Other Slow-Growing Nontuberculous Mycobacteria

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ABSTRACT The list of clinically important slow-growing nontuberculous mycobacteria (NTM) continues to expand as new species are identified and older ones are found to be pathogenic. Based on pigment production, the strains may be classified as photochromogenic, scotochromogenic, or unpigmented. Some of these organisms are not newly discovered but have heretofore been considered virtually nonpathogenic. Previously, many were regarded as contaminants when isolated from clinical specimens. Ubiquitous in nature, many NTM have been isolated from groundwater or tap water, soil, house dust, domestic and wild animals, and birds. Most infections result from inhalation or direct inoculation from environmental sources. They are not spread from person to person. The infections may be localized or disseminated. In most cases, the optimal regimen or duration of therapy has not been firmly established. The results of in vitro susceptibility testing may be used to select a therapeutic regimen. Many experts recommend clarithromycin with companion drugs such as rifampin and ethambutol for most, but not all, slowly growing species. Aminoglycosides, clofazimine, fluoroquinolones, linezolid, pyrazinamide, or trimethoprim-sulfamethoxazole also may be effective against some strains. Immunocompetent patients with clinically significant infections with NTM usually should receive 18 to 24 months of therapy. Infected immunocompromised patients, particularly those with disseminated infection, probably should receive therapy as long as their immune systems remain impaired. Some of the species discussed include Mycobacterium aliense, M. celatum, M. gordonae, M. haemophilum, M. kyorinense, M. malmoense, M. simiae complex, M. szulgai, M. terrae complex, M. ulcerans, and M. xenopi.

MICROBIOLOGY

The list of clinically important slow-growing nontuberculous mycobacteria (NTM) continues to expand as new species are identified and older ones are found to be pathogenic. More detailed information on these organisms (including many not covered in this chapter) can be found in a number of excellent reviews (1–5). As a group, these mycobacteria currently cause fewer infections than those species discussed in previous chapters. Some of these organisms are not newly discovered but have heretofore been considered virtually nonpathogenic. Previously, many were regarded as contaminants when isolated from clinical specimens. Timpe and Runyon established that these organisms could cause disease in humans and classified them based on pigment production, growth rate, and colonial characteristics. Photochromogens (group I) grow slowly on culture media (>7 days). Their colonies change from a buff shade to bright yellow or orange after exposure to light. Scotochromogens (group II) also grow slowly but demonstrate pigmented colonies when incubated in the dark. Group III mycobacteria grow slowly and lack pigment in the dark or light. Rapid growers (group IV) also lack pigment, but they grow in culture within 3 to 5 days. Collectively, these four groups have been called the “atypical mycobacteria,” NTM, mycobacteria other than tubercle bacilli, or “potentially pathogenic environmental mycobacteria.” Molecular techniques such as DNA probes, real-time PCR, and gene amplification and restriction length polymorphism are useful tools for rapid identification of NTM (5–7).
EPIDEMIOLOGY
Ubiquitous in nature, many NTM have been isolated from ground or tap water, soil, house dust, domestic and wild animals, and birds. Despite their wide distribution, some species are more common in certain geographic locations. Most infections, including those that are hospital acquired, result from inhalation or direct inoculation from environmental sources. Ingestion may be the source of infection for children with NTM cervical adenopathy and for patients with AIDS whose disseminated infection may begin in the gastrointestinal tract. These infections are not considered contagious, since person-to-person transmission is undocumented.

PATHOPHYSIOLOGY
The pathogenic potentials for human disease vary among NTM. As a group, these organisms are less virulent for humans than Mycobacterium tuberculosis or M. bovis and may colonize body surfaces or secretions without causing disease. However, because of reports of invasive disease with such organisms, all mycobacteria should be considered potentially pathogenic. This is especially true when they are isolated from patients with immunocompromising conditions such as AIDS, cystic fibrosis, inhaled steroid therapy, hematologic malignancies, hematopoietic stem cell transplantation, and solid-organ transplantation. Patients receiving antitumor necrosis factor alpha are at increased risk for mycobacterial infections. Other immunologic deficits, such as genetic defects, and other structural lung diseases, such as bronchiectasis, may be associated with disease. In general, disease is slowly progressive, and histopathologic findings resemble those seen in tuberculosis.

DIAGNOSIS
The steps taken to diagnose tuberculosis generally apply to NTM infections. Standardized, specific skin test antigens for NTM, however, are unavailable. In addition, colonization of asymptomatic individuals and environmental contamination of specimens can yield positive cultures in the absence of clinical infection. Experts have suggested clinical and microbiologic criteria for diagnosis of pulmonary NTM infection. Clinical criteria include, in addition to exclusion of other diagnoses, “pulmonary symptoms, nodular or cavitary opacities on chest radiograph, or a high-resolution computed tomography scan that shows multifocal bronchiectasis with multiple small nodules.” Findings needed to meet microbiologic criteria include positive cultures from two expectorated sputum samples or a positive culture from one bronchial wash or lavage fluid. An alternative microbiologic criterion is the combination of a positive culture and characteristic histopathology. However, for less common pathogens, such as those discussed in this chapter, the pertinence of these criteria is only “assumed, not proven.” Review of 28 specimens from which Mycobacterium simiae was isolated, indeed, revealed patients who met accepted criteria for diagnosis yet who remained clinically stable without treatment.

Extrapulmonary or disseminated disease is confirmed by isolation of the organism from normally sterile body fluids, closed sites, or lesions, with exclusion of environmental contamination of specimens. Owing to the possibility of infection due to fastidious mycobacteria known to cause infections in skin, joints, and bone, specimens from these sources require supplemented media and lower incubation temperatures. Radiometric culture systems, DNA probes, and PCR assays have increased the speed and accuracy of laboratory diagnosis of pulmonary and extrapulmonary mycobacterial infections, but susceptibility testing is not standardized for all NTM species.

CLINICAL DISEASE
Slowly growing NTM cause a broad spectrum of diseases. It should be noted that therapeutic approaches continue to evolve and therefore remain controversial. Many conventional antituberculosis agents have little or no activity against these organisms. Some treatment regimens contain new agents or older antimicrobials newly found to have activity against mycobacteria. Although general guidelines exist for the therapy of infections caused by some of these organisms, in most cases, the optimal regimen or duration of therapy has not been firmly established. The results of in vitro susceptibility testing may be used to select a therapeutic regimen. Many experts recommend a macrolide-containing regimen with companion drugs such as rifampin and ethambutol for most, but not all, slowly growing species. Immunocompetent patients with clinically significant NTM infections usually should receive 18 to 24 months of therapy. Infected immunocompromised patients, particularly those with disseminated infection and AIDS, probably should receive therapy as long as their immune systems remain impaired.
**TABLE 1** Slow-growing nontuberculous mycobacterial infection sites and etiologic species

<table>
<thead>
<tr>
<th>Site of infection</th>
<th>Most common</th>
<th>Less frequent</th>
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<tbody>
<tr>
<td>Skin or soft tissue</td>
<td><em>M. marinum</em> (I), <em>M. ulcerans</em> (III)</td>
<td><em>M. avium</em> complex (III), <em>M. celatum</em> (II), <em>M. gordonae</em> (II), <em>M. haemophilum</em> (III), <em>M. kansasii</em> (I), <em>M. malmoense</em> (III), <em>M. szulgai</em> (I/II), <em>M. terrae</em> complex (III)</td>
</tr>
<tr>
<td>Bones and joints</td>
<td><em>M. marinum</em> (I), <em>M. terrae</em> complex (III), <em>M. ulcerans</em> (III)</td>
<td><em>M. branderi</em> (III), <em>M. haemophilum</em> (III), <em>M. lacus</em> (III), <em>M. lentiflavum</em> (II), <em>M. simiae</em> complex (I), <em>M. szulgai</em> (I/II), <em>M. xenopi</em> (II)</td>
</tr>
<tr>
<td>Central nervous system</td>
<td><em>M. kansasii</em> (I), <em>M. avium</em> complex (III)</td>
<td><em>M. malmoense</em> (III), <em>M. gordonae</em>, <em>M. haemophilum</em> (III), <em>M. simiae</em> complex (I), <em>M. terrae</em> complex (III)</td>
</tr>
<tr>
<td>Eye</td>
<td><em>M. avium</em> complex (III), <em>M. szulgai</em> (I/II)</td>
<td><em>M. gordonae</em> (I), <em>M. haemophilum</em> (III), <em>M. kansasii</em> (I)</td>
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*Note: I, photochromogen; II, scotochromogen; III, nonpigmented.*

**Mycobacterium alsiense**

*M. alsiense* is a slowly growing scotochromogen that was first isolated from pulmonary specimens from two patients, one from Denmark and one from Italy (18). Its phylogenetic position is unclear. It shares the 16S rRNA gene with *M. gordonae* and *M. asiaticum* but also is genetically similar to additional Mycobacterium species (3). The two reported strains were isolated from elderly immunocompetent individuals who had pulmonary disease. Treatment with rifampin (combined with ethambutol in one case and clarithromycin in the other) resulted in radiographic improvement in both patients (18).

**Mycobacterium celatum**

*M. celatum* is a slowly growing, nonchromogenic species with biochemical and morphologic characteristics resembling those of *M. avium-M. intracellulare* and *M. xenopi*. It has been reported as a cause of pulmonary and disseminated infections in patients with AIDS (19–21) or primary immunodeficiency (22). It rarely causes infections in immunocompetent patients (23). *M. celatum* isolates have shown variable susceptibilities to antimicrobial agents, suggesting that different groups of strains may represent separate clones (24). Most isolates are resistant to rifampin. Treatment regimens have included various combinations of clarithromycin, ciprofloxacin, pyrazinamide, ethambutol, rifabutin, clofazimine, and amikacin.

**Mycobacterium gordonae**

Also known as the “tap water bacillus,” the scotochromogen *M. gordonae* (formerly *Mycobacterium aquae*) is ubiquitous in the environment. It is commonly isolated from soil and water sources, including tap water. Its presence in water accounts for its association with numerous nosocomial pseudoinfections and pseudoepidemics (25–29). A molecular probe is commercially available which consists of DNA specific for a sequence of 16S rRNA (2). *M. gordonae* has long been considered among the least pathogenic mycobacteria, and its presence in clinical cultures is commonly attributed to specimen contamination or even host colonization. In a review of the literature, Weinberger et al. (28) concluded that 24 published cases met their criteria for a documented infection caused by *M. gordonae*. These included five cases of disseminated disease with pulmonary and hepatic involvement in four patients, bone marrow involvement in three, and renal and central nervous system involvement in two each. Four of these five patients had no underlying immunodeficiency, and one had AIDS. Localized sites of infection in the remaining 19 patients included the lung (eight cases), soft tissue (seven cases), peritoneum (three cases), and cornea (one case) (28). Rare cases of meningitis have been reported, some of which were associated with ventriculoatrial shunt infections (4, 30). A number of reports suggest that *M. gordonae* can not only colonize but also cause disease in patients with AIDS (31–33). Optimal therapy for a documented *M. gordonae* infection remains undefined. The majority of isolates tested have been resistant in vitro to isoniazid and pyrazinamide, whereas many are susceptible to ethambutol, rifampin, clarithromycin, linezolid, and the fluoroquinolones (2).
Mycobacterium haemophilum

*M. haemophilum*, the “blood-loving” mycobacterium, is nonpigmented, fastidious, and slow growing. It grows optimally at 30°C, a lower temperature than that preferred by most mycobacteria. It requires hemin or ferric ammonium citrate for growth (34). Initially identified as the cause of cutaneous ulcerating lesions in an Israeli woman with Hodgkin’s disease (35), *M. haemophilum* has been isolated from patients worldwide (34, 36, 37). Many cases have involved patients who are immunocompromised by conditions such as organ or bone marrow transplantation, lymphoma, or AIDS (38). Reports have described cases in immunocompetent children (39). Disease can be focal or widespread. Cutaneous and subcutaneous lesions are most common. They frequently overlie joints and can be nodular, cystic, or papular. Typically, they evolve from papules to pustules and can form deep ulcers that may be painful. Skin findings have been misdiagnosed as leprosy (40). Other infections have included bacteremia, septic arthritis, osteomyelitis, pneumonitis, sinusitis, endophthalmitis, and lymphadenitis (34, 36, 41). There are no standard guidelines for treatment of *M. haemophilum* infections. However, most experts recommend some combination of clarithromycin, ciprofloxacin, and a rifamycin for a duration of 12 to 24 months (34). Immunocompromised adults with multiple cutaneous lesions or osteomyelitis have been successfully treated with regimens that included ciprofloxacin, clarithromycin, and rifampin (42–44). The addition of granulocyte-macrophage colony-stimulating factor along with a reduction in immunosuppressive treatment was associated with successful treatment of systemic disease in a cardiac transplant patient (41). Immunocompetent children with localized lymphadenitis may do well with excisional therapy alone (36).

Mycobacterium kyorinense

Formerly called *M. celatum* type 2, *M. kyorinense* is now recognized as a novel, slow-growing nonchromogenic species (45). Like *M. celatum*, *M. kyorinense* is characterized by two copies of the 16S rRNA gene. This is an exceptional condition among the slow growers, since they typically contain one copy (5). Reported cases have involved pulmonary infections in immunocompetent patients from Japan (46), Brazil (47), and Australia (48), the last report describing one patient with cavitary pulmonary pneumonia (48). The species is susceptible *in vitro* to amikacin, clarithromycin, and fluoroquinolones but is resistant to rifampin. Successful therapy has been reported with clarithromycin and levofloxacin (5, 46).

Mycobacterium malmoense

*M. malmoense*, first described in 1977, is nonchromogenic and slow growing, often requiring at least 6 weeks for primary isolation. It appears to be distributed worldwide and has been isolated from natural waters in Finland and soil in Japan. This organism was initially linked to chronic pulmonary infection in British and northern European adults who had underlying lung disease (49). An association with coal workers’ pneumoconiosis has been described (50). Reports of extrapulmonary disease have described cervical lymphadenopathy, particularly in children, and tenosynovitis (51, 52). Disseminated infection with involvement of the skin, gastrointestinal tract, or lymph nodes has been reported for patients with leukemia or AIDS (52). Most isolates are susceptible to ethambutol, and many are susceptible to rifampin and streptomycin. A regimen effective against *M. avium-M. intracellulare* has been recommended for initial therapy (2). A 2-year triple-drug regimen that included rifampicin, ethambutol, and high-dose clarithromycin (or ciprofloxacin) was well tolerated and successfully eradicated the organism at 6 months after completion of therapy in 14 patients with pulmonary infections (53). A patient with AIDS and disseminated infection was treated successfully with rifabutin, clofazimine, and isoniazid (52). Success in three cases was reported with rifampin, ethambutol, and clarithromycin (54).

Mycobacterium simiae Complex

The *M. simiae* complex is comprised of numerous species, including *M. simiae*, *M. europaeum*, *M. florentinum*, *M. genavense*, *M. heidelbergense*, *M. interjectum*, *M. intermedium*, *M. kubicae*, *M. lentiflavum*, *M. monteforense*, *M. palustre*, *M. parascrofulaceum*, *M. parvum*, *M. saskatchewanense*, *M. sherrisii*, *M. shigaense*, *M. stomatopae*, *M. tilburgii*, and *M. triplex* (5). *M. simiae* was first isolated from a colony of *Macaca* (rhesus) monkeys (35). A slowly growing photochromogen, its colonies may be only weakly pigmented even after prolonged exposure to light. Unlike other NTM, it produces niacin and thus may be confused with *M. tuberculosis*. Unlike all other known mycobacteria, *M. simiae* contains 16S rRNA sequences that are similar to those found both in slowly growing mycobacteria and in rapidly growing strains. The organism has been isolated from human feces (56) and also from water (57), which has been implicated in pseudo-outbreaks (2). Members of the *M. simiae* complex can colonize the respiratory tract, and the lung is the most commonly reported site of infection. Most cases of pulmonary
Other Slow-Growing Nontuberculous Mycobacteria

Mycobacterium szulgai

M. szulgai is scotochromogenic at 37°C but photochromogenic at 25°C. The organism has been isolated from patients worldwide, but its presumed environmental source has not been identified (63). Most reported cases have involved lung disease indistinguishable from that caused by M. tuberculosis. Other sites of infection have included the bursa, tendon sheaths, bones, lymph nodes, skin, and urinary tract (66–68). M. szulgai is susceptible to most antituberculosis agents in vitro (2). Therapeutic regimens with combinations of two or more drugs, including a macrolide, and 12 months of sputum negativity have been successful (2, 4, 65). A patient with AIDS who had pulmonary infection caused by multidrug-resistant M. szulgai responded to therapy with isoniazid, ethambutol, rifampin, and pyrazinamide. The isolate was resistant in vitro to isoniazid, kanamycin, capreomycin, and cycloserine but susceptible to ethambutol, rifampin, and ciprofloxacin (69). A study performed in the Netherlands found that 12 months of rifampin, ethambutol, and clarithromycin led to favorable outcomes without bacteriological relapse (70). Combination therapy based on in vitro susceptibilities for at least 4 to 6 months is recommended for extrapulmonary M. szulgai disease (2).

Mycobacterium terrae Complex

Members of the Mycobacterium terrae complex, including M. terrae, M. algericum, M. arupense, M. engbaekii, M. heraklionense, M. hiberniae, M. kumamotonense, M. longobardum, M. minnesotaense, M. nonchromogenicum, M. paraterrae, M. senuense, and M. sinense (5), are slow-growing and, with few exceptions, nonchromogenic mycobacteria. Within the M. terrae complex, M. arupense and M. kumamotonense are the most frequently isolated species, while M. terrae (which was originally confused with M. arupense) is very rare (71). M. paraterrae, a slowly growing scotochromogenic, is genetically related to M. terrae complex (72). Reports have associated these interrelated species with pulmonary infection (73–77) and bone and joint infections (78–81). M. nonchromogenicum should be considered in patients who present with tenosynovitis of the hand that is refractory to routine antibiotics and exacerbated by steroid therapy (82). Although members of the Mycobacterium terrae complex are often resistant to many conventional antituberculosis agents, susceptibility patterns of isolates vary within and between species. Individual isolates may be susceptible to ethambutol, rifabutin, macrolides, linezolid, sulfonamides, or fluoroquinolones (2, 4). Excision with or without antimicrobial therapy may be curative for cutaneous infections due to M. nonchromogenicum.

Mycobacterium ulcerans

M. ulcerans is a slowly growing, unpigmented mycobacterium that grows best at 25 to 33°C, requiring 8 to 12 weeks of incubation and optimally recovered with egg yolk supplementation (2). It produces a heat-stable toxin and is the cause of a chronic necrotizing skin infection (Bairdsdale ulcer or Buruli ulcer). M. ulcerans infection was first described in Australia but subsequently has been reported in central and west Africa, Mexico, Central and South America, Southeast Asia, and the central Pacific. Analysis of 16S rRNA sequences of isolates from three continents revealed three subgroups corresponding to the continent of origin (83). Although unproven, it is a generally accepted assumption that the environment is the source of the organism. The majority of reported infections have occurred in persons living near rivers or stagnant bodies of water. Inoculation appears to occur via trauma to the skin. The trauma may be minor and unrecognized by the patient. Infection has followed a snakebite (84) or human bite (85), gunshot wound, and vaccination (86).

Most lesions occur on the distal parts of a limb and typically begin as a painless papule or subcutaneous swelling. In several weeks, the lesion becomes a shallow ulcer with a necrotic base and undermined margins. Prominent involvement of subcutaneous tissue follows, and satellite ulcers and nodules can develop. Ulcers vary in severity and size and may involve joints. Healed lesions leave stellate scars with retraction, and patients with large ulcers may have permanent deformity and disability. Most lesions are widely ulcerated when they are detected and require extensive surgical excision and skin grafting.

The pathogenesis of the ulcer is associated with the production of mycolactone, a diffusible polyketide cytotoxin with immunomodulating properties (87).
Patients with active ulcers have impaired production of Th1, Th2, and Th17 cytokines upon stimulation with mitogenic agents. These immunological defects are detected early in the disease and resolve after successful therapy (88).

Early diagnosis and treatment improve outcome. Clumps of acid-fast bacilli are usually visible in material taken from skin lesions, but primary culture of *M. ulcerans* may take several months. The efficacy of antimicrobials has been disappointing, particularly when they are used late in the course of disease. They may be helpful early or when coupled with surgical excision. Regimens have included streptomycin and dapson with or without ethambutol and various combinations of trimethoprim-sulfamethoxazole, rifampin, ethambutol, and clarithromycin (89). Isoniazid, rifampin, and ethambutol for 2 months followed by rifampin and clarithromycin for 5 months was successful therapy in a human immunodeficiency virus-infected patient who had a cutaneous ulcer with infection extending to the underlying fascia (90). An open-label randomized trial conducted in Ghana compared two regimens for early, limited *M. ulcerans* infections (91). In this study, 73 of 76 patients who received streptomycin and rifampin for 8 weeks and 68 of 75 patients who received 4 weeks of streptomycin and rifampin followed by 4 weeks of rifampin and clarithromycin had healed lesions at 1 year after the start of treatment. A subsequent study in Ghana monitored clinical and microbiological responses to therapy with rifampin and streptomycin for 2 weeks followed by rifampin plus clarithromycin for 6 weeks in patients with Buruli lesions of less than 15 cm in maximum diameter. Treatment was successful, with complete healing in 93% of patients, and there were no recurrences within 1 year of follow-up (92). Preventive efforts may help reduce the incidence of disease. Wearing long pants appeared to be protective in a case-control study involving Côte d’Ivoire patients who had lower-extremity lesions (93).

*Mycobacterium xenopi*

*M. xenopi*, first isolated from a toad, is a scotochromogen that grows optimally at 43°C. The organism is able to grow at 45°C and has been recovered from hot-water generators and storage tanks. It is frequently found in both cold and hot water samples from taps and showers. Failure to grow at temperatures below 28°C likely explains its absence in samples from water treatment plants, reservoirs, and distribution systems. A cluster of bronchoscopy-associated *M. xenopi* pseudo-infections was linked to use of tap water for cleaning bronchoscopes (94). Human exposure to the organism can occur via aerosolization and inhalation or ingestion. *M. xenopi* appears to have a variable geographic distribution. It has been recovered frequently from clinical specimens in Wales, southern England, the northwest coast of Europe, and Toronto, Canada. It was rarely isolated in the United States prior to the AIDS epidemic (1). Detection of *M. xenopi* in clinical specimens may require prolonged incubation at 37°C or incubation at higher temperatures.

*M. xenopi* is increasingly recognized as a cause of pulmonary infection. In immunocompetent patients, clinical illness typically occurs as an indolent, often cavitary, lung infection in middle-aged men who have underlying chronic pulmonary diseases (95–97). Less commonly it may infect the spine (98) or joints (99). In a series of seven cases of arthritis, all patients had a history of invasive procedures, and the authors noted that an earlier outbreak of 58 cases of spinal infection was attributed to contamination of an instrument with tap water (100). *M. xenopi* infections in immunocompromised patients are being reported more frequently. Solid-organ transplantation (101, 102) and AIDS increase the risk of pulmonary and disseminated disease (95, 103, 104). Infections with *M. xenopi* have shown variable responses to drug therapy (105). Recommendations for initial therapy include isoniazid, a rifamycin, ethambutol, and clarithromycin with or without an initial course of streptomycin or the use of moxifloxacin (2, 4). Pyrazinamide and ciprofloxacin have been included in some successful regimens (95, 100).

**Rare NTM Pathogens**

A growing number of other uncommon slow-growing NTM are being isolated from clinical specimens. Among them are *M. arosiense*, *M. asiaticum*, *M. bohemicum*, *M. bouchedurhonense*, *M. branderi*, *M. conspicuum*, *M. europaeum*, *M. fragae*, *M. gastrae*, *M. heidelbergense*, *M. insubricum*, *M. intermedium*, *M. koreense*, *M. lentiflavum*, *M. mantenii*, *M. marseillense*, *M. noviomagense*, *M. nebraskense*, *M. paraffinicum*, *M. parafortuitum*, *M. paraseudolense*, *M. phlei*, *M. riyadhense*, *M. seoulense*, *M. shinjukuense*, *M. shimoidei*, *M. sitophilum*, *M. thermoresistibile*, *M. timonense*, *M. tusciae*, *M. vulneris*, and *M. yongonense* (3).

Previously considered nonpathogenic saprophytes or environmental contaminants, many of these strains have been implicated as rare causes of pulmonary, extrapulmonary, or disseminated infections (1, 3). We can expect to see an increase in the clinical significance...
of these NTM, particularly in patients who have AIDS or other conditions which diminish host defenses.

REFERENCES


Other Slow-Growing Nontuberculous Mycobacteria


