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Biochemical Differentiation and Comparison of *Desulfovibrio* Species and Other Phenotypically Similar Genera

Yumi A. Warren,[1] Diane M. Citron,[1]* C. Vreni Merriam,[1] and Ellie J. C. Goldstein[1,2]

R. M. Alden Research Laboratory, Santa Monica, California 90404,[1] and UCLA School of Medicine, Los Angeles, California 90095[2]

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Seventeen human clinical isolates representing four species of *Desulfovibrio* were characterized using 16S rRNA gene sequences and tests for catalase, indole, nitrate, bile, urease, formate-fumarate stimulation, desulfoviridin, motility, and hydrogen sulfide production, plus susceptibility to antimicrobial agents. Eighty additional strains representing 10 phenotypically similar genera (*Bilophila*, *Selenomonas*, *Capnocytophaga*, *Campylobacter*, *Bacteroides*, *Sutterella*, *Anaerobiospirillum*, *Dialister*, *Veillonella*, and *Mobiluncus*) were included for comparison. All *Desulfovibrio* species produced H_2S and were desulfoviridin positive, and all *Desulfovibrio* species except *D. piger* were motile. The four *Desulfovibrio* species could be distinguished from each other using tests for catalase, indole, nitrate, urease, and growth on bile, with the following results (positive [+], negative [−], growth [G], and no growth [NG]): for *D. piger*, −, −, −, −, and G, respectively; for *D. fairfieldensis*, +, −, +, −, and G, respectively; for *D. desulfuricans*, −, −, +, +, and NG, respectively; and for *D. vulgaris*, −, +, −, −, and G, respectively. Resistance to the 10-μg colistin disk separated the *Desulfovibrio* species from most of the other genera, which were usually susceptible. These simple tests were useful for characterizing the *Desulfovibrio* species and differentiating them from other phenotypically similar genera.

Sulfate-reducing bacteria are a diverse group of organisms that include *Desulfovibrio*, *Desulfomicrobium*, *Desulfobulbus*, *Desulfobacter*, *Desulfococcus*, *Desulfosarcina*, *Desulfobacterium*, *Desulfonema*, *Desulfotomaculum*, and *Thermodesulfobacterium*. This group of organisms has a variety of morphologies, biochemical properties, and nutritional requirements. With the exception of *Desulfovibrio* spp., they are found only in the natural environment (4, 30).

Desulfovibrio is one of the first genera described and the most thoroughly studied genus among the sulfate-reducing bacteria (4, 5). These are sulfate-reducing, nonfermenting, anaerobic, gram-negative bacilli characterized by the presence of a pigment, desulfoviridin, which fluoresces red in alkaline pH and blue-green in acid pH under long-wavelength UV light (15, 28) and by a strong sulfurous odor in broth media.

Desulfovibrio spp. are ubiquitous, found in the environment, such as soil, water, and sewage, as well as in the digestive tracts of animals and humans (4, 5, 17, 20, 25, 27, 31). Two of the *Desulfovibrio* species, *D. piger* and *D. fairfieldensis*, have never been isolated from outside the human body and can be considered natural inhabitants of the intestinal tract, where sulfates abound (10, 17, 19). The organisms are usually recovered from mixed cultures and may cause human infections. They have been isolated from a variety of sources, such as brain abscess (18, 21), periodontal pocket (2, 16), blood (11, 18, 22, 25), appendix (1), liver (26, 29), urine (14), and colon and bowel (6, 17, 18, 30, 31). Bacteremia appears to be the only instance where a *Desulfovibrio* sp. was the sole pathogen (11, 25).

The organisms grow very slowly, taking 3 to 5 days to produce tiny transparent, nonhemolytic colonies on anaerobic blood agar plates and therefore are easily missed or overgrown in mixed cultures (17, 18). Yet, identification of this group is important because similar organisms, such as *Campylobacter* or *Bilophila wadsworthia*, have quite different resistance patterns (3). Genetic methods, such as 16S rRNA gene sequencing (8, 9, 14, 29), have shown that among sulfate-reducing bacteria are organisms that are phenotypically similar yet phylogenetically diverse (4, 27). This is also true in reverse, as in the case of phenotypically dissimilar "*Desulfomonas pigra*," found by 16S rRNA gene sequencing to be closely related to *Desulfovibrio* spp. and hence reclassified into that genus (19).

Since genetic methods are not routinely employed in clinical laboratories, more-practical ways to identify fastidious organisms are still required. Commercial kits, such as the Rapid ANA II system (Remel, Kansas City, MO), Vitek ANI card (bioMerieux, St. Louis, MO), and API 20A (bioMerieux), are available, but *Desulfovibrio* species are generally nonreactive and/or are not included in the database (14, 22, 29). In one study, the BBL Crystal anaerobe identification system (Becton Dickinson, Sparks, MD) correctly identified all *Desulfovibrio* spp. and *Bilophila* sp. to the genus level. The genus identification was based on positive reactions for catalase and on hydrolysis of L-methionine and escosyl; however, the system failed to identify individual species (3).

Our laboratory has acquired 17 human strains of *Desulfovibrio* spp. over the past several years, 15 from intra-abdominal infections and two from blood cultures. Using those strains, we set about to devise a practical system for clinical laboratories to identify *Desulfovibrio* spp. to the species level and to differen-

* Corresponding author. Mailing address: R. M. Alden Research Lab, 2001 Santa Monica Blvd., Suite 685W, Santa Monica, CA 90404. Phone: (310) 453-7820. Fax: (310) 453-7670. E-mail: d.m.citron@verizon.net.

tiate them from other similar gram-negative bacilli. We also performed antimicrobial susceptibility testing on the *Desulfovibrio* isolates and compared the results to those of *Bilophila wadsworthia*.

MATERIALS AND METHODS

The organisms studied were *Desulfovibrio* spp. (17), *Bilophila wadsworthia* (11), *Selenomonas* spp. (6), *Capnocytophaga* spp. (6), *Campylobacter* spp. (6), *Bacteroides ureolyticus* (8), *Sutterella wadsworthensis* (8), *Anaerobiospirillum* spp. (7), *Dialister pneumosintes* (5), *Veillonella* spp. (8), *Mobiluncus* spp. (5), and 10 unnamed organisms. We also included ATCC strain 7757, originally classified as *Desulfovibrio desulfuricans* but subsequently reidentified as *D. vulgaris* (5).

All *Desulfovibrio* isolates and 26 of the other strains were extracted and amplified, using a universal primer and QIAGEN kits (QIAGEN, Inc., Valencia, CA), as previously described (9, 17, 18, 31). The DNA products were sent to a reference laboratory (Laguna Scientific Laboratory, Laguna Beach, CA) for sequencing. The sequences received were analyzed using BLASTN 2.2.9. and were compared with all the available bacterial sequences from the GenBank database. Through 16 S rRNA gene sequencing, our *Desulfovibrio* isolates were identified as 10 strains of *D. fairfieldensis*, three strains of *D. desulfuricans*, two strains of *D. piger*, and two strains of *D. vulgaris*.

All isolates were taken from 20% skim milk frozen at −70°C and transferred at least twice on brucella agar plates (Hardy Diagnostics, Santa Maria, CA) before testing. They were finally subcultured on brucella agar plates with kanamycin (1,000-μg), vancomycin (5-μg), and colistin (10-μg) differential antibiotic disks (Hardy Diagnostics, Santa Maria, CA). Further tests included catalase, spot indole, nitrate reduction, growth on bile, urease, growth stimulation with formate/fumarate, motility, desulfoviridin, and SIM (sulfide-indole-motility) medium tubes, as described in the *Wadsworth-KTL Anaerobic Bacteriology Manual* (13).

Nitrate disks (Hardy Diagnostics, Santa Maria, CA) were placed on the first quadrant of each inoculated plate and incubated in the anaerobic chamber for 72 h. After the addition of 1 drop each of nitrate reagents A and B (Anaerobe Systems, Morgan Hill, CA), the appearance of a red color indicated nitrate reduction to nitrite. If there was no color change to red, zinc powder was added. If still no color change occurred, other methods were used to confirm the test result. The alternative methods were prereduced anaerobically sterilized (PRAS) nitrate broth (Anaerobe Systems) and Rapid nitrate disks (Key Scientific Products, Round Rock, TX). The nitrate broth tubes were inoculated from a 0.5 McFarland turbidity standard inoculum suspension in brucella broth and incubated until they showed turbidity. Rapid nitrate disks were inoculated according to the package insert. The same nitrate reagents (reagents A and B) were used for all methods.

Bile growth tests employed two methods: differential bile disks (Hardy Diagnostics) and PRAS bile broth (Anaerobe Systems). Bile disks were placed on the second quadrant where more-distinct colonies could be seen. Bile broth was inoculated from the same inoculum suspension as the nitrate broth.

Urea disks (Hardy Diagnostics) were placed in tubes, which were inoculated with a heavy suspension of the isolate, and incubated up to several days to detect weak reactions. After 24 h, a positive reaction gave a pale pink color, and a negative test appeared orange. No additional positive reactions were detected after 24 h.

Motility tests were also performed using two methods, one method using wet mount and the other using motility tube media with an indicator (Hardy Diagnostics). The presence of desulfoviridin pigment was tested by swiping the colonies with a cotton-tipped swab and then adding 1 drop of 2 N NaOH directly onto the swab. The reaction was immediately observed in a dark box under UV light at 365 nm. Red fluorescence indicated a positive reaction.

Susceptibility testing was performed by agar dilution according to the procedure described in CLSI (formerly NCCLS) M11-A6 (24).

RESULTS

Biochemical test results for the different species are shown in Table 1. The desulfoviridin pigment was observed by red fluorescence and detected in both *Desulfovibrio* spp. and *Bilophila wadsworthia*, and the SIM medium tubes for both genera turned black within several days, indicating H_2S production. It was observed that *D. desulfuricans* and *D. vulgaris* turned the SIM tubes completely black in 1 day. On the other hand, *D. piger* and *D. fairfieldensis* started turning black in 3 days; in general, *D. fairfieldensis* required the longest time to turn black.

Desulfovibrio spp. and *Bilophila* sp. could be differentiated by the 10-μg colistin differential disk pattern, which showed resistance for the former and susceptibility for the latter. Of the *Desulfovibrio* spp., *D. piger* was distinguished by the lack of motility, whereas the other three species were motile. All 10 *D. fairfieldensis* strains produced catalase, unlike the other *Desulfovibrio* spp., which were all catalase negative. Distinguishing features of all three *D. desulfuricans* strains were an inability to grow on bile and positive urease results after 24 h. Both *D. vulgaris* clinical isolates, as well as the ATCC 7757 strain, were the only species that were indole positive.

The nitrate test result was recorded as positive if any one of the three methods tested positive. All 10 *D. fairfieldensis* strains and all three *D. desulfuricans* strains were nitrate positive. Eleven of these 13 strains were positive with the initial nitrate disk tests, and two were positive only with the rapid test. Using nitrate disks, it was our observation that the growth had to be very heavy to have positive results for nitrite and that the addition of zinc powder to a negative test did not give a strong red color. The rapid nitrate test was the most sensitive of the three methods and gave only one false-negative result.

The ability to grow on bile was easiest to see on blood agar with bile disks. Most of the strains that grew on bile grew poorly in PRAS peptone-yeast broth with 20% bile.

The semisolid motility agar failed to show motility of the *Desulfovibrio* strains, and their motility was best demonstrated by microscopic examination of a wet mount. Three of four *Selenomonas flueggei* strains and one of four *Anaerobiospirillum thomasii* strains did not show any sign of motility at room temperature or at 37°C. Since these are motile organisms, they apparently lost their flagella during frozen storage. Although *Capnocytophaga* spp. are gram-negative bacilli, half the strains tested were susceptible to the vancomycin differential disk, which might indicate borderline susceptibility and suggests a gram-positive-like cell wall.

ATCC strain 7757 showed biochemical test results consistent with those of *D. vulgaris* as expected, positive for indole and bile growth tests and negative for all the other tests.

Four groups of organisms were categorized as unnamed organism groups I, II, III, and IV. The five organisms in unnamed organism group I were originally thought to be *D. piger*, with which they were morphologically consistent. They showed similar gram stain morphologies and were susceptible to kanamycin and resistant to vancomycin and colistin differential disks, typical for the genus. However, they were negative for the desulfoviridin pigment and H_2S production in SIM medium tubes. The PCR sequences of all five strains were similar to each other but did not match any sequences in the GenBank database. The other five strains were also phenotypically similar to one of the comparator genera but did not match exactly and thus were sequenced to determine their identity.

The list of antimicrobials and their MICs are summarized in Table 2. All strains of *Desulfovibrio* spp. and *Bilophila* sp. were susceptible to chloramphenicol and metronidazole, most were susceptible to imipenem and clindamycin, and in general, they were resistant to penicillin. *D. fairfieldensis* was significantly

TABLE 1. Biochemical reactions for *Desulfovibrio* and similar organisms

Organism or group	No. tested	Drug resistance[a]			Biochemical reaction (% positive)									Source of isolate (no. of isolates)
		K	V	C	Catalase	Indole	Nitrate	Growth on bile	Urea	F/F[b]	Motility	Desulfoviridin	SIM[c]	
Desulfovibrio piger	2	S	R	R	0	0	0	100	0	0	0	100	100	Peritoneal fluid (2)
Desulfovibrio fairfieldensis	10	S	R	R	100	0	100	100	0	0	100	100	100	Peritoneal fluid (5), abdomen (2), pelvis (1), blood (1), colorectum (1)
Desulfovibrio desulfuricans	3	S	R	R	0	0	100	0	100[d]	100	100	100	100	Peritoneal fluid (2), blood (1)
Desulfovibrio vulgaris	3	S	R	R	0	100	0	100	0	0	100	100	100	Peritoneal fluid (1), abdomen (1), ATCC 7757
Bilophila wadsworthia	11	S/R	R	S	100	0	100	100	36	0	0	100	100	Peritoneal fluid (8), abdomen (2), appendix (1)
Selenomonas flueggei	4	S	R	R	0	0	100	0	0	0	25	0	0	Tongue (4)
Selenomonas infelix	2	S	R	S/R	0	0	100	0	0	0	100	0	0	Tongue (2)
Capnocytophaga ochracea	2	S	R	R	0	0	0	0	0	0	0	0	0	Oral (1), blood (1)
Capnocytophaga gingivalis	2	S	S	R	0	0	0	0	0	0	0	0	0	Oral (1), sputum (1)
Capnocytophaga canimorsus	2	S	S/R	R	50	0	0	0	0	0	0	0	0	Blood (1), human bite (1)
Campylobacter showae	2	S	R	S	0	0	100	0	0	100	100	0	0	Human bite (1), blood (1)
Campylobacter gracilis	3	S	R	S	0	0	100	0	0	100	0	0	0	Tongue (1), tonsil (1), pelvis (1)
Campylobacter curvus	1	S	R	S	0	0	100	0	0	0	100	0	0	Vagina (1)
Bacteroides ureolyticus	8	S	R	S	0	0	100	25	100	100	0	0	0	Skin (5), pelvic fluid (2), unknown (1)
Sutterella wadsworthensis	8	S	R	S	0	0	100	100	0	100	0	0	0	Peritoneal fluid (4), abdomen (2), perirectum (1), unknown (1)
Anaerobiospirillum thomasii	4	S	R	S/R	0	0	0	50	0	0	75	0	0	Stool (4)
Anaerobiospirillum succiniciproducens	3	S	R	S/R	0	0	0	0	0	0	100	0	0	Blood (1), unknown (2)
Dialister pneumosintes	5	S	R	R	0	0	0	0	0	0	0	0	0	Peritoneal fluid (3), abdomen (2)
Veillonella spp.	8	S	R	S	17	0	100	0	0	0	0	0	0	Peritoneal fluid (2), abdomen (2), tongue (2), human Bite (1), unknown (1)
Mobiluncus curtisii	3	S	S	R	0	0	100	0	0	0	100	0	0	Vagina (3)
Mobiluncus mulieris	2	S	S	R	0	0	0	0	0	0	100	0	0	Vagina (2)
Unnamed organism groups														
I	5	S	R	R	0	0	0	100	0	0	0	0	0	Peritoneal fluid (5)
II	2	S	R	R	0	0	50	0	0	0	0	0	0	Skin (2)
III	1	S	R	S	0	0	0	0	0	0	0	0	0	Appendix (1)
IV	2	S	R	S	0	0	100	50	0	0	0	0	0	Peritoneal fluid (2)

[a] Resistance (R) or susceptibility (S) to 1 mg of kanamycin (K), 5 μg of vancomycin (V), or 10 μg of colistin (C).
[b] F/F, sodium formate/sodium fumarate stimulation.
[c] Sulfide-indole-motility medium for detection of H_2S.
[d] Positive after 24 h.

more resistant than the other three *Desulfovibrio* spp. and also *Bilophila* sp.: all 10 *D. fairfieldensis* strains were resistant to piperacillin-tazobactam and ceftriaxone, and most were resistant to ticarcillin-clavulanic acid, cefoxitin, and ertapenem. *D. vulgaris* appeared to be the most susceptible species in this genus, with our two strains susceptible to all the antimicrobials tested, including penicillin. The nitrocefin disk test for beta-lactamase gave variable results, with only three strains among *Desulfovibrio* spp. being positive.

DISCUSSION

Phylogenetically related by DNA profile, *Desulfovibrio* spp. and *Bilophila* sp. are found in similar sites of the human body (23) and also share phenotypic characteristics. In fact, before *Bilophila* sp. was first described, positive tests for the desulfoviridin pigment and H_2S production would have classified the organism as *Desulfovibrio* spp. However, the two genera have different mechanisms for sulfate reduction (15, 23, 26).

Loubinoux et al. (18) reported somewhat different biochemical reactions from ours for some of their isolates. Firstly, two out of four strains identified as *D. desulfuricans* in their study were catalase and nitrate positive, whereas all three of our strains of *D. desulfuricans* were catalase negative. Secondly, the four strains of *D. fairfieldensis* in their study were described as catalase positive and nitrate negative, whereas all 10 of our strains of *D. fairfieldensis* were consistently both catalase and

TABLE 2. In vitro susceptibilities of *Desulfovibrio* species and *Bilophila wadsworthia* to 14 antimicrobial agents

Species and antimicrobial	MIC (μg/ml)[a]		
	Range	50%	90%
Desulfovibrio fairfieldensis (*n* = 10)			
Penicillin	4–>4	>4	>4
Ampicillin-sulbactam	4–32	16	32
Amoxicillin-clavulanic acid	2–8	8	8
Piperacillin-tazobactam	>128–>128	>128	>128
Ticarcillin-clavulanic acid	0.125–>128	128	128
Cefoxitin	32–128	64	128
Ceftriaxone	128–>128	>128	>128
Levofloxacin	0.5–>16	1	>16
Moxifloxacin	0.5–>16	0.5	>16
Ertapenem	4–>32	>32	>32
Imipenem	0.125–2	1	1
Chloramphenicol	4–8	6	8
Clindamycin	0.125–>32	0.25	0.25
Metronidazole	0.125–0.5	0.25	0.25
Desulfovibrio piger (*n* = 2)			
Penicillin	1–>4		
Ampicillin-sulbactam	0.25–4		
Amoxicillin-clavulanic acid	0.125–1		
Piperacillin-tazobactam	32–128		
Ticarcillin-clavulanic acid	2–4		
Cefoxitin	2–4		
Ceftriaxone	2–4		
Levofloxacin	2–4		
Moxifloxacin	2–4		
Ertapenem	0.25–4		
Imipenem	0.06–0.125		
Chloramphenicol	4–8		
Clindamycin	≤0.06–0.5		
Metronidazole	0.125–0.25		
Desulfovibrio desulfuricans (*n* = 3)			
Penicillin	4–>4		
Ampicillin-sulbactam	1–1		
Amoxicillin-clavulanic acid	0.25–0.5		
Piperacillin-tazobactam	32–64		
Ticarcillin-clavulanic acid	2–4		
Cefoxitin	128–>128		
Ceftriaxone	4–16		
Levofloxacin	0.5–2		
Moxifloxacin	0.125–2		
Ertapenem	0.25–0.5		
Imipenem	0.25–0.25		
Chloramphenicol	4–8		
Clindamycin	0.25–1		
Metronidazole	0.125–0.25		
Desulfovibrio vulgaris (*n* = 3)			
Penicillin	0.25–0.25		
Ampicillin-sulbactam	0.125–0.125		
Amoxicillin-clavulanic acid	≤0.06–≤0.06		
Piperacillin-tazobactam	≤0.06–>128		
Ticarcillin-clavulanic acid	0.5–64		
Cefoxitin	1–4		
Ceftriaxone	0.5–16		
Levofloxacin	≤0.06–0.125		
Moxifloxacin	≤0.06–≤0.06		
Ertapenem	≤0.015–0.25		
Imipenem	0.125–1		
Chloramphenicol	1–4		
Clindamycin	0.125–0.125		
Metronidazole	≤0.06–≤0.06		
Bilophila wadsworthia (*n* = 11)			
Penicillin	0.5–>4	4	>4
Ampicillin-sulbactam	1–>32	2	8
Amoxicillin-clavulanic acid	0.5–>32	1	8
Piperacillin-tazobactam	2–>128	8	>128
Ticarcillin-clavulanic acid	0.125–>128	0.25	64
Cefoxitin	4–>128	16	128
Ceftriaxone	0.125–>128	1	4
Levofloxacin	0.25–2	0.5	1
Moxifloxacin	0.125–2	0.25	1
Ertapenem	≤0.015–>32	0.03	0.5
Imipenem	0.125–>32	0.25	1
Chloramphenicol	2–8	4	8
Clindamycin	≤0.06–>32	0.25	1
Metronidazole	≤0.06–0.25	0.125	0.25

[a] 50% and 90%, MICs at which 50 and 90% strains were inhibited, respectively.

nitrate positive. The reason for this discrepancy is not clear but may be due to differences in test methods.

Loubinoux et al. (17) suggested that *D. fairfieldensis*, *D. desulfuricans*, and *D. piger* were the three species that have been isolated from human sources. Our study and Johnson et al. (12) have reported *D. vulgaris* from human sources as well.

The first known case of *Desulfovibrio desulfuricans* infection was reported by Porschen et al. in 1977 (25), and the strain was described as catalase positive but urease and nitrate negative (the nitrate reaction was too weak to be considered positive). According to our own test results, however, *D. desulfuricans*, as identified by sequence matching to a level greater than 99%, is characterized by positive urease and nitrate tests. *D. fairfieldensis* was the only species that was both catalase and nitrate positive, and therefore it is more likely that the 1977 case is actually the first reported instance of infection by *D. fairfieldensis*. This biochemical characterization would agree with the finding by Dzierzewicz et al., who analyzed *D. desulfuricans* through fatty acid profiles (7) and genetic fingerprinting (8) and found great homogeneity among the intestinal strains of this organism, making the identification of Porschen's isolate unlikely.

Except for isolates from case reports (11, 14, 18, 22, 29), to date, there are few studies that report antimicrobial susceptibilities for *Desulfovibrio* organisms. Lozniewski et al. (20) tested 16 isolates and found that all were susceptible to imipenem and metronidazole and resistant to penicillin, piperacillin-tazobactam, and cefoxitin. Although which species were tested was not indicated in their report, the susceptibility results match those we obtained with *D. fairfieldensis*.

Finally, our own results agree with those of Devereux et al., who suggested that 16S RNA showed *D. desulfuricans* ATCC 7757 to be closely related to *D. vulgaris* (5). This is also borne out by the MICs for that strain, which, like other *D. vulgaris* strains, are the least resistant of the *Desulfovibrio* organisms.

Loubinoux et al. (18) suggested that *Desulfovibrio fairfieldensis* may be the most potentially pathogenic among all *Desulfovibrio* species, and our study found that this species was the most common isolate from intra-abdominal specimens, as well as being the most antimicrobial resistant. Because of variation in susceptibility patterns, it is therefore clinically important to identify *Desulfovibrio* spp. to the species level.

Conclusion. More sulfate-reducing organisms will likely be isolated from human sources in the future, and their identification properties may become more complex. Currently, identification of human isolates of *Desulfovibrio* spp. to the species

level can be accomplished by performing gram stain (typically the organisms stain bipolar), differential disks, catalase, indole, nitrate, urease, motility, H_2S production in SIM medium, and detection of a desulfoviridin pigment. The last test mentioned is the key reaction to distinguish *Desulfovibrio* spp. and five organisms in unnamed organism group I that were phenotypically very similar to *Desulfovibrio*. Additional studies are currently under way to further characterize and name the unidentifiable strains.

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