
Charles Darwin University

Short Report:

Melioidosis as a Consequence of Sporting Activity

Hill, Audrey; Mayo, Mark; Kaestli, Mirjam; Price, Erin; Richardson, Leisha Jade; Godoy, Daniel; Spratt, Brian; Currie, Bart

Published in:
American Journal of Tropical Medicine and Hygiene

DOI:
[10.4269/ajtmh.12-0744](https://doi.org/10.4269/ajtmh.12-0744)

Published: 01/08/2013

Document Version
Publisher's PDF, also known as Version of record

[Link to publication](#)

Citation for published version (APA):

Hill, A., Mayo, M., Kaestli, M., Price, E., Richardson, L. J., Godoy, D., Spratt, B., & Currie, B. (2013). Short Report: Melioidosis as a Consequence of Sporting Activity. *American Journal of Tropical Medicine and Hygiene*, 89(2), 365-366. <https://doi.org/10.4269/ajtmh.12-0744>

General rights

Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal

Take down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

Short Report: Melioidosis as a Consequence of Sporting Activity

Audrey A. Hill, Mark Mayo, Mirjam Kaestli, Erin P. Price, Leisha J. Richardson, Daniel Godoy,
Brian G. Spratt, and Bart J. Currie*

Menzies School of Health Research, Charles Darwin University, Darwin, Northern Territory, Australia; Department of Infectious Disease Epidemiology, Imperial College, London, United Kingdom; Infectious Diseases Department, Northern Territory Medical Program, Royal Darwin Hospital, Darwin, Northern Territory, Australia

Abstract. In the tropical city of Darwin, Northern Territory, Australia, dry season soil sampling cultured *Burkholderia pseudomallei* from 7 (70%) of 10 sports fields. However, during the 23 years of the Darwin Prospective Melioidosis Study, only 5 (0.6%) of 785 melioidosis cases have been attributed to infection from sports fields. In one soccer player with cutaneous melioidosis, *B. pseudomallei* cultured from the player was identical by multilocus sequence typing and multilocus variable-number tandem repeat analysis with an isolate recovered from soil at the location on the sports field where he was injured. Melioidosis is uncommon in otherwise healthy sports persons in melioidosis-endemic regions but still needs consideration in persons with abrasion injuries that involve contact with soil.

Athletes can be infected by pathogens through inoculation events caused by cuts and abrasions resulting from sporting activities.¹ A notable example is the methicillin-resistant *Staphylococcus aureus* outbreak in a professional American football team that was associated with turf-abrasion inoculation.² With the globalization of sporting competitions, the rate of returning athletes with diseases endemic to tropical regions is expected to increase, such as occurred with the Eco-challenge 2000 event in Malaysian Borneo, where leptospirosis developed in > 80 of the endurance athletes after contact with contaminated water and soil.^{3,4} Melioidosis is another disease that is endemic in tropical regions, potentially placing athletes at risk from inoculation events through broken skin and contact with contaminated soil or surface water.^{5,6}

In the early 1970s, *Burkholderia pseudomallei*, the causative bacterium of melioidosis was found in surface water samples of five sports fields in Singapore⁷ after a potential sporting connection in two of ten cases described from neighboring Malaysia.⁸ In Darwin in northern Australia, *B. pseudomallei* levels in the upper soil layers increase during the wet season (October–April), and *B. pseudomallei* is often cultured from surface water.⁹ The first use of molecular genotyping to link clinical isolates of *B. pseudomallei* to epidemiologically related environmental strains was published in 1994 and used ribotyping.¹⁰ However, ribotyping has limited specificity in its ability to discriminate between closely related strains of *B. pseudomallei*. Subsequently, pulsed-field gel electrophoresis was shown to be superior to ribotyping and was used to link two separate clonal clusters of melioidosis in northern Australia to contamination of the community water supplies with *B. pseudomallei*.^{11,12} Multilocus sequence typing (MLST) has more recently become the global standard for epidemiologic investigations of melioidosis, with > 1,000 *B. pseudomallei* sequence types (STs) identified worldwide.¹³

Early in the 2005 Darwin dry season, a soccer player had chronic cutaneous melioidosis of the lower leg after a grass-abrasion incident 2.5 months earlier on a water-logged Darwin soccer field. *Burkholderia pseudomallei* was cultured from the leg ulcer (isolate MSHR2080) and also from pus from incision and drainage of an ipsilateral inguinal lymph node abscess (MSHR2078). The patient made a full recovery after therapy for

two weeks with intravenous ceftazidime plus oral trimethoprim/sulfamethoxazole, followed by three additional months of oral trimethoprim/sulfamethoxazole. Environmental sampling was undertaken as a response to the incident and nine *B. pseudomallei* isolates were cultured from soil samples taken at various locations on the soccer field, including one isolate (MSHR2188) from the location where the patient identified that the abrasion incident had occurred.

Multilocus sequence typing was performed on the strains from this study, and patient strains MSHR2078 and MSHR2080 were typed as ST36. ST36 was also found in four of nine soccer field soil samples, including MSHR2188, and the remaining 5 isolates typed as ST144. ST36 has been found in clinical and environmental samples elsewhere in the Northern Territory. Therefore, to obtain greater genotypic resolution of the soccer field and clinical isolates, we performed multilocus variable-number tandem repeat analysis (MLVA) on the six ST36 isolates by using four-locus MLVA (MLVA-4) as described.¹⁴ Multilocus variable-number tandem repeat analysis can discriminate within a single MLST ST and thus can help further establish the relatedness of isolates that have temporal and spatial proximity to each other.¹⁵ MSHR2078 and MSHR2080 strains from the patient were identical at all four MLVA loci to MSHR2188 retrieved from soil at the location of the abrasion incident. In contrast, the three ST36 environmental isolates from different locations of the same field contained 1–3 MLVA locus mismatches when compared with MSHRs 2078, 2080 and 2188. The 100% MLST and MLVA match between clinical and environmental isolates supports the field soil being the source of the soccer player's infection.

The Darwin Prospective Melioidosis Study began in October 1989, and in the 23 years until October 2012, there have been 785 culture-confirmed cases of melioidosis identified at Royal Darwin Hospital. We previously documented that in 22% of cases there was a specific exposure scenario that was considered the likely infecting event, with most events being abrasion or laceration inoculations.¹⁶ Despite this finding, during 23 years there have been only 5 (0.6%) of 785 cases (including the one described in this report) where the documented suspected infecting event involved an injury on a Darwin sports field: three from soccer, one from rugby, and one from Australian rules football.

Field sports are especially popular during the Darwin dry season, when there is less surface water and *B. pseudomallei* is less abundant at the soil surface.⁹ Nevertheless, irrigation of

* Address correspondence to Bart J. Currie, Menzies School of Health Research, PO Box 41096, Casuarina, Darwin, Northern Territory 0811, Australia. E-mail: bart@menzies.edu.au

sports fields during the dry season is considered likely to increase survival of *B. pseudomallei* in upper soil layers. We therefore investigated the prevalence of *B. pseudomallei* at ten Darwin grassed sports fields during the dry season of 2010, and collected ten soil samples per field. Using culture,¹⁷ we detected *B. pseudomallei* at 7 (70%) of 10 sports fields. This prevalence was higher than our previous finding of 27% (38 of 141) *B. pseudomallei* positive sites (environmentally disturbed and undisturbed sites) in the 2006 dry season ($P = 0.008$, by two-tailed Fisher's exact test), and with 16% (16 of 100) positive sports field soil samples overall, the prevalence was similar to the prevalence we found in soil samples from irrigated gardens (17%, 11 of 65).⁹ The mean pH of each positive field was within the optimal pH range for *B. pseudomallei* survival and growth (5.0–6.0)¹⁸ and electrical conductivity measures of salt content were low, which is consistent with previous environmental studies of conditions favorable for *B. pseudomallei*.¹⁷

Although there may be some under-ascertainment of inoculating event histories, we have identified only five cases of sports field-related melioidosis in Darwin in over 20 years, despite the high incidence of melioidosis in Darwin, the high prevalence of *B. pseudomallei* in sports field soil, and the large numbers of adults and children partaking in organized sports. This low number reflects the opportunistic nature of melioidosis, a disease that predominantly affects those with defined medical risk factors such as diabetes and hazardous alcohol use, and where severe disease is relatively uncommon in healthy persons.¹⁶ Although cutaneous melioidosis without disseminated disease occurs in healthy persons,¹⁹ most melioidosis cases diagnosed in the United States and Europe are in returned travelers or residents from melioidosis-endemic regions who have recognized risk factors such as diabetes and cystic fibrosis.²⁰ The rarity of severe melioidosis in those without risk factors contrasts with leptospirosis, where severe disease is well recognized to occur in exposed healthy travelers participating in outdoor activities such as sports.^{3,4} Nevertheless fatal co-infection with both melioidosis and leptospirosis has also been recently described.²¹

In conclusion, melioidosis is uncommon in otherwise healthy sports persons in melioidosis-endemic regions. However, this disease needs to be suspected in persons with abrasion injuries that involve contact with soil.

Received December 12, 2012. Accepted for publication January 11, 2013.

Published online June 3, 2013.

Acknowledgments: We are grateful to Linda Ward and Alex Humphrey for collating case data and Daniel Gal, Glenda Harrington, Ian Harrington, and Darcy Tupper-Creed for advice and assistance with soil sampling.

Financial support: This study was supported by project grants from the Australian National Health and Medical Research Council. Daniel Godoy and Brian G. Spratt were supported by Wellcome Trust grant WT089472.

Authors' addresses: Audrey A. Hill, Mark Mayo, Mirjam Kaestli, Erin P. Price, Leisha J. Richardson, and Bart J. Currie, Menzies School of Health Research, Casuarina, Darwin, Northern Territory, Australia, E-mails: audrey.hill@menzies.edu.au, mark.mayo@menzies.edu.au, mirjam.kaestli@menzies.edu.au, erin.price@menzies.edu.au, leisha.richardson@menzies.edu.au, and bart@menzies.edu.au. Daniel Godoy and Brian G. Spratt, Department of Infectious Disease Epidemiology, Imperial College, London, UK, E-mails: d.godoy@outlook.com and b.spratt@imperial.ac.uk.

REFERENCES

1. Kozarsky P, 2006. The body of knowledge for the practice of travel medicine. *J Travel Med* 13: 251–254.
2. Kazakova SV, Hageman JC, Matava M, Srinivasan A, Phelan L, Garfinkel B, Boo T, McAllister S, Anderson J, Jensen B, Dodson D, Lonsway D, McDougal LK, Arduino M, Fraser VJ, Killgore G, Tenover FC, Cody S, Jernigan DB, 2005. A clone of methicillin-resistant *Staphylococcus aureus* among professional football players. *N Engl J Med* 352: 468–475.
3. Sejvar J, Bancroft E, Winthrop K, Bettinger J, Bajani M, Bragg S, Shutt K, Kaiser R, Marano N, Popovic T, Tappero J, Ashford D, Mascola L, Vugia D, Perkins B, Rosenstein N, 2003. Leptospirosis in “Eco-Challenge” athletes, Malaysian Borneo, 2000. *Emerg Infect Dis* 9: 702–707.
4. Pappas G, Papadimitriou P, Siozopoulou V, Christou L, Akritidis N, 2008. The globalization of leptospirosis: worldwide incidence trends. *Int J Infect Dis* 12: 351–357.
5. Cheng AC, Currie BJ, 2005. Melioidosis: epidemiology, pathophysiology, and management. *Clin Microbiol Rev* 18: 383–416.
6. Dance DA, 2000. Melioidosis as an emerging global problem. *Acta Trop* 74: 115–119.
7. Thin RN, Groves M, Rapmund G, Mariappan M, 1971. *Pseudomonas pseudomallei* in the surface water of Singapore. *Singapore Med J* 12: 181–182.
8. Thin RN, Brown M, Stewart JB, Garrett CJ, 1970. Melioidosis: a report of ten cases. *Q J Med* 39: 115–127.
9. Kaestli M, Mayo M, Harrington G, Ward L, Watt F, Hill JV, Cheng AC, Currie BJ, 2009. Landscape changes influence the occurrence of the melioidosis bacterium *Burkholderia pseudomallei* in soil in northern Australia. *PLoS Negl Trop Dis* 3: e364.
10. Currie B, Smith Vaughan H, Golledge C, Buller N, Sriprakash KS, Kemp DJ, 1994. *Pseudomonas pseudomallei* isolates collected over 25 years from a non-tropical endemic focus show clonality on the basis of ribotyping. *Epidemiol Infect* 113: 307–312.
11. Inglis TJ, Garrow SC, Henderson M, Clair A, Sampson J, O'Reilly L, Cameron B, 2000. *Burkholderia pseudomallei* traced to water treatment plant in Australia. *Emerg Infect Dis* 6: 56–59.
12. Currie BJ, Mayo M, Anstey NM, Donohoe P, Haase A, Kemp DJ, 2001. A cluster of melioidosis cases from an endemic region is clonal and is linked to the water supply using molecular typing of *Burkholderia pseudomallei* isolates. *Am J Trop Med Hyg* 65: 177–179.
13. Godoy D, Randle G, Simpson AJ, Aanensen DM, Pitt TL, Kinoshita R, Spratt BG, 2003. Multilocus sequence typing and evolutionary relationships among the causative agents of melioidosis and glanders, *Burkholderia pseudomallei* and *Burkholderia mallei*. *J Clin Microbiol* 41: 2068–2079.
14. Currie BJ, Haslem A, Pearson T, Hornstra H, Leadem B, Mayo M, Gal D, Ward L, Godoy D, Spratt BG, Keim P, 2009. Identification of melioidosis outbreak by multilocus variable number tandem repeat analysis. *Emerg Infect Dis* 15: 169–174.
15. Price EP, Hornstra HM, Limmathurotsakul D, Max TL, Sarovich DS, Vogler AJ, Dale JL, Ginther JL, Leadem B, Colman RE, Foster JT, Tuanyok A, Wagner DM, Peacock SJ, Pearson T, Keim P, 2010. Within-host evolution of *Burkholderia pseudomallei* in four cases of acute melioidosis. *PLoS Pathog* 6: e1000725.
16. Currie BJ, Ward L, Cheng AC, 2010. The epidemiology and clinical spectrum of melioidosis: 540 cases from the 20 year Darwin prospective study. *PLoS Negl Trop Dis* 4: e900.
17. Draper AD, Mayo M, Harrington G, Karp D, Yinfoo D, Ward L, Haslem A, Currie BJ, Kaestli M, 2010. Association of the melioidosis agent *Burkholderia pseudomallei* with water parameters in rural water supplies in Northern Australia. *Appl Environ Microbiol* 76: 5305–5307.
18. Palasatien S, Lertsirivorakul R, Royros P, Wongratanacheewin S, Sermswan RW, 2008. Soil physicochemical properties related to the presence of *Burkholderia pseudomallei*. *Trans R Soc Trop Med Hyg* 102 (Suppl 1): S5–S9.
19. Gibney KB, Cheng AC, Currie BJ, 2008. Cutaneous melioidosis in the tropical top end of Australia: a prospective study and review of the literature. *Clin Infect Dis* 47: 603–609.
20. Wiersinga WJ, Currie BJ, Peacock SJ, 2012. Melioidosis. *N Engl J Med* 367: 1035–1044.
21. Hin HS, Ramalingam R, Chunn KY, Ahmad N, Ab Rahman J, Mohamed MS, 2012. Fatal co-infection: melioidosis and leptospirosis. *Am J Trop Med Hyg* 87: 737–740.