cases reported to health authorities. The chronic form of paracoccidioidomycosis will probably develop in some of these patients.

This study underscores the need for paracoccidioidomycosis surveillance, especially in the context of environmental alterations enhanced by climate change and affected by construction, deforestation, and other human interventions. Enhanced surveillance will more fully identify relative risks of different human enterprises and facilitate interventions for at-risk populations to reduce and prevent future outbreaks of paracoccidioidomycosis.

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Mycobacterium shimoidei, a Rare Pulmonary Pathogen, Queensland, Australia

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Nontuberculous mycobacteria are human pathogens with increasing incidence and prevalence worldwide. *Mycobacterium shimoidei* is a rare cause of pulmonary disease, with only 15 cases previously reported. This series documents an additional 23 cases of *M. shimoidei* from Queensland, Australia, and highlights the pathogenicity and clinical role of this species.

Nontuberculous mycobacteria (NTM) are prominent human pathogens, with >150 species reported worldwide (*1*). *Mycobacterium shimoidei* is a slow-growing NTM that was first isolated in Japan in 1968, successfully gaining species status in 1975 (*2*). Since then, only 15 cases have been reported worldwide (*3–10*).

In Queensland, Australia, NTM is a reportable condition, requiring all isolates to be reported to the Queensland Mycobacterium Reference Laboratory. This series examines all *M. shimoidei* cases in Queensland during January 1, 2000– December 31, 2014.

We extracted data from the Queensland Notifiable Condition System with ethics approval obtained from the Metro North Human Research Ethics Committee (HREC/15/QPCH/65). Confirmatory testing was conducted at the Queensland Mycobacterium Reference Laboratory using 2 methods: the Hain Genotype CM/AS Line Probe Assays (Hain Lifescience, Nehren, Germany) and 16S rRNA sequencing.

We obtained clinical information from treating physicians and patient medical records. We recorded each isolate as being likely clinically significant, possibly significant, or unlikely significant, and as being consistent or not with NTM lung disease according to the 2007 American Thoracic Society and Infectious Disease Society of America criteria.

Specimens from 23 patients (35 total isolates) cultured *M. shimoidei* in Queensland during the study period. Individual clinical characteristics, treatment, and outcomes can be seen in the Table. Previously reported cases are summarized in the online Technical Appendix (https://wwwnc.cdc.gov/EID/article/23/11/17-0999-Techapp1.pdf).

Sixteen (69.6%) patients were male (mean ± SD age 66.2 ± 12.6 years), consistent with previous case reports (*3–10*). Nine (39.1%) were classified as being likely clinically significant and 7 (30.4%) possibly significant. Ten patients (43.5%) met the 2007 American Thoracic Society and Infectious Disease Society of America criteria for having NTM lung disease. All isolates were cultured from respiratory specimens, with 15 (65.2%) isolated from sputum; 2 (8.7%) from bronchial washings; 2 (8.7%) from both bronchial washings and sputum; and 2 (17.4%) from lung tissue either with computed tomography–guided biopsy or at autopsy. Only 4 (17.4%) specimens were smear-positive by microscopy.

Table. Clinical characteristics, treatment, and outcomes of *Mycobacterium shimoidei* isolates, Queensland, Australia*

Specimen (isolates)	Age, y/sex	Significant	Signs/ symptoms	Radiology	Concurrent conditions	Management (time)	Outcome
Sp and Br (×4)	60/M	Likely	C, Sp, WL	Cavities, nodule	COPD, asthma	Observed	Stable
LTis (×1)	56/M	Likely	Died	Unknown	Unknown	None	Died
Sp (×1)	75/F	Likely	C, Sp, WL	Cavities, nodules	COPD, HF, AF, GERD	None	Died of other cause
Sp (×3)	72/M	Likely	C, D, WL	Cavity, nodules	COPD, bronchiectasis, IHD	Observed	Died of lung disease
LTis (×1)	62/F	Likely	C, WL, NS	Cavity	None	INH, RFP, PZA, EMB (6 mo)	Stable
Sp (×2)	68/M	Likely	C, Sp, H, WL, Fa	Cavities, consolidation	COPD, aspergillus, HTN	CLA, MFX, SMX (12 mo)	Improved
Sp and Br (×4)	70/M	Likely	C, Sp, CP	Cavities	Lung cancer, COPD, bronchiectasis	CLA, RIF, EMB (12 mo)	Died of lung disease
LTis (×1)	77/F	Likely	C, WL, Fa	Cavity, nodules	COPD, GERD	CLA, RFP, EMB (18 mo)	Improved
Sp (×3)	68/M	Likely	C, Sp, WL	Cavity, consolidation	COPD, RA, anemia	Observed	Stable
Br (×1)	76/M	Possibly	D, WL	Nodules	COPD, anemia	None	Unknown
Br (×1)	84/M	Possibly	C, Sp	Mass, effusion	Lung cancer, GERD	Observed	Died of lung disease
Sp (×1)	84/M	Possibly	C, D, Fa	Consolidation	COPD, bronchiectasis	Observed	Improved
Sp (×1)	29/M	Possibly	C, D, WL	Nodules	CF, bronchiectasis	AMK, CFX, AZA, CFZ (24 mo)	Improved
Sp (×1)	74/F	Possibly	C, Sp	Nodules, consolidation	Bronchiectasis	Observed	Improved
Sp (×5)	84/F	Possibly	C, Sp, H, WL	Nodules	Bronchiectasis, type 2 diabetes, HTN	CLA (2 mo)	Improved
Sp (×1)	58/M	Possibly	C, Sp	Normal	Obesity, HTN	Observed	Stable
Sp (×1)	57/M	Unlikely	Unknown	Unknown	Unknown	Unknown	Unknown
LTis (×1)	55/F	Unlikely	Unknown	Unknown	Unknown	Unknown	Unknown
Sp (×1)	67/M	Unlikely	Unknown	Unknown	Unknown	Unknown	Unknown
Sp (×1)	60/M	Unlikely	C, D	Normal	Asthma	None	Unknown
Sp (×1)	59/F	Unlikely	C	Normal	Asthma, GERD	None	Unknown
Sp (×1)	73/M	Unlikely	Unknown	Unknown	Unknown	Unknown	Unknown
Sp (×1)	54/M	Unlikely	Unknown	Unknown	Unknown	Unknown	Unknown

*AF, atrial fibrillation; AMK, amikacin; AZA, azithromycin; Br, bronchoscopic washing; C, cough; CF, cystic fibrosis; CFX, cefoxitin; CFZ, clofazimine; CLA, clarithromycin; COPD, chronic obstructive pulmonary disease; CP, chest pain; D, dyspnea; EMB, ethambutol; Fa, fatigue; GERD, gastroesophageal reflux disease; H, hemoptysis; HF, heart failure; HTN, hypertension; IHD, ischemic heart disease; INH, isoniazid; LTis, lung tissue; MFX, moxifloxacin; NS, night sweats; PZA, pyrazinamide; RA, rheumatoid arthritis; RFP, rifampin; RIF, rifabutin; SMX, sulfamethoxazole, Sp, sputum; WL, weight loss.

The most common symptoms were cough or sputum (16; 69.6%); weight loss (9; 39.1%); dyspnea (5; 21.7%); fevers or sweats (4; 17.4%); and fatigue (2; 8.7%). Cough and sputum predominated in previous cases, but not weight loss (*3–10*). Radiology demonstrated cavitary disease in 9 patients (39.1%). Similar to our cohort, 9 of the 15 previously reported cases had cavities, highlighting a potentially distinguishing feature of *M. shimoidei* lung disease (*3–7*).

The most common associated concurrent conditions were obstructive airway disease (10; 43.5%), bronchiectasis (6; 26.1%), gastroesophageal reflux disease (4; 17.4%), and malnutrition (3; 13.0%). Underlying chronic lung disease was also present in previously reported cases and included chronic obstructive pulmonary disease, past tuberculosis, pneumoconiosis, and bronchiectasis (*3–10*).

Although 16 patients (69.5%) were deemed to have either likely or possibly clinically significant disease, only 6 (26.0%) underwent medical treatment, with 7 (30.4%) being actively observed. These low treatment numbers may reflect a lack of knowledge in relation to *M. shimoidei*; however, they may also be an indirect result of the underlying comorbidities and poor functional status of infected patients.

When medical treatment was offered, however, 5 of the 6 patients improved or had stable disease, with the sixth patient dying of lung cancer while undergoing antimicrobial therapy. Of the 7 patients who were observed, 3 remained stable, 2 improved, and 2 died of either chronic lung disease or progression of their *M. shimoidei* infection. In comparison, 6 of the 15 previous cases in the literature improved with medical treatment, with 4 dying during treatment and 1 remaining stable with observation alone (*3–10*). Although this relatively high death rate may reflect the nature of the patients' comorbidities, it still highlights the clinical significance of *M. shimoidei* if isolated.

Although none of the Queensland cohort underwent drug susceptibility testing, review of previous cases suggests that a combination of rifabutin, ethambutol, and clarithromycin may be an effective drug regimen, with moxifloxacin/levofloxacin, sulfamethoxazole, pyrazinamide, and linezolid as other potential agents (*3–7*).

Our study has several limitations. First, it is a retrospective case series with data extracted from a passive surveillance system. Even though all laboratory-confirmed cases were captured, it is possible that not all patients with *M. shimoidei* infection received this diagnosis or were able to provide an appropriate specimen for identification. Futhermore, due to both the clinical characteristics being reported by various treating physicians and a large proportion not having complete clinical or follow-up data available, we may have captured inaccurate or inconsistent data.

This case series highlights the clinical significance and pathogenicity of *M. shimoidei.* Cases have been isolated only from respiratory specimens, occur predominantly in male patients with underlying chronic lung disease, and commonly present with cavitary disease. Although illness and death are associated with *M. shimoidei* infection, a reasonable outcome can be achieved with treatment. Possible drug regimens involve a combination of rifabutin, ethambutol, and clarithromycin, with moxifloxacin/levofloxacin, sulfamethoxazole, pyrazinamide, and clofazimine also potentially being useful. Increased recognition and understanding of this pathogenic organism are necessary to improve patient outcomes.

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The Breadth of Viruses in Human Semen

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Zika virus RNA is frequently detected in the semen of men after Zika virus infection. To learn more about persistence of viruses in genital fluids, we searched PubMed for relevant articles. We found evidence that 27 viruses, across a broad range of virus families, can be found in human semen.

The finding by Atkinson et al. that Zika virus RNA is frequently detected in the semen of men after infection (*1*) highlights our knowledge gaps regarding the persistence of viruses in genital fluids, especially semen. Replicating Zika virus (*2*), like Ebola and Marburg viruses (*3*), has been isolated from semen and has been sexually transmitted. However, it is probable that many more viruses capable of causing viremia (presence of virus in the blood) can be found in semen. Seeding to the male reproductive tract may frequently occur in the context of viremia because the blood–testes/deferens/epididymis barriers are imperfect barriers to viruses, especially in the presence of systemic or local inflammation (*4*). Virus may persist even if incapable of replicating within the male reproductive tract because the testes are immunologically privileged (*4*); that is, within the testes, the immune response is restricted to enable the survival of sperm, which are immunogenic. Virus may also be transmitted to semen as a result of survival and replication within the accessory glands (*5*).

To investigate the breadth of viruses in semen, we performed a PubMed search by using the terms "virus* AND semen OR sperm* OR seminal." We imposed no date or language restrictions. This search returned 3,818 results. We screened the titles, abstracts, and full text articles for data that described detection of viruses in semen by nucleic acid amplification or detection, antigen detection, replication in cell culture, or replication in an animal system. We restricted the results to viruses capable of causing viremia. Where we found evidence for virus in semen, we then searched PubMed for evidence of sexual transmission by using the terms "(name of virus) AND sex* AND Transm*."

Our search revealed that 27 viruses that can result in viremia have been found in human semen (Table). For many of these, data on sexual transmission are lacking. Of these 27 viruses, many cause chronic or latent infection (e.g., HIV virus, cytomegalovirus). However, several cause acute infections, including Lassa fever, Rift Valley fever, and chikungunya viruses. Of those causing acute infections, only Zika and Ebola viruses have been systematically screened for in semen (i.e., in case series or cohort studies rather than case reports). These 27 viruses come from diverse families, suggesting that the presence of many viruses in semen is unlikely to be exclusively dependent on specific or conserved viral epitopes, ability of virus to replicate within the male reproductive tract, or common mechanisms of immune evasion. Other factors that may also influence whether viruses exist in semen are level of viremia, inflammatory mediators (altering blood-barrier permeability), systemic immunosuppression, male reproductive tract immune responses, presence of sexually transmitted diseases, and virus structural stability. In mammals, numerous viruses are detectable in semen, including viruses that can cause disease in humans, such as Japanese encephalitis virus, foot and mouth disease virus, parainfluenza virus, and paravaccinia virus (*6*). Several other viruses that result in viremia can cause orchitis and have been detected in human testes, suggesting the possibility that these viruses may also be detectable in semen. These viruses include influenza virus, lymphocytic choriomeningitis virus, phlebotomus fever virus, cocksackie B virus, echovirus, dengue virus, systemic acute respiratory syndrome virus, parvovirus, smallpox virus, vaccinia virus, and rubella virus (*7*).

Given these findings, the following questions need to be addressed: which viruses are shed and remain viable in semen, for how long, and at what concentrations? The answers to these questions have implications for risks for sexual transmission and, therefore, embryonic infection, congenital disease, miscarriage, and effects on epidemiologic and transmission models. The presence of virus in the male reproductive tract may increase the risk for acquisition of sexually transmitted infections and may reduce male fertility through spermatogonial stem cell infection or local inflammation. Infection of spermatozoa could result in transmission of virus-induced mutations to subsequent

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Mycobacterium shimoidei, a Rare Pulmonary Pathogen, Queensland, Australia

Technical Appendix Table. Clinical characteristics, treatment, and outcomes of previously reported *Mycobacterium shimodei* isolates*

Author, year	Country	Age, y, sex	Symptoms	Radiology	Comorbidities	Management	Outcome
Tsukumura et al., 1975 (*1*)	Japan	56/M	Unknown	Cavity	Unknown	Unknown	Died of lung disease
Rusch-Gerdes et al., 1985 (*2*)	Germany	77/M	F	Cavity	Silicosis	INH, PRO, RFP, SM, ISO (3 mo)	Improved
Chromyc et al., 1989 (*3*)	Canada	84/M	C, S	Consolidation	COPD, pneumoconiosis	Unknown	Died of lung disease
Tortoli and Simonetti, 1989 (*4*)	Italy	68/M	U	Cavity	Old TB, Addison's	INH, SM, EB, RFP, KM, PAS (4 mo)	Died of lung disease
Miller et al., 1991 (*5*)	Canada	65/M	F	Mass	COPD, lung Ca	Unknown	Unknown
Furrer,et al.,1994 (*6*)	Switzerland	34/M	U	Unknown	HIV	None	Died of other cause
Heller et al., 1996 (*7*)	France	48/F	A	Cavity, nodules	Old TB	SM, EMB, CLA, RIF; EM, CPFX, RIF (18 mo)	Improved
Auregan et al., 1997 (*8*)	Madagascar	43/F	H	Cavity	Old TB	INH, RFP, PZA, EMB, SM (17 mo)	Died of lung disease
Goudge et al., 1998 (*9*)	Australia	75/M	C, S, WL, NS	Cavity, nodules	COPD, previous lobectomy	EMB, PZA, CLA, RIF (5 wk)	Died of lung disease
Mayall et al., 1999 (*10*)	Australia	53/F	C, S, WL	Cavity	COPD, fibrosis, esophageal Ca	INH, RFP, PZA, EMB (10 d)	Died of lung disease
Koukila-Kahkola et al., 2000 (*11*)	Finland	78/F	C, S	Normal	None	Observation	Stable
Sundman et al., 2000 (*12*)	Sweden	59/F	F, CP, D	Cavity	COPD	CPFX, CLDM; MDZ, TFX (6 wk)	Improved
Takayama et al., 2005 (*13*)	Japan	68/M	F, C	Consolidation	COPD	RFP, EMB, CAM, PZA, CPFX (6 mo)	Improved
Kanaji et al., 2011 (*14*)	Japan	83/M	C, S	Cavity	COPD	CLA, RFP, EMB (18 mo)	Improved
Galizzi et al., 2013 (*15*)	Italy	53/F	C, H	Nodules	Bronchiectasis	AMK, RIF, EMB, CLA (18 mo)	Improved

* A, asymptomatic; AMK, amikacin; C, cough; CLA, clarithromycin; CLDM, clindamycin; COPD, chronic obstructive pulmonary disease; CPFX, ciprofloxacin; D, dyspnea; EMB, ethambutol; F, fever; H, hemoptysis; INH, isoniazid; KM, kanamycin; MDZ, metronidazole; NS, night sweats; PAS, para-aminosalicylic acid; PRO, prothionamide; PZA, pyrazinamide; RFP, rifampin; RIF, rifabutin; SM, streptomycin; Sp, sputum; TB, tuberculosis; TFX, trifolitoxin; U, unknown; WL, weight loss

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