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Comprehensive Study of *Corynebacterium freneyi* Strains and Extended and Emended Description of *Corynebacterium freneyi* Renaud, Aubel, Riegel, Meugnier, and Bollet 2001[▽]

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In 2001, *Corynebacterium freneyi* was described as a new fermentative, α-glucosidase-positive *Corynebacterium* species related to *C. xerosis* based on data from three strains. During a review of our extensive culture collection we encountered 18 additional *C. freneyi* strains and further characterized them in detail. Thirteen of the 18 strains were isolated from female genital tract specimens without any obvious disease association. Phenotypically, *C. freneyi* can be easily differentiated from *C. xerosis* by its distinct wrinkled colonies whereas nearly all other routinely applied phenotypic tests do not allow a unanimous separation of *C. freneyi* from *C. xerosis*. Restriction length polymorphism analysis using CfoI of the 16S-23S rRNA gene intragenic spacer definitively allows differentiation between the two species. Surprisingly, comparative 16S rRNA gene analysis does not discriminate between *C. freneyi* and *C. xerosis* because the designated type strain of *C. freneyi* is not the most representative strain for this species. The present report also includes detailed data on the antimicrobial susceptibility pattern of *C. freneyi* presented here for the first time. Based on the large number of additional *C. freneyi* strains from our culture collection, we provide an extended and emended species description of *C. freneyi*.

During the last 15 years, clinical microbiologists have witnessed a massive increase in the number of medically relevant species of corynebacteria from 13 in 1993 to 40 in 2007 (5). One of the recently newly described species is *Corynebacterium freneyi* (13). This taxon was characterized by Renaud et al. in 2001 based on data from three strains (13). Up to now, only one additional *C. freneyi* strain has appeared in the relevant literature (2). *C. freneyi* is a fermentative, α-glucosidase-positive *Corynebacterium* species closely related to *C. xerosis* (13).

While screening the extensive bacterial species culture collection of one of the authors (G. Funke) we encountered 18 additional *C. freneyi* strains. As the result of a comprehensive study of these 18 clinical isolates by applying biochemical, chemotaxonomical, and molecular genetic methods, this paper presents an extended and emended description of *C. freneyi*.

MATERIALS AND METHODS

Strains. The 18 clinical strains included in the present study (see Table 1) were isolated in our routine clinical laboratory or referred to our reference laboratory during a 3-year period. The strains were definitively not linked epidemiologically.

Biochemical profiles. The methods applied were described in detail before (9). All strains were grown on Columbia base sheep blood agar plates (BD, Heidelberg, Germany) and incubated at 35°C in ambient air. The commercial API Coryne (RAPID Coryne) and API ZYM (both from bioMérieux, Marcy l'Etoile, France) systems were used following the instructions of the manufacturers. The API 50 CH system (bioMérieux) applying CHB medium (bioMérieux) was used for incubation for up to 120 h.

Antimicrobial susceptibility testing. The recently established CLSI standard for determination and interpretation of antimicrobial MICs for *Corynebacterium* spp. (3) has been applied. Briefly, by use of a broth microdilution method (prepared in our laboratory), bacterial cells with an inoculum equivalent to a 0.5 McFarland standard were grown in cation-adjusted Mueller-Hinton broth with lysed horse blood and incubated for up to 48 h. Reading of MICs was done by two independent researchers.

Cellular fatty acid analysis. The methods for preparation of the bacterial cells and analysis of the cellular fatty acid (CFA) profiles have been described before (15).

Molecular genetic investigations. Analysis of the complete 16S rRNA gene sequences was performed according to a published protocol (6). Full-length (>1,350 bp) 16S rRNA gene sequences were determined for each clinical strain by aligning the resulting sequences by use of a Lasergene 5 package (DNASTAR Inc., Madison, WI). Analysis of the restriction fragment length polymorphism (RFLP) of the 16S-23S rRNA gene intragenic spacer region was performed by use of restriction enzyme CfoI and following the methods outlined by Aubel et al. (1).

Nucleotide sequence accession numbers. The GenBank accession numbers of the complete 16S rRNA gene sequences of all 18 clinical isolates included in the present study are given in Table 1. Strains 3078, 3172, 3228, 3229, and 3527 mentioned in this report have been deposited in the Culture Collection of the University of Göteborg, Sweden, under accession numbers CCUG 54465, 54466, 54467, 54468, and 54469, respectively.

RESULTS

Table 1 lists the origins of the 18 clinical *C. freneyi* strains which were further characterized in the present study. The ages of the patients were in a range from 18 to 77 years, and the number of female patients was much higher than the number of the male patients (14 versus 4). Interestingly, 13 of 14 specimens from females came from the genital tract.

The extensive biochemical profiling data obtained from the 18 clinical and 3 reference *C. freneyi* strains are given in Table 2. *C. freneyi* strains are consistently positive for α-glucosidase and alkaline phosphatase. Activity of phosphoamidase was stronger in *C. freneyi* than in *C. xerosis* strains, whereas weak cystine arylamidase activity was detected in *C. xerosis* but not in *C. freneyi*. Only one *C. freneyi* strain (strain 3171) was negative

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TABLE 1. Strains included in the present study

Species and strain no.[a]	Origin of strain	Patient age (yr), sex[b]	16S rRNA gene GenBank accession no.
C. xerosis			
ATCC 373^T	Ear discharge		X84446
ATCC 7711	NK[c]		
C. amycolatum			
CIP 103452^T	Human skin		
NCTC 7243	Infant nose		
C. freneyi			
CIP 106767^T	Pus from a toe		AJ292762
ISPB 16799604	Subcutaneous abscess fistula		
ISPB 20395347	Varicose ulcer		
22	Vaginal swab	19, f	EF462397
2319	Uterine cervix	27, f	EF462398
2484	Testicle swab	50, m	EF462399
2586	Outer ear canal	38, m	EF462400
3008	Vaginal swab	31, f	EF462401
3017	Vaginal swab	18, f	EF462402
3034	Vaginal swab	30, f	EF462403
3077	Leg	77, m	EF462404
3078	Vaginal swab	35, f	EF462405
3171	Vaginal swab	70, f	EF462406
3172	Vaginal swab	24, f	EF462407
3228	Cervical swab	71, f	EF462408
3229	Duodenal biopsy	48, f	EF462409
3296	Vaginal swab	33, f	EF462410
3297	Urine	63, m	EF462411
3437	Cervical swab	18, f	EF462412
3526	Vaginal swab	73, f	EF462413
3527	Vaginal swab	24, f	EF462414

[a] ATCC, American Type Culture Collection; CIP, Collection de Institut Pasteur; NCTC, National Type Culture Collection; ISPB, Institut des Sciences Pharmaceutiques et Biologiques, Lyon, France.
[b] Data only for the clinical strains of the present study. f, female; m, male.
[c] NK, not known.

for leucine arylamidase activity. Nineteen of 21 *C. freneyi* strains fermented glucose at 42°C, and all strains grew at 20°C.

The following cellular fatty acids were detected at more than 1% of total CFAs: $C_{15:0}$, 4% (mean); $C_{16:0}$, 26%; $C_{17:1\omega8c}$, 4%; $C_{17:0}$, 17%; $C_{18:1\omega9c}$, 21%; and $C_{18:0}$, 23%. Tuberculostearic acid was not detected.

Table 3 outlines the antimicrobial susceptibility pattern of *C. freneyi*. All 21 strains tested were susceptible to doxycycline, gentamicin, linezolid, meropenem, rifampin, and vancomycin, with gentamicin and rifampin exhibiting extremely low MICs for all *C. freneyi* strains.

Table 4 and Table 5 show the overall numbers and exact details of 16S rRNA gene base pair mismatches between all 18 clinical *C. freneyi* strains and the comparative sequence AJ 292762 of the *C. freneyi* CIP 106767^T type strain and sequence X84446 of the of *C. xerosis* ATCC 373^T type strain. In general, the 18 clinical strains had similar numbers of mismatches with either *C. freneyi* or *C. xerosis*. The 16S rRNA gene homology of the 18 clinical strains was in a range from 98.9 to 99.3% (mean, 99.0%) for *C. freneyi* and in a range from 98.7 to 99.1% (mean, 98.8%) for *C. xerosis*.

In order to clarify the exact genetic status (i.e., species identity) of the 18 clinical strains, RFLP analysis of the amplified 16S-23S rRNA gene intragenic spacer region was performed. Figure 1 shows the RFLP patterns for five clinical strains which undoubtedly represented *C. freneyi* strains. The RFLP patterns for the remaining 13 clinical strains were identical to those of the other five clinical strains (data not shown).

DISCUSSION

None of the 18 clinical *Corynebacterium* strains included in the present study that exhibited dry colonies, fermentative metabolism, and α-glucosidase positivity turned out to be *C. xerosis*, indicating that this species is extremely rarely encountered in clinical specimens, as reported previously (4, 7).

Of the 4 *C. freneyi* strains for which studies have appeared in the literature (2, 13), none came from urogenital specimens, whereas 13 of the 18 strains described in the present report came from the female genital tract. This finding certainly cannot be explained by increased awareness on the part of the plate-reading personnel, since 12 of the 18 strains were referred to our laboratory from other independent laboratories. The limited patient clinical data available did not indicate a clear disease association with *C. freneyi* or any pathogenic potential. The prevalence of *C. freneyi* in the female genital tract is presently not known.

The most striking feature of *C. freneyi* strains is their distinctive macroscopic morphology (Fig. 2), showing dry, buff, and rough colonies with a wrinkled surface which strongly reminds the clinical microbiologist of a *Mycobacterium tuberculosis* culture on Middlebrook 7H10 agar or of certain *Tsukamurella* species. Some of the plate-reading personnel in our laboratory often described *C. freneyi* colonies as "folded-up colonies." The majority of *C. freneyi* strains had a whitish-grayish color, but two strains (strains 3077 and 3437) showed a yellowish pigment (which had not been reported previously), as constantly reported from examinations of the few true published *C. xerosis* strains (7), making the distinction between these two species even more complicated. However, strongly wrinkled or folded colonies are seen neither in *C. xerosis* nor in the morphologically related dryish *C. amycolatum* strains (when applying the above-mentioned incubation conditions), making *C. freneyi* easily recognizable in the routine clinical laboratory.

Because of the larger number of *C. freneyi* strains in the present study ($n = 18$) compared to the initial *C. freneyi* study ($n = 3$), we were able to detect the following biochemical features not described previously. *C. freneyi* strains are variable for fermentation of lactose and glycogen. A minority of *C. freneyi* strains (2 of 21) are able to produce acid from mannitol. In addition, the present report outlines API ZYM data on *C. freneyi* for the first time.

C. freneyi strains can be differentiated from the recently described, phylogenetically related *C. hansenii* species (14) by the fact that it gives positive results for α-glucosidase and alkaline phosphatase activity whereas *C. hansenii* is negative for these reactions. Furthermore, the majority of *C. freneyi* strains ferment glucose at 42°C whereas *C. hansenii* does not.

For CFA analysis, the MIDI system (MIDI Inc., Newark, DE) tentatively identified some fatty acids as pentadecanoic acid ($C_{15:0}$), margaric acid ($C_{17:0}$), and *cis*-heptadec-8-enoic acid ($C_{17:1\omega8c}$) but these components may have been degradation products of mycolic acids cleaved at the temperature pro-

TABLE 2. Biochemical properties of *C. freneyi* and *C. xerosis*

Category	% Positive reactions[a]		Category	% Positive reactions[a]	
	C. freneyi (n = 21)	*C. xerosis* (n = 2)		*C. freneyi* (n = 21)	*C. xerosis* (n = 2)
Nitrate reduction	67	50	Inuline	(14)	0
Urea hydrolysis	0	0	Melezitose	(19)	0
Esculin hydrolysis	0	0	D-Raffinose	(10)	0
Pyrazinamidase	100	100	Amidon	(5)	0
Pyrrolidonyl arylamidase	0	0	Xylitol	(29)	0
Gelatinase	0	0	β-Gentibiose	(33)	50
CAMP	0	0	D-Turanose	100	100
Lipophilia	0	0	D-Lyxose	(14)	50
			D-Tagatose	(100)	(50)
Fermentation			D-Fucose	(29)	50
Glucose	100	100	L-Fucose	(19)	0
Maltose	100	100	D-Arabitol	(24)	0
Sucrose	100	100	L-Arabitol	(19)	0
Mannitol	10	0	Gluconate	(43)	0
Xylose	0	0	2-Keto-Gluconate	0	0
Ribose	(100)	100	5-Keto-Gluconate	100	100
Lactose	48	0			
Glycogen	(48)	0	Further enzymatic activities[b]		
Glycerol	0	50	Alkaline phosphatase	100 (m)	100 (w)
Erythritol	0	0	Esterase (C4)	100 (m)	100 (m)
D-Arabinose	0	0	Esterase lipase (C8)	100 (s)	100 (s)
L-Arabinose	(5)	0	Lipase (C14)	14 (w)	50 (w)
Adonitol	(10)	(50)	Leucine arylamidase	95 (s)	100 (s)
β-Methyl-xyloside	(5)	0	Valine arylamidase	0	0
Galactose	95	50	Cystine arylamidase	0	100 (w)
D-Fructose	100	100	Trypsin	0	0
D-Mannose	100	100	Chymotrypsin	0	0
L-Sorbose	10	0	Acid phosphatase	19 (w)	100 (w)
Rhamnose	10	0	Phosphoamidase	100 (m)	100 (w)
Dulcitol	(10)	50	α-Galactosidase	0	0
Inositol	(19)	50	β-Galactosidase		
Sorbitol	(14)	50	pH 5.4	0	0
α-Methyl-D-mannoside	(10)	0	pH 7.4[c]	0	0
α-Methyl-D-glucoside	(10)	0	β-Glucuronidase	0	0
N-Acetyl-glucosamine	(10)	0	α-Glucosidase		
Amygdaline	(10)	0	pH 5.4	100 (s)	100 (s)
Arbutine	(24)	0	pH 7.4[c]	100 (s)	100 (s)
Salicine	(10)	50	β-Glucosidase	0	0
Cellobiose	(19)	0	*N*-Acetyl-β-glucosaminidase	0	0
Melibiose	(10)	0	α-Mannosidase	0	0
Trehalose	100	100	α-Fucosidase	0	0

[a] Values in parentheses represent weak acid production after 120 h incubation. (w), hydrolyzed substrate (approximately 5 nmol); (m), hydrolyzed substrate (approximately 20 nmol); (s), hydrolyzed substrate (>40 nmol).
[b] As determined by use of the API ZYM system.
[c] As determined using the API Coryne system.

TABLE 3. Antimicrobial susceptibility pattern of *C. freneyi* strains[a]

Antimicrobial agent	MIC (μg/ml)			No. (%) of isolates in indicated category		
	Range	50%	90%	Susceptible	Intermediate	Resistant
Cefotaxime	0.12–8	0.5	1	20 (95)	0 (0)	1 (5)
Ciprofloxacin	0.06–2	0.12	0.25	20 (95)	1 (5)	0 (0)
Clindamycin	0.015–>32	0.06	>32	18 (86)	0 (0)	3 (14)
Doxycycline	0.12–4	0.25	1	21 (100)	0 (0)	0 (0)
Erythromycin	≤0.015–>32	0.12	>32	13 (62)	1 (5)	7 (33)
Gentamicin	≤0.06	≤0.06	≤0.06	21 (100)	0 (0)	0 (0)
Linezolid	0.5–2	2	2	21 (100)	0 (0)	0 (0)
Meropenem	≤0.03–1	0.06	1	21 (100)	0 (0)	0 (0)
Penicillin	0.06–32	0.25	8	18 (86)	0 (0)	3 (14)
Rifampin	≤0.015–0.03	≤0.015	0.03	21 (100)	0 (0)	0 (0)
Vancomycin	0.5–1	1	1	21 (100)	0 (0)	0 (0)

[a] The 21 strains listed in Table 1 were tested.

duced in the injection port of the system (300°C). Normally, cleaved mycolic acids account for only 2% to 10% of all CFAs in *Corynebacterium* spp. A similar observation of an even higher percentage of cleaved mycolic acids has been reported for another *Corynebacterium* species, *C. auris* (8).

Since 2006, a CLSI standard has been available for performance and interpretation of antimicrobial susceptibility testing of *Corynebacterium* species by use of a microdilution method. Therefore, the antimicrobial susceptibility data of the present study are more significant than the data of Renaud et al. obtained using an unvalidated commercial system (API Staph gallery) or a disk diffusion method (13). In addition, in the publication by Renaud et al., the interpretation standard used remained unclear. The present study outlines MIC data for *C. freneyi* for the first time. As observed for many other *Corynebacterium* spp. (5, 10, 11), β-lactams show good activity against

TABLE 4. 16S rRNA gene sequence comparison between the *C. freneyi* ISPB 6695110 type strain and the 18 clinical strains

Alignment position	Position in *C. freneyi*	Base in *C. freneyi*	Event in comparison to *C. freneyi* result	No. of bases in the majority of the 18 clinical strains	No. of bases in the minority of the 18 clinical strains[a]
19	19	T	Point mutation	16 C	2 T (2586, 3526)
72	Between 71 and 72		Insertion	18 C	
94	Between 92 and 93		Insertion	18 C	
138	141	G	Point mutation	15 G	3 A (3077, 3297, 3437)
180	178	T	Point mutation	17 T	1 A (3296)
304	Between 301 and 302		Insertion	18 A	
926	923	C	Point mutation	17 T	1 Y (22)
929	926	G	Point mutation	14 C	3 S (22, 3008, 3034), 1 G (3297)
930	927	G	Point mutation	14 C	2 G (22, 3297), 2 S (3008, 3034)
931	928	C	Point mutation	14 G	3 C (3008, 3034, 3297), 1 S (22)
940	937	K	Point mutation	14 T	3 G (3008, 3034, 3297), 1 K (22)
941	938	T	Point mutation	14 G	3 K (22, 3008, 3034), 1 T (3297)
942	939	C	Point mutation	14 G	2 C (3034, 3297), 2 S (22, 3008)
943	940	C	Point mutation	18 T	
1055	1052	G	Point mutation	13 G	3 T (3077, 3297, 3437), 2 K (22, 3296)
1341	1338	T	Deletion		

[a] Numbers in parentheses represent strain designations.

C. freneyi strains. In contrast, in the related *C. amycolatum* strains the activity of β-lactams is often limited (11). As with many other *Corynebacterium* spp. (5, 10, 11), resistance to erythromycin is the most frequently encountered form of single-substance resistance. Both linezolid and vancomycin are active against *C. freneyi* strains, as seen with all other true corynebacteria (10). A peculiar feature of *C. freneyi* is the consistently very low MICs for gentamicin, which have not been detected in studies of many other true corynebacteria.

Unfortunately, Renaud et al. (13) did not give any precise 16S rRNA data with respect to gene homology between *C. freneyi* and *C. xerosis* strains. Our 16S rRNA gene data obtained using a very reliable double-stranded DNA reading approach (with multiple overlapping partial sequences) indicate that our 18 *C. freneyi* strains share on average only 99.0% 16S rRNA gene homology with the *C. freneyi* type strain. However, the 16S rRNA gene homology within our 18 *C. freneyi* strains often reached 100% (i.e., within 8 of 18 strains, not a single 16S rRNA gene base pair mismatch was seen), indicating that the *C. freneyi* type strain might not be the most representative *C. freneyi* strain. Our sequencing data confirm that *C. freneyi* and *C. xerosis* are the most closest related phylogenetic neighbors presently known, although they are distinct species with a relatively high level (13% to 23%) of DNA-DNA homology (13).

Another molecular genetic method for discerning closely related *Corynebacterium* species is sequencing of the *rpoB* gene (12). Khamis et al. (12) demonstrated that for either the complete or a partial *rpoB* gene sequence the similarity of *C. xerosis* and *C. freneyi* was greatest (>95.0%) among all closely related

TABLE 5. 16S rRNA gene sequence comparison between the *C. xerosis* ATCC 373 type strain and the 18 clinical strains

Alignment position	Position in *C. xerosis*	Base in *C. xerosis*	Event in comparison to *C. xerosis* result	No. of bases in the majority of the 18 clinical strains	No. of bases in the minority of the 18 clinical strains[a]
4	4	N	Point mutation	15 G	
17	17	N	Point mutation	17 G	
19	19	C	Point mutation	15 C	2 T (2586, 3526)
27	27	C	Point mutation	17 G	
130	130	C	Point mutation	18 T	
138	138	G	Point mutation	16 G	2 A (3077, 3437)
155	Between 154 and 155		Insertion	18 T	
180	179	T	Point mutation	17 T	1 A (3296)
217	216	N	Point mutation	18 G	
926	925	C	Point mutation	17 T	1 Y (22)
929	928	G	Point mutation	14 C	3 S (22, 3008, 3034)
930	929	G	Point mutation	14 C	2 S (3008, 3034)
931	930	C	Point mutation	14 G	1 S (22)
940	939	G	Point mutation	14 T	1 K (22)
941	940	T	Point mutation	14 G	3 K (22, 3008, 3034)
942	941	C	Point mutation	14 G	2 S (22, 3008)
943	942	C	Point mutation	18 T	
1055	1054	A	Point mutation	15 G	2 T (3007, 3437), 2 K (22, 3296)
1182	1181	G	Point mutation	18 A	
1205	1204	G	Point mutation	18 T	

[a] Numbers in parentheses represent strain designations.

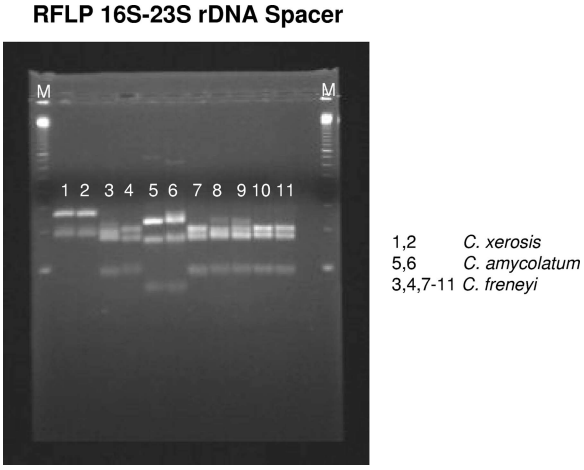

FIG. 1. Lane 1, *C. xerosis* ATCC 373^T; lane 2, *C. xerosis* ATCC 7711; lane 3, *C. freneyi* CIP 106767^T; lane 4, *C. freneyi* ISPB 16799604; lane 5, *C. amycolatum* CIP 103452^T; lane 6, *C. amycolatum* NCTC 7243; lanes 7 to 11, clinical strains 3078, 3172, 3228, 3229, and 3527, respectively; lanes M, 100-bp ladder (Amersham/GE Healthcare, Buckinghamshire, United Kingdom).

Corynebacterium species, indicating again that sequencing approaches directed at identification of *C. freneyi* may not lead to unanimous identification.

Using another molecular genetic technique, RFLP analysis of the 16S-23S rRNA gene intragenic spacer, we definitively assigned all 18 of our clinical isolates to the species *C. freneyi*. However, for the routine clinical laboratory the simple morphological and biochemical features outlined above are certainly sufficient to conclusively identify *C. freneyi* strains.

Based on the large number of strains characterized in the present study, we provide an extended and emended description of *C. freneyi*.

Extended and emended description of *Corynebacterium freneyi* Renaud et al. 2001, corr. Funke & Frodl 2008. *Corynebacterium freneyi* (fre'ney.i. N.L. gen. n. *freneyi* of Freney, to honor Jean Freney, a contemporary French microbiologist).

The description given below is based on the results of studying 21 strains. Cells are gram positive, non-spore-forming, and nonmotile. They are typical club-shaped rods. Colonies are whitish-grayish (19 of 21 strains) or yellowish (2 of 21 strains), dry, and rough, with a distinct wrinkled morphology (see Fig. 2). Colonies are 1 to 2 mm in diameter after 48 h of incubation on a blood-enriched medium. The edges are irregular. Growth

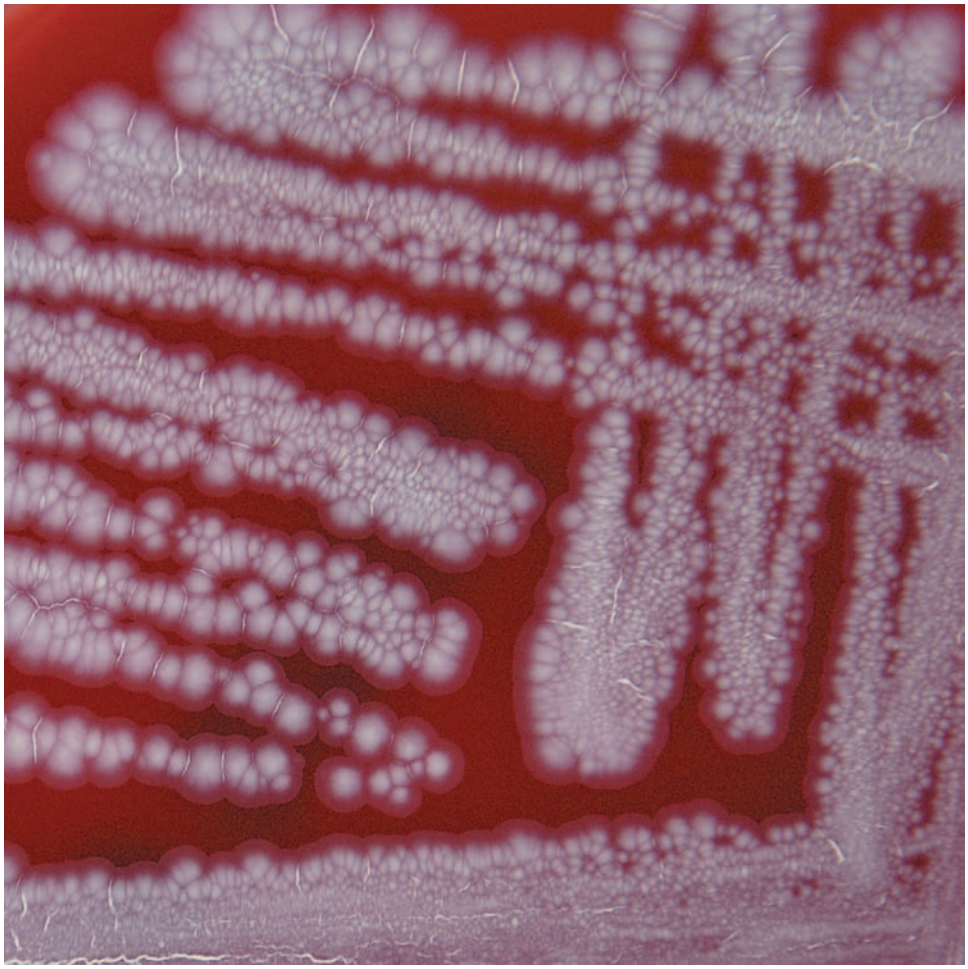

FIG. 2. Colonies of *C. freneyi* on sheep blood agar plates after 48 h incubation at 35°C. Note the wrinkled colonies.

is not enhanced in a medium containing lipids. The organism is catalase positive. Reduction of nitrates is variable. The strains express α-glucosidase, pyrazinamidase, alkaline phosphatase, esterase (C4), esterase lipase (C8), leucine arylamidase (20 of 21 strains), and phosphoamidase. They do not produce pyrrolidonyl arylamidase, β-glucuronidase, β-galactosidase, *N*-acetyl-β-glucosaminidase, valine arylamidase, cystine arylamidase, trypsin, chymotrypsin, α-galactosidase, α-mannosidase, or α-fucosidase. They do not hydrolyze esculin, gelatin, or urea. The CAMP reaction is negative. They produce acid from glucose, maltose, sucrose, galactose (20 of 21 strains), D-fructose, D-mannose, trehalose, D-turanose, and 5-keto-gluconate. Ribose and D-tagatose acidifications are slow and weak. The results of fermentation of lactose and glycogen are variable, and very few strains (2 of 21) ferment mannitol. Xylose, glycerol, erythritol, D-arabinose, and 2-keto-gluconate are not fermented. The cell wall contains *meso*-diaminopimelic acid, arabinose, galactose, and mycolic acids.

The type strain, ISPB 6695110^T (= CIP 106767^T = DSM 44506^T), was isolated from pus from a toe.

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