Leptotrichia

For a long time, Leptotrichia were considered opportunistic pathogens until recent reports that indicated that they may be pathogenic.

From: Atlas of Oral Microbiology, 2015

Related terms:

Streptococcus, Lactobacillus, Fusobacterium, Microbiome, Prevotella, Bacterium, Microflora, Clustered Regularly Interspaced Short Palindromic Repeat, Genus

View all Topics

Supragingival Microbes

In Atlas of Oral Microbiology, 2015

3.2.1 Leptotrichia

Leptotrichia is a gram-negative anaerobic bacillus and is a very commonly observed genus in the human oral cavity. The genus Leptotrichia was first found in 1896 and was named leptothrix as it was isolated from the rabbit uterus. For a long time, *Leptotrichia* were considered opportunistic pathogens until recent reports that indicated that they may be pathogenic. *Leptotrichia* can be isolated from the oral cavity and are mainly found in bacterial biofilms. It can also separate from the vagina and the uterus of pregnant women.

The *Leptotrichia* cell measures 0.8–1.5 μm × 5–20 μm. They can be straight or curved rod shapes. The ends of the cell (either one or both ends) can be sharp or rounded. Cells normally organize as pairs or in a chain (Figure 3.20(A)–(C)). The cells do not produce spores and are nonmotile. Fresh cultures can be stained gram-positive. Under the light microscope, both gram-negative and gram-positive cells can be observed on a single slide.

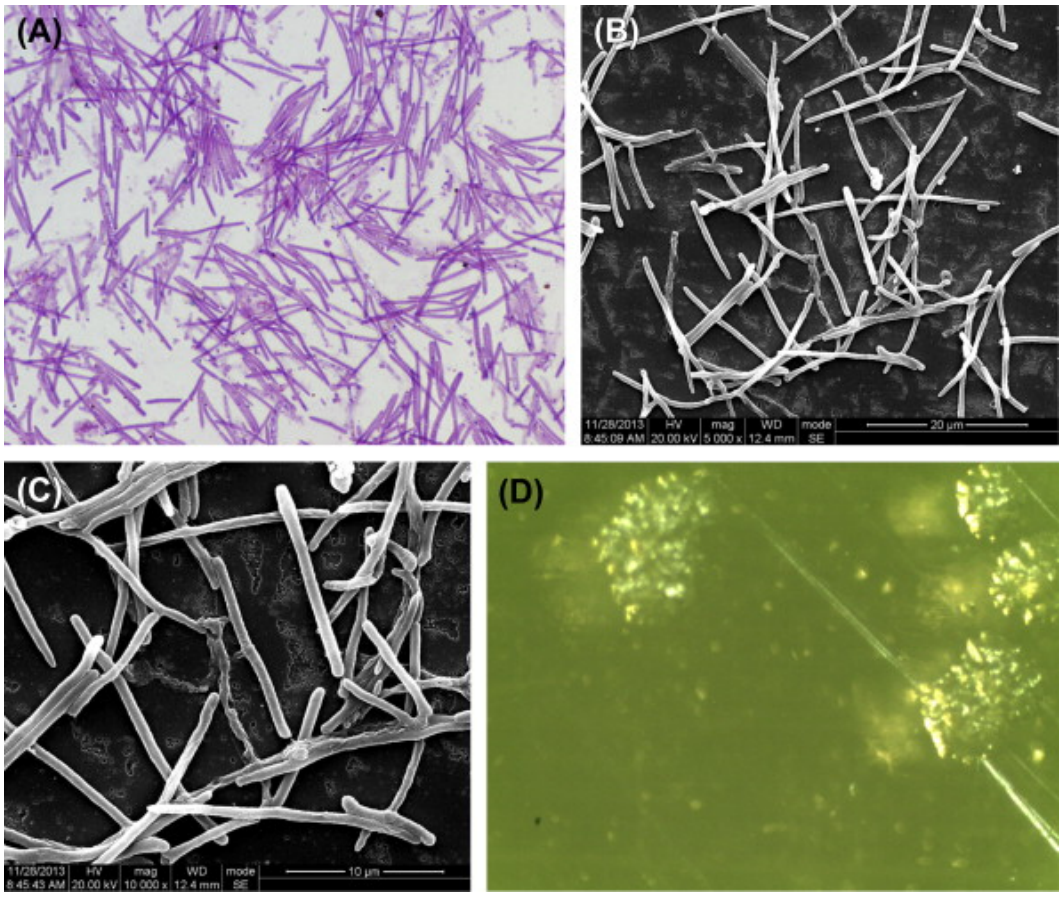

Figure 3.20. (A) *Leptotrichia* cells (Gram stain). (B) *Leptotrichia* cells (SEM). (C) *Leptotrichia* cells (SEM). (D) *Leptotrichia* colonies.

After culturing in anaerobic blood agar for 1–2 days, *Leptotrichia* can form 1–2-mm, raised, and transparent colonies with smooth and filamentous edges (Figure 3.20(D)). Sometimes polymorphous colonies are also formed.

Leptotrichia grow best under anaerobic conditions. Cultures require 5–10% CO_2. The ideal temperature for culture growth is between 35 °C and 37 °C, while *Leptotrichia* cells stop growing when temperatures drop below 25 °C. The ideal pH for culturing these cells is between pH 7.0 and 7.4. Growth is not inhibited by 20% bile.

Leptotrichia is biochemically active. It can ferment amygdalin, cellobiose, fructose, glucose, maltose, mannose, melezitose, salicin, sucrose, and trehalose to produce acid. The terminal products of lactose and starch fermentation are variable. *Leptotrichia* does not ferment arabinose, dulcitol, glycerol, inositol, inulin, mannitol, melibiose, raffinose, rhamnose, ribose, sorbitol, and xylose. The cells do not produce indole, catalase, urease, H_2S, phospholipase, and ammonia gas.

The percent GC in *Leptotrichia* DNA is 25% (by Tm or Bd). The type strain is ATCC14201.

> Read full chapter

Microbiology, Inflammation, and Viral infections

Prabodh K. Gupta, Cindy McGrath, in Comprehensive Cytopathology (Third Edition), 2008

Leptotrichia buccalis

These microbes, also known as just Leptotrichia or Leptothrix, are Gram-negative, non-spore-forming anaerobic organisms. They occur in the oral and vaginal cavities as very thin, segmented, large, filamentous structures. Occasionally, branching may be observed (Fig. 7.35). Morphologically they may be indistinguishable from certain forms of Doederlein's bacillus. Most frequently (75–80%), cases of Leptotrichia have concomitant *T. vaginalis* infection. Numerous other infective organisms, including *Candida* and *G. vaginalis*, may occur in the presence of *L. buccalis* infection.

Bibbo and Wied[13] made an investigative study on the prevalence of *Leptotrichia* in cervicovaginal smears. They observed *Leptotrichia* organisms in 75% cases with trichomonads, 1.5% with Doederlein's bacillus, and about 1% among patients with fungal or BV infection. Nearly half (47%) of the 1,000 patients studied were oral contraceptive users. Pregnancy and menopause were other physiologic features, followed by the postpartum state, that were often associated with the presence of *L. buccalis* in cervical smears. Sometimes acute inflammatory changes may be observed in the presence of *Leptotrichia*.

> Read full chapter

The Gastric Microbiome in Benign and Malignant Diseases

Thais Fernanda Bartelli, ... Emmanuel Dias-Neto, in Microbiome and Metabolome in Diagnosis, Therapy, and other Strategic Applications, 2019

General Dysbiosis in Gastric Cancer

Other taxa enriched in gastric cancer patients are Lactococcus, Veillonella, Fusobacterium, and Leptotrichia. Analysis of microbial metabolic output in those patients identified an increase in lactic acid–producing bacteria, enrichment of short-chain

fatty acid production pathways, and enrichment of proinflammatory oral bacterial species [31].

Dysbiosis in intestinal metaplasia and gastric cancer subjects has been characterized, as compared to superficial or atrophic gastritis. Additionally, oral bacteria, such as Peptostreptococcus *stomatis* and *Dialister pneumosintes*, had significantly higher abundance in gastric cancer as compared to the other stages analyzed, and together with Streptococcus *anginosus*, *Parvimonas micra* and *Slackia exigua*, could differentiate gastric cancer patients from superficial gastritis cases [32].

Whether the gastric mucosal dysbiosis is the causative agent or a consequence of altered mucosal physiology/tumor microenvironment is yet to be fully uncovered. Controlled in vivo experiments, through the introduction of specific known bacteria, will be required to elucidate their role [33]. Nonetheless, gastric cancer has been subdivided into four molecular subtypes by the TCGA (The Cancer Genome Atlas) [34], one of them defined according to the presence of EBV. Future studies might well consider the results of gastric microbiome analysis [33].

> Read full chapter

Bacteroides

Sheila Patrick, in Molecular Medical Microbiology (Second Edition), 2015

Classification and Taxonomic Position

The genus Bacteroides, having initially been a repository for any Gram-negative strictly anaerobic, non-spore-forming, rod-shaped bacteria that were neither Fusobacterium spp. nor Leptotrichia spp., has been refined over the years. The revised taxonomic position of the *Bacteroides* and related genera as detailed in the second edition of *Bergey's Manual of Systematic* Bacteriology is based on phylogenetic analyses of 16S rRNA gene sequences [6].

In the wider taxonomic picture, *Bacteroides* fall into the new phylum Bacteroidetes, formerly *Cytophaga-Flexibacter-Bacteroides* or *Cytophaga-Flavobacterium-Bacteroides* (CFB). Bearing in mind the diverse phenotypes of the bacteria within this phylum, which includes obligate aerobes of the genus Flavobacterium with growth temperature ranges of 0–30°C, this is perhaps an unexpected taxonomic association. Interestingly, this phylum is phylogenetically divergent from other eubacteria. The phylum Proteobacteria, which contains Gram-negative bacteria such as *Escherichia coli* and Pseudomonas aeruginosa and the phylum Firmicutes, which contains

Gram-positive bacteria such as Clostridium spp. and Staphylococcus spp., are taxonomically more closely related to each other than to the Bacteroidetes [7].

The genus *Bacteroides* sensu stricto and the '*B. fragilis* group', as *B. fragilis* and related species were sometimes referred to, has changed significantly with the addition of novel species and the reclassification of others (Table 51.2). Of note is the reclassification of *B. distasonis* and *B. merdae* to the new genus Parabacteroides on the basis of 16S rRNA gene sequence data [8]. The genus *Bacteroides* contains a further eight misplaced species which will no doubt be subject to future reclassification when valid new genera are defined. The reader is therefore cautioned to determine the current taxonomic status of species referred to as '*Bacteroides*' in the earlier as well as current literature and is directed to *Bergey's Manual* for details of the taxonomic history and current status of the genus *Bacteroides.*

Table 51.2. Position of *Bacteroides* and *Parabacteroides* sensu stricto in the Phylum Bacteroidetes[a]

Phylum XIV	**Bacteroidetes**		
Class I	Bacteroidia		
Order I	Bacteroidales		
Family I	*Bacteroidaceae*		
Genus	*Bacteroides*		*Parabacteroides*
Species	*fragilis*[c] *(type species)*	*acidifaciens*	*distasonis*[b,c]
	caccae[b,c]	*coprocola*[c]	*merdae*[b,c]
	eggerthii[b,c]	*coprosuis*	*goldsteinii*[c]
	ovatus[b,c]	*dorei*[c]	*johnsonii*[c]
	stercoris[b,c]	*finegoldii*[c]	
	thetaiotaomicron[b,c]	*helcogenes*	
	uniformis[b,c]	*intestinalis*[c]	
	vulgatus[b,c]	*massiliensis*[c]	
		nordii[c]	
		plebius[c]	
		pyogenes	
		salyersiae[c]	
		suis	
		tectus	
		uniformis[c]	

Note: B. capillosus, cellulosolvens, coagulans, galacturonicus, pectinophilus, polypragmatus, splanchnicus *and* xylanolyticus *are incertae sedis.*

a As defined in Bergeys's Manual of Systematic Bacteriology *2nd Edition.*

b Formerly Bacteroides sensu stricto.

c *Bacteroides* spp. isolated from humans.

B. fragilis strains can be divided into two divisions on the basis of DNA homology [9], ribotyping, analysis of PCR-generated fragment patterns, insertion sequence content [10] and small-subunit rDNA sequencing [11]. The authors of these studies also noted that *B. fragilis* DNA homology division I lack the *cfiA* gene (also called *ccrA*) which encodes a metallo-□-lactamase (Ambler class B enzyme). This enzyme confers resistance to the majority of □-lactam antibiotics (e.g. carbapenems, imipenem and meropenem) and also is not susceptible to inhibitors such as clavulanic acid, sulbactam and tazobactam. This gene was identified in about 3% of isolates. Only about a third of the strains examined, however, expressed this □-lactamase; the gene being silent in the remainder of these strains. The *cfiA* gene, and indeed other genes in *Bacteroides* spp. (see below), is activated by insertion sequences (IS) which encode the *B. fragilis* consensus promoter (reviewed in [12]). Interestingly, three insertion sequences (*IS4351, 942* and *1186*), which provided promoter sequences for the transcription of *cfiA*, were also identified. It is possible to distinguish between division I and II *B. fragilis* by detection of the *cfiA* encoded metallo-□-lactamase by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) [13]. Division I strains, which represent the majority of clinical isolates, lack the *cfiA* gene but carry the *cepA* gene which encodes an active site serine □-lactamase, related to the Ambler class A enzymes (see 'Antimicrobial Agents' below for further discussion).

> Read full chapter

Precision Medicine

Joel Faintuch, Jacob J. Faintuch, in Microbiome and Metabolome in Diagnosis, Therapy, and other Strategic Applications, 2019

Rheumatological and Autoimmune Conditions

Behçet's disease is not ubiquitous; however, similarities with Crohn's disease have prompted microbiome investigations in the gut as well as oral cavity [40,41]. Fecal samples revealed depletion of Roseburia and *Subdoligranulum* along with low bu-

tyrate production. Oral metagenomics pointed to a different direction with abundant Haemophilus parainfluenzae and depleted *Alloprevotella rava* and Leptotrichia spp. Although encouraging, these signatures will require confirmation before they can be translated to clinical practice.

Gout is a condition with solid metabolomic credentials, represented by derangements of purine metabolism and uric acid. However, they stem from the host, not the microbiome. In this sense it is somewhat surprising that Bacteroides, Porphyromonadaceae, *Rhodococcus*, *Erysipelatoclostridium*, and Anaerolineaceae were upregulated in fecal samples [42]. Although a mechanistic explanation is lacking, integration between metabolome and microbiome shifts could emerge in future studies.

Rheumatoid arthritis is another challenge. In early disease an expansion of Prevotella *copri* has been registered [43]. In a more heterogeneous cohort an abundance of Actinobacteria, along with reduction in conventional phyla, was the most visible shift. At the genus level, higher proportions of Collinsella, Eggerthella, and *Faecalibacterium* occurred. Collinsella was associated with proinflammatory cytokine production, as well as gut permeability disruption, and disease severity [44].

> Read full chapter

Reprogramming the Genome: Applications of CRISPR-Cas in Non-mammalian Systems Part A

Lakkakula Satish, ... Ariel Kushmaro, in Progress in Molecular Biology and Translational Science, 2021

7.3.1 SHERLOCKv2

SHERLOCKv2 is a multiplexed, rapid and attomole sensitive method for nucleic acid detection from clinical specimens within an hour.[78] SHERLOCKv2 comprises the following distinguished characteristic features,

1. 3.5-fold higher signal amplification by combining an auxiliary CRISPR-associated protein, Csm6 with Cas13 (Fig. 5B).
2. Multiplexed nucleic acid detection at 2 aM sensitivity in a four-channel single-reaction using orthogonal CRISPR-Cas enzymes (Fig. 5C).
3. Lateral flow readout.

In multiplexing, more than one Cas enzymes are used namely, *Lwa*Cas13a (Leptotrichia *wadei* F0279), *Psm*Cas13b (*Prevotella* sp. MA2016), *Cca*Cas13b (*Capnocytophaga canimorsus* Cc5) and *Lba*Cas13a (*Lachnospiraceae* bacterium NK4A179). Gootenberg et al.[30] have demonstrated AU, UC, AC and GA dinucleotide cleavage property of *Lwa*Cas13a, *Psm*Cas13b, *Cca*Cas13b and *Lba*Cas13a respectively and verified following multiplexing in SHERLOCKv2,

(i) Detection of synthetic ssRNAs of ZKV and Dengue virus in HEX and FAM channel respectively in a single reaction.
(ii) *Lwa*Cas13a uridine reporter in Cy5 channel, *Psm*Cas13b adenine reporter in FAM channel and *As*Cas12a ssDNA reporter in HEX channels for detecting ssDNA target, ZIKV ssRNA and DENV ssRNA respectively, in a single reaction.
(iii) Polynucleotide reporters for *Psm*Cas13b in FAM channel, *Lwa*Cas13a in TEX channel, *Cca*Cas13b in Cy5 channel and *As*Cas12a in HEX channel for detecting ssRNA, ZIKV, DENV and dsDNA respectively, in a single reaction.

Apart from multiplexing, SHERLOCKv2 also uses Csm6 endonuclease of type III CRISPR system to enhance the signal intensity in a reaction. Csm6 is activated by cyclic adenylate or linear adenine homopolymer with terminal 2′, 3′ cyclic phosphate. Activated Csm6 cleaves the ssDNA reporter molecules result in amplified signals in SHERLOCKv2. Cas13 (*Lwa*Cas13a and *psm*Cas13b) collateral activity generates hydroxylated 5′ end and 2′, 3′-cyclic phosphate end cleaved products. These cleaved products efficiently activate *Enterococcus italicus* Csm6 (*Ei*Csm6), *Lactobacillus salivarius* Csm6 (*Ls*Csm6) and *Thermus thermophilus* Csm6 (*Tt*Csm6) proteins. When target activated Cas13 system cleaves Degradation of poly A and poly U homopolymers produce high quantity of hydroxylated 5′ and 2′, 3′-cyclic phosphate end-products. Hence, SHERLOCKv2 accounts FQ coated poly A and poly U ssRNA as a substrate for Cas13 *trans* cleavage and the cleaved products activate *Ei*Csm6 and *Ls*Csm6 effector molecules. Activated Csm6 effectors cleave $(A)_6$ reporters which amplifies signal intensities in the reaction.[30]

Further, SHERLOCKv2 equipped with FAM—biotin coated ssRNA reporter and anti-FAM antibody—gold nanoparticle conjugates (AFabGNP) based lateral flow analysis (LFA). The AFabGNPs are coated at the conjugation pad of lateral flow strip. Absence and presence of target nucleic acids in the sample leaves the intact and cleaved FAM-biotin reporter molecules in the reaction. Intact FAM- biotin reporters absorb AFabGNPs from conjugation pad and accumulate them at first line and prevent binding with protein A on second line. Whereas, cleaved reporter molecules reduce accumulation of AFabGNP at first line and release signal on second line. Gootenberg et al.[30] have demonstrated the detection of ZIKV and DENGUE ssRNA within 90 min with 2 aM sensitivity.

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The Microbiome at Other Mucosal Sites

Paul A. Cardenas, William O.C. Cookson, in Mucosal Immunology (Fourth Edition), 2015

Dental Disease

Tooth decay with dental caries is one of the most common human diseases. It has been suggested that they first resulted from a shift in the oral microbiome as early humans made the transition from hunter-gathering to farming (Adler et al., 2013). Several genera of bacteria may cause caries, including Streptococcus, Veillonella, Bifidobacterium, Actinomyces, Granulicatella, Leptotrichia, and *Thiomonas* (Kaur et al., 2013; Ling et al., 2010; Wade, 2013). The importance of diet has been demonstrated by observations that breast-fed infants have a lower prevalence of these pathogenic genera and a higher abundance of Lactobacillus spp. (Holgerson et al., 2013; Ling et al., 2010).

Endodontic disease of the soft tissues inside of the tooth is another common oral bacterial infection with a polymicrobial origin (Peciuliene et al., 2008; Sundqvist, 1994). Anaerobic proteolytic bacteria are most commonly related to endodontic infections and can produce complications that can diverge from teeth loosening to systemic infections. The phyla most commonly related to this disease are Firmicutes, Bacteroidetes, and Actinobacteria (Munson et al., 2002). Microbiome analysis on samples from endodontic infection patients has shown a higher occurrence of Prevotella and Fusobacterium and less abundance of *Streptococcus* spp. (Hsiao et al., 2012). In addition, in the same study, Granulicatella adiacens, Eubacterium *yurii*, Prevotella melaninogenica, *Prevotella salivae*, Streptococcus mitis, and Atopobium *rimae* were significantly more common in root infections (Hsiao et al., 2012). Uncommon genera of Firmicutes also have been associated with root canal infections, specifically Dialister *pneumosintes* and other *Dialister* spp. (Munson et al., 2002).

> Read full chapter

Infectious Disorders of the Lower Genital Tract

Thing Rinda Soong, ... Alvaro C. Laga, in Diagnostic Gynecologic and Obstetric Pathology (Third Edition), 2018

Clinical Background

Various terms have been used to describe bacterial vaginosis (BV), including non-specific vaginitis, Haemophilus vaginitis, Corynebacterium vaginitis, Gardnerella *vaginalis* vaginitis, and anaerobic vaginosis. Bacterial vaginosis represents a complex shift in the normal vaginal flora. It is characterized by a reduction in the prevalence and concentration of hydrogen peroxide–producing lactobacilli and an increase in the prevalence and concentration of a number of microbes that have been implicated in the disease process, including Gardnerella vaginalis, Atopobium and Mobiluncus spp., Mycoplasma hominis, anaerobic gram-negative rods belonging to the genera Prevotella, Porphyromonas, and Bacteroides, as well as Peptostreptococcus, Leptotrichia, and *Sneathia* spp. and BV-associated bacterium 1 (BVAB1) to BVAB3.[34,35] It is thought that BV results from the presence of a multispecies vaginal biofilm in which *G. vaginalis* is the predominant species.[36] *G. vaginalis* has been detected in culture samples from nearly all symptomatic women with BV and in approximately 50% of the vaginal microflora of healthy women.[35]

It has been estimated that about 20% to 30% of reproductive age women worldwide suffer from BV. Risk factors include the number of lifetime sex partners, early age of sexual debut, regular douching, and being a commercial sex worker.[35] Associations of BV with other sexually transmitted diseases and health issues have been reported, (e.g., HIV, herpes simplex virus type 2 [HSV2], chlamydia, gonorrhea),[35] as well as preterm births, and pelvic inflammatory disease.[37] Although causality and underlying biologic mechanisms have not been established, studies have suggested that BV is a potential risk factor of acquisition of other sexually transmitted infections (STIs), particularly in high-risk women.

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Bacterial Vaginosis-Associated Bacteria

Elisa Margolis, David N. Fredricks, in Molecular Medical Microbiology (Second Edition), 2015

Clinical Diagnosis of Bacterial Vaginosis

In clinical practice, BV is diagnosed on the basis of the Amsel criteria, specifically the presence of three of the following four changes in the vaginal discharge: thin greyish homogeneous discharge, amine odour when potassium hydroxide is added to secretions (positive 'whiff test'), a vaginal pH greater than 4.5, and bacteria coating the surface of epithelial cells (clue cells) when viewed by wet-mount microscopy

(>20% of the total cells on 100× magnification) [64]. Modern molecular techniques have allowed for the Amsel score components to be characterized microbiologically; for clue cells there is a positive association with the presence of Leptotrichia/*Sneathia* spp., *A. vaginae*, BVAB1 or *G. vaginalis* [65] and an amine odour is associated with *A. vaginae* or BVAB1 [66]. While this diagnostic test can be done quickly and routinely in primary care clinics, there is interobserver variability between clinicians [67].

A more definitive diagnostic approach for BV is the Nugent criteria based on the proportion of different bacterial morphotypes on a Gram-stained slide of vaginal secretions with a score of 7 or higher indicating BV (normal score being≤3) [68]. The weighted score computed using these criteria is presumed to reflect the relative abundance of: lactobacilli, *G. vaginalis*, Prevotella, Porphyromonas, and curved Gram-negative rods. The Nugent score is widely regarded as the gold standard for BV diagnosis in research studies but is considered cumbersome in comparison to the Amsel criteria in clinical practice.

Inconsistencies exist between diagnosis of BV by the Amsel and Nugent criteria, with a much larger number of asymptomatic women being diagnosed as having BV by the Nugent criteria [69]. The Nugent criteria can be inaccurate due to morphological similarities between different bacterial species (see Fig. 83.2). For example, the lactic acid-producing *L. iners* (one of the most frequent Lactobacillus isolates in healthy women) and *A. vaginae* (another lactic acid producer) are small rods easily confused with small Gram-positive or Gram-variable rods; this confusion could inflate the Nugent score. The importance and classification of intermediate vaginal microflora (Nugent score of 4–6), which is not dominated by *Lactobacillus* spp. but which does contain other genera known to produce lactic acid (e.g. Megasphaera, Streptococcus, Atopobium spp.), complicate the clinical diagnosis [1]. In addition, some healthy women may also have a pH that is higher than 4.5 (part of the Amsel criteria), a common occurrence during menses or in post-menopausal women. The difficulty of both over- and underdiagnosis of BV has led to alternative point-of-care diagnostics (based on vaginal pH, bacterial sialidase activity and amine compounds), but (similar to molecular diagnostics discussed below) they have not been widely used [70].

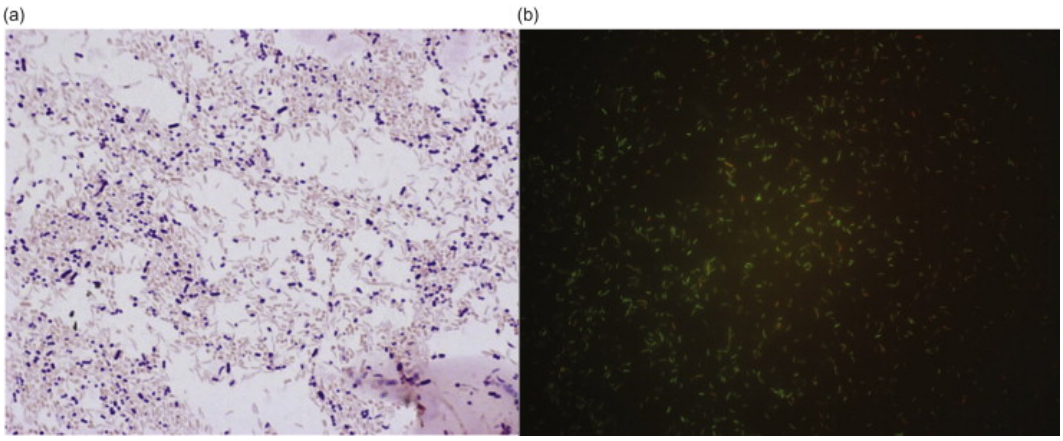

Figure 83.2. Morphologic similarities between different bacteria species associated with BV (BVAB). A representative Gram stain (a) of a vaginal discharge from a women with BV (a Nugent score of 10). A fluorescent in situ hybridization stain (b) highlighting that curved Gram-negative rods, which had previously been thought to be mostly *Mobilincus* sp. (stained in red), are more likely to be BVAB1 (stained in green).

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Urethritis, Vulvovaginitis, and Cervicitis

Paula K. Braverman, in Principles and Practice of Pediatric Infectious Diseases (Fifth Edition), 2018

Bacterial Vaginosis.

BV is the most common cause of vaginitis in postpubertal women, affecting approximately one third of women.[63,66–68] BV represents a disruption in the normal vaginal flora, with a decrease in lactobacilli and overgrowth of a variety of primarily anaerobic organisms. BV-associated organisms include *G. vaginalis*, genital mycoplasmas *(M. hominis), U. urealyticum*, Bacteroides, Prevotella, Porphyromonas, Peptostreptococcus, Fusobacterium, and Mobiluncus species.[63,64,69–71] Although many patients with BV have moderate to heavy concentrations of vaginal Gardnerella species, detection of this organism is not diagnostic because vaginal colonization is common in patients without BV and not specific for the diagnosis.[63,64,71]

New technologies employing amplification of ribosomal RNA are being used to characterize bacterial species that are not identified by culture [64,70–72] They include three clostridial bacteria, Leptotrichia and Megasphaera species, and Eggerthella-like bacteria.[70–72] In one study, detection of these noncultivatable bacteria had excellent sensitivity and specificity for diagnosing BV compared with standard diagnostic criteria.[71]

In a multivariate analysis of NHANES data from 2001 through 2004, risk factors for BV included a higher number of lifetime sex partners, douching, low educational achievement, and being non-Hispanic black.[68] BV has also been associated with poverty, smoking, having a female sex partner, high body mass index, and previous pregnancy.[68,73,74] The prevalence of BV among women attending STI clinics is higher (30% to 37%) than among college students (4% to 15%)[7] and the prevalence of BV in a nationally representative sample of 14-19 year old women was 18.5%.[68]

BV is more common among adolescents and young adults who are sexually active and have multiple sexual partners. However, designating BV as an STI has been controversial because some studies have found BV in sexually inexperienced females.[64,66,68,75,76] One study of women entering the military found that 19% of subjects denying a history of vaginal intercourse met criteria for BV compared with 28% who had been active sexually.[75] Another study questioned the accuracy of sexual histories having failed to find BV in truly sexually inexperienced college students.[77] Studies have shown a concordance between the presence of *G. vaginalis* in the urethra of male sex partners of women diagnosed with BV but did not find this organism in male controls.[64] Sexual activity, multiple sexual partners, receptive oral sex, and vaginal insertion of sex toys that were not cleaned are associated with BV, whereas the use of condoms appears to be protective.[64,73,74,77–80] Treatment of sex partners does not appear to prevent recurrence.[64] However, a systematic review of randomized, controlled trials of male partner treatment for BV criticized these studies for having methodologic flaws.[81]

Up to one third of women experience recurrent episodes of BV within 3 months of treatment.[66] In one study, factors associated with recurrence over a 12-month period included a history of BV and having a female sex partner or a regular sex partner.[82] Recurrence may be related to reinfection from an infected partner or to failure to re-establish lactobacilli dominance with persistence of pathogenic bacteria.[63,67,72] Some investigators have postulated that the recurrence of BV is related to persistence of a biofilm containing *G. vaginalis* and other organisms that adheres to the epithelial cells and provides protection from systemic and topical antibiotics.[72,83–85]

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