Pathogenic Bacteria & Yeast

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	GI Symptoms
Aeromonas *Aeromonas hydrophilia* *Aeromonas caviae* *Aeromonas veronii* *Aeromonas jandaei* *Aeromonas schuberti*	*Aeromonas* is a facultatively anaerobic, Gram-negative rod.[1] *Aeromonas* species share many biochemical properties with *Vibrio* species and were jointly classified in the *Vibrionaceae* family until genotypic information provided new insights.[2] (P)	Aeromonads normally inhabit the aquatic environment, though they have been isolated from a variety of foods, such as fish, meat, milk, and vegetables. The foodborne isolations are predominantly *A. hydrophilia.*[1]	Aeromonads possess virulence factors, such as enterotoxins, cytotoxins, and hemolysins. They have the ability to adhere to and invade cells, and produce various enzymes that are regarded as pathogenic mechanisms.[3]	*Aeromonas* has been associated with a wide variety of human infectious diseases, including gastroenteritis, wound infections, septicemia, respiratory infections, and urinary tract infections.[2] However, *Aeromonas* is most commonly associated with gastrointestinal enteropathy. Symptoms include watery diarrhea (with a self-limiting course), fever, abdominal pain, vomiting, bloody diarrhea, and possible secondary dehydration.[2]
Bacillus anthracis	*B. anthracis* is a spore-forming, Gram-positive bacterium which causes anthrax.[4] In humans, there are three major forms of anthrax as delineated by the spore exposure route: cutaneous, gastrointestinal, and inhalational.[5] (P)	*B. anthracis* spores primarily infect grazing animals, but humans may be exposed to anthrax through the handling of infected animals and animal products or tainted meat consumption.[4]	Spores are ingested and germinate within the GI tract epithelium. *B. anthracis* then uses a toxin called anthrolysin to disrupt the GI barrier.[6]	GI anthrax can present clinically as either intestinal or, less commonly, oropharyngeal infection. The incubation period is typically 1-6 days. Intestinal anthrax manifests with ileal or cecal ulcerations. Illness begins with anorexia, nausea, vomiting, and fever; this progresses to severe abdominal pain, hematemesis, melena, and/or frank blood in the stool.[6]
Bacillus cereus	*B. cereus* is a Gram-positive, aerobic (or facultative aerobic), spore-forming, rod-shaped bacterium.[7] (PP)	*B. cereus* is ubiquitous in soil and freshwater environments in all temperate zones. It is capable of contaminating many food products, including rice, chicken, vegetables, spices, and dairy products.[7]	*B. cereus* produces several toxin types: hemolysin, phospholipase, cereulide (emetic toxin), and enterotoxins. The incubation time averages 12 hours, and the duration of signs/symptoms is between 12-24 hours.[7]	*B. cereus* infectious symptoms include gastroenteritis and vomiting, but the illness is self-limiting and usually lasts less than 24 hours.[7]

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Bacillus species	*Bacillus* species are Gram-positive aerobic (or facultatively aerobic) rods.[8] Most human non-anthracis *Bacillus* spp. infections are caused by *B. cereus*. Not all isolates are associated with disease. Many *Bacillus* species are used in spore- and soil-based probiotics, such as *B. subtilis, B. coagulans,* and *B. licheniformis.*[9] (PP)	*Bacillus* organisms are widely distributed in the environment, though the primary habitats are soil and water. Many *Bacillus* species are beneficial and used in probiotics and in biocidal environmental insecticides.[9,10]	Different *Bacillus* species produce various extracellular products, including antimicrobial substances, enzymes, pigments, and toxins. Except for a select few species, most *Bacillus* species have no pathogenic potential and are not associated with disease.[8]	*Bacillus* infection is not always pathogenic and often asymptomatic. Infections caused by the *Bacillus* species include self-limiting gastroenteritis (*B. cereus*), localized infections due to trauma, ocular infections, and rarely systemic illness as seen in *B. anthracis*.[8]
Campylobacter spp. *Campylobacter jejuni* *Campylobacter coli*	*Campylobacter* species are non-spore-forming, Gram-negative, helical, rod-shaped, or curved bacteria.[11] *Campylobacter* genus belongs to the family *Campylobacteraceae.*[12] (P)	*Campylobacter* has a world-wide distribution and international travel is a risk factor for infection. *Campylobacter* is a confirmed foodborne bacterial pathogen. Infection occurs after consumption of contaminated food, particularly poultry, unpasteurized milk, and water.[12,13]	*Campylobacter*'s helical shape and flagella are thought to be responsible for their ability to colonize the intestinal tract, and for adhesion and invasion into epithelial cells.[11] Additionally, cytotoxin production leads to cell death, damage to mucosal surfaces, and subsequent diarrhea.[14] The onset of symptoms usually occurs 24-72 hours following ingestion.[12]	*C. jejuni* and *C. coli* are established causes of gastroenteritis world-wide. *C. jejuni* can also lead to autoimmune conditions like Guillain-Barre' syndrome and Miller Fischer syndrome. Patients with *C. jejuni* or *C. coli* experience acute watery or bloody diarrhea, weight loss, and abdominal cramping.[12] Many *Campylobacter* species are known pathogens associated with a wide range of gastrointestinal conditions, including inflammatory bowel disease, Barrett's esophagus, and colorectal cancer. They have also been known to cause extra-gastrointestinal manifestations, including bacteremia, lung infections, brain abscesses, meningitis, and reactive arthritis.[12]

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Candida spp. *Candida albicans* *Candida* species, not *albicans* *Candida auris* *Candida dubliniensis* *Candida famata* *Candida glabrata* *Candida guilliermondii* *Candida krusei* *Candida lusitaniae* *Candida parapsilosis* *Candida pseudotropicalis* *Candida rugosa* *Candida stellatoidea* *Candida tropicalis* *Candida zeylanoides*	*Candida* spp. have commonly been identified as part of the healthy human mycobiome. Host defense interruption, or immunocompromise, is required for them to act as pathogens.[15] *Candida albicans* is the most prevalent among the *Candida* spp.[15] (PP)	Fungi, including *Candida*, are ubiquitous in our environment and are part of natural foods and industrial processes, including antibiotic production, bread, cheese, alcoholic beverages, decomposing natural debris, fruits, and soil nutrients.[16] *Candida* is present in the gut of up to 70% of healthy adults, but certain factors, including diabetes, antibiotics, antacid, and steroid inhaler use, promote overgrowth.[17] *Candida* growth in the GI tract is positively correlated with carbohydrate consumption.[18]	*Candida* pathogenesis depends on virulence factor expression, like germ tube formation, adhesions, phenotypic switching, biofilm formation, and hydrolytic enzyme production. Most *Candida* disease processes are primarily due to biofilm formation.[15] During overgrowth, *Candida* produces pseudohyphae that push their way into the intestinal lining, destroying cells and brush borders, and may eventually send toxic metabolic by-products through the intestinal wall into the blood.[19] High-level *Candida* colonization is frequently observed in ulcer and IBD patients. This may in part reflect common treatments for these conditions. In addition, the presence of *Candida* delays healing and exacerbates disease.[20]	As noted, most patients are asymptomatic, and *Candida* is considered a commensal organism. Depending on the host's immune status and comorbidities, symptoms will vary. *Candida* overgrowth in the GI tract has been shown to cause diarrheal illness.[21] Other GI symptoms sometimes seen include thrush, bloating, gas, intestinal cramps, rectal itching, and altered bowel habits.[22] Some generalized symptoms of patients with yeast infections include chronic fatigue, mood disorders, and malaise.[22]

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Citrobacter spp. *Citrobacter amalonaticus* *Citrobacter braakii* *Citrobacter freundii* *Citrobacter youngae* *Citrobacter koseri/diversus*	*Citrobacter* are Gram-negative, non-spore-forming, facultatively anerobic bacilli. *Citrobacter* fall within the *Enterobacteriaceae* family.[23] *Citrobacter* is considered a commensal bacteria; however, depending on the clinical picture, it is also known to be an opportunistic pathogen.[24] (PP)	*Citrobacter* species are found in water, soil, food, and commonly in the human intestinal tract.[23] *Citrobacter* infections can also be nosocomial.[23]	Although considered a commensal, some *Citrobacter* isolates have virulent toxins, such as Shiga-like toxins, heat-stable toxins, and cholera B toxin B subunit homologs.[25]	*Citrobacter* is most often asymptomatic but can cause diarrhea.[24]
Clostridium difficile	*C. difficile* is an anaerobic, Gram-positive, spore-forming, toxin-producing bacillus.[26] (P/PP) *See GI Symptoms column Genova measures *C. difficile* toxin via EIA. A prerequisite for *C. difficile* EIA toxin testing is a stool consistency of 7 on the Bristol stool scale, whereby the sample takes the shape of the container. Clinical relevance is determined by the presence of toxin A/B. When these toxins are present, correlation with patient symptoms is recommended.[27]	*C. difficile* spores are frequently found in healthcare facilities, and are found in lower levels in the environment and food supply. Infection can be nosocomial or community transmitted.[26]	*C. difficile* spores are resistant to heat, acid, and antibiotics. They colonize the large intestine and release two protein exotoxins (A, B). These exotoxins cause colonocyte death, barrier function loss, and neutrophilic colitis.[26] Colonization is prevented by barrier properties of the microbiota; weakening of this barrier by antibiotics is the major risk factor for disease.[26,28]	Not all colonized patients develop symptoms.[27] A majority of infants are colonized with C. *difficile* and are asymptomatic.[26] When present, *C. difficile* infection presents with bloody and non-bloody diarrhea, fever, abdominal pain, vomiting, ileus, and dehydration. Toxic megacolon and peritonitis are significant complications of advanced infections.[26] Of note, many successfully treated patients will continue to test positive for weeks or months after symptom resolution; additional treatment is neither required nor effective.[26]

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Cryptococcus albidus *Cryptococcus gattii* *Cryptococcus humicolus* *Cryptococcus laurentii* *Cryptococcus luteolus* *Cryptococcus neoformans*	*Cryptococcus* is a fungus. Although there are more than 30 species of *Cryptococcus*, only two commonly affect humans and animals: *C. neoformans and C. gattii*.[29] 95% of cryptococcal infections are caused by *C. neoformans*.[30] (PP)	Cryptococcosis has a worldwide distribution. Cryptococcosis occurs through the inhalation of fungal cells from soil, plants, and decaying natural materials, though zoonotic transmission is possible. The yeast may incidentally enter the gastrointestinal tract, though this is less likely.[30]	There are prominent virulence factors attributed to *Cryptococcus,* including capsule formation, thermotolerance, and melanin pigment production, which protects the yeast from host oxidative stresses. An effective host immune response is common, using helper T cell reactions; therefore, any weakening of that response allows *Cryptococcus* to survive and thrive.[30]	Cryptococcal infection primarily affects the lungs or central nervous system, though GI tract infection causing diarrhea is increasing among immunocompromised patients (HIV/AIDS).[29]
Edwardsiella tarda	*E. tarda* is a Gram-negative, facultatively anaerobic rod.[31] It is a member of the *Enterobacteraceae* family. (PP)	*E. tarda* exists widely in nature and is isolated from lakes, streams, seawater, and aquatic animals/fish.[31] Infection results from the consumption of contaminated meat/fish, though human infection is rare.[32]	Pathogenicity of *E. tarda* is associated with many virulence factors, such as hemolysins, which enable the bacteria to have access to essential nutrient elements in order to colonize.[33]	Gastroenteritis, with fever and vomiting, is the most common symptom of *E. tarda* infection, ranging from mild secretory enteritis to chronic enterocolitis. Symptoms can be self-limiting; however, extraintestinal manifestations can include systemic abscesses and septicemia.[32,33]
Enterobacter cloaceae	*E. cloaceae* is a Gram-negative, non-spore-forming, enteric bacilli belonging to the *Enterobacteriaceae* family. *Enterobacteriaceae* are not considered primary human pathogens, but are capable of causing opportunistic infections.[34] (PP)	*Enterobacter* have a ubiquitous environmental distribution (trees, plants, crops, soil, water, and foods). They are also part of the normal flora of the GI tract.[34] It can also be a common nosocomial infection.[35]	*Enterobacter's* ability to form biofilms and to secrete various cytotoxins, such as enterotoxins and hemolysins, contribute to its pathogenicity.[35]	Most patients with an *E. cloaceae* infection are asymptomatic. However, when present, symptoms can include nausea, vomiting, diarrhea, and abdominal cramps.[36]

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Escherichia coli 0157:H7 Shiga toxin producing	*E. coli* is a Gram-negative, rod-shaped, facultative anaerobe. Most *E. coli* harmlessly colonize the GI tract as normal flora. However, some strains have evolved and acquired virulence factors, which are characterized by serotypes. *E.coli 0157:H7* has become one of the most virulent foodborne pathogens.[37] (P)	*E. coli 0157* is transmitted to humans through contaminated food and water, directly between persons, and through contact with animals. The most common reservoir is cattle, and the most frequently identified mode of transmission is through ground beef consumption.[38]	*E. coli 0157's* ability to induce injury is a result of its ability to produce Shiga toxin, which is cytotoxic. Additionally, it produces other proteins which aid in the attachment and colonization in the intestinal wall and can lyse red blood cells to liberate iron to support its own metabolism. It should be noted that there are other organisms which can also produce Shiga-like toxin. The characteristic histopathological lesions caused by *E. coli 0157:H7* are called attaching and effacing (A/E) lesions. Microvilli are effaced and bacteria adhere to the epithelium.[37]	Signs and symptoms associated with Shiga-toxin producing *E. coli 0157* include bloody diarrhea, stomach cramping, and vomiting. This can progress to hemolytic uremic syndrome and death.[38]
Geotrichum species *Geotrichum candidum* *Geotrichum capitum*	*Geotrichum* is a eukaryotic, aerobic, Gram-positive, non-capsulated fungus. *Geotrichum* is considered a common commensal in the human GI tract, though opportunistic infections are seen in immunocompromised patients.[39] (PP)	*Geotrichum* is ubiquitous and is commonly found on fruits, vegetables, cheeses, mil, soil, water, air, and in the human digestive tract.[40] Transmission is through inhalation of fungal cells or ingestion of contaminated foods.[39]	*Geotrichum* infection is rare and, in general, *Geotrichum* has low virulence. In patients with normal immunity, it is not pathogenic.[41]	Clinical manifestations are very similar to candidiasis. Many patients are asymptomatic; when present, symptoms include diarrhea, abdominal pain, and mucus in the stool.[39]
Hafnia alvei	*H. alvei* is a Gram-negative, facultatively anaerobic bacillus that belongs to the *Enterobacteriaceae* family. Though rare, it is considered an opportunistic pathogen.[42] (PP)	*H. alvei* is most commonly isolated from vacuum-packed meat, raw milk, raw fish, and other foods. Transmission is via ingestion of contaminated foods, but nosocomial infections have been seen.[42]	*H. alvei* pathogenicity is in biofilm formation and cellulose production; this aids in colonization and mediates cell-cell interaction. It also produces adhesins and toxins which contribute to symptoms and antimicrobial resistance.[42]	*H. alvei*'s clinical relevance is not clear. It has been isolated from feces in asymptomatic patients, yet is also known to cause gastroenteritis, necrotizing enterocolitis, and extra-intestinal illnesses.[43]

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Hansenula anomala Also known as *Pichia anomala* and *Wickerhamomyces anomalus*	*H. anomala/W. anomalus* is an ascomycete yeast.[44] Although useful in food processing, it has been shown to be a very rare opportunistic and nosocomial pathogen in humans, mainly neonates and immunocompromised patients.[44,45] (PP)	*H. anomala/W.anomalus* is frequently found in natural environments (plants, soil, fruit, animals) and is useful in wine fermentation.[45] It also has antimicrobial properties and has been used as a biocontrol agent. It can be found on the skin and as normal flora in the human gastrointestinal tract.[44]	*H. anomala/W. anomalus* are classed as biosafety level 1 by the European Food Safety Authority, and there are no reports in the literature regarding hazardous mycotoxin formation or allergic reactions to spores from this yeast. However, rare isolates from immunocompromised patients are emerging with no clear specific pathogenicity.[44]	*H. anomala/W. anomalus* are considered normal flora and very rarely cause disease, but they have been known to cause sepsis, fungal arthritis, pneumonia, and endocarditis in immunocompromised patients.[46]
Helicobacter pylori	*H. pylori* is a Gram-negative, aerophilic bacterium. *H. pylori* infection is one of the most common chronic bacterial infections affecting humans.[47] (P) Genova uses an enzyme immunoassay platform that utilizes antibodies to detect *H. pylori* antigen present in the stool sample.	*H. pylori* infection is chronic and is usually acquired in childhood. The exact means of infection is not clear.[47]	After entering the host stomach, *H. pylori* uses its urease activity to neutralize the acidic environment. It has a flagella-mediated motility to help it move toward the gastric epithelium. Specific bacterial adhesin proteins lead to colonization and persistent infection. It finally releases effector proteins and toxins causing host tissue damage.[48]	*H. pylori* is an important cause of peptic ulcer disease (PUD) and gastric cancer. It may also have a role in functional dyspepsia, ulcer risk in patients taking low-dose aspirin or starting NSAID therapy, unexplained iron deficiency anemia, and idiopathic thrombocytopenic purpura (ITP).[47] According to the American College of Gastroenterology, the indications to test for *H. pylori* include active PUD, a history of PUD, low-grade mucosa-associated lymphoid tissue (MALT) lymphoma, or endoscopic early gastric cancer. Patients initiating chronic aspirin or NSAID treatment, those with unexplained iron deficiency, and patients with ITP, should be tested.[47] Patients with typical GERD symptoms without a history of PUD, need not be tested for *H. pylori*; however, those who are tested and found to be infected should be treated.[47]

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Klebsiella oxytoca *Klebsiella pneumoniae*	*Klebsiella* are non-motile, Gram-negative rods that belong to the *Enterobacteriaceae* family. *Klebsiella* bacteria are considered commensal but act as opportunistic bacteria in the GI tract. *Klebsiella* is a leading cause of hospital-acquired infections.[49] (PP)	*Klebsiella* is part of the normal intestinal flora. The environment likely acts as a reservoir for human acquisition, either as colonization or infection. It is frequently found in water, sewage, soil, and plant surfaces.[50]	*Klebsiella* possesses virulence factors, such as a capsule, lipopolysaccharides, and pili. *Klebsiella* translocates across the intestinal epithelium via a transcellular mechanism by active bacterial invasion. This allows it to penetrate the intestinal barrier and enter systemic circulation causing extraintestinal disease.[51] Cytotoxins produced by *Klebsiella oxytoca* are associated with antibiotic-associated hemorrhagic colitis (AAHC).[52] Ankylosing spondylitis and Crohn's disease have been shown to be triggered by *Klebsiella pneumoniae*. Increased starch consumption by genetically susceptible patients (HLA-B27 allelotypes) could trigger disease by enhancing the growth of *Klebsiella* in the gut. The cross-reactive antibodies between *Klebsiella* and AS/Crohn's trigger inflammatory cascades, such as the complement system, as well as producing various cytokines causing pathologic changes .[53]	*Klebsiella* can asymptomatically colonize the GI tract. However, depending on host factors and immunocompetence, it may cause diarrhea and systemic illnesses.[49,50]
Listeria monocytogenes	*Listeria* is a Gram-positive, facultative intracellular bacterium.[54] (P)	*Listeria* is ubiquitous in the environment. It is the causative agent of Listeriosis, a rare but fatal foodborne disease.[54,55]	*Listeria* can cross several physiological barriers, including the intestinal epithelium and placenta, and survive in multiple cell types. Following internalization into the host cell, the bacterium escapes its membrane-bound vacuole using the toxin listeriolysin. It then replicates within the cytosol and can multiply and spread from cell to cell.[55]	Ingestion of *L. monocytogenes*-contaminated food by immune-competent individuals is often limited to gastroenteritis that resolves in a few days, with pathogenic clearance from the intestine.[54] Severe complications include systemic dissemination causing septicemia, meningitis, and chorioamnionitis; all are associated with high mortality.[54]

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Moellerella wisconsensis	*Moellerella wisconsensis* is a Gram-negative bacilli from the *Enterobacteriaceae* family.[56] (PP)	*M. wisconsensis* has been recovered from various sources, such as water, food, and animals.[56] Isolation of this bacteria in clinical samples is very rare. The majority of *M. wisconsensis* isolates from human clinical samples have been from stool, though bronchial aspirates, biliary samples, and peritoneal exudates have been seen.[56,57]	Pathogenicity is unclear due to the scarcity of human infection.	Though rare, isolated case reports show that *M. wisconsensis* has been associated with diarrhea.[56,57]
Morganella morganii	*M. morganii* is a facultative anaerobic, Gram-negative, enteric bacterium which belongs to the *Enterobacteriaceae* family. *M. morganii* is an opportunistic pathogen often isolated as a cause of nosocomial infections in adults.[43] (PP)	*M. morganii* is found in the environment and colonizes the human intestinal tract as part of the normal flora.[58]	*Morganella* produces a urease that predisposes to encrustation of urinary catheters. It may also produce a hemolysin, which enhances virulence by lysing erythrocytes.[58]	Although *Morganella* is part of the normal intestinal flora, it has been implicated in various diseases, including diarrhea, urinary tract infections, and wound infections. Serious infections, like meningitis in AIDS patients, have been reported.[43]
Pichia ohmeri More recently known as *Kodamaea ohmeri*	*K. ohmeri* is a fungus that belongs to the *Saccharomycetes* family, which acts as a very rare opportunistic pathogen.[59] (PP)	*K. ohmeri* is widely used in the food industry for the fermentation of fruits, pickles, and rinds.[59] In the past, *Kodamaea ohmeri* was considered a food contaminant, but is now recognized as an emerging opportunistic pathogen in immunocompromised patients.[60]	Pathogenicity is not yet clearly defined due to the rarity of human infection.	*K. ohmeri* infection is rarely reported to cause human infection, with only isolated case reports seen in the literature; these are primarily in infants and immunocompromised patients.[60-62] Systemic fungemia has been rarely seen in association with indwelling catheters, phlebitis, wound infections, endocarditis, and outbreaks in intensive care units.[60]
Plesiomonas shigelloides	*P. shigelloides* is an anerobic, Gram-negative bacillus, belonging to the *Enterobacteriaceae* family. (P)	*Plesiomonas* is a global pathogen with worldwide distribution. It is most often isolated in aquatic environments. Infection occurs primarily by undercooked freshwater fish consumption.[63]	*Plesiomonas* contains a Shigella phase I antigen, cholera-like toxins, hemolysins, and cytotoxic lipopolysaccharides.	*P. shigelloides* causes gastroenteritis, which ranges from a secretory enteritis to a cholera-like diarrhea. Extraintestinal manifestations can occur with bacteremia and sepsis.[63]

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Providencia alcalifaciens	*P. alcalifaciens* is a Gram-negative rod that belongs to the *Enterobacteriaceae* family. It is usually considered to be a commensal bacteria, but can also be an opportunistic pathogen and a cause of traveler's diarrhea.[64] (PP)	*P. alcalifaciens* is found throughout the environment, and as a commensal bacteria in the large intestine. Food contamination and human transmission has been shown to be via the fecal-oral route, lack of sanitation, and poor food storage.[64]	*P. alcalifaciens* has lipopolysaccharides that cause epithelial barrier dysfunction and endothelial apoptosis.[65]	Although often considered a commensal bacteria, *P. alcalifaciens* has been shown to cause diarrhea.[64]
Proteus mirabilis *Proteus penneri* *Proteus vulgaris*	*Proteus* is Gram-negative bacteria belonging to the *Enterobacteriaceae* family. *Proteus* spp. are considered opportunistic pathogens, isolated from urine, stool, and wounds.[66,67] *Proteus* are a common cause of nosocomial infections in patients with impaired immunity. (PP)	*Proteus* is widespread in the environment and considered part of the normal GI flora. *Proteus* spp. are found in soil or water habitats and are often regarded as indicators of fecal contamination.[67]	The chemical structure of *Proteus*' lipopolysaccharides plays an important role in how it adapts to the environment and its pathogenicity. In impaired immunity, *Proteus* bacteria become opportunistic. Cross infection with the urinary tract is common.[67]	*Proteus* species in the stool are considered normal flora, but have been shown to cause diarrheal illness.[67]
Pseudomonas aeruginosa	*P. aeruginosa* is a Gram-negative aerobic bacilli. Although seen as part of the normal healthy intestinal flora, it is considered a potential pathogen. It is generally not a common cause of infectious diarrhea in a healthy host. Patients with chronic disease, chronic antibiotic use, or immunocompromise are at highest risk for infection.[68] (PP)	*Pseudomonas aeruginosa* is readily found in the environment (soil and water) and in the healthy gastrointestinal tract.	*P. aeruginosa* induces pro-inflammatory responses and anti-microbial peptides within intestinal epithelial cells. It also has cytotoxic activity. Disruption of the intestinal epithelial protective mechanisms allow for disease progression.[69]	Most patients are asymptomatic, though *P. aeruginosa* can cause mild diarrhea. A rare complication is Shanghai Fever, which is characterized by fever, diarrhea, and sepsis. *P. aeruginosa* has also been associated with antibiotic-related diarrhea.[68]

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Pseudomonas pseudomallei Also known as *Burkholderia pseudomallei*	*P. pseudomallei* is a Gram-negative, aerobic, saprophytic bacillus. It causes the rare, often fatal disease, melioidosis.[70] There are fears that *P. pseudomallei* can be used as a biological weapon.[71] (P)	*Pseudomonas/Burkholderia pseudomallei* is widespread in South, Central, and North America; it is also common in Southeast Asia. Infection occurs through contact with soil and water in endemic areas through inhalation, skin inoculation, or ingestion.[70]	*P. pseudomallei* possesses several secretion systems essential for its dissemination. Pathogenicity is due to its endotoxins inducing apoptosis. Most infections occur in the lung, though systemic disease is possible.[71] It is likely to be consumed in water and food in settings where the organism is present in the environment. It can colonize the gastrointestinal tract without clinical features for months or years.	Most symptoms of melioidosis are pulmonary, though colonization, shedding, and carriage through the GI tract are possible. Systemic disseminated abscesses are common.[71]
Rhodotorula spp. *Rhodotorula glutinis* *Rhodotorula rubra*	*Rhodotorula* is a saprophytic yeast. Previously considered non-pathogenic, it has emerged as an opportunistic pathogen. (PP)	*Rhodotorula* is a common, ubiquitous yeast that is found in air, soil, lakes, ocean water, food, and beverages.[72]	It has been shown that *Rhodotorula* species are able to form biofilms which may play a role in its pathogenicity. Antibiotics and cytotoxic agent exposure increases intestinal colonization and mucosal damage.	Isolation from non-sterile sites, like skin and stool, are more commonly contaminant or colonization. Specific gastrointestinal symptoms are not well studied. Systemic infections and fungemia are possible in immunocompromised patients.[73]
Saccharomyces cerevisiae	*Saccharomyces cerevisiae* and *Saccharomyces boulardii* are two closely related strains of non-spore-forming yeast which are nearly identical at the molecular level. Classically considered a safe, nonpathogen, *S. cerevisiae* can cause disease in immunocompromised patients.[74] (PP)	*S. cerevisiae* commonly colonizes the human respiratory, gastrointestinal, and urinary tracts. *S. cerevisiae* is found in many niches in the environment, but is commonly known as baker's yeast, and is frequently used in the industrial fermentation of bread, beer, and wine.[74] *S. cerevisiae* is also commercially available as a nutritional supplement and is used to treat antibiotic-related diarrhea and IBS.[75]	*S. cerevisiae* uses adhesin proteins to penetrate disrupted epithelial or endothelial barriers. Most fungal pathogens display resistance to the reactive oxygen species used by human cells to resist infection.[74]	Immunosuppression can lead to *S. cerevisiae* infection, though indwelling catheters, chronic antibiotic therapy, and nosocomial spread are common risk factors. *S. cerevisiae* infection can cause a wide variety of clinical syndromes, such as fungemia, pneumonia, abscess, esophagitis, and fever. It has been associated with Crohn's disease and ulcerative colitis.[76]

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Salmonella typhii *Salmonella* species *S. arizonae* *Salmonella group A, B, C, D, E, E+G, C+D* *S. paratyphi A, B, C*	*Salmonella* is a facultative intracellular, Gram-negative bacteria within the *Enterobacteriaceae* family. It is the causative agent of human typhoid fever.[77,78] (P)	Humans are typically infected with *Salmonella* after consuming food or drinking water contaminated with bacteria, and transmission is often fecal-oral.[77]	After oral ingestion, *Salmonella* invades the epithelial cells in the distal ileum and invades Peyer's patches. *Salmonella* travels via the afferent lymphatics to gain access to the blood and systemic tissues.[77]	Depending on the serotype, *Salmonella* symptoms can vary from a self-limiting gastroenteritis and diarrhea, to systemic infection with fever, respiratory distress, hepatic and splenic complications, and neurologic damage.[78]
Serratia marcescens	*Serratia* are non-spore-forming, Gram-negative rods, and are part of the *Enterobacteriaceae* family. *S. marcescens* is an opportunistic pathogen, which is generally thought not to be pathogenic in the intestine, but is emerging as a frequent nosocomial infectious agent.[79,80] (PP)	*Serratia* species are ubiquitous in the environment, and found in water, soil, plants, insects, humans, and other animals.[81] Infection is acquired through ingestion of contaminated food or contact with hospital equipment and personnel.[80]	*S. marcescens* has the potential for adhesion, invasion, cytotoxicity, perturbation of intestinal barrier function, cytokine release, and alteration of cellular morphology.[80]	Patients most at risk for *S. marcescens* infection include those with immunocompromise, patients on broad spectrum antibiotics, or hospitalized patients subjected to invasive instrumentation/catheters. Most patients are asymptomatic carriers, though *S. marcescens* infection symptoms may include diarrhea and rarely necrotizing enterocolitis.[80]
Shigella species *Shigella boydii* *Shigella dysenteriae* *Shigella flexnari* *Shigella sonnei*	*Shigella* are Gram-negative pathogenic bacteria that belong to the Enterobacteriaceae family.[82] *Shigella* is the causative organism of Shigellosis, accounting for the majority of dysentery worldwide.[82] (P)	*Shigella* species are transmitted via the fecal-oral route. They are easily transmitted by personal contact with an infected person or consumption of contaminated food or water.[83] *Shigella* species are geographically stratified based on the level of economic development in a given country. *S. flexneri* is the primary infectious species in the developing world, whereas *S. sonneri* rates increase with economic development. *S. boydii* is restricted to Bangladesh and Southeast Asia. S. *dysenteriae* occurs sporadically worldwide.[82]	The *Shigella* bacteria invades colonic mucosa, then can multiply causing epithelial cell death, and spread laterally to cause mucosal ulcers, bleeding, and inflammation.[83]	Symptoms of shigellosis include fever, bloody diarrhea, and abdominal cramping. Infection is usually restricted to the gastrointestinal tract, though extra-intestinal manifestations (reactive arthritis, hemolytic-uremic syndrome, and neurologic complications) can be seen.[83]

Pathogenic Bacteria & Yeast

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	GI Symptoms
Staphylococcus aureus	*S. aureus* in the GI tract is a commensal Gram-positive bacterium, which can be responsible for opportunistic toxigenic infections.[84] (PP)	*S. aureus* is a common cause of food-borne disease. Though ubiquitous in the environment, and a commensal found on the skin, nasopharynx, and gastrointestinal tract, it can be transmitted via contaminated food or water consumption.[85] Fecal carriage is considered an important risk factor for hospital- and community-acquired infections.[86]	*S. aureus* produces varying enterotoxins and contains several virulence genes.[87]	Asymptomatic fecal *S. aureus* carriage is common. However, *S. aureus* GI infection symptoms include nausea, vomiting, and abdominal cramping, with or without diarrhea.[85] The foodborne illness can be self-limiting with resolution after 24-48 hours. Severe disease often requires hospitalization.[87] Colonization with *S. aureus* increases the risk of systemic infection and bacteremia.[84]
Trichosporon species *Trichosporon beigelii* *Trichosporon pullulans*	*Trichosporon* are amorphic fungi. Though considered a commensal yeast, they are increasingly recognized as opportunistic pathogens in immunocompromised individuals.[88] (PP)	*Trichosporon* fungi are commonly found in nature and can reside harmlessly as commensals on the skin and in healthy individuals' gastrointestinal tracts.[88]	*Trichosporon's* ability to invade the skin and other tissues includes several virulence factors, including yeast-to-hyphae transition, biofilm formation, lipases and proteases, and cell wall plasticity.[88]	*Trichosporon* is a commensal yeast in the GI tract and is usually asymptomatic. Changes in nutrient availability may influence *Trichosporon* spp. abundance and diversity and underlie gut mycobiome dysbiosis. This can potentially lead to inflammatory pathologies, such as inflammatory bowel disease. Invasive and systemic trichosporonosis is seen in immunocompromised hosts.[88]
Vibrio cholerae	*Vibrio cholerae* is a Gram-negative, facultative anaerobic bacterium that is responsible for epidemic cholera, a severe diarrheal disease.[89,90] (P)	*V. cholerae* naturally inhabits aquatic environments. Epidemic cholera is transmitted to humans by contaminated water and food consumption.[89] Cholera is associated with unsanitary conditions and countries with poor infrastructure.[90]	*V. cholerae* are ingested and colonize the intestinal mucosa using adhesin proteins and mucinase enzymes. The incubation period is between 12 hours and 5 days. Once a certain concentration of cells is reached, enterotoxin cascades are produced. After being shed, cells can be found in a hyperinfectious state, which make secondary infection to others prevalent.[89]	When mild, cholera symptoms are often indistinguishable from other diarrheal causes. However, more commonly, patients develop severe dehydration or die due to acute watery diarrhea.[90]
Vibrio fluvialis	*V. fluvialis* is a Gram-negative rod known to be pathogenic in humans.[91] (P)	*V. fluvialis* occurs widely in the aquatic environment. It is one of the emerging foodborne pathogens throughout the world. *V. fluvialis* is often associated with raw or undercooked fish consumption.[92]	Upon ingestion into the GI tract, the prevalent virulence factors in *V. fluvialis* infection are hemolysin and cytotoxins.[92]	*V. fluvialis* is found to be associated with cholera-like diarrhea. Rare complications include biliary tract infection, suppurative cholangitis, peritonitis, and other extraintestinal manifestations.[92]

Pathogenic Bacteria & Yeast

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	GI Symptoms
Vibrio furnissii	*V. furnissii* is a Gram-negative rod. Initially it was assigned and named as a subgroup of *V. fluvialis*, but it is now considered a separate species. It is considered pathogenic, but rare.[93] (P)	*V. furnissii* is ubiquitous in aquatic marine environments. Infection is associated with ingestion of contaminated seafood, or exposure to coastal waters.[93]	Flagellum are one virulence factor in *Vibrio* infections, in addition to proteases, hemagglutinins, and hydrolytic exoenzymes.[93]	*V. furnissii* has been associated with gastroenteritis in humans.[93]
Vibrio hollisae Now reclassified as *Grimontia hollisae*	*G. hollisae* is a Gram-negative, aerobic, rod-shaped bacteria, which belongs to the Vibrionaceae family.[94] (P)	Infection usually follows the ingestion of raw, undercooked, or contaminated seafood.[94]	*G. hollisae* shares a pathogenic gene cluster with the entire *Vibrio* genus. It releases a thermostable hemolysin toxin, which is absorbed in the intestines after ingestion.[95]	*G. hollisae* causes severe gastroenteritis, hypovolemia, and septicemia. It is associated with hepatotoxicity.[95]
Vibrio metschnikovii	*V. metschnikovii* is a Gram-negative rod. It is a very rare species with only a small number of cases reported.[96] (P)	Nonhuman sources include shrimp, crab, birds, water, sewage, and other seafood.[96]	As with other members of the *Vibrio* genus, hemolysin and cytotoxins contribute to pathogenicity.[96]	Presentation includes diarrhea and vomiting, though infections with *V. metschnikovii* can be fatal in patients with significant comorbidities.[96]
Vibrio mimicus	*V. mimicus* is a Gram-negative rod closely related to *V. cholerae*.[97] (P)	The natural habitat of *V. mimicus* is similar to *V. cholera*—the aquatic ecosystem. Infection usually occurs from the consumption of infected seafood.[98]	Many *V. mimicus* virulence factors have been identified, including enterotoxin, hemolysin, proteases, and hemagglutinin.[97]	*V. mimicus* gastroenteritis is characterized by diarrhea, nausea, vomiting, abdominal cramping, and fever. However, unlike *V. cholerae*, it is not associated with cholera epidemics since most isolates do not produce cholera toxin.[98]
Vibrio parahaemolyticus	*V. parahaemolyticus* is a Gram-negative rod belonging to the Vibrionaceae family. (P)	*V. parahaemolyticus* grows in warm, low salinity marine water and is the most prevalent food poisoning bacterium associated with seafood consumption.[99]	The vast majority of *V. parahaemolyticus* strains have hemolysin, causing hemolysis in the initiation of disease.[99]	Infection usually causes acute gastroenteritis and is generally self-limiting. Common characteristics include abdominal cramps, nausea, headaches, diarrhea, fever, and chills.[99]

Genus/Organism	Description	Habitat/Sources of Isolation	Pathogenicity	GI Symptoms
Yersinia enterocolitica *Yersinia pseudotuberculosis*	*Yersinia* is a Gram-negative bacillus belonging to the *Enterobacteriaceae* family. Genus *Yersinia* includes three bacteria that cause human pathology: *Y. enterocolitica, Y. pseudotuberculosis,* and *Y. pestis.* *Y. pestis* causes plague and is transmitted via flea bites. *Y. enterocolitica* and *Y. pseudotuberculosis* cause gastroenteritis and are mainly transmitted via contaminated food and water.[100] (P)	Yersiniosis has been detected on all continents. *Yersinia enterocolitica* has been associated with contamination of a variety of foods, including milk and milk products, raw meats, poultry, eggs, vegetables, seafood, and others. *Yersinia* species are able to propagate in vacuum-packed foods and at refrigeration temperatures.[101]	Following ingestion, approximately 10% of bacteria survive the acidic gastric environment and translocate the gut barrier, which compromises the Peyer's patches in the small bowel and lymphoid follicles in the large bowel. *Yersinia* then drains to neighboring lymph nodes and possibly the portal blood stream.[101] It has been postulated that *Yersinia* species contribute to the occurrence or persistence of gut inflammation in Crohn's disease.[101]	*Y. enterocolitica* and *Y. pseudotuberculosis* can both cause acute watery or bloody diarrhea and gastroenteritis. Although gastroenteritis from *Yersinia* is often self-limiting, some patients develop chronic infections, such as reactive arthritis, erythema nodosum, glomerulonephritis, or myocarditis.[100]

TREATMENT RESOURCES:

The decision to treat potentially pathogenic organisms should be based on the patient's clinical presentation.
The following resources provide valuable insight into the clinical management of pathogenic and potentially pathogenic bacteria and yeast:

- Sanford Guide – infectious disease treatment guidelines: https://www.sanfordguide.com/
- Johns Hopkins Antibiotic Guide – subscription service for in depth information on pathogens, treatment, and clinical implications: https://www.hopkinsguides.com/hopkins/index/Johns_Hopkins_ABX_Guide/All_Topics/A
- PubMed – literature search engine for up to date clinical and treatment information: https://www.ncbi.nlm.nih.gov/pubmed/
- Mayo Clinic – conditions search engine: https://www.mayoclinic.org/
- Merck Manual – treatment and clinical implications of infectious diseases: https://www.merckmanuals.com/professional

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