

Most *Corynebacterium xerosis* Strains Identified in the Routine Clinical Laboratory Correspond to *Corynebacterium amycolatum*

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Received 28 November 1995/Returned for modification 22 January 1996/Accepted 2 February 1996

A comprehensive study was performed on 25 bacterial clinical isolates originally identified as *Corynebacterium xerosis*. Three reference strains of *C. xerosis* were also included in the study. On the basis of a variety of phenotypic characteristics tested, all strains could be divided into two separate clusters: reference strains ATCC 373 (the type strain of *C. xerosis*) and ATCC 7711 showed yellow-pigmented, dry, rough colonies, fermented 5-keto-gluconate, exhibited strong leucine arylamidase and α -glucosidase activities, produced lactate as the major end product of glucose metabolism, were susceptible to most of the 19 antimicrobial agents tested, and showed an inhibition zone around disks containing the vibriocidal compound O/129. In contrast, the remaining 26 strains including reference strain NCTC 7243 as well as all clinical isolates formed white-grayish, dry, slightly rough colonies, did not ferment 5-keto-gluconate, exhibited only weak leucine arylamidase and no α -glucosidase activity, produced large amounts of propionic acid as the end product of glucose metabolism, and were resistant to most antimicrobial agents tested, including O/129. Chemotaxonomic (cellular fatty acids, mycolic acids, and G+C content) and molecular genetic (16S rRNA gene sequence) investigations revealed that the strains of the second cluster unambiguously belonged to the species *C. amycolatum*. Our data suggest that most strains reported in the literature as *C. xerosis* are probably misidentified and correspond to *C. amycolatum*.

Within the last few years numerous publications have focused on the disease associations of coryneform bacteria as well as on the taxonomy of this heterogeneous group of bacteria. The reason for this is that clinical microbiologists and clinicians have become more and more aware of the pathogenic potential of coryneform bacteria, especially in immunocompromised patients. In recent years, one of the most frequently reported coryneform bacteria causing infections in humans has been *Corynebacterium xerosis* (2, 13, 16, 18, 19, 26–28). On the other hand, there are some reports of the heterogeneity of what was generally thought to be *C. xerosis* (6). The most recent and valuable study came from Coyle and coworkers (7), who demonstrated multiple taxa within commercially available reference strains of *C. xerosis*. In order to clarify the identity of *C. xerosis* strains isolated from clinical specimens, 25 strains originally identified as *C. xerosis* and referred to two reference laboratories in Europe and North America for identification were studied by applying phenotypic as well as molecular genetic methods. Three reference strains of *C. xerosis* were also included in the study for comparative investigations. Some lipophilic strains available as *C. xerosis* reference strains (7) were not included in the present investigations since a recent study demonstrated that these strains actually belong to some other already established taxa (17). *C. xerosis* strains were identified according to the Hollis-Weaver guide for the identification of gram-positive bacteria (14) and not according to the criteria given in Bergey's manual (6); i.e., *C. xerosis* strains produced acid from maltose. Most surprisingly, all 25 clinical strains examined turned out to be *C. amycolatum*. Therefore, our observations cast doubt on the iden-

ties of most strains of so-called *C. xerosis* reported in the literature.

MATERIALS AND METHODS

Strains, media, and growth conditions. The strains studied are listed in Table 1. The reference strains used for comparative investigations were obtained from the American Type Culture Collection (ATCC; Rockville, Md.) and the National Collection of Type Cultures (NCTC; London, United Kingdom). The other strains were referred to the Department of Medical Microbiology (DMMZ), University of Zürich, Zürich, Switzerland, and the Special Bacteriology Laboratory, Laboratory Centre for Disease Control (LCDC), Ottawa, Ontario, Canada, between 1989 and 1995. For examination of the CAMP reaction, *Staphylococcus aureus* ATCC 25923 was used. All strains were cultured on Columbia agar (Difco, Detroit, Mich.) with 5% sheep blood (SBA) at 37°C in a 5% CO₂ atmosphere.

Biochemical tests. Preparation of the media used for biochemical characterization of the strains was done as described by Nash and Krenz (20). All biochemical tests were carried out at 37°C. Testing of the other biochemical characteristics given in Table 2 has been outlined in detail previously (11, 12). Hydrolysis of starch was tested on Mueller-Hinton agar plates after 48 h of incubation by flooding the plates with a 1:5 dilution of Lugol's iodine solution. The commercial API (RAPID) Coryne system was used as recommended by the manufacturer (API bioMérieux, Marcy l'Etoile, France); readings of the carbohydrate fermentation reactions were done after 24 and 48 h of incubation. The activities of leucine arylamidase and α -glucosidase were tested (apart from examination with the API Zym and the API Coryne systems) with Rosco tablets (Rosco, Taastrup, Denmark) by preparing a bacterial suspension with turbidity equivalent to that of a McFarland no. 3 standard and incubation for 18 h as recommended by the manufacturer.

Antimicrobial susceptibility patterns. The susceptibilities of the 28 strains included in the present study to 19 antimicrobial agents known to have activity against coryneform bacteria were tested according to the guidelines for performance and interpretation of the National Committee for Clinical Laboratory Standards (21, 22). For interpretation of susceptibility to amoxicillin-clavulanic acid, ampicillin, oxacillin, and penicillin, the categories for staphylococci were used. MICs were tested by the agar dilution procedure (Mueller-Hinton agar supplemented with 5% sheep blood). For susceptibility to the vibriocidal compound O/129, disks with 150 μ g of O/129 (Oxoid, Basingstoke, United Kingdom) were placed on Mueller-Hinton agar supplemented with 5% sheep blood after streaking the plates with a suspension with turbidity equivalent to that of a McFarland no. 0.5 standard. Growth inhibition zones were observed after 24 h of incubation at 37°C.

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TABLE 1. Strains included in the study

Strain group and no.	Yr of isolation	Source
<i>C. xerosis</i>		
ATCC 373 (type strain)	1924	Ear discharge
ATCC 7711	1940	NK ^a
<i>C. amycolatum</i> primarily misidentified as <i>C. xerosis</i>		
NCTC 7243	1947	Infant nose
DMMZ 103	1991	Blood culture
DMMZ 323	1992	Blood culture
DMMZ 1171	1994	Intravascular catheter
DMMZ 1303	1993	Deep wound, lower leg
DMMZ 1318	1994	Easy flow secretion
DMMZ 1373	NK ^b	Urine
DMMZ 1422	1995	Retroauricular fistula
DMMZ 1423	1995	Fistula, upper leg
DMMZ 1480	1995	Deep wound, lower leg
DMMZ 1502	1995	Blood culture
DMMZ 1503	1995	Blood culture
DMMZ 1525	1995	Intravascular catheter
DMMZ 1528	1995	Intravascular catheter
DMMZ 1532	1995	Intravascular catheter
DMMZ 1563	1995	Abscess
DMMZ 1566	1995	Perianal abscess
DMMZ 1618	1995	Urine
DMMZ 1624	1995	Intravascular catheter
DMMZ 1668	1995	Intravascular catheter
DMMZ 1680	1995	Intravascular catheter
DMMZ 1788	1995	Blood culture
LCDC 89-0826	1989	Back wound
LCDC 91-0077	1991	Sternal bone fragments
LCDC 92-0042	1992	Aspirate from shoulder
LCDC 92-0043	1992	Blood culture

^a NK, not known; the strain was originally from the University of Maryland and was deposited in ATCC.

^b NK, year not known.

Gas-liquid chromatography. Determination of volatile and nonvolatile fatty acids from the fermentation of glucose was performed as described previously (11). For analysis of cellular fatty acids, cells were grown on Columbia agar base (Oxoid) supplemented with 5% defibrinated sheep blood at 35°C for 24 h in a 5% CO₂ incubator. Fatty acid derivatization and gas-liquid chromatography of the cellular fatty acids were done as outlined before (4), except that we used MIDI software, version 3.8.

Chemotaxonomic investigations. For the detection of the diamino acid of the cell wall peptidoglycan and of the presence of mycolic acids, the methods outlined by Schaal (24) were applied. Cell wall components were separated by thin-layer chromatography and were then visualized. The method for determination of the G+C contents of the bacterial DNAs was as given in an earlier report (12).

16S rRNA gene sequence analysis. A large fragment of the 16S rRNA gene was amplified from DNA by PCR with universal primers pA (positions 8 to 28; *Escherichia coli* numbering) and pH (positions 1542 to 1522). The amplified product was sequenced directly by using primers to conserved regions of the rRNA. Sequencing was performed with a PRISM Dyedeoxy Terminator Cycle Sequencing Kit (Applied Biosystems, Warrington, United Kingdom), and the reaction products were electrophoresed with an Applied Biosystems model 373A automatic DNA sequencer according to the manufacturer's protocols (Applied Biosystems). Sequences were compared by pairwise analysis (9).

RESULTS

Thirteen of the 25 clinical strains were isolated from blood cultures or from intravascular catheters. Many other strains came from infections which were anatomically closely related to the skin. Two cases of urinary tract infections were noted, but none of the clinical strains originated from a respiratory tract specimen.

Macroscopic morphology separated all strains examined

TABLE 2. Biochemical characteristics of the strains studied

Reaction	Result for <i>C. xerosis</i> (n = 2)		% Positive reactions for <i>C. amycolatum</i> (n = 26)
	ATCC 373	ATCC 7711	
Catalase	+	+	100
Motility	-	-	0
Nitrate reduction	+	-	96
Hydrolysis of			
Urea	-	-	0
Esculin	-	-	0
Gelatin	-	-	0
Starch	+	(+) ^a	100
Casein	-	-	0
Tyrosine	-	-	0
Xanthine	-	-	0
CAMP reaction	-	-	0
Fermentation of ^b :			
Glucose	+	+	100
Maltose	+	+	100
Sucrose	+	+	100
Mannitol	-	-	0
Xylose	-	-	0
Ribose	+	+	92
Lactose	-	-	0
Glycogen	-	-	0
5-Keto-gluconate	+	+	0
Activity of ^c :			
Pyrazinamidase ^d	+	+	100
Pyrrolidonyl arylamidase ^d	-	-	0
Alkaline phosphatase	+(w)	+(w)	100 (s)
Esterase (C ₄)	+(m)	+(m)	100 (m)
Esterase lipase (C ₈)	+(s)	+(s)	100 (s)
Lipase (C ₁₄)	-	+(w)	92 (w)
Leucine arylamidase	+(s)	+(s)	85 (w)
Valine arylamidase	-	-	0
Cystine arylamidase	+(w)	+(w)	100 (w)
Trypsin	-	-	0
Chymotrypsin	-	-	0
Acid phosphatase	+(w)	+(w)	100 (w)
Phosphoamidase	+(w)	+(w)	92 (w)
α-Galactosidase	-	-	0
β-Galactosidase	-	-	0
β-Glucuronidase	-	-	0
α-Glucosidase	+(s)	+(s)	0
β-Glucosidase	-	-	0
n-Acetyl-β-glucosaminidase	-	-	0
α-Mannosidase	-	-	0
α-Fucosidase	-	-	0

^a Parentheses indicate weak activity only.

^b All fermentation reactions except for that for 5-keto-gluconate were determined at 48 h of incubation; the reaction for 5-keto-gluconate was recorded at 120 h.

^c As measured with the API Zym system. (w), approximately 5 nmol of substrate hydrolyzed; (m), approximately 20 nmol of substrate hydrolyzed; (s), >40 nmol of substrate hydrolyzed.

^d As measured with the API Coryne system.

into two groups (Table 1): all clinical strains grew as white-grayish, dry, slightly rough colonies of 1 to 2 mm in diameter after 48 h of incubation; these strains were referred to as *C. xerosis*-like (later shown to be *C. amycolatum*) strains. The other group (referred to as the *C. xerosis* group) contained the

type strain of *C. xerosis* (ATCC 373) and reference strain ATCC 7711 only. These two strains showed yellow-pigmented (after 48 h of incubation on SBA at 37°C), dry, and rough colonies reminiscent of rapidly growing mycobacteria. Microscopically, no differences were observed between the two groups of strains since both showed typical club-shaped, non-partially acid-fast, gram-positive organisms, as observed for *Corynebacterium* spp. in general.

None of the key reactions usually applied in the routine identification of coryneform bacteria (nitrate reduction, urea hydrolysis, esculin hydrolysis, fermentation of glucose, maltose, sucrose, mannitol, and xylose, and the CAMP test) were found to differentiate the two groups of bacteria (Table 2). It should be noted that reference strains ATCC 7711 and NCTC 7243 were unable to reduce nitrate (as also reported by others [7]), whereas all other strains reduced nitrate. When applying the API (RAPID) Coryne system, we observed that both strains of the *C. xerosis* group exhibited strong α -glucosidase activity which was not present in strains of the *C. xerosis*-like group. This difference was confirmed by testing α -glucosidase activity with the API Zym gallery as well as with Rosco tablets. Furthermore, we found that in the 26 strains of the *C. xerosis*-like cluster, leucine arylamidase activity was absent or only weak, whereas the other two strains showed very strong activity of this enzyme. In addition, the testing of all 28 strains included in the present study with the API Zym system indicated stronger activity of alkaline phosphatase in the strains of the *C. xerosis*-like cluster than in the two other strains. The API 50 CH system did not provide further differential reactions between the two groups of organisms except that the two strains of the *C. xerosis* group produced acid from 5-keto-gluconate, which was not observed for any strain of the other group. All *C. xerosis*-like strains produced large amounts of propionic acid, whereas the two other strains produced lactate as their major end product of the glucose metabolism.

Table 3 outlines the different API (RAPID) Coryne profiles and identifications for all 28 strains tested after 24 and 48 h of incubation. It should be noted that 24 and 25 of 26 *C. xerosis*-like strains were identified with very good scores as *C. xerosis* after 24 and 48 h of incubation, respectively.

The majority of the *C. xerosis*-like strains were multiresistant, as revealed by the MICs at which 50% (MIC₅₀s) and 90% (MIC₉₀s) of strains are inhibited (Table 4). Tetracycline, teicoplanin, and vancomycin were the only antimicrobial agents whose MIC₉₀s were below the breakpoints recommended by the National Committee for Clinical Laboratory Standards (22). In contrast, the two strains of the *C. xerosis* group were susceptible to nearly all antimicrobial agents tested. Another easy-to-perform test which clearly separated the two groups was the resistance of all *C. xerosis*-like strains to the vibriocidal compound O/129; both strains of the *C. xerosis* group showed inhibition zones.

Cellular fatty acid analysis revealed qualitatively similar patterns for both *C. xerosis* and *C. xerosis*-like strains (Table 5). However, quantitative differences in the cellular fatty acid patterns were noted, with significantly lower amounts of palmitic acid but higher amounts of oleic acid in *C. xerosis* strains compared with the amounts in the *C. xerosis*-like strains. In the analyses of *C. xerosis* strains, not all resulting peaks could be assigned to certain cellular fatty acids. These unidentifiable peaks (at or near equivalent chain lengths 15.000, 16.8, and 17.000) represented cleaved mycolic acids. In contrast, no peaks suggestive of cleaved mycolic acids were detected in the *C. xerosis*-like strains.

All 28 strains studied contained *meso*-diaminopimelic acid in their peptidoglycan. In both *C. xerosis* strains, mycolic acids

TABLE 3. API Coryne profiles of the strains studied

Incubation time, strain, and numerical code (no. of strains)	Results by the API Coryne system			
	Identification	% Identified ^a	T index	Profile acceptance
24 h				
<i>C. xerosis</i> (n = 2)				
2110305 (ATCC 7711)	<i>Corynebacterium</i> sp. strain G-2	71.8	0.43	Doubtful
	<i>C. striatum</i>	23.3	0.35	
3110325 (ATCC 373)	<i>C. xerosis</i>	99.8	0.77	Very good
<i>C. amycolatum</i> (n = 26)				
2100125 (NCTC 7243)	<i>C. minutissimum</i>	97.1	0.92	Good
3100105 (1)	<i>C. striatum</i>	97.1	1.00	Good
3100125 (14)	<i>C. xerosis</i>	99.7	0.95	Very good
3100325 (10)	<i>C. xerosis</i>	99.6	1.00	Very good
48 h				
<i>C. xerosis</i> (n = 2)				
2110325 (ATCC 7711)	<i>C. minutissimum</i>	53.2	0.50	Doubtful
	<i>C. xerosis</i>	46.7	0.49	
3110325 (ATCC 373)	<i>C. xerosis</i>	99.8	0.77	Very good
<i>C. amycolatum</i> (n = 26)				
2100325 (NCTC 7243)	<i>C. minutissimum</i>	97.9	1.00	Good
3100125 (2)	<i>C. xerosis</i>	99.7	0.95	Very good
3100325 (23)	<i>C. xerosis</i>	99.6	1.00	Very good

^a Identification percentages of less than 3.0 were not reported.

could be detected, but in the *C. xerosis*-like strains, no mycolic acids were found, even after repeated attempts. The only species within the genus *Corynebacterium* not containing mycolic acids is *C. amycolatum* (5, 6, 23). Determination of the G+C contents of the two *C. xerosis* strains revealed values of 66 to 69 mol%; in contrast, the *C. xerosis*-like strains possessed significantly lower amounts of G+C (55 to 60 mol%).

To further investigate the possible affinity between *C. xerosis*-like strains and *C. amycolatum*, phylogenetic analyses were performed. A large fragment of the 16S rRNA genes of five representative clinical isolates (strains DMMZ 323, DMMZ 1171, DMMZ 1303, DMMZ 1318, and DMMZ 1422) and strain NCTC 7243 was amplified by PCR and sequenced. Approximately 1,400 bases were determined for each strain, and pairwise analyses showed the six strains to be highly related to each other and to the type strain of *C. amycolatum* (strain NCFB 2768 [5]). Considerably lower sequence relatedness (<98%) was shared with *C. xerosis* ATCC 373, to which the 16S rRNA gene sequence of reference strain ATCC 7711 was identical. These findings demonstrate that the *C. xerosis*-like group of strains do not correspond to *C. xerosis* but are representatives of the species *C. amycolatum*.

DISCUSSION

Phenotypic, chemotaxonomic, and molecular genetic investigations revealed that none of the 25 clinical isolates studied

TABLE 4. Antimicrobial susceptibility patterns of the strains studied^a

Substance	MIC ($\mu\text{g/ml}$)				
	<i>C. xerosis</i> (n = 2)		<i>C. amycolatum</i> (n = 26)		
	ATCC 373	ATCC 7711	Range	50%	90%
Amoxicillin-clavulanic acid	≤ 0.03	0.06	0.13–>64	1	64
Ampicillin	0.125	0.125	0.25–>64	2	>64
Ceftriaxone	0.25	0.125	0.5–>64	1	>64
Cefuroxime	0.06	0.06	0.25–>64	0.5	>64
Cephalothin	0.06	0.06	0.13–>64	1	>64
Chloramphenicol	8	8	16–32	32	32
Ciprofloxacin	2	2	4–>64	>64	>64
Clindamycin	2	4	0.25–>64	>64	>64
Erythromycin	0.125	8	≤ 0.03 –>64	>64	>64
Gentamicin	≤ 0.03	0.125	4–>64	16	32
Imipenem	≤ 0.03	≤ 0.03	≤ 0.03 –>64	0.5	>64
Oxacillin	1	0.5	2–>64	16	>64
Penicillin	0.06	0.06	0.25–>64	2	>64
Rifampin	≤ 0.03	≤ 0.03	≤ 0.03 –>64	>64	>64
Sparfloxacin	2	4	≤ 0.03 –32	32	32
Teicoplanin	0.125	0.5	0.13–0.5	0.5	0.5
Tetracycline	1	2	0.5–32	1	2
Trimethoprim-sulfamethoxazole	16	16	>64	>64	>64
Vancomycin	0.25	0.25	0.13–0.5	0.25	0.25

^a Susceptibility to O/129 was determined by the disk diffusion method (see text). The *C. xerosis* strains were susceptible to O/129 (inhibition zone, 12 to 28 mm), and the *C. amycolatum* strains were resistant.

belonged to the species represented by the *C. xerosis* type strain (ATCC 373). The reference strain ATCC 7711 is a true *C. xerosis* strain, whereas the reference strain NCTC 7243 belongs to *C. amycolatum*.

For the routine clinical laboratory, we recommend use of the following criteria for differentiating between *C. xerosis* and *C. amycolatum* strains: dry, rough colonies of fermentative coryneform bacteria with yellowish pigment should raise the suspicion of *C. xerosis*, whereas *C. amycolatum* exhibits dry colonies which are never pigmented; susceptibility to O/129 differentiates the susceptible *C. xerosis* strains from the resistant *C. amycolatum* strains; the activity of α -glucosidase also provides a clear-cut distinction between *C. xerosis* (positive reaction) and *C. amycolatum* (negative reaction) strains. Further means of differentiation include the detection of major amounts of propionic acid (*C. amycolatum*) rather than lactic acid (*C. xerosis*) as an end product of glucose fermentation and assays for the presence (*C. xerosis*) or absence (*C. amycolatum*) of mycolic acids. Different G+C contents had been reported before for "*C. xerosis*" strains (6), which can now be explained by the fact that *C. amycolatum* strains (55 to 60 mol% G+C) have a significantly lower G+C content than authentic *C. xerosis* strains (66 to 69 mol%). Subtle quantitative ($C_{16:0}$ and $C_{18:1\omega 9cis}$) and qualitative (presence [*C. xerosis*] or absence [*C. amycolatum*] of poorly cleaved mycolic acids) differences may also assist in the differentiation of these taxa.

In our experience, *C. xerosis* is extremely rarely found in clinical specimens, whereas *C. amycolatum* is probably the most frequently encountered nonlipophilic *Corynebacterium* species in clinical specimens. Because *C. amycolatum* is variable for many biochemical reactions such as nitrate reduction, urea hydrolysis, and fermentation of maltose and sucrose (3, 5), it seems most likely that many more *C. amycolatum* strains are misidentified as *C. striatum* or *C. minutissimum*, as outlined by Barreau et al. (3). We therefore emphasize that every strain of fermenting coryneform bacteria with dry colonies should raise the suspicion of *C. amycolatum*.

The misidentification of *C. amycolatum* as *C. xerosis* is al-

lowed by the Hollis-Weaver charts (14) (which could not possibly contain *C. amycolatum* because this species was proposed later by Collins and coworkers [5]) since none of the 33 biochemical features given in the tables separates *C. amycolatum* from *C. xerosis*. The commercial API (RAPID) Coryne system may also misidentify *C. amycolatum* strains as *C. xerosis* (Table 3); its database states that only 4% of all *C. xerosis* strains are α -glucosidase positive (1). Therefore, it seems likely that the database for *C. xerosis* was constructed with *C. amycolatum* strains. α -Glucosidase activity is a good marker for *C. xerosis* strains because only a few other *Corynebacterium* species pathogenic for humans (*C. diphtheriae*, *C. ulcerans*, *C. pseudotuberculosis*, and *C. glucuronolyticum*) exhibit activity of this enzyme, and all of these strains can be readily distinguished from *C. xerosis* (1, 6, 10). It is important to note that nitrate reduction in authentic *C. xerosis* strains is variable, which is in contrast to the data given both in the Hollis-Weaver charts (14) as well as in the API (RAPID) Coryne database (1).

TABLE 5. Cellular fatty acid patterns of the strains studied

Component	% Cellular fatty acids (mean \pm SD [range]) ^a		
	<i>C. xerosis</i> (n = 2)		<i>C. amycolatum</i> (n = 24) ^b
	ATCC 373	ATCC 7711	
$C_{14:0}$			1 \pm 1 (0–1)
$C_{16:1\omega 9cis}$	2	1	2 \pm 1 (1–2)
$C_{16:0}$	12	16	26 \pm 2 (24–31)
$C_{17:0}$			3 \pm 2 (1–7)
Feature 6 ^c	1	2	2 \pm 1 (2–3)
$C_{18:1\omega 9cis}$	54	46	36 \pm 3 (33–39)
Feature 7 ^d	2	2	1 \pm 1 (0–2)
$C_{18:0}$	16	27	29 \pm 3 (24–35)

^a Amounts of less than 1% were not reported.

^b Two of the 26 *C. amycolatum* strains were not included in this analysis.

^c Feature 6 contains $C_{18:2\omega 6cis}$, $C_{18:2\omega 9cis}$, and $C_{18:0antiso}$.

^d Feature 7 contains $C_{18:1\omega 7cis}$, $C_{18:1\omega 9trans}$, and $C_{18:1\omega 12trans}$.

Coyle et al. (7) assigned 3 of the 10 "*C. xerosis*" reference strains examined in their study to the species *C. striatum*, 4 of the 10 strains belonged to some lipophilic *Corynebacterium* species (7, 17), and this report demonstrated that strain NCTC 7243 is *C. amycolatum*. In contrast to our results, Coyle et al. (7) assigned strains ATCC 373 and ATCC 7711 to different hybridization groups, i.e., separate species. However, our data indicating 100% homology within the 16S rRNA genes of both strains demonstrate that they most likely belong to the same species. The reason for this discrepancy is unclear. Profiles of whole-cell protein electrophoresis as well as the mycolic acid patterns were almost identical for strains ATCC 373 and ATCC 7711, thereby reinforcing the close relation between the two strains (8, 15).

Like *C. jeikeium* and *C. urealyticum*, many *C. amycolatum* strains are resistant to multiple antibiotics. The molecular basis for this feature of *C. amycolatum* strains is not known at present. Nearly all multiresistant *C. amycolatum* strains remained susceptible to tetracycline, an observation that was also reported by Soriano et al. (25) for their so-called *C. xerosis* strains.

We conclude that the majority of strains designated "*C. xerosis*" in the literature may have been, in fact, misidentified *C. amycolatum* strains. True *C. xerosis* strains are, in our experience, extremely rarely encountered in clinical specimens. Therefore, for future publications on *C. xerosis*, performance of the minimal set of tests outlined above seems to be necessary to ensure the identity of the tested strains as authentic *C. xerosis* strains.

ACKNOWLEDGMENTS

We recognize A. von Graevenitz for an early awareness of the problem and for a careful review of the manuscript. V. Pünter and J. Winstanley provided expert technical assistance.

This study was supported by the European Community (CT93-0119 and CT94-3098), the Jubiläumsspende der Universität Zürich, and the Hochschulverein Zürich. G.F. acknowledges support by a grant from the Sassella-Stiftung (Zürich, Switzerland).

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