ANTIMICROBIAL AGENTS AND CHEMOTHERAPY, Dec. 1996, p. 2874–2878 Vol. 40, No. 12
0066-4804/96/$04.00+0

NOTES

Antimicrobial Susceptibility Patterns of Some Recently Established Coryneform Bacteria

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Received 17 January 1996/Returned for modification 26 April 1996/Accepted 22 September 1996

The susceptibility patterns of 480 isolates representing six recently defined species of coryneform bacteria (*Corynebacterium amycolatum* [*n* = 101], *Corynebacterium auris* [*n* = 48], *Corynebacterium glucuronolyticum* [*n* = 86], *Brevibacterium casei* [*n* = 50], *Dermabacter hominis* [*n* = 49], and *Turicella otitidis* [*n* = 146]) to 17 antimicrobial agents were determined by an agar dilution method. Most significantly, for *C. amycolatum* strains the MICs at which 90% of isolates are inhibited were ≥32 μg/ml for nearly all agents. However, all 480 strains examined were susceptible to glycopeptide antibiotics.

Coryneform bacteria have been recognized with increasing frequency as opportunistic pathogens in recent years (6). Apart from this, many new species have been described as a result of the increasing number of taxonomic investigations with this heterogeneous group of organisms. However, data on the antimicrobial susceptibility patterns of recently described species (or species recognized as clinically significant) are very scanty. The Department of Medical Microbiology at the University of Zürich has, over the last few years, collected a large number of isolates belonging to some recently defined species of coryneform bacteria. The intention of the present study was to provide comprehensive antimicrobial susceptibility data for six species for the first time.

Brevibacterium casei was defined in 1983 by Collins et al. (5) but was only shown in 1994 to represent the majority of *Brevibacterium* strains isolated from clinical specimens (9). *Corynebacterium amycolatum* (the only true *Corynebacterium* species lacking mycolic acids) was established in 1988 (4), and in 1993, Barreau and coworkers (1) suggested that some strains identified as *Corynebacterium striatum*, *Corynebacterium minutissimum*, and CDC coryneform group F-2 and I-2 bacteria in the routine clinical laboratory actually represent misidentified *C. amycolatum* strains. *Dermabacter hominis* was also established in 1988 (16) and was shown in 1994 (13) to comprise the former CDC coryneform group 3 and 5 bacteria. In 1994, *Turicella otitidis* was described from patients with otitis media (12), as was *Corynebacterium auris* in 1995 (11). Finally, *Corynebacterium glucuronolyticum* was isolated from male patients with genitourinary infections, and its description appeared in the literature in 1995 (8, 15).

The 480 isolates used in this study were collected by the Department of Medical Microbiology at the University of Zürich between 1990 and 1995, but mainly between 1993 and 1995. The isolates were identified by published methods (4, 8–13). About 120 of the isolates studied were referred to our institution for identification from different laboratories located throughout Europe and North America. From the available data, we had no indication that the patients' isolates were epidemiologically linked. All strains were kept in 10% skim milk at −70°C until further use.

The antibiotics used were kindly provided as powders for in vitro studies by the following companies in Switzerland: amoxicillin-clavulanic acid and ampicillin (SmithKline Beecham, Thörishaus), ceftriaxone (Hoffmann-LaRoche, Basel), cefuroxime sodium (Glaxo, Schönbühl), cephalothin, erythromycin, and vancomycin (Lilly, Bern), chloramphenicol (Parke-Davis, Baar), ciprofloxacin and oxacillin (Bayer, Zürich), clindamycin (Upjohn, Brüttisellen), fosfomycin (Boehringer Mannheim, Rotkreuz), imipenem (Merck Sharpe & Dohme, Glattbrugg), penicillin G (Hoechst, Zürich), rifampin (Ciba-Geigy, Basel), teicoplanin (Marion Merrell Dow, Horgen), and tetracycline (Lederle, Adliswil). Gentamicin was purchased from Sigma Chemical Co. (St. Louis, Mo.). All antibiotics were dissolved and diluted as recommended by the guidelines of the National Committee for Clinical Laboratory Standards (NCCLS) (20).

The MICs were determined by an agar dilution method (20) with Mueller-Hinton agar supplemented with 5% sheep blood. About 10^4 CFU per strain was placed on the surfaces of the plates with a 96-point inoculator (Dynatech, Embrach, Switzerland). Incubation was carried out at 35°C in ambient air for 24 h. *Staphylococcus aureus* ATCC 29213 and *Enterococcus faecalis* ATCC 29212 served as controls. MICs were defined by no visible growth after 24 h. "Breakpoints" for susceptibility were defined as follows: ceftriaxone, ≤8 μg/ml; cefuroxime sodium, ≤8 μg/ml; cephalothin, ≤8 μg/ml; chloramphenicol, ≤8 μg/ml; ciprofloxacin, ≤1 μg/ml; clindamycin, ≤0.5 μg/ml; erythromycin, ≤0.5 μg/ml; gentamicin, ≤4 μg/ml; imipenem, ≤4 μg/ml; rifampin, ≤1 μg/ml; teicoplanin, ≤8 μg/ml; tetracycline, ≤4 μg/ml; and vancomycin, ≤4 μg/ml. It is, however, important to note that the breakpoints applied are similar to NCCLS values (21) but that breakpoints have not been defined by NCCLS to include coryneform bacteria. By a pragmatic approach, breakpoints for staphylococci (21) were applied to amoxicillin-clavulanic acid (≤4/2 μg/ml), ampicillin (≤0.25 μg/ml), penicillin (≤0.12 μg/ml), and oxacillin (≤2 μg/ml).

The results of the susceptibility testing (i.e., 8,160 MIC de-

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TABLE 1. MICs of 17 antimicrobial agents for the six recently defined coryneform bacteria

Organism (no. of isolates)	Antimicrobial agent	MIC (μg/ml)		
		Range	50%	90%
C. amycolatum (101)	Amoxicillin-clavulanic acid	0.06–>64	4	>64
	Ampicillin	0.06–>64	4	>64
	Ceftriaxone	0.125–>64	1	>64
	Cefuroxime sodium	0.125–>64	0.5	>64
	Cephalothin	0.06–>64	0.25	>64
	Chloramphenicol	1–64	16	32
	Ciprofloxacin	≤0.03–>64	4	>64
	Clindamycin	0.125–>64	>64	>64
	Erythromycin	≤0.03–>64	>64	>64
	Gentamicin	0.06–>64	0.25	32
	Imipenem	≤0.03–>64	0.5	>64
	Oxacillin	0.25–>64	8	>64
	Penicillin G	0.06–>64	0.25	>64
	Rifampin	≤0.03–>64	≤0.03	>64
	Teicoplanin	0.125–1	0.25	0.5
	Tetracycline	0.25–>64	0.5	2
	Vancomycin	0.125–0.5	0.25	0.25
C. auris (48)	Amoxicillin-clavulanic acid	0.5–2	1	2
	Ampicillin	1–4	2	4
	Ceftriaxone	4–16	8	16
	Cefuroxime sodium	0.5–2	1	2
	Cephalothin	0.125–0.5	0.25	0.5
	Chloramphenicol	1–4	2	4
	Ciprofloxacin	≤0.03–0.25	0.06	0.125
	Clindamycin	0.06–>64	0.5	>64
	Erythromycin	≤0.03–>64	0.5	>64
	Gentamicin	≤0.03–1	0.125	0.25
	Imipenem	0.125–1	0.25	1
	Oxacillin	4–32	16	32
	Penicillin G	0.5–2	1	2
	Rifampin	≤0.03–0.06	≤0.03	0.06
	Teicoplanin	0.125–0.25	0.125	0.25
	Tetracycline	0.125–1	0.5	1
	Vancomycin	0.125–0.25	0.125	0.25
C. glucuronolyticum (86)	Amoxicillin-clavulanic acid	≤0.03–0.25	0.06	0.06
	Ampicillin	≤0.03–0.25	0.06	0.125
	Ceftriaxone	≤0.03–4	0.25	2
	Cefuroxime sodium	0.06–1	0.125	0.5
	Cephalothin	≤0.03–1	0.06	0.125
	Chloramphenicol	0.06–8	2	4
	Ciprofloxacin	0.06–16	0.25	8
	Clindamycin	≤0.03–>64	2	>64
	Erythromycin	≤0.03–>64	0.25	16
	Gentamicin	≤0.03–8	0.06	1
	Imipenem	≤0.03–0.5	0.06	0.125
	Oxacillin	≤0.03–4	0.25	1
	Penicillin G	≤0.03–0.25	0.06	0.125
	Rifampin	≤0.03	≤0.03	≤0.03
	Teicoplanin	0.125–0.5	0.5	0.5
	Tetracycline	0.5–64	32	32
	Vancomycin	0.06–0.25	0.25	0.25
B. casei (50)	Amoxicillin-clavulanic acid	4–32	8	16
	Ampicillin	4–16	8	8
	Ceftriaxone	0.5–32	4	8
	Cefuroxime sodium	2–16	4	16
	Cephalothin	2–16	8	16
	Chloramphenicol	2–64	32	32
	Ciprofloxacin	0.5–4	2	2
	Clindamycin	0.06–8	4	4
	Erythromycin	0.125–16	2	8
	Gentamicin	0.25–4	0.5	1
	Imipenem	0.5–16	2	8
	Oxacillin	16–64	16	32
	Penicillin G	1–4	1	2

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TABLE 1—*Continued*

Organism (no. of isolates)	Antimicrobial agent	MIC (μg/ml)		
		Range	50%	90%
	Rifampin	≤0.03	≤0.03	≤0.03
	Teicoplanin	0.25–1	0.5	1
	Tetracycline	0.125–1	0.5	1
	Vancomycin	0.125–0.5	0.25	0.25
D. hominis (49)	Amoxicillin-clavulanic acid	≤0.03–4	0.5	4
	Ampicillin	≤0.03–4	0.5	2
	Ceftriaxone	≤0.03–8	0.5	4
	Cefuroxime sodium	≤0.03–8	0.25	4
	Cephalothin	≤0.03–1	0.125	1
	Chloramphenicol	0.5–32	2	32
	Ciprofloxacin	0.25–64	2	4
	Clindamycin	≤0.03–>64	0.25	>64
	Erythromycin	≤0.03–>64	1	>64
	Gentamicin	0.5–64	1	8
	Imipenem	≤0.03–4	0.5	2
	Oxacillin	0.5–32	2	16
	Penicillin G	0.06–4	0.25	2
	Rifampin	≤0.03–32	≤0.03	≤0.03
	Teicoplanin	0.06–0.25	0.06	0.125
	Tetracycline	0.5–32	2	16
	Vancomycin	0.125–0.5	0.25	0.5
T. otitidis (146)	Amoxicillin-clavulanic acid	≤0.03	≤0.03	≤0.03
	Ampicillin	≤0.03	≤0.03	≤0.03
	Ceftriaxone	≤0.03–0.25	0.125	0.25
	Cefuroxime sodium	≤0.03–0.125	0.06	0.125
	Cephalothin	≤0.03	≤0.03	≤0.03
	Chloramphenicol	0.25–2	1	2
	Ciprofloxacin	0.06–0.25	0.125	0.125
	Clindamycin	≤0.03–>64	0.125	32
	Erythromycin	≤0.03–>64	≤0.03	>64
	Gentamicin	≤0.03	≤0.03	≤0.03
	Imipenem	≤0.03	≤0.03	≤0.03
	Oxacillin	≤0.03–0.5	0.125	0.25
	Penicillin G	≤0.03	≤0.03	≤0.03
	Rifampin	≤0.03	≤0.03	≤0.03
	Teicoplanin	0.125–1	0.25	0.5
	Tetracycline	≤0.03–1	0.25	0.25
	Vancomycin	0.125–0.5	0.25	0.5

terminations) are summarized in Table 1. All MICs could usually be read without difficulty after 24 h of incubation. Only for *C. amycolatum* isolates that were resistant to β-lactam antibiotics (MIC, >64 μg/ml) was growth more easily observed after 48 h; nevertheless, the MICs for these isolates were also readable after 24 h. The MICs for the two control strains were within the accepted ranges for each of the antimicrobial agents.

For all strains tested the MICs of both teicoplanin and vancomycin were ≤1 μg/ml. The MICs of teicoplanin tended to be 1 twofold dilution higher than those of vancomycin.

About 40% of the *C. amycolatum* isolates were resistant to β-lactam antibiotics (MICs, >64 μg/ml). The MICs of chloramphenicol, ciprofloxacin, clindamycin, and erythromycin, at which 50% of isolates are inhibited (MIC_{50}s) were above the applied susceptibility breakpoints, as were the MIC_{90}s of gentamicin and rifampin. Tetracycline, however, showed good activity against the *C. amycolatum* isolates.

All *C. auris* isolates could be considered resistant to penicillins. The MICs of cephalothin were lower than those of ceftriaxone. All *C. auris* isolates could be classified as susceptible to chloramphenicol, ciprofloxacin, gentamicin, rifampin, and tetracycline, whereas for about 30% of the isolates the MICs of either clindamycin or erythromycin were >64 μg/ml (most often in parallel).

For the *C. glucuronolyticum* isolates the MICs of all β-lactam antibiotics were relatively low. All isolates were susceptible to chloramphenicol (MIC, ≤4 μg/ml). Rifampin showed excellent activity against *C. glucuronolyticum* isolates, whereas the MIC_{50} of tetracycline was 32 μg/ml. The MICs of ciprofloxacin, clindamycin, and erythromycin for *C. glucuronolyticum* strains covered a broad range.

The *B. casei* isolates exhibited decreased susceptibilities to all β-lactam antibiotics tested (i.e., MIC_{50}s, ≥1 μg/ml) as well as to nearly all other antimicrobial agents except gentamicin, rifampin, and tetracycline.

The MICs of cephalosporins were ≤8 μg/ml for *D. hominis* strains, whereas the MIC_{90}s of the penicillins were close to the applied levels for resistance. The activities of chloramphenicol, ciprofloxacin, clindamycin, erythromycin, gentamicin, and tetracycline against *D. hominis* were also limited, as revealed by their MIC_{90}s. Again, rifampin showed excellent activity.

The MICs of the β-lactams were extremely low for the *T. otitidis* isolates tested. For all of these strains the MICs of

chloramphenicol (≤2 μg/ml), ciprofloxacin (≤0.25 μg/ml), gentamicin (≤0.03 μg/ml), rifampin (≤0.03 μg/ml), and tetracycline (≤1 μg/ml) were very low. About 25% of all *T. otitidis* isolates were resistant to clindamycin and erythromycin (MICs, ≥32 μg/ml). Again, resistance to these two antimicrobial agents was, in almost every case, present in the same isolates.

It should be noted that the β-lactamase inhibitor clavulanic acid did not influence the MICs of any of the isolates resistant to penicillins.

For all isolates tested fosfomycin MICs were >64 μg/ml (2); however, for *D. hominis* isolates fosfomycin MICs were 16 to 64 μg/ml (data not shown).

No comprehensive data regarding the antimicrobial susceptibility patterns of the six species examined in this study could be found in the literature. The limited data given for 26 *C. amycolatum* isolates (10), 10 *C. auris* isolates (11), 17 *C. glucuronolyticum* isolates (8), 12 *Brevibacterium* spp. isolates (14), and 15 *D. hominis* isolates (13) were confirmed in the present study with a much larger number of strains. In contrast, no data on the MICs for *T. otitidis* have been published so far.

Multiresistance has been well documented in *C. jeikeium* and *C. urealyticum* strains (6, 25), and our data add *C. amycolatum* to the list of *Corynebacterium* species with reduced susceptibility to many antimicrobial agents. Moreover, in our experience, *C. amycolatum* is the most frequently isolated nonlipophilic *Corynebacterium* species encountered in clinical specimens. *C. amycolatum* strains may be misidentified as *C. striatum*, *C. minutissimum* (1), or *Corynebacterium xerosis* (10) if tests for the detection of mycolic acids are not performed; therefore, it is suggested that some of the multiresistant *C. striatum*, *C. minutissimum*, and *C. xerosis* strains reported in the literature (19, 25, 27–29) represent, in fact, *C. amycolatum* strains, because in our experience, true *C. striatum*, *C. minutissimum*, and *C. xerosis* strains are almost never multiresistant. Zapardiel et al. (31) reported difficulties in reading agar dilution MICs as well as Etest inhibition zones for some *C. xerosis* and CDC group F isolates, which may indicate that some of their isolates actually belonged to the species *C. amycolatum*, which may grow slowly in the case of β-lactam resistance (see above). The molecular basis for the resistance of *C. amycolatum* isolates to many antibiotics is not known at present.

B. casei isolates were also resistant to many antimicrobial agents, which has also been mentioned in case reports for a few unspecified *Brevibacterium* isolates (17, 18). Again, the molecular basis of the reduced susceptibility of *B. casei*, in particular to β-lactams, is not known, but it should be the subject of future investigations.

Oxacillin exhibited, in comparison with the other β-lactams, reduced activity against the six species tested, as has also been described for *Corynebacterium diphtheriae* and *Listeria monocytogenes* (3, 25). The most frequent resistance observed within all six species was to clindamycin and erythromycin. This type of resistance has also been demonstrated in *C. diphtheriae* (23) and *C. striatum* (22) strains which were carrying the *erm*Cd gene encoding an rRNA methylase (24). Tauch et al. (26) have recently demonstrated that the *erm*Cx gene derived from an R plasmid of a so-called "*C. xerosis*" isolate shared 99% homology with the *erm*Cd gene. In our opinion, it is not unlikely that, in particular, *C. auris* (and *T. otitidis*) isolates may also carry rRNA methylases, and this will be the subject of future investigations.

For nearly all isolates tested, the MICs of rifampin were either very low (≤0.03 μg/ml) or very high (>64 μg/ml) (25 isolates only), which may be explained by mutations significantly altering the β subunit of the bacterial DNA-dependent RNA polymerases (7). Resistance to fosfomycin in coryneform bacteria has been well documented before (25) and has served as a basis for its use in semiselective media for coryneform bacteria (30). However, the susceptibility of *D. hominis* to fosfomycin concentrations of ≥16 μg/ml should be kept in mind when semiselective media for coryneforms are used.

Penicillins are very useful antibiotics in the treatment of infections caused by coryneform organisms because the MICs of penicillins are very low for many strains. NCCLS does not provide specific breakpoints of penicillins for coryneform bacteria. However, it would be desirable to have specific breakpoints for susceptibility for coryneform bacteria (at least for this class of antimicrobial agents), e.g., in order to answer the question of whether penicillins might be used to treat infections caused by coryneform bacteria for which MICs are elevated (i.e., 0.5 to 2 μg/ml).

All strains were susceptible to glycopeptide antibiotics, which would justify their use as first-line drugs against serious infections caused by coryneform bacteria. However, because of the emergence of glycopeptide resistance in other gram-positive organisms, mainly in *Enterococcus* species, it would be prudent to alter the antibiotic regimen when the results of antimicrobial susceptibility testing become available in order to reduce the use of glycopeptides. Our data may facilitate the use of certain antibiotics in the empiric treatment of infections caused by the species included in this study.

This study was funded by the Hochschulverein, Zürich, Switzerland. G.F. acknowledges support by a grant from the Sassella-Stiftung, Zürich, Switzerland.

REFERENCES

1. **Barreau, C., F. Bimet, M. Kiredjian, N. Rouillon, and C. Bizet.** 1993. Comparative chemotaxonomic studies of mycolic acid-free coryneform bacteria of human origin. J. Clin. Microbiol. **31:**2085–2090.
2. **Barry, A. L., M. A. Pfaller, P. C. Fuchs, F. C. Tenover, L. B. Reller, S. D. Allen, D. J. Hardy, and E. H. Gerlach.** 1993. Interpretive criteria and quality control parameters for determining bacterial susceptibility to fosfomycin tromethamine. Eur. J. Clin. Microbiol. Infect. Dis. **12:**352–356.
3. **Chambers, H. F., and H. C. Neu.** 1995. Penicillins, p. 233–246. *In* G. L. Mandell, J. E. Bennett, and R. Dolin (ed.), Principles and practice of infectious diseases. Churchill Livingstone, New York.
4. **Collins, M. D., R. A. Burton, and D. Jones.** 1988. *Corynebacterium amycolatum* sp. nov. a new mycolic acid-less *Corynebacterium* species from human skin. FEMS Microbiol. Lett. **49:**349–352.
5. **Collins, M. D., J. A. E. Farrow, M. Goodfellow, and D. E. Minnikin.** 1983. *Brevibacterium casei* sp. nov. and *Brevibacterium epidermidis* sp. nov. Syst. Appl. Microbiol. **4:**388–395.
6. **Coyle, M. B., and B. A. Lipsky.** 1990. Coryneform bacteria in infectious diseases: clinical and laboratory aspects. Clin. Microbiol. Rev. **3:**227–246.
7. **Farr, B. M.** 1995. Rifamycins, p. 317–329. *In* G. L. Mandell, J. E. Bennett, and R. Dolin (ed.), Principles and practice of infectious diseases. Churchill Livingstone, New York.
8. **Funke, G., K. A. Bernard, C. Bucher, G. E. Pfyffer, and M. D. Collins.** 1995. *Corynebacterium glucuronolyticum* sp. nov. isolated from male patients with genitourinary infections. Med. Microbiol. Lett. **4:**204–215.
9. **Funke, G., and A. Carlotti.** 1994. Differentiation of *Brevibacterium* spp. encountered in clinical specimens. J. Clin. Microbiol. **32:**1729–1732.
10. **Funke, G., P. A. Lawson, K. A. Bernard, and M. D. Collins.** 1996. Most *Corynebacterium xerosis* strains identified in the routine clinical laboratory correspond to *Corynebacterium amycolatum*. J. Clin. Microbiol. **34:**1124–1128.
11. **Funke, G., P. A. Lawson, and M. D. Collins.** 1995. Heterogeneity within human-derived Centers for Disease Control and Prevention (CDC) coryneform group ANF-1-like bacteria and description of *Corynebacterium auris* sp. nov. Int. J. Syst. Bacteriol. **45:**735–739.
12. **Funke, G., S. Stubbs, M. Altwegg, A. Carlotti, and M. D. Collins.** 1994. *Turicella otitidis* gen. nov., sp. nov., a coryneform bacterium isolated from patients with otitis media. Int. J. Syst. Bacteriol. **44:**270–273.
13. **Funke, G., S. Stubbs, G. E. Pfyffer, M. Marchiani, and M. D. Collins.** 1994. Characteristics of CDC group 3 and group 5 coryneform bacteria isolated from clinical specimens and assignment to the genus *Dermabacter*. J. Clin. Microbiol. **32:**1223–1228.
14. **Gruner, E., G. E. Pfyffer, and A. von Graevenitz.** 1993. Characterization of

Brevibacterium spp. from clinical specimens. J. Clin. Microbiol. **31:**1408–1412.
15. **International Journal of Systematic Bacteriology.** 1995. Validation of the publication of new names and new combinations previously effectively published outside the IJSB: list no. 55. Int. J. Syst. Bacteriol. **45:**879–880.
16. **Jones, D., and M. D. Collins.** 1988. Taxonomic studies on some human cutaneous coryneform bacteria: description of *Dermabacter hominis* gen. nov., sp. nov. FEMS Microbiol. Lett. **51:**51–56.
17. **Kaukoranta-Tolvanen, S. S. E., A. Sivonen, A. A. I. Kostiala, P. Hormila, and M. Vaara.** 1995. Bacteremia caused by *Brevibacterium* species in an immunocompromised patient. Eur. J. Clin. Microbiol. Infect. Dis. **14:**801–804.
18. **Lina, B., A. Carlotti, V. Lesaint, Y. Devaux, J. Freney, and J. Fleurette.** 1994. Persistent bacteremia due to *Brevibacterium* species in an immunocompromised patient. Clin. Infect. Dis. **18:**487–488.
19. **Lortholary, O., A. Buu-Hoi, J. Y. Fagon, J. Pierre, M. Slama, L. Gutmann, and J. F. Acar.** 1993. Mediastinitis due to multiply resistant *Corynebacterium xerosis*. Clin. Infect. Dis. **16:**172.
20. **National Committee for Clinical Laboratory Standards.** 1993. Methods for dilution antimicrobial susceptibility tests for bacteria that grow aerobically, 3rd ed. Approved standard. NCCLS document M7-A3. National Committee for Clinical Laboratory Standards, Villanova, Pa.
21. **National Committee for Clinical Laboratory Standards.** 1993. Minimum inhibitory concentration (MIC) interpretive standards (μg/ml) for organisms other than *Haemophilus*, *Neisseria gonorrhoeae*, and *Streptococcus pneumoniae*. NCCLS document M7-A3. National Committee for Clinical Laboratory Standards, Villanova, Pa.
22. **Roberts, M. C., R. B. Leonard, A. Briselden, F. D. Schoenknecht, and M. B. Coyle.** 1992. Characterization of antibiotic-resistant *Corynebacterium striatum* strains. J. Antimicrob. Chemother. **30:**463–474.
23. **Schiller, J., N. Groman, and M. Coyle.** 1980. Plasmids in *Corynebacterium diphtheriae* and diphtheroids mediating erythromycin resistance. Antimicrob. Agents Chemother. **18:**814–821.
24. **Serwold-Davis, T. M., and N. B. Groman.** 1988. Identification of a methylase gene for erythromycin resistance within the sequence of a spontaneously deleting fragment of *Corynebacterium diphtheriae* plasmid pNG2. FEMS Microbiol. Lett. **56:**7–14.
25. **Soriano, F., J. Zapardiel, and E. Nieto.** 1995. Antimicrobial susceptibilities of *Corynebacterium* species and other non-spore-forming gram-positive bacilli to 18 antimicrobial agents. Antimicrob. Agents Chemother. **39:**208–214.
26. **Tauch, A., F. Kassing, J. Kalinowski, and A. Pühler.** 1995. The *Corynebacterium xerosis* composite transposon Tn*5432* consists of two identical insertion sequences, designated IS*1249*, flanking the erythromycin resistance gene *ermCX*. Plasmid **34:**119–131.
27. **Tumbarello, M., E. Tacconelli, A. Del Forno, S. Caponera, and R. Cauda.** 1994. *Corynebacterium striatum* bacteremia in a patient with AIDS. Clin. Infect. Dis. **18:**1007–1008.
28. **van Bosterhaut, B., R. Cuvelier, E. Serruys, F. Pouthier, and G. Wauters.** 1992. Three cases of opportunistic infection caused by propionic acid producing *Corynebacterium minutissimum*. Eur. J. Clin. Microbiol. Infect Dis. **11:**628–631.
29. **Wallet, F., C. H. Marquette, and R. J. Courcol.** 1994. Multiresistant *Corynebacterium xerosis* as a cause of pneumonia in a patient with acute leukemia. Clin. Infect. Dis. **18:**845–846.
30. **Wichmann, S., C. H. Wirsing von Koenig, E. Becker-Boost, and H. Finger.** 1984. Isolation of *Corynebacterium* group JK from clinical specimens with a semiselective medium. J. Clin. Microbiol. **19:**204–206.
31. **Zapardiel, J., E. Nieto, M. I. Gegundez, I. Gadea, and F. Soriano.** 1994. Problems in minimum inhibitory concentration determinations in coryneform organisms: comparison of an agar dilution and the Etest. Diagn. Microbiol. Infect. Dis. **19:**171–173.