalosporin such as ceftriaxone. Penicillin G and ceftriaxone are equally effective although ceftriaxone's dosing schedule often makes it more attractive. Supportive therapy is important for those who require hospitalisation.

Typhoid and paratyphoid fever

The enteric fevers, typhoid and paratyphoid fever caused by *Salmonella typhi* and *paratyphi* A, B or C respectively, are common global infections with particularly high incidence in Asia, Africa, the Caribbean and central and south America. Travel to India carries the highest risk of infection[20,21] and not surprisingly, increased rates of infection are seen in rural areas with poor sanitation. Interestingly, deciding not to follow pre-travel advice surrounding food and water precautions increase the risk tenfold[22] and travellers visiting friends and relations are at particularly high risk due to poor pre-travel consultation rates, poor food hygiene and low vaccination rates.[23]

The enteric fevers, typhoid and paratyphoid fever are common global infections with particularly high incidence in Asia, Africa, the Caribbean and central and south America.

Enteric fevers are characterised by insidious onset of fever, which usually increases steadily in the first few days, and nonspecific symptoms which occur after an incubation period of 14 - 21 days (range 3 - 60). Headache, non-productive cough and prostration are common. Abdominal symptoms are varied and may include constipation rather than diarrhoea, although co-infecting pathogens may influence this. Hepatosplenomegaly may be evident and rose spots (2 - 4 mm blanching erythematous maculopapular lesions) are classically described on the abdomen and chest. Like relative bradycardia, another sign commonly said to be present in typhoid fever, rose spots are commonly absent. If untreated, enteric fever may progress to cause complications with high morbidity and mortality (up to 30%). Erosion of Peyer's patches leading to intestinal perforation and gastrointestinal bleeding are the two most feared complications. Typhoid encephalopathy may occur in 10 - 15% of patients in endemic countries. These complications are rare in travellers. Laboratory investigations are nonspecific. Leucopenia, thrombocytopenia and mild anaemia may be present and eosinopenia occurs secondary to glucocorticoid action.

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Diagnosis of enteric fever relies on blood culture, whose sensitivity in the first week of illness is between 80% and 100%, but declines incrementally thereafter week by week, and correlates with the accumulation of bacteria in Peyer's patches.[24] The Widal test for enteric fever, based on measurement of agglutinating antibodies against O and H antigens, suffers from poor sensitivity and specificity and although still commonly employed in developing countries, is a poor test and should not be used. A number of companies are developing rapid diagnostic tests for typhoid fever such as Typhidot-M, which detects IgM antibodies, but these still need further evaluation before recommendations on their use can be made.

Treatment of typhoid and paratyphoid fever in a timely manner reduces mortality rates to <1%. Due to the rise of quinolone resistance of *S. typhi* in endemic areas such as India, empiric treatment with a third-generation cephalosporin should be instigated until sensitivities are known. A fluoroquinolone may be substituted once sensitivities are available. Another option is azithromycin, which has been shown to be equally efficacious to quinolones in uncomplicated typhoid fever.[25] Even with appropriate antibiotic therapy, defervescence of fever in typhoid cases is delayed and classically takes 3 - 5 days. Prevention measures for typhoid fever are discussed in Table II.

Acknowledgements

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In a nutshell

- The relentless increase in tourism, adventure travel and mass sporting events leads to increasing travel-related infections.
- The major bacterial travel-related infections are bacterial causes of travellers' diarrhoea (TD) and skin infections, as well as rickettsial diseases.
- Enterotoxigenic *E. coli* is the commonest cause of TD.
- Prevention of TD involves avoidance of non-potable water and foods prepared with poor hygiene, and use of bismuth subsalicylate or non-absorbable antimicrobials such as rifaximin.
- Prophylactic antibiotics are generally not recommended for preventing TD.
- African tick-bite fever is the commonest cause of rickettsial disease in returning travellers.
- Protective clothing, DEET-based repellents and permethrin-treated clothing are the best methods for preventing tick exposure.
- Travellers involved in water sports, including white water rafting, are at increased risk of leptospirosis, which should be on the differential diagnosis for any traveller presenting with febrile jaundice and renal dysfunction.
- Typhoid and paratyphoid fever are particularly prevalent in India and Asia and travellers to high-risk destinations should receive typhoid vaccination.
- Defervescence of fever in typhoid and paratyphoid fever is often delayed by 3 - 5 days after instigation of appropriate antibiotics.

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Single Suture

Lead poisoning a risk for regular game eaters

Fragments of lead that are too small to be picked out during a meal may be enough to poison people who regularly eat game shot with small lead. Debbie Pain and colleagues of the Wildfowl and Wetlands Trust in Slimbridge, Gloucestershire, bought wild-shot birds from supermarkets, game dealers and butchers across the UK. They X-rayed them and then cooked them in either wine, cider or a pH-neutral preparation such as cream sauce.

Only after cooking – apparently traditional with game – did they pick out the larger, visible lead fragments. However, subsequent analysis of the deboned, pulverised meat revealed that just over 3 meals of the game bird woodcock in a week would take a 70 kg person over the lead threshold set by United Nations bodies for most farmed animals. Red grouse, partridge and pheasant hit the limit with about 10 meals a week. Wood pigeon and mallard eaters, however, would have to eat 24 - 30 servings per week.

The X-rays suggest that the problem is small unnoticed pieces of shot. According to Grainger Hunt of the Perigrine Fund in California, people all over the world eat birds killed with lead shotgun pellets.

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