Accuracy of commercial systems for identification of *Burkholderia pseudomallei* varies substantially (10). In conclusion, scientists must be aware of the potential misidentification of *B. pseudomallei* by automated identification systems, especially those in regions to which *B. pseudomallei* is not endemic.

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**References**

1. Chewapreecha C, Holden MT, Vehkala M, Valimaki N, Yang Z, Harris SR, et al. Global and regional dissemination and evolution of *Burkholderia pseudomallei*. Nat Microbiol. 2017;2:16263. https://doi.org/10.1038/s41564-017-00279-5

2. Kiratisin P, Santanirand P, Chantratita N, Kaewdaeng S. Accuracy of commercial systems for identification of *Burkholderia pseudomallei* versus *Burkholderia cepacia*. Diagn Microbiol Infect Dis. 2007;59:277–81. https://doi.org/10.1016/j.diagmicrobiol.2007.06.013

3. Kobayashi H, Seike S, Yamaguchi M, Ueda M, Takahashi E, Kinoshita R, et al. *Aeromonas sobria* serine protease decreases epithelial barrier function in T84 cells and accelerates bacterial translocation across the T84 monolayer in vitro. PLoS One. 2019;14:e0221344. https://doi.org/10.1371/journal.pone.0221344

4. Lipsitz R, Garges S, Aurigemma R, Baccam P, Blaney DD, Cheng AC, et al. Workshop on treatment of and postexposure prophylaxis for *Burkholderia pseudomallei* and *B. mallei* Infection, 2010. Emerg Infect Dis. 2012;18:e2. https://doi.org/10.3201/eid1812.12068

5. Godoy D, Randle G, Simpson AJ, Aanensen DM, Pitt TL, Kinoshita R, et al. Multilocus sequence typing and evolutionary relationships among the causative agents of melioidosis and glanders, *Burkholderia pseudomallei* and *Burkholderia mallei*. J Clin Microbiol. 2003;41:2068–79. https://doi.org/10.1128/JCM.41.5.2068-2079.2003

6. Kamthan A, Shaw T, Mukhopadhyay C, Kumar S. Molecular analysis of clinical *Burkholderia pseudomallei* isolates from southwestern coastal region of India, using multi-locus sequence typing. PLoS Negl Trop Dis. 2018;12:e0006915. https://doi.org/10.1371/journal.pntd.0006915

7. Zong Z, Wang X, Deng Y, Zhou T. Misidentification of *Burkholderia pseudomallei* as *Burkholderia cepacia* by the VITEK 2 system. J Med Microbiol. 2012;61:1483–4. https://doi.org/10.1099/jmm.0.041525-0

8. Hoffmaster AR, AuCoin D, Baccam P, Baggett HC, Baird R, Bhengsri S, et al. Melioidosis diagnostic workshop, 2013. Emerg Infect Dis. 2015;21.

9. Zakharova IB, Lopasteyksaya YA, Toporkov AV, Viktorov DV. Influence of biochemical features of *Burkholderia pseudomallei* strains on identification reliability by Vitek 2 System. J Glob Infect Dis. 2018;10:7-10. https://doi.org/10.4103/jgid.jgid_39_17

10. Lau SK, Sriddhar S, Ho CC, Chow WN, Lee KC, Lam CW, et al. Laboratory diagnosis of melioidosis: past, present and future. Exp Biol Med (Maywood). 2015;240:742–51. https://doi.org/10.1177/1535370215583801

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**Autochthonous Case of Pulmonary Histoplasmosis, Switzerland**

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DOI: https://doi.org/10.3201/eid2703.191831

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A 48-year-old man in Switzerland sought treatment for a 1-year history of progressive dyspnea, cough, 20-kg weight loss, and increased sweating; he was receiving oxygen therapy. Results of previous consultations had been inconclusive. An HIV screening test was negative. Medical history included hyperreflexia, depression, and chronic hepatitis B. The man had stopped cocaine inhalation and heroin consumption 20 years earlier but continued smoking cigarettes and cannabis. Regular medications included omeprazole and trimipramine. Except for a short trip to Greece and Italy many years before, the patient reported no foreign travel.

In the absence of travel history to an endemic area, histoplasmosis was not initially considered at the time this patient sought treatment. A prolonged diagnostic process and delayed treatment initiation had meanwhile resulted in significant deterioration of health, including need for home oxygen therapy, and loss of ability to work. Meanwhile, the patient was cachectic and had clubbing on his fingers and toes. Spirometry revealed nearly normal dynamic lung volumes. Forced expiratory volume was 3 L (75%) and forced vital capacity 4.1 L (83%), but diffusion capacity was severely impaired; diffusion capacity for carbon monoxide was 20%. A 6-minute walking test was limited to 400 m (59% predicted), initial oxygen saturation dropping from 90% to 78%.

A chest computed tomography (CT) scan showed a diffuse reticulonodular pattern with predominantly upper lung opacifications and bronchiectases indicating fibrotic lung disease (Figure, panels A, B). Reversed halo signs and right upper lobe nodules were found. Bronchoscopy results including bronchoalveolar lavage were unremarkable. Initial sampling with microbiological screening was negative.

Differential diagnoses included toxic lung damage or other interstitial lung disease, (e.g. atypical presentation of Langerhans cell histiocytosis or sarcoidosis). A wedge biopsy showed predominantly upper-lobe fibrosis and multiple, confluent, necrotizing granulomas harboring yeasts, establishing the diagnosis of pulmonary histoplasmosis (Appendix Figure, https://wwwnc.cdc.gov/EID/article/27/3/19-1831-App1.pdf).

A qualitative immunodiffusion test (IMMY, https://www.immy.com) was positive for antibodies in plasma, but an antigen immunoassay for Histoplasma in urine (IMMY) was negative; a beta-1,3-D glucan test (Fungitell, https://www.fungitell.com) was highly positive (>500 pg/mL; limit <80 pg/mL).
pneumonia prophylaxis. At 3-month follow-up, the patient had improved considerably. Repeated spirometry was nearly normal, showing persistent impairment of diffusion capacity. Follow-up chest CT scan (Figure 1, panels C, D) showed regression of ground-glass opacities and micronodules; the reversed halos had disappeared. Overall, optimal treatment duration remains unclear (5), but because of probable underlying preexisting lung disease, persistent pathological findings from CT, and continued desaturation under exercise, continuing treatment for >12 months seemed necessary.

The source of infection for this patient remains speculative. However, possible risk exposures were guano from flying bats in the garden (6), previous use of organic fertilizer possibly containing histoplasma (7), and regular work-related unpacking of fruits and spices from straw-filled boxes from West Africa, although H. capsulatum var. capsulatum is less common in that region (8).

In addition to previous findings of histoplasmosis in badgers (9), this case confirms the likely environmental occurrence of H. capsulatum in Switzerland. Although diagnoses of autochthonous histoplasmosis have been rare, and few autochthonous cases have been described (10), our finding of a probable autochthonous case of chronic pulmonary histoplasmosis in an immunocompetent male in Switzerland highlights the incomplete understanding of histoplasmosis endemicity and indicates that it has likely been underestimated in Europe.

About the Author
Ms. Schmiedel has a masters degree in epidemiology and a diploma in tropical medicine from Cayetano Heredia Universidad in Lima, Peru, and has completed specialized training in infectious diseases and internal medicine. She currently works as a senior infectious disease consultant at Hôpital du Jura (affiliated with Basel University Hospital) and has a strong interest in infection control and tropical medicine. Ms. Büchi has a masters degree in immunology and microbiology from Bern University in Switzerland and is studying to become an internist at the Inselspital in Bern. She has a primary research interest in bloodstream infection.

References
1. Hage CA, Ribes JA, Wengenack NL, Baddour LM, Assi M, McKinsey DS, et al. A multicenter evaluation of tests for diagnosis of histoplasmosis. Clin Infect Dis. 2011;53:448–54. https://doi.org/10.1093/cid/cir435
2. Kauffman CA. Histoplasmosis: a clinical and laboratory update. Clin Microbiol Rev. 2007;20:115–32. https://doi.org/10.1128/CMR.00027-06
3. Wheat LJ, Conces D, Allen SD, Blue-Hnidy D, Loyd J. Pulmonary histoplasmosis syndromes: recognition, diagnosis, and management. Semin Respir Crit Care Med. 2004;25:129–44. https://doi.org/10.1055/s-2004-824898
4. Marchiori E, Melo SMD, Vianna FG, Melo BSD, Melo SSD, Zanetti G. Pulmonary histoplasmosis presenting with the reversed halo sign on high-resolution CT scan. Chest. 2011;140:789–91. https://doi.org/10.1378/chest.11-0055
5. Wheat LJ, Freifeld AG, Kleiman MB, Baddley JW, McKinsey DS, Loyd JE, et al.; Infectious Diseases Society of America. Clinical practice guidelines for the management of patients with histoplasmosis: 2007 update by the Infectious Diseases Society of America. Clin Infect Dis. 2007;45:807–25. https://doi.org/10.1086/521259
etymologia

Histoplasma capsulatum [his’tə-plāz’ma kăp’sə-lā’təm]

Monika Mahajan

In 1905, Samuel Taylor Darling serendipitously identified a protozoan-like microorganism in an autopsy specimen while trying to understand malaria, which was prevalent during the construction of the Panama Canal. He named this microorganism Histoplasma capsulatum because it invaded the cytoplasm (plasma) of histiocyte-like cells (Histo) and had a refractive halo mimicking a capsule (capsulatum), a misnomer. Histoplasma capsulatum, a dimorphic fungus, now belongs to Kingdom Fungi and causes histoplasmosis (Darling’s disease) through inhalation of spores found in soil and bird droppings. The fungus thrives in the central and eastern parts of United States, especially around the Ohio and Mississippi River valleys, and in South America, Africa, Asia, and Australia. Three varieties exist globally: H. capsulatum var. capsulatum, H. capsulatum var. duboisii, and H. capsulatum var. farciminosum.

Sources
1. Darling ST. A protozoon general infection producing pseudotubercules in the lungs and focal necrosis in the liver, spleen, and lymphnodes. JAMA. 1906;46:1283. https://doi.org/10.1001/jama.1906.62510440037003
2. Hagan T. The discovery and naming of histoplasmosis: Samuel Taylor Darling. JAMA. 1903;46:1905-7 [cited 2020 Nov 19]. http://www.antimicrobe.org/hisphoto/history/%20of%20Histoplasmosis-Darling.asp
3. Histoplasmosis, types of diseases, fungal diseases, CDC [cited 2020 Aug 21]. https://www.cdc.gov/fungal/diseases/histoplasmosis/
4. Ramsey TL, Applebaum AA. Histoplasmosis “darling.” Am J Clin Pathol. 1942;12:85–94. https://doi.org/10.1093/ajcp/12.2.85
5. Slavin MA, Chakrabarti A. Opportunistic fungal infections in the Asia-Pacific region. Med Mycol. 2012; 50:18–25. https://doi.org/10.3109/13693786.2011.602989

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DOI: https://doi.org/10.3201/eid2703.ET2703
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Appendix

Appendix Figure. Diagnostic wedge-biopsy establishing diagnosis of pulmonary histoplasmosis. A) (hematoxylin and eosin staining; 20× magnification) Overview of the diagnostic wedge-biopsy showing emphysema, fibrosis, and necrotizing granulomas. B) Higher magnification (hematoxylin and eosin staining; 400×) highlights the necrosis and the surrounding histiocytic wall. C) (400× magnification) Grocott silver staining discloses the ovoid histoplasma organisms (black staining).