Report of Invasive Rhodococcus equi Infections in Taiwan, with an Emphasis on the Emergence of Multidrug-Resistant Strains

Po-Ren Hsueh, Chien-Ching Hung, Lee-Jeng Teng, Ming-Chih Yu, Yu-Chi Chen, Hua-Kung Wang,* and Kwen-Tay Luh

From November 1995 to October 1997, seven patients with invasive infections due to Rhodococcus equi were treated in Taiwan. Four patients had pulmonary lesions, and one each of the remaining three patients had a recurrent Port-A-Cath (Kabi-Pharmacia, North Ryde, New South Wales, Australia)–related bacteremia, a primary bacteremia, and a brain abscess. Three patients had underlying hematologic malignancies, and one each of the remaining four patients had diabetes mellitus, Waldenström’s macroglobulinemia, long-term use of steroids, and AIDS. The 13 isolates of R. equi recovered from these patients were identified by using API Coryne System (bioMérieux, Marcy l’Etoile, France), VITEK GPI card (bioMérieux Vitek, Hazelwood, MO), supplemental biochemical tests, and cellular fatty acid chromatograms. Susceptibilities of these isolates to 16 antimicrobial agents, with use of the agar dilution method, varied; among them, amikacin and trimethoprim–sulfamethoxazole were the most active agents. Different random amplified polymorphic DNA (RAPD) patterns of isolates from different patients documented the lack of epidemiological relatedness of the causative organisms of these infections. This study confirms the emergence of multidrug-resistant R. equi infection in Taiwan and documents the relapsing or reactivating nature of this infection.

Since the first description of human infection due to Rhodococcus equi (formerly Corynebacterium equi) in 1967 [1], this organism has been increasingly recognized as an opportunistic pathogen of clinical importance among immunocompromised hosts, particularly those infected with HIV [2–5]. R. equi are aerobic, nonmotile, nonspore-forming, pleomorphic, gram-positive and partially acid-fast bacilli (AFB) [6]. They are widely distributed among livestock and livestock environments [2, 5, 6]. The incidence of human infection due to R. equi may be underestimated because the organisms are commonly misidentified as a normal flora or as other contaminants, because of incomplete or inappropriate identification [2, 5, 6]. Pleuropulmonary infection, commonly presenting as chronic and relapsing cavitary pneumonia, is the most common clinical manifestation of infection due to R. equi [2–5, 7]. Extrapulmonary R. equi infections most likely represent secondary foci due to hematogenous dissemination [8–11]. Reports of cases of extrapulmonary infection without pulmonary involvement are rare [2, 5, 9, 11–13]. Frequently, infected patients have a history of contact with farm animals (especially horses and foals), contaminated soil, or manure [2, 5, 6]. Most infections due to Rhodococcus species are community acquired [1–13]; however, nosocomial infections or hospital outbreaks have been reported [14, 15].

From November 1995 to October 1997, we identified seven patients with invasive infections due to R. equi. To understand the clinical characteristics and outcome of infections due to R. equi in Taiwan and the probable epidemiological relatedness, we reviewed the medical records of the seven patients and studied the microbiological characteristics of 12 isolates recovered from these patients, including antimicrobial susceptibilities, cellular fatty acid profiles, and molecular typing.

Patients and Methods

Clinical data. Records of all clinical specimens submitted for culture to the microbiology laboratory at National Taiwan University Hospital from January 1987 to October 1997 that were positive for Rhodococcus species were reviewed. During this period, 13 R. equi isolates were recovered from various specimens obtained from six patients who were treated at the hospital from November 1995 to October 1997. The other one isolate was recovered from one patient who was treated at Taiwan Provincial Chronic Disease Control Bureau (Taipei) in March 1997. Relevant clinical and demographic information concerning these patients was obtained retrospectively through a review of medical records. This information included basic patient data such as age and sex, underlying diseases, types of infection, associated conditions (implantation of indwelling...
devices, chemotherapy, and bone marrow transplantation), source and date of isolation, antimicrobial therapy, and outcome.

**Bacterial isolates.** The isolates were initially identified from colonial and microscopic morphology, properties on Kinyoun staining, and biochemical characteristics determined by using the API Coryne system (bioMérieux, Marcy l’Etoile, France), API ZYM, and the VITEK GPI card (bioMérieux Vitek, Hazelwood, MO) [6]. These isolates were confirmed as *R. equi* with use of supplemental tests including the presence of *equi* factor (CAMP test); catalase, urease, lipase, and oxidase reactions; ortho- nitrophenyl-β-d-galactopyranoside (ONPG); growth on MacConkey agar; and hydrolysis of xanthine, tyrosine, hypoxanthine, and gelatin, as described by Prescott [2].

**Cellular fatty acid analysis.** For cellular fatty acid analysis, the isolates were incubated in trypticase soy agar (BBL Becton Dickinson Microbiology Systems, Cockeysville, MD). Procedures for preparation and identification of fatty acid methyl esters and the instrument used were the same as the previous description [16]. The software library used to identify the *Rhodococcus* species was TSA, version 3.9 (Microbial ID, Newark, DE). A major peak of the esterified fatty acid was defined as an area percentage ≥3% of the area of the total esterified fatty acid of the isolate; a minor peak was defined as an area percentage <3% of the total esterified fatty acid of the isolate. The similarity index (ranges, 0–1) was defined as the closeness of a match of the unknown bacterium to a library entry. A similarity index of >0.6 was defined as an excellent match.

**Antimicrobial susceptibility testing.** MICs of 16 antimicrobial agents for the 13 isolates of *R. equi* were determined according to the National Committee for Clinical Laboratory Standards (NCCLS) guidelines [17]. The following antimicrobial agents were obtained from the corresponding manufacturers as standard reference powders of known potency for laboratory use: penicillin G, ampicillin/sulbactam, cefuroxime, trimethoprim-sulfamethoxazole (TMP-SMZ), erythromycin, rifampin, and vancomycin (Sigma Chemical, St. Louis); amoxicillin/clavulanic acid (Beecham Research Laboratories, Brentford, England); ceftriaxone (Roche Laboratories, Nutley, NJ); aztreonam and amikacin (Bristol-Myers Squibb Laboratories, New York); imipenem (Merck Sharp & Dohme, West Point, PA); minocycline (Lederle Laboratories, Pearl River, NY); clarithromycin (Abbott Laboratories, Abbott Park, IL); ofloxacin (Daichi Pharmaceutical, Tokyo, Japan); and ciprofloxacin (Bayer, Leverkusen, Germany).

The MICs of the antimicrobial agents that were tested ranged from 0.03 μg/mL to 256 μg/mL. *Staphylococcus aureus* American Type Culture Collection (ATCC) 29213, *Escherichia coli* ATCC 25922, and *Pseudomonas aeruginosa* ATCC 27853 were used as control strains. Because the resistant and susceptible MIC breakpoints for *Rhodococcus* species are not defined by the NCCLS, the percentage of isolates susceptible or resistant to antimicrobial agents was classified presumptively by application of the susceptibility breakpoints used for organisms other than *Haemophilus* species, *Neisseria gonorrhoeae*, and streptococci [17]. The NCCLS criteria for staphylococci were adopted for defining susceptibilities to penicillin and amoxicillin/clavulanic acid. The presence of β-lactamases among these isolates was determined with use of a cefinase disk (BBL Microbiology Systems).

**Random amplified polymorphic DNA (RAPD) analysis.** All isolates were incubated in brain-heart infusion broth (Difco Laboratories, Detroit, MI) for 36–48 hours at 35°C in ambient air. Bacterial genomic DNA was extracted by using a commercial kit (Puregene; Gentra Systems, Inc., Minneapolis). The random amplified polymorphic DNA (RAPD) assay, generated by arbitrarily primed PCR (APPCR), was performed using the following two arbitrary oligonucleotide primers: H3 (5′-AGA-CGCCA-3′) and H4 (5′-GGAAGTCGCC-3′) (OPERON Technologies, Inc., Alameda, CA). The conditions for PCR followed the previous description [16].

**Definitions.** Infections that developed ≥72 hours after admission were regarded as nosocomial, whereas those infections that developed earlier were considered community acquired. Antimicrobial therapy given within 48 hours of admission was considered appropriate if at least one of the agents administered was active against the *R. equi* isolates in vitro. A response to antibiotic therapy was defined as complete resolution of all clinical and microbiological signs and symptoms of infection, except in cases of pneumonia. Among the patients with pneumonia, response was defined as resolution of all symptoms, sputum and blood cultures negative for *R. equi*, and significant improvement in condition, demonstrated by chest radiographs. A response to initial antimicrobial therapy (initial response) was determined at the point when the initial regimen was discontinued.

Antibiotypes of the isolates were considered different if the MICs for at least two of the antimicrobial agents tested were at least a two-dilution discrepancy. Both faint and intense bands were included for the interpretation of RAPD patterns [16]. Patterns differing by more than one band were considered different; otherwise, they were considered identical. Isolates were defined as the same strain or considered to be derived from a single clone if they had identical antibiotypes and identical RAPD patterns [16]. A relapse was defined as recurrence of an infection due to the same *R. equi* clone.

**Results.**

**Clinical features.** Clinical characteristics of the seven patients with invasive *R. equi* infections are shown in table 1. All patients had underlying diseases. Three of them had hematologic malignancies, and one had AIDS. None of the patients had a known history of contact with any animal sources. Two patients (patients 3 and 6) had had more than one admission to the same ward within an interval of 3 months for scheduled chemotherapy before the documentation of *R. equi* infection,
Table 1. Clinical characteristics of seven patients in Taiwan with invasive infections due to Rhodococcus equi.

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Age (y)/sex</th>
<th>Underlying disease/associated condition</th>
<th>Clinical diagnosis</th>
<th>Source of isolation (isolate)*</th>
<th>Date of isolation (d/mo/y)</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>27/M</td>
<td>AIDS</td>
<td>Thoracic empyema (CA)</td>
<td>Empyema fluid (A)</td>
<td>3/11/95</td>
<td>Clm, Rif, TMP-SMZ</td>
<td>Survived</td>
</tr>
<tr>
<td>2</td>
<td>67/F</td>
<td>Waldenström’s macroglobulinemia</td>
<td>Brain abscess (CA)</td>
<td>Aspirate of abscess (B), drain tube</td>
<td>31/12/96</td>
<td>Cpfx, Imi, Vm; and surgical drainage</td>
<td>Died</td>
</tr>
<tr>
<td>3</td>
<td>58/F</td>
<td>AML and Port-A-Cath disease (CA)</td>
<td>Catheter-related bacteremia (CA or N)</td>
<td>Blood (C1)</td>
<td>14/6/96</td>
<td>Vm</td>
<td>Relapsed</td>
</tr>
<tr>
<td>4</td>
<td>80/F</td>
<td>DM, intracranial hemorrhage</td>
<td>Primary bacteremia (N)</td>
<td>Blood (D)</td>
<td>28/7/96</td>
<td>Amp/Sub, Gm</td>
<td>Survived</td>
</tr>
<tr>
<td>5</td>
<td>58/F</td>
<td>COPD, steroid use</td>
<td>Cavitary pneumonia (CA)</td>
<td>Lung aspirate (E)</td>
<td>18/3/97</td>
<td>Cpfx, Amik</td>
<td>Survived</td>
</tr>
<tr>
<td>6</td>
<td>47/M</td>
<td>AML/BMT, Port-A-Cath disease (CA)</td>
<td>Cavitary pneumonia (CA)</td>
<td>Lung aspirate (F1), sputum (F2)</td>
<td>23/7/97</td>
<td>Ofx, Clm</td>
<td>Relapsed</td>
</tr>
<tr>
<td>7</td>
<td>49/M</td>
<td>Non-Hodgkin’s lymphoma/Port-A-Cath disease (CA)</td>
<td>Cavitary pneumonia, infiltrative liver disease (CA)</td>
<td>Liver biopsy (G)</td>
<td>28/8/97</td>
<td>Vm, Cpfx</td>
<td>Survived</td>
</tr>
</tbody>
</table>

NOTE. Amik = amikacin; AML = acute myelocytic leukemia; Amp/Sub = ampicillin/sulbactam; BMT = bone marrow transplantation; CA = community acquired; Cpfx = ciprofloxacin; Clm = clarithromycin; COPD = chronic obstructive pulmonary disease; DM = diabetes mellitus; Gm = gentamicin; Imi = imipenem; N = nosocomial; Ofx = olfoxacin; Rif = rifampicin; TMP-SMZ = trimethoprim-sulfamethoxazole; Vm = vancomycin.

* See figure 1.

and they also had the Port-A-Cath (Kabi-Pharmacia, North Ryde, New South Wales, Australia) implants. Patient 3 had positive cultures of a blood specimen collected via a Port-A-Cath and peripheral veins on three occasions. Gram-stained smear of the blood aspirated from the removed Port-A-Cath tip disclosed many gram-positive bacilli that were later identified as R. equi. Patient 4 acquired nosocomial R. equi septicemia 1 month after admission, and positive cultures were documented for two sets of blood specimens. Among the four patients who had pulmonary lesions, one patient (patient 7) had a liver biopsy specimen positive for R. equi but had no microbiological documentation of R. equi from the lung lesions. The other three patients (patients 1, 5, and 6) recovered, and their lung lesions disappeared after antibiotic treatment.

There was recurrence of infection in two patients with initial responses: patient 6 developed fulminant pneumonia 2 weeks after treatment, and patient 3 had an additional two episodes of bacteremia within 5 months after treatment. All patients were treated with antimicrobial agents for 2–4 months, and all survived, except one patient (patient 2) who died 2 days after treatment of acute lung edema unrelated to R. equi infection.

_Bacterial isolates._ All isolates were gram-positive and weakly positive for AFB with use of modified Kinyoun staining. The typical cyclic microscopic morphology was found on trypticase soy agar supplemented with 5% sheep blood (BBL Becton Dickinson Microbiology Systems). The colonial morphology of _R. equi_ isolates was variable: one type (three isolates) was pink and mucoid, and the other type (ten isolates) was coral colored and nonmucoid. All isolates were catalase, lipase, and urease positive and oxidase negative. They all produced _equi_ factor on interaction with _S. aureus_. None of the isolates hydrolyzed xanthine, tyrosine, or hypoxanthine, and all were negative for the ONPG reaction. Results of API ZYM gave enzyme profiles that were characteristic for _R. equi_ [2].

_Cellular fatty acid chromatograms._ Figure 1 shows the cellular fatty acid chromatogram for _R. equi_ isolates. All isolates had major peaks for the following fatty acids: 2-hydroxy-13-methyltetra decanoic acid (i-2-OH-15:0), hexadecanoic acid (16:0), cis-9-octadecanoic acid (18:1 w9c), and 10-methyloctadecanoic acid methyl ester (10 Me 18:0), and minor peaks for myristic acid (14:0), pentadecanoic acid (15:0), and octadecanoic acid (18:0). The similarity indices of these isolates with the identification of _Rhodococcus_ species ranged from 0.7 to 0.8.

_Antimicrobial susceptibilities._ MICs of the 16 antimicrobial agents for the isolates are shown in table 2. Seven antibiotic types were identified among the 13 isolates. Isolates from different patients possessed different antibiotic types; otherwise, antibiotic types from isolates recovered from the same patients (isolates C1–C4 from patient 3 and isolates F1–F4 from patient 6) were identical. All isolates were susceptible to amikacin and TMP-SMZ. Susceptibilities to the other 14 agents were
Invasive R. equi Infections in Taiwan

Growth media, and rifampin (MICs, >256 µg/mL). Isolates E, F1–F4, and G were β-lactamase positive; the other isolates were β-lactamase negative.

RAPD patterns. RAPD patterns for the 13 isolates by the primers H3 and H4 are shown in figure 2. Isolates from different patients had different RAPD patterns. Identical RAPD patterns were found for the four isolates (C1–C4) from patient 3, and the four isolates (F1–F4) from patient 6.

Discussion

This retrospective study of the clinical features of the first seven Taiwanese patients with infections due to R. equi and the microbiological characterization of the 13 isolates from these patients disclosed four important points. (1) To our knowledge, we have reported the first presence of R. equi in Taiwan, and this organism should be considered an emerging pathogen in this geographic area. (2) We documented invasive infections in humans due to R. equi strains that are highly resistant to extended-spectrum β-lactams, imipenem, macrolides, and rifampin, for which reports are rare [2–4]. (3) This is the first application of phenotypic and genotypic methods to confirm the relapsing or reactivating nature of infection due to R. equi among immunocompromised hosts and to rule out nosocomial spread of this organism. (4) We report the first case of Port-A-Cath±related bacteremia due to R. equi, as well as the first case of brain abscess among patients who are not HIV infected [18, 19].

For definitive identification and adequate differentiation of all species of Rhodococcus, an extensive panel of biochemical and enzymatic characteristics is needed, and such analyses are not feasible in routine clinical microbiology laboratories [2, 5, 6]. Cellular fatty acid analysis by gas-liquid chromatography has been useful for the species-level identification of a large number of bacteria [16]. However, as shown in this study, the application of this analysis for identifying R. equi is limited. Previous studies have shown that ribotype analysis could be

![Figure 1.](https://example.com/figure1.png) Cellular fatty acid chromatogram for Rhodococcus equi isolates showing major peaks for the following fatty acids: 2-hydroxy-13-methyltetradecanoic acid (i2-OH-15:0), hexadecanoic acid (16:0), cis-9-octadecenoic acid (18:1 w9c), and 10-methyloctadecanoic acid methyl ester (10 Me 18:0), and minor peaks for myristic acid (14:0), pentadecanoic acid (15:0), and octadecanoic acid (18:0). SP = solvent peak.

<table>
<thead>
<tr>
<th>Patient no./isolate</th>
<th>Pen G</th>
<th>Amp/Subb</th>
<th>Amox/CA</th>
<th>Cfur</th>
<th>Ctri</th>
<th>Atm</th>
<th>Imi</th>
<th>Mino</th>
<th>Em</th>
<th>Clm</th>
<th>Rif</th>
<th>Ofx</th>
<th>Cpfx</th>
<th>Vm</th>
<th>Amik</th>
<th>TMP-SMZ</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/A</td>
<td>0.25</td>
<td>0.25</td>
<td>1</td>
<td>2</td>
<td>4</td>
<td>0.5</td>
<td>0.5</td>
<td>0.06</td>
<td>1</td>
<td>0.03</td>
<td>&lt;0.03</td>
<td>0.03</td>
<td>0.03</td>
<td>0.5</td>
<td>0.25</td>
<td>&lt;0.03</td>
</tr>
<tr>
<td>2/B</td>
<td>0.12</td>
<td>0.12</td>
<td>0.06</td>
<td>1</td>
<td>0.5</td>
<td>0.5</td>
<td>0.06</td>
<td>2</td>
<td>1</td>
<td>0.25</td>
<td>4</td>
<td>0.12</td>
<td>0.06</td>
<td>1</td>
<td>0.12</td>
<td>&lt;0.03</td>
</tr>
<tr>
<td>3/C1</td>
<td>0.5</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0.03</td>
<td>0.25</td>
<td>0.12</td>
<td>0.12</td>
<td>2</td>
<td>0.06</td>
<td>0.03</td>
<td>2</td>
<td>0.25</td>
<td>0.06</td>
</tr>
<tr>
<td>4/D</td>
<td>0.25</td>
<td>0.5</td>
<td>1</td>
<td>2</td>
<td>4</td>
<td>0.5</td>
<td>0.06</td>
<td>2</td>
<td>1</td>
<td>0.03</td>
<td>&lt;0.03</td>
<td>0.03</td>
<td>0.03</td>
<td>0.5</td>
<td>0.25</td>
<td>0.5</td>
</tr>
<tr>
<td>5/E</td>
<td>64</td>
<td>16</td>
<td>8</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>4</td>
<td>8</td>
<td>32</td>
<td>32</td>
<td>&gt;256</td>
<td>1</td>
<td>0.5</td>
<td>32</td>
<td>1</td>
<td>0.5</td>
</tr>
<tr>
<td>6/F1</td>
<td>&gt;256</td>
<td>8</td>
<td>2</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>64</td>
<td>4</td>
<td>16</td>
<td>16</td>
<td>&gt;256</td>
<td>32</td>
<td>64</td>
<td>4</td>
<td>1</td>
<td>0.5</td>
</tr>
<tr>
<td>7/G</td>
<td>256</td>
<td>16</td>
<td>8</td>
<td>32</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>32</td>
<td>8</td>
<td>32</td>
<td>16</td>
<td>&gt;256</td>
<td>0.5</td>
<td>0.19</td>
<td>128</td>
<td>1</td>
<td>0.5</td>
</tr>
</tbody>
</table>

NOTE. Amox/CA = amoxicillin/clavulanic acid; Amp/Subb = ampicillin/subbactam; Amik = amikacin; Atm = aztreonam; Clm = clarithromycin; Cpfx = ciprofloxacin; Cfur = cefuroxime; Ctri = ceftriaxone; Em = erythromycin; Imi = imipenem; Mino = minocycline; Ofx = ofloxacin; Pen G = penicillin G; Rif = rifampin; TMP-SMZ = trimethoprim-sulfamethoxazole; Vm = vancomycin.

* Ratio of antibiotic concentrations of TMP to SMZ is 1:19.
In summary, the first seven cases of Rhodococcus equi infection have been confirmed in different geographic areas, including the patient with AIDS (patient 1) who had no contact with livestock or soil that was heavily contaminated with livestock. Most of the patients with R. equi infection had a significant history of contact with livestock or soil that was heavily contaminated with livestock. Although the soil habits of this organism and the associated zoonoses have been confirmed in different geographic areas, no such data existed in Taiwan.

Most infections due to Rhodococcus species are community acquired [1–13]. Nosocomial infections associated with the use of indwelling devices or due to contaminated closed-system packed RBCs have been reported [14, 15]. Two of the patients in our study (patients 6 and 7) were admitted to a ward that was occupied previously by another patient (patient 3) with R. equi infection, and they acquired the infection several days after admission. Given this situation and the rarity of this organism, the occurrence of a nosocomial outbreak might have been possible. However, the different antibiotypes and RAPD patterns excluded this possibility. Patient 4 had been hospitalized 1 month before identification of R. equi bacteremia; this suggested the possibility of nosocomial acquisition of this organism. Unfortunately, surveillance cultures of the hospital environment were not performed.

Previous reports have shown that R. equi is susceptible to ampicillin/sulbactam, amoxicillin/clavulanic acid, gentamicin, erythromycin, tetracycline, rifampin, TMP-SMZ, imipenem, and vancomycin [2, 5, 20, 21]. The organism is usually less susceptible to penicillin, ampicillin, cephalosporins, or quinolones [2, 4, 5, 20, 21]. Our susceptibility results partly support the previous findings. It is of interest that three of the seven strains from patients with community-acquired infections had inherent concomitant resistance to all β-lactams, aminoglycosides, macrolides, and rifampin. This finding is unique to isolates from other geographic areas.

For selection of appropriate agents to treat R. equi infections, both the in vivo and in vitro properties of the antimicrobial agents should be considered [21]. One of the patients (patient 6) had a relapse of infection after treatment with vancomycin, which has good in vitro efficacy but a low level of uptake by macrophages, although the relapse might have been due in part to the persistence of the organism in the Port-A-Cath. However, patient 4 had relapse of infection after treatment with ofloxacin and clarithromycin, which achieve higher intracellular concentrations. The identical antibiotypes for repeated isolates from each of these two patients with relapsing infections indicates that the failure of therapy was not attributable to acquisition of resistance to the agents used. It is suggested that combination therapy is more effective than monotherapy, because the former might reduce the mutation frequencies of resistance to antimicrobial agents [2–5, 20, 21]. Our study further confirms that an initial treatment regimen consisting of a combination of at least two agents with bactericidal activity and intracellular activity might be crucial for adequate treatment.

Previous studies have shown that the mortality associated with R. equi infection is higher for HIV-infected patients than for non-HIV-infected patients [3, 4]. The frequent simultaneous opportunistic infections that occur among HIV-infected patients and patients with deep-tissue infections treated with antibiotics alone, without surgical intervention, might have contributed to the higher mortality [4]. In the present study, all patients with R. equi infection recovered, including the patient with AIDS (patient 1) who had no concomitant infection, although one patient (patient 7) had multiple cavitary lung lesions and had not undergone repeated aspiration or surgical drainage.

In summary, the first seven cases of R. equi infection in Taiwan, which all occurred during a 2-year period, were epide-
miologically unrelated. Given that the incidence of this opportunistic human pathogen is increasing, medical microbiologists will need to improve upon techniques used to identify this pathogen, rather than dismiss the organism as a contaminant. In addition, physicians need to learn how to treat invasive R. equi infections appropriately, particularly in areas where isolates that are resistant to conventional antimicrobial agents are not unusual.

References