

Bacterial lifestyle shapes pangenomes

Supplementary Material 2: Species' lifestyle details

We categorised species' lifestyle using a combination of BacDive, Bergey's manual (2nd edition), IMG/JGI genome metadata and the literature. Below is all the information we collected from these sources for each species.

Acetobacter pasteurianus

From Bergey's manual on Acetobacter genus: "Acetobacter species occur in flowers, fruits, palm wine, vinegar, kefir, and fermented foods and can cause infections in grape wine, sake, tequila, cocoa wine, cider, beer, and fermented meat. Acetobacter is not known to have any pathogenic effect toward humans and animals."

On A. pasteurianus: "found in different kefir grains as milk adapted biotype". Described as non-motile or motile with petrichous flagella.

Commonly found associated with plants and plant products, including sugar rich substrates such as fruits, flowers and vegetables - widely used in production of fermented foods.¹ Described as endophytic (in link [here](#)) – means lives within plant for at least part of lifestyle without causing apparent disease. Usually isolated from plants – therefore, will put host, but facultative since isolated from fermented plant products and used in industry too etc.

Strains isolated from Thailand were from plants including orange, banana, watermelon, apple, heliconia flower, cantaloup, strawberry, jackfruit, avocado, star fruit, periwinkle flower (table 1)². The highest grouping of these is Eudicots, which is a class.

Bergey: no spores.

Acinetobacter baumannii

From Bergey's Manual on Acinetobacter genus: "Most frequently saprophytic, occurring naturally in soil, water, sewage, and foods such as raw vegetables. Can also reside, possibly indigenously, on the human skin and in the human respiratory tract. Can cause nosocomial infections such as bacteremia, secondary meningitis, pneumonia, and urinary tract infections in humans... naturally occur in soil and water, and are present in sewage... Although considered normally nonpathogenic, acinetobacters are causative agents of nosocomial infections, particularly in debilitated individuals and those residing in intensive care units." On A. baumannii: "Isolated from human clinical specimens or the natural environment. Most Acinetobacter strains isolated from nosocomial infections belong to this species."

Causes opportunistic hospital infections in humans, has planktonic and biofilm forms.³ Another paper also describes it as an opportunistic pathogen, predominantly in hospitals.⁴ Found almost exclusively in hospital environment, unlike rest of species in genus; some evidence also in vet clinics infecting animals, and on vegetables, aquaculture and soil; unclear if contaminated from primary hospital environment, or if exists as natural reservoirs.^{4,5}

“according to most authors, *A. baumannii* being considered ubiquitous or highly prevalent in nature is an important misconception due to the difficulties encountered in accurately identifying this organism.”⁵ However, paper argues that community infection often occurs (outside hospitals), and argues its 16S rRNA has been sequenced in soil in many countries, vegetables, surfaces like game consoles and tables, up to 10% of humans carry on skin (though other studies found <1%), and infections in vet animals (horses), and in human lice.⁵

“In conclusion, it has been demonstrated by molecular identification that *A. baumannii* is actually present outside the hospital. Several arguments are consistent with the involvement of extra-hospital reservoirs of *A. baumannii* in the occurrence of community-acquired infections.”⁵

Does not have motility, so cannot swim – but can move along surfaces via twitching motility.

Capsules present. Do not form spores.

Acinetobacter pittii

Not in Bergey’s 2005 manual (named in 2011).

First described in 2011: type strain isolated from cerebrospinal fluid of human, non-motile.⁶ IMG/JGI strains isolated from blood, urine, wastewater, sputum. Often isolated from blood.⁷

Nosocomial pathogen rarely involved in community infections.⁸ Otherwise not much seems known – described as rare.

Do not form spores.

Aeromonas hydrophila

Also known as *A. caviae*.

Bergey’s manual for *A. hydrophila*: “Isolated from fresh and marine waters, diseased fish and poikilothermic aquatic animals (e.g., frogs with red leg disease), and warm-blooded animals; associated with both extraintestinal and diarrheal disease in humans.” Listed in table BXII.182 as motile.

From Bergey's description of *Aeromonas* description:

"The presence of *Aeromonas* species in foods most likely reflects contact of these foods with water, as reflected in the name of the type species, *A. hydrophila*, which means "water-loving".

"While initially believed to be an opportunistic organism capable of infecting only immunocompromised individuals, a body of evidence now indicates that *Aeromonas* is a primary cause of extraintestinal illness and is strongly associated with gastrointestinal disease."

"Before the aeromonads were recognized as human pathogens, they were commonly isolated from poikilothermic animals, particularly amphibians, reptiles, and fish."

"*Aeromonads* are also found with some frequency to cause various types of diseases in birds and domestic animals including pneumonia, peritonitis, and various localized infections. Various species of fish develop hemorrhagic disease, ulcerative disease, furunculosis, red sore disease, and septicemia, resulting from infections with both motile and nonmotile *Aeromonas* species."

Described as opportunistic pathogen; predominantly free-living in marine, but can cause broad pathogenicity.^{9,10}

Does not form spores.

Alteromonas mediterranea

Genus described as motile, via single flagella in Bergey. In 2nd edition *A. mediterranea* not listed as a species. Seems it was named in 2015. Genus requires salt to grow – found in marine environments. However, says "little is known about specific ecology" of the genus. Commonly found in Mediterranean sea, and probably common on tropical and subtropical waters.

Type strain on *A. mediterranea* was isolated from Adriatic Sea.¹¹

Literature & IMG/JGI suggests overlap with *A. macleodii*, which was only described species in Bergey.

All IMG/JGI samples from aquatic marine environments.

Do not form spores.

***Bacillus* genus notes**

From Bergey: "most species have little or no pathogenic potential and are rarely associated with disease in humans or other animals; an exception is *Bacillus anthracis*, the agent of anthrax; several other species may cause food poisoning and opportunistic infections, and strains of *Bacillus thuringiensis* are pathogenic to invertebrates."

“For example, *Bacillus anthracis* and *Bacillus mycoides* are nonmotile, while most *Bacillus cereus* strains are motile. The flagella of *Bacillus thuringiensis* may bind to insect cells and be important in virulence.”

“The ability to form endospores in aerobic conditions has been a defining character of the genus *Bacillus* since the 1920s, and has been applied in all editions of the Manual.”

“Most *Bacillus* species are saprophytes widely distributed in the natural environment, but some species are opportunistic or obligate pathogens of animals, including humans, other mammals, and insects. *Bacillus anthracis* is, to all intents and purposes, an obligate pathogen of animals and humans.”

Speaks about general issue of whether spore means actually lives/ thrives in that environment. Heat treatment usually done to isolates to activate spores. Therefore, just because isolated there, doesn't mean common there.

Bacillus amyloliquefaciens

Bergey's manual lists 'inner tissue of plants', paper/paperboard, as its habitats. Later states it has been isolated from inner tissues of healthy plants. “This organism has been isolated from a wide range of environments, including industrial amylase fermentations, foods and soil. It is phenotypically similar to *Bacillus subtilis*.”

Listed as motile in Table 3, page 73 of Firmicutes vol. Source listed as soil and industrial amylase fermentations. Can colonise roots and promote plant growth by inhibiting pathogens.¹² Also lives in rhizosphere.¹² “intimately associated with the plant since strains of *B. amyloliquefaciens* could be isolated in reasonably high titres from stems, leaves and pods of oilseed rape plants one month after initial treatment of the seed... isolated from cotton plant, but did not colonise barley or oilseed rape readily in lab.”¹³

Page 47 lists ecology, page 48 lists habitats.

Forms spores.

Bacillus anthracis

Obligate pathogen of animals. Bergey: “Generally considered to be an obligate pathogen; if it ever multiplies in the environment, it probably only does so rarely. Spores remain viable in soil for many years and their persistence does not depend on animal reservoirs.” Non-motile from table 3. “Virulence genes are carried by plasmids pX01 (toxins) and pX02 (capsule); these plasmids may be transmissible to other members of the *Bacillus cereus* group.”

Source: “blood of animals and humans suffering from anthrax, from anthrax carcasses, and from animal products and soil contaminated with spores of the organism.”

Page 43 has section on anthrax. Primarily a disease of herbivores, including sheep, cattle, goats and horses. Spores get into spleen via lymphatic system, where they produce toxin and cause septicemia – spores then exit the carcass.

Spores.

Bacillus cereus

Bergey: causes food borne illness an opportunistic infection. Ubiquity means these are not uncommon. When causing diarrhea, toxins produced in small intestine. Can also cause infection to eye after trauma. Based on list of conditions it can cause, seems to be capable of causing infections in many parts of body, either in immunocompromised people or through wound entry. Also causes infections in domestic animals, such as cattles.

Motile in table 3. Ecology: “as well as being a common organism in the natural environment and foods, *Bacillus cereus* frequently contaminates domestic kitchen environments.” Isolated from: soil, emperor moth caterpillars, feathers, honey bee & greater moth frass, inner tissues of plants, leather, milk, poultry manure, paper, sheep fleece, gemstones.

“Endospores are very widespread in soil, in milk and other foods, and in many other environments.” For actual vegetative cells multiply readily in food and may cause food poisoning, and occasionally causes opportunistic infections.

However, it can also promote plant growth by killing other pathogens, and can live in the rhizosphere.¹⁴ Also found in bird feathers.

Spores.

Bacillus licheniformis

Bergey: “Widely distributed in soil and many other environments, including milk and other foods, and clinical and veterinary specimens. Vegetative growth may occur readily in foods held at 30–50 °C. Occasionally reported as an opportunistic pathogen in man and other animals, and as a cause of food poisoning.”

Habitats include emperor moths, tissues of plants, feathers, leather, paper, gemstones etc. Literature suggests soil & bird feathers two of its main environments – it degrades beta keratin.¹⁵

Bacillus pumilus

Bergey: “Source: soil and many other environments, including foods, and clinical and veterinary specimens”. In addition to soil, isolated from: moth caterpillars, feathers, inner tissues off plants, leather, poultry manure, paper. “toxigenic strains of *Bacillus pumilus* have been isolated in association with food-borne illness and from clinical and environmental specimens.” Associated with bovine mastitis, and causes rare opportunistic infections in humans.

Described on NCBI species as ubiquitous soil organism. Says it colonises root zone of some plants.

Spores.

Bacillus subtilis

Bergey: “Endospores are very widespread in soil, dust and on vegetation, and in many other environments. The vegetative organisms participate in the early stages of the breakdown of organic matter.” and “The natural habitat of *Bacillus subtilis* is soil, which contains a wide range of carbohydrates and polysaccharides from micro- organisms, plants and animals, and so it can utilize a wide range of such substrates and possesses a large number of enzymes which degrade polysaccharides.” Page 45 says implicated in rare food poisoning in humans, but only when bacterial load ingested was very high, and mainly vomiting. Similar habitats to above species, inc moth, feathers, plants etc.

Also thought to be normal inhabitant of mammalian and insect guts, and marine sponges.¹⁶⁻¹⁸ Very diverse, capable of growth in many environments – however, hard to distinguish between where spores are vs where it grows... current evidence suggests “appears to grow in diverse environments including soils, on plant roots, and within the GI tract of animals.”¹⁹

Spores.

Bacillus thuringiensis

Bergey’s habitats listed: invertebrates, sheep fleece. Pathogenic to invertebrates.

“*Bacillus thuringiensis* strains produce crystalline, proteinaceous, parasporal bodies within the sporangia, and the insecticidal activities of many of these δ -endotoxins have made the organism one of the most widely produced and studied bacteria in bio- technology... The δ -endotoxin genes are designated with the “cry” prefix to indicate that their product proteins are crystalline; they are nearly always located on large conjugative plasmids.”

“They are known as δ -endotoxins or insecticidal crystal proteins, and are protoxins which may be toxic for certain insects and other invertebrates including flatworms, mites, nematodes and protozoa. The ability to synthesize parasporal bodies is plasmid borne...”

“Endospores are very widespread in soil and many other environments, and this organism has been isolated from all continents, including Antarctica. Although numerous strains are toxic to invertebrates, this property has not been demonstrated in many other strains. Natural epizootics do not seem to occur, and it has been suggested that the natural habitat of this organism is soil.”

Host range is insects and inverts, so phylum arthropoda, but flatworms are phylum platyhelminths, and nematodes also different phylum. So Animalia is range i.e. kingdom.

Spores or cells of *B. thuringiensis* are taken up by larvae via ingestion or by injury of cuticle. The toxins are then solubilised in the gut, and then they digest the insect, and then form spores.²⁰

Spores.

Bacillus velezensis

Promotes plant growth. Suppresses growth of pathogens inc bacteria, fungi and nematodes.²¹ Live in rhizosphere, as well as soil, plants, marine and animal intestine – some strains are endophytes, while others cannot colonise plant roots.²² Isolated from calf gut in Kyiv.

Spores.

Bifidobacterium animalis

Bergey on *Bifidobacterium* genus: “Gram-stain-positive, non-acid-fast, nonsporeforming, and nonmotile... anaerobic... present in the intestine of man, various animals, and honey bees. Also found in sewage, fermented milk products, and human clinical samples.” Considered obligate anaerobes.

On pathogenicity of genus: “*Bifidobacteria* have been isolated occasionally from clinical material and, although implicated in some opportunistic infections they are generally regarded as non- pathogens.”

On ecology of genus: “*Bifidobacteria* are typically found in the alimentary tracts of human infants and adults. Many factors control the number and the composition of microbial populations in different regions of the gastrointestinal tract.”

This species is similar to *B. longum* which lives in humans, and have been referred to as subspecies of each other in previous literature. Currently: “subsequently allotted to a DNA

homology group of bifidobacteria isolated from chickens, rats, rabbits, and sewage and proposed as the distinct species, *Bifidobacterium animalis* comb. nov.”

Bergey: “found in calves, chickens, rabbits, feces of rats, sewage, and fermented milk.” Can be identified as *B. animalis* if found in animal habitats, ferments arabinose and xylose, and also ferments both lactose and salicin, but does not ferment gluconate.

“*Bifidobacterium animalis* strains were subdivided into the subspecies *animalis* and *lactis* by Masco et al. (2004). This taxonomic rearrangement has been supported by genotypic and phenotypic data. *Bifidobacterium animalis* subsp. *animalis* contains strains isolated from rat feces, whereas *Bifidobacterium animalis* subsp. *lactis* includes strains isolated from chicken and rabbit feces, and from fermented milk and sewage.”

No spores.

Bifidobacterium breve

Bergey: “the type strain was isolated from the feces of a human infant.” Only found in humans, common in infants. Also found in breast milk.²³

No spores.

Bifidobacterium longum

Bergey: “a similar ecological distribution is shared by *Bifidobacterium longum* subsp. *longum* and *Bifidobacterium longum* subsp. *infantis*, which are particularly found in the gastrointestinal tracts of humans and infants, respectively; *Bifidobacterium longum* subsp. *suis* is typically found in the pig gastrointestinal tract.”

No spores.

Bordetella holmesii

Bergey on genus: “Gram negative. Nonmotile or motile by peritrichous flagella. Strictly aerobic... Mammalian and avian parasite and pathogen. Most species localize and multiply among the epithelial cilia of the respiratory tract... *Bordetella* species are isolated from warm-blooded animals, including humans. Until recently, the habitat of this genus was thought to be limited to the respiratory tract of the host. However, *B. holmesii* and *B. hinzii* have been isolated from human blood”

“Tang et al. (1998) have described a series of *Bordetella holmesii*-like strains associated with septicemia, endocarditis, and respiratory failure in humans. These isolates are phenotypically consistent with *B. holmesii* and have 99.8% 16S rRNA gene sequence similarity. Additional studies will be required to determine the taxonomic status of these strains.”

Bordetella holmesii is non-motile (TABLE BXII.b.33. of Bergey Proteobacteria, part C, page 668).

Bergey on species “Strains have been isolated from blood and upper respiratory tract cultures of humans. The type strain was isolated from human blood.”

Genus lives both extracellular and intracellular in host, and *B. holmesii* is emerging human pathogen which is host-restricted²⁴ Not a huge amount known about this species, since often mis-identified.²⁵ Described as obligate human pathogen.²⁶

No spores.

Bordetella pertussis

Bergey: “Parasitic, pathogenic, found only in the respiratory tract of humans, where it is the cause of pertussis (whooping cough).”

Bergey says non-motile (same table as *B. holmesii*), but 2019 paper says can be motile and express flagella – so will put both.²⁷ It has evolved to an obligate lifestyle in only humans.

Brucella abortus

Now merged with *Brucella melitensis* - some debate over whether moving *Brucella* to monospecific genus was a good idea, so will keep species in dataset.^{28,29}

Bergey on genus: “Gram negative. Nonmotile; do not produce flagella. Aerobic... Intracellular parasites, transmissible to a wide range of animal species including man...”

Pathogenicity: “*Brucella* species are pathogenic for a wide variety of animals, frequently producing generalized infections with a bacteremic phase followed by localization in the reproductive organs and the reticuloendothelial system. Infection in the pregnant animal often results in placental and fetal infection and this frequently causes abortion. The organisms may localize in mammary tissue and can be excreted in the milk. Because all the main species of meat- and milk-producing domesticated animals are susceptible to brucellosis and act as sources of human infection, the economic impact of the disease is enormous.”

“Typically, growth in vivo is intracellular and the organisms can survive within both granulocytes and monocytes. Infections in the natural host are rarely lethal and often mild, with clinical manifestations occurring mainly in the pregnant animal. Nevertheless, localization can occur in a wide range of organs with production of a variety of lesions.”

Ecology: “Although easily cultivated in vitro, under natural conditions *Brucella* species behave as obligate parasites and do not pursue an existence independent of their animal hosts. However, under suitable conditions of temperature and humidity they can persist in the environment, for example in soil or surface water, for long periods. Their distribution is worldwide, apart from the few countries from which they have been successfully eradicated.”

Bergey on *B. abortus*: “The usual natural hosts are cattle and other bovidae. Horses, camels, sheep, deer, dogs, man, and other species may also be infected. Placentitis and abortion are usually produced in the pregnant animal. Pathogenic for laboratory animals including rabbits, guinea pigs, and mice; the guinea pig is probably the most susceptible. Rough strains are usually avirulent, but mice retain the organisms in the spleen for some time after inoculation.”

Described as facultative intracellular pathogen, since can survive outside cells and occasionally in environment.

Brucella melitensis

Bergey: “The usual natural hosts are sheep and goats, but other species, including cattle, pigs, and humans, may be infected. Smooth cultures are usually pathogenic for the guinea pig and the mouse.” Very similar to *abortus*.

No spores.

Brucella suis

Bergey: “Biovars 1, 2, and 3 are naturally pathogenic for pigs. Biovar 2 also naturally infects hares. Strains resembling biovar 3 have also been isolated from various species of rodents. Biovar 4 is naturally pathogenic for reindeer (Davy-dov, 1961). Biovar 5 has only been isolated from rodents. All biovars, with the possible exception of biovar 2, are pathogenic for humans. Other species, including dogs, horses, and many species of rodents, may also be infected. In the natural hosts, generalized infections are produced, with localizing lesions, particularly in the genitalia. The testes, epididymides, and seminal vesicles are usually severely affected in the male. Metritis, placentitis, and abortion are produced in the pregnant female.”

No spore.

Buchnera aphidicola

Obligate intracellular endosymbiont, provides tryptophan to its aphid host.³⁰ Lives inside specialised cells called bacteriocytes.

Non-motile and don't form spores.

Burkholderia cenocepacia

Bergey on genus: “Motile by means of one or, more commonly, several polar flagella. One species (*Burkholderia mallei*) lacks flagella and is nonmotile... Over one-half of the species are pathogenic for plants or animals (including humans).”

B. cenocepacia not in Bergey 2010 version – *B. cepacia* has now been split into many species.

It is an opportunistic pathogen, primarily affecting CF patients.³¹ Members of the *B. cepacia* complex are widespread in the environment.

Motile, widespread in environment, particularly in rhizosphere, and is an opportunistic pathogen.³² Paper says can be isolated from soil, such as maize rhizosphere, and “*B. cenocepacia* can be associated with plants, including onions, sugarcane, maize, wheat, and legumes.”

Lives inside macrophages, infects intracellularly when inside mammalian host.³³

Burkholderia mallei

Bergey: “Parasitic on horses and donkeys, in which it causes glanders and farcy. The infection is transmissible to humans and to other animal species.”

“*B. mallei* is a highly evolved obligate parasite of horses, mules, and donkeys with no other known natural reservoir.”³⁴

Lost many genes for soil survival and is now adapted to life inside mammalian cells.³⁵

No spores.

Burkholderia pseudomallei

Bergey: “Isolated from human and animal cases of melioidosis and from soil and water in tropical regions, particularly Southeast Asia. Probably a soil organism and accidental pathogen, causing melioidosis.”

“The environmental bacterium *Burkholderia pseudomallei* causes melioidosis, an important endemic human disease in tropical and sub-tropical countries. This bacterium occupies broad ecological niches including soil, contaminated water, single-cell microbes, plants and infection in a range of animal species.... The bacterium has a broad range of ecological niches, and can be isolated from soil, surface water, amoebae, plants and infected humans and other animals in many tropical and sub-tropical regions... *B. pseudomallei* can be found in the stool of some infected humans and experimental murine models. This provides a potential mechanism for

human-to-environmental transmission and the possibility of repeated passage through the human host. Serial passage of *Burkholderia cenocepacia* in a long-term chronic airway infection model in mice has been shown to increase bacterial fitness¹³. Based on this observation, the natural passage of *B. pseudomallei* through humans, other animals or its natural predators such as soil amoebae might have enhanced and maintained selection pressure for pathogenicity in a subset of the population.”³⁶

Infects and multiplies intracellularly in tomato plant and caused disease – described as intracellular pathogen.³⁷

No spore.

Burkholderia thailandensis

Bergey: “Motile... Type strain was isolated from a rice field soil sample in central Thailand.”

Close to *B. pseudomallei*, but only rarely causes disease in humans or animals, requires inoculation of many more cells to be pathogenic.³⁸

“*Burkholderia thailandensis* is a member of the *Burkholderia pseudomallei* complex and is generally considered nonpathogenic... *B. thailandensis*, like *B. pseudomallei*, naturally occurs in the environment (e.g., in moist soils) and is associated with tropical or subtropical climate.”³⁹

Campylobacter coli

On *C. coli* and *C. jejuni*: “These bacteria are a common component of the intestinal microbiota of numerous bird and mammal species and cause disease in humans, typically via consumption of contaminated meat products, especially poultry meat.... Since the 1970s, *C. coli* and *C. jejuni* have been isolated from a wide range of wild and domesticated bird and mammal species, in which, typically, they are thought to cause few if any disease symptoms. Humans are usually infected by the consumption of contaminated food (especially poultry meat), water, milk, or contact with animals or animal feces.”⁴⁰

“Most of what is known about these species comes from isolates obtained from humans with disease, the food chain, and the agricultural environment. It is, however, important to note that such isolates are by no means representative of natural *Campylobacter* populations, and it is becoming increasingly apparent that much of the diversity present among the *Campylobacters* is in strains that colonize wild animals. Increasing numbers of novel genotypes are being found as *Campylobacter* populations are analyzed in different animal species, especially wild birds.”⁴⁰

“Asymptomatic carriage of *C. jejuni* and *C. coli* is thought to be rare in humans, especially among people in industrialized countries, suggesting that humans are not a primary host for

these organisms in these settings and that people are sporadically, and frequently pathologically, infected via the food chain from animal reservoir hosts.”

Motile, non-spore.

Campylobacter fetus

Bergey: “Pathogenic. A cause of abortion and infertility in cattle. Transmitted venereally. Found in the vaginal mucus of infected cows, the semen and prepuce of bulls, and in the placenta and tissues of aborted bovine fetuses. Pathogenic for cattle, guinea pigs, hamsters, and embryonated chicken eggs. Rarely isolated from human blood. Not pathogenic for rabbits, mice, or rats when injected intraperitoneally. Will not multiply in the intestinal tract of man and animals.”

“Our results support a broad view of the natural history of *C. fetus* where the bacterium originally colonized humans, likely the gut microbiota. Then within the past 10,500 years *C. fetus* likely began to colonize and adapt to cattle, giving rise to a modern day bovine lineage that displays significant genomic distinctions from the other genomes belonging to the human lineages, linked to host-microbe interactions and genome stability.”⁴¹

“*Campylobacter fetus* are important animal and human pathogens and the two major subspecies differ strikingly in pathogenicity. *C. fetus* subsp. *venerealis* is highly niche-adapted, mainly infecting the genital tract of cattle. *C. fetus* subsp. *fetus* has a wider host-range, colonizing the genital- and intestinal-tract of animals and humans.”⁴²

Chlamydia muridarum

Bergey on genus: “Coccoid, nonmotile, obligate intracellular organisms, 0.2–1.5 μm in diameter that parasitize and multiply in the cytoplasm of eukaryotic cells within membrane bound vacuoles, termed inclusions, by a unique developmental cycle... Chlamydiae cause a variety of diseases in humans, other mammals, and birds. Cultivation of Chlamydia in cell-free medium has not been achieved. They may be propagated in laboratory animals, the yolk sac of chicken embryos, or in cell culture. Chlamydiae require their hosts for low-molecular-weight synthetic intermediates that they use to synthesize DNA, RNA, proteins, and lipids. The genome size is among the smallest of prokaryotes, $1\text{--}1.3 \times 10^6$ bp.”

Bergey says forms things similar to spores: “Elementary bodies are 0.2–0.4 μm in diameter, have electron-dense DNA condensed with protein and a few ribosomes, and are surrounded by rigid trilaminar cell walls resistant to sonic lysis. Elementary bodies are infectious. Reticulate bodies are 0.6–1.5 μm in diameter, have less dense, fibrillar nuclear material, more ribosomes, and plastic trilaminar walls that are sensitive to sonic lysis. Reticulate bodies are not infectious.”

Spore-like structures.

C. muridarum only infects hamsters & mice, i.e. family Muridae.

Chlamydia psittaci

Bergey: Natural host is birds, “*Chlamydia psittaci* is comprised of six serovars, A–F, of avian chlamydial isolates (Andersen, 1991, 1997) that cause latent and clinically overt infections of the intestinal and respiratory tracts in many species of birds. Upon exposure of birds to crowding and stress, these infections often become severe, with diarrhea, air sacculitis, pneumonia, and fatal systemic spread with pericarditis and splenomegaly.”

Infects wide range of bird species, and rarely humans and other mammals.⁴³

“*C. psittaci* is an obligate intracellular bacterium replicating within a non-acidified vacuole, termed an inclusion. Within the inclusion, *C. psittaci* undergoes a unique biphasic developmental cycle alternating between the EB, which guarantees extracellular survival and infection of host cells and RB, which is responsible for intracellular replication and generation of infectious progenitor bacteria.... Today, *C. psittaci* has been demonstrated in about 465 bird species comprising 30 different bird orders”⁴⁴

Chlamydia trachomatis

Infects only humans. Similar to other *Chlamydia* species.⁴⁵

Bergey: “All *Chlamydia trachomatis* isolates have come from humans. *Chlamydia trachomatis* isolates are classified into three biovars, ocular, genital, and lymphogranuloma venereum (LGV) on the basis of tissue tropism, pathogenesis in humans and laboratory animals, and their biological characteristics in cell cultures.”

Citrobacter freundii

Bergey on genus: “Conform to the general definition of the family Enterobacteriaceae. Usually not encapsulated. Gram negative. Usually motile by peritrichous flagella. Facultatively anaerobic, having both a respiratory and a fermentative type of metabolism... Occur in the feces of humans and some animals; probably normal intestinal inhabitants. Sometimes pathogenic and often isolated from clinical specimens as opportunistic pathogens. Can also be found in soil, water, sewage, and food.”

Ecology: “As intestinal inhabitants of humans and animals, citrobacters are excreted into the environment and found in sewage, water, and soil. Apart from this, nothing is known about the ecological role of *Citrobacter* species in the environment.”

On *C. freundii*: “Found in humans and animals including mammals, birds, reptiles, amphibians, and fish. Also found in soil, water, sewage, and food. Often found in clinical specimens such as urine, throat, sputum, blood, and wound swabs as an opportunistic or secondary pathogen.”

“These organisms are found in soil, water, intestinal tract of animals, and in human clinical samples. Members of the genus *Citrobacter* are gram-negative, non-sporing rods... *C. freundii* with gastroenteritis, neonatal meningitis, and septicemia. It is known to cause health care associated infections of the urinary tract, respiratory tract, blood, and other normally sterile sites in the body. The chief cause is a weak and attenuated immune system and functioning of the body.”⁴⁶

No spores.

Clostridioides difficile

Synonym with *Clostridium difficile*.

Bergey on genus: “Motile or nonmotile. When motile, cells usually are peritrichous. The majority of species form oval or spherical endospores that usually distend the cell.”

On species (referred to as *Clostridium difficile*), it is usually motile and produces spores, “Pseudomembranous colitis in humans is caused by over- growth of the organism in the colon, usually after the flora has been disturbed by antimicrobial therapy. A very similar disease can be produced in hamsters and several other rodents by administration of antibiotics, but rats and mice are not affected. Although *Clostridium difficile* has been reported to cause cecitis in rabbits and hares, *Clostridium spiroforme* appears to be a more common cause of the disease in these animals.”

Sources listed by Bergey: “marine sediment, soil, sand, the hospital environment; camel, horse, and donkey dung; feces of dogs, cats, and domestic birds; the human genital tract; feces of humans without diarrhea; and rarely from blood and pyogenic infections in humans and animals.”

Clostridium botulinum

Bergey: There are different strain types. For types A & B: “soil, marine and lake sediments; animal, bird, and fish intestines; food (particularly improperly preserved vegetables, meat, and fish). (The types isolated reflect those present in the soil or sediment of the area. *Clostridium botulinum* toxin type F has been implicated in one case of infant botulism, but otherwise only types A and B have been reported. These two types also are most frequently isolated in outbreaks of food poisoning and cases of wound botulism.”

Types E & some B & F: “soil, marine and lake sediments, food, fish, birds, mammals.”

Types C & D: “feces and carcasses of animals and birds, soil, lake mud, rotting vegetation.”

Type G: “Culture supernatants are toxic to mice. Monkeys, chickens, guinea pigs, and mice are susceptible to the toxin; sheep and dogs are resistant. Source: soil, human autopsy.”

Most commonly found in the soil.⁴⁷

“Clostridium botulinum comprises a diverse group of botulinum toxin-producing anaerobic rod-shaped spore-forming bacteria that are ubiquitously distributed in soils and aquatic sediments. Decomposition of plants, algae, and animals creates anaerobic environments that facilitate growth of *C. botulinum*, which may then enter into food webs leading to intoxication of animals.... Clostridium botulinum is ubiquitously present in the environment in soils, dust, and the marine and freshwater sediments of wetlands, rivers, and lakes. Spores in soil may be mobilized by surface waters in heavy rain, or dust carried away by wind... The bacterium has been found in the intestinal tract of healthy fish, birds, and mammals. *C. botulinum* serotype E does not multiply in the fish gut (Bott et al., 1968), and fish fed 500,000 spores per day (in pellets) did not acquire botulism”⁴⁸

Also reported to be found in high numbers in decaying floating algal mats, including in vegetative form, and plants & algae may play a role. Insects also act as vectors for transmission, particularly aquatic instars or larvae. ⁴⁸

No spores.

Corynebacterium diphtheriae

Bergey on genus: Gram-positive, non-sporeforming, all species non-motile.

Pathogenicity, ecology, misc: “Corynebacterium diphtheriae, Corynebacterium ulcerans, and Corynebacterium pseudo-tuberculosis are the only species which may produce diphtheria toxin (DT), a potent exotoxin which plays a significant role in pathogenicity. The resultant disease, diphtheria, has historically been associated with significant morbidity and mortality in humans and animals, prior to universal use of an efficacious vaccine in countries around the world with higher socio-economic means.... It has been known since the 1950s that nontoxigenic strains of Corynebacterium diphtheriae could become toxigenic after infection by temperate bacteriophages and a one-to-one correlation exists between the presence of the tox gene mechanism on a bacteriophage and subsequent DT production.”

Ecology: “There are at time of writing, ~51 medically relevant species have been described that cause occasional infections in humans or are transmitted to humans by zoonotic means. Most are deemed to be rare opportunistic pathogens. Some species thought to be part of the common skin flora, such as Corynebacterium amycolatum, Corynebacterium jeikeium... Corynebacterium species can cause significant infection or appear to be commensals in animals or birds. Virulence factors and mechanisms of pathogenicity remain understudied...”

Source of species: “clinical specimens from humans or animals.”

“Even though the organisms commonly exist in different body sites of humans including the skin, upper respiratory tract, gastrointestinal tract, and urogenital tract, relatively few are pathogenic. The most known of these is the etiologic agent of diphtheria, *C. diphtheriae*... Diphtheria is a disease characterized by local inflammation, mainly in the upper respiratory tract, caused by *C. diphtheria*. *C. diphtheria* infection is typically characterized by a local inflammation, usually in the upper respiratory tract, associated with toxin-mediated cardiac and neural disease.”⁴⁹

Narrow host range, usually only infects humans: “Our report is the first on the isolation of *C. diphtheriae* from a wildlife animal without any previous human contact. In contrast, the very few unambiguous publications on *C. diphtheriae* in animals referred to livestock or pet animals with close human contact. *C. diphtheriae* carriage in animals has to be considered as an exceptionally rare event.”⁵⁰

“Unlike other diphtheroids (coryneform bacteria), which are ubiquitous in nature, *C. diphtheriae* is an exclusive inhabitant of human mucous membranes and skin. Spread is primarily by airborne respiratory droplets, direct contact with respiratory secretions of symptomatic individuals, or exudate from infected skin lesions. Asymptomatic respiratory tract carriage is important in transmission. In areas where diphtheria is endemic, 3–5% of healthy individuals can carry toxigenic organisms, but carriage is exceedingly rare when diphtheria is rare. Skin infection and skin carriage are silent reservoirs of *C. diphtheriae*, and organisms can remain viable in dust or on fomites for up to 6 mo. Transmission through contaminated milk and through an infected food handler has been proved or suspected.”⁵¹

One paper says ‘Recognised as obligate human pathogen’⁵², even though also reported to live on surfaces for 6 months.⁵³

Corynebacterium glutamicum

Bergey: “*Corynebacterium glutamicum* (environment, production of glutamic acid)... source: sewage.”

“*Corynebacterium glutamicum* is a non-pathogenic bacterium widely used in industrial amino acid production and metabolic engineering research... The non-spore-forming Gram-positive bacterium *Corynebacterium glutamicum*, a non-pathogenic species in the *Corynebacterium* genus, has been widely used for the industrial production of amino acids, because of its numerous and ideally suited attributes...”⁵⁴

“*Corynebacterium glutamicum* is a predominantly aerobic, rod-shaped, Gram-positive soil bacterium which is generally recognized as safe.”⁵⁵

Most genomes will be from industry, so be careful – many will be clones. Not much known, except lives in soil and is non-pathogenic.

While mostly thought as extracellular pathogen, growing evidence it can invade cells.⁵⁶

No spores.

Corynebacterium pseudotuberculosis

Bergey: “infections in sheep, goats, horses, and other warm- blooded animals; occasionally linked to disease in humans.... *Corynebacterium pseudotuberculosis* (ovine caseous lymphadenitis and other sheep or goat diseases)”

“*Corynebacterium pseudotuberculosis* is an important animal pathogen. It is the etiological agent of a disease that is commonly called caseous lymphadenitis (CLA) or cheesy gland. This disease is found in all the world’s major sheep and goat production areas, causing significant economic losses.”⁵⁷

“This microorganism is a facultative intracellular pathogen that exhibits pleomorphic forms, such as coccoids and filamentous rods... non-sporulating, non-capsulated and non-motile bacterium... Though *C. pseudotuberculosis* was originally identified as the causative microorganism of CLA in sheep and goats, this bacterium has also been isolated from other species, including horses, in which it causes ulcerative lymphangitis and pigeon fever in cattle, camels, swine, buffaloes, and humans... The most frequent form of the disease, external CLA, is characterized by abscess formation in superficial lymph nodes and in subcutaneous tissues. These abscesses can also develop internally in organs, such as the lungs, kidneys, liver and spleen, characterizing visceral ”⁵⁷

Will categorise as obligate, since needs host. Likely the facultative in above paper referred to the intracellular, not pathogenicity i.e. it can survive outside cells, but host is main part of its lifestyle.

Corynebacterium ulcerans

Bergey on species: “*Corynebacterium ulcerans* (goats, pigs, cattle, horses, cats, dogs, otters, and other)... Source: animals and humans. Infection of farm animals or their milk can result in the transmission of infection to humans. Infections from companion pets have been documented to be transferred to and cause disease in humans.”

“*Corynebacterium ulcerans* (*C. ulcerans*) may cause diphtheria in humans and can be carried by a wide range of animal species including dairy cows and, more recently, dogs and cats that have been increasingly involved in zoonotic transmission.”⁵⁸

“By contrast, the *C. ulcerans* reservoir is thought to be animals. Cases of *C. ulcerans* have been reported after consumption of raw dairy products and contact with cattle, pigs, and domestic pets. *C. ulcerans* diphtheria person-to-person transmission has been proposed but has yet to be confirmed.”⁵⁹

Coxiella burnetii

Bergey on genus: “Strictly intracellular bacteria, usually 0.2–0.4 μm 0.4–1.0 μm . Best stained by Gimenez staining. They have no flagella or capsule. They live in close natural association with arthropod and vertebrate hosts. The genus includes *Coxiella burnetii*—the agent of Q fever—and endosymbionts of ticks and aquatic invertebrates. *C. burnetii* grows well in cultured cell lines and in the yolk sac of chicken embryos, where it undergoes a cycle of development with formation of an endospore-like body.”

On animal infection: “Livestock—including cattle, sheep and goats—and pets are major reservoirs of *C. burnetii*. The pathogen can be found in urine, feces, milk, and birth products of these animals and transmitted to humans by these substances as well as by aerosols. Animals are usually not harmed by *C. burnetii*, although association of the organisms with infertility, abortion, low birth weight and death of newborns, in animal hosts has been reported.... Rats, mice, guinea pigs, rabbits, and monkeys have been used in laboratory studies; infection can produce no symptoms, can produce fever and granulomas, or can lead to death.”

“*Coxiella burnetii* is an obligate intracellular pathogen that causes acute and chronic Q fever. *C. burnetii* grows within a eukaryotic host cell in a vacuole highly similar to a phagolysosome. Found worldwide, this environmentally stable pathogen is maintained in nature via chronic infection of ruminants. Aerosol-mediated infection of humans results in infection and usurpation of alveolar macrophages... exhibits a bi-phasic life cycle consisting of an environmentally stable small cell variant (SCV) and replicative large cell variant (LCV) of approximately 0.3 and 2.0 μm in size, respectively [kind of like spore?] Although *C. burnetii* constitutes a single species, numerous isolates have been harvested from diverse disease settings and mammalian reservoirs.”⁶⁰

“Evidence for infection exists in a multitude of vertebrate and invertebrate hosts [3]. Infected domestic livestock, especially goats, sheep and dairy cattle are responsible for the vast majority of human Q fever cases. These zoonotic reservoirs shed *Coxiella* into the environment that can fuel human outbreaks.”⁶¹

“Q fever is a zoonosis with a worldwide distribution. The reservoir is large and includes many wild and domestic mammals, birds, and arthropods such as ticks. Babudieri, in a large review, reported that *C. burnetii* was detected in virtually all the animal kingdoms. However, domestic ruminants represent the most frequent source of human *C. burnetii* infection... Over 40 tick species are naturally infected with *C. burnetii*... ticks may play a significant role in the transmission of coxiellosis among the wild vertebrates, especially in rodents, lagomorphs, and

wild birds.... Birds may also be infected, and *C. burnetii* was isolated from pigeons, chickens, ducks, geese, and turkeys... Anti-*C. burnetii* antibodies have been found in snakes and tortoises in India, but *C. burnetii* has not been isolated from these animals.”⁶²

Cutibacterium acnes

Synonym of *Propionibacterium acnes* (how it is listed in Bergey).

Bergey on *Propionibacterium* genus: “Gram-stain-positive, nonmotile, non-acid-fast, non-sporeforming.”

Bergey on species: “Source: the bacterium primarily colonizes the sebaceous gland-rich areas of human skin, but the organism is also found in the mouth, as well as the genito-urinary tract and large intestine. In relation to infections, it has been recovered from comedones of acne vulgaris, wounds, blood, pus, soft tissue abscesses, the surface of indwelling medical devices, and eye infections. Levels of *Propionibacterium acnes* colonization on the skin vary from person-to-person and from the area of the body sampled.”

“*Propionibacterium acnes* is a major inhabitant of adult human skin, where it resides within sebaceous follicles, usually as a harmless commensal although it has been implicated in acne vulgaris formation... The genome sequence offers insights into the traits that favor *P. acnes* as a ubiquitous commensal on human skin.”

Dehalococcoides mccartyi

Isolated from rivers and sediment, particularly those contaminated with halogens – also isolated from sludge and wastewater.⁶³

IMG/JGI has isolates from: groundwater, freshwater, sediment, river/lotic (flowing water).

Description of genus: “Cells are non-motile, non-spore-forming, non-pigmented and disc-shaped.” “Cells stain Gram-indifferent and peptidoglycan in the cell wall is absent.” Type strain of species: “isolated from an anaerobic reactor seeded with digester sludge from a wastewater treatment plant at Ithaca.”⁶³

Vol I of Bergey is not searchable, and not much on Chloroflexi phyla (page 430). Also no mention of actual species or genus.

Elizabethkingia anopheles

Bergey on genus: “Nonmotile. Flagellar motility, gliding movement, and swarming growth have not been reported... As they are ubiquitous in soil and freshwater, strains of

Elizabethkingia meningoseptica occasionally occur in the hospital environment and clinical specimens; they are opportunistic pathogens of humans and various animals.”

But nothing on this species. Was defined in 2011, so after publication of this Bergey Edition.

Isolated from midgut of mosquito *Anopheles gambiae*, from insects kept in captivity but initially originating from McCarthy Island, The Gambia.⁶⁴

Description of species: “aerobic Gram-reaction-negative, non-motile, non-spore-forming rods.”⁶⁴

“Flavobacteria (members of the family Flavobacteriaceae) dominate the bacterial community in the *Anopheles* mosquito midgut. One such commensal, *Elizabethkingia anophelis*, is closely associated with *Anopheles* mosquitoes through transstadial persistence (i.e., from one life stage to the next)...” seems different species associated with different species or mosquito – unclear if these are *E. anopheles* or other species from genus.⁶⁵

For example, a different species, *Elizabethkingia meningoseptica*, as found in *Anopheles stephensi*.⁶⁶

Unknown if facultative or obligate, but given can be reared in water and then the mosquitoes feed, seems unlikely to be obligate.

Really not much known about its natural habitat, so most lifestyle traits are unknown.

Can cause opportunistic infections in humans, and cause of a number of mosquito-borne outbreaks.⁶⁷

Not clear if commensal in mosquito, or uses mosquito as vector to get to mammals, or both.

Enterobacter cloacae

Bergey on genus: Motile by peritrichous flagella, gram negative. “*Enterobacter* species are found in the natural environment including water, sewage, vegetables, and soil.... Nitrogen-fixing strains of *E. cloacae* have been isolated from the roots of dryland and wetland rice. The nitrogen-fixing *E. cloacae* strains isolated belong to *E. cloacae* genomic group 5. A strain identified as *E. cloacae* suppresses sporangium germination in *Pythium ultimum* (the cause of seed and root diseases of a wide variety of plants). This is achieved by a competitive utilization of fatty acids present in the exudate of germinating. Some strains identified as *E. cloacae* are endophytic symbionts of corn.”

On species sources: “Occurs in water, sewage, soil, meat, and hospital environments and on the skin and in the intestinal tracts of man and animals as a commensal. May cause nosocomial infections.”

Opportunistic pathogen, part of normal flora of gut and widely distributed in the environment.⁶⁸

Enterobacter cloacae complex (Ecc) species are widely distributed opportunistic pathogens mainly associated with humans and plants.⁶⁹

Also found in wild migratory birds – isolated from gut.⁷⁰

“*E. cloacae* has been isolated from food, especially from samples of formula containing plants, raw vegetables, and roots, as well as from drinking water.”⁷¹

Enterobacter hormaechei

“*Enterobacter hormaechei* is commonly considered a causative pathogen for nosocomial infections and it does not usually cause diseases in animals. However, researchers have recently dissociated the pathogenic *Enterobacter hormaechei* from foxes and piglets. Here, the *Enterobacter hormaechei* was first found to be associated with respiratory disease in unweaned calves in China.... *E. hormaechei* is widespread in many environmental niches. It is commonly considered a causative pathogen of nosocomial infections, and it does not usually cause diseases in animals other than humans.”⁷²

Bergey says ‘isolated from clinical specimens’.

Can’t find any specific niches, beyond usual *Enterobacter* spp.

In IMG/JGI, isolated from: humans, insect gut (though only 2 genomes, and seem the same), mine(‘decant and tailings from uranium mine’), oil contaminated soil. Vast majority from humans though.

Enterococcus faecalis

Bergey on genus: “Gram-positive. Cells are ovoid, occur singly, in pairs, or in short chains, and are frequently elongated in the direction of the chain. Nonsporeforming. Strains of some species may be motile by scanty flagella.”

“*Enterococcus faecalis* represents the most commonly isolated enterococcal species from human clinical material (80–90%) followed by *Enterococcus faecium* (8–16%).”

“More species are found in animals. Poultry is commonly inhabited by *Enterococcus faecium*, *Enterococcus cecorum*, *Enterococcus faecalis*, *Enterococcus hirae*, and *Enterococcus durans*;

in contrast *Enterococcus gallinarum* and *Enterococcus avium* are rare... *Enterococcus faecalis* is a prevailing species in rectum and tonsils of dogs and cats.”

“Enterococci can also be isolated from food, plants, soil, and water. Although these bacteria are considered to be only a temporary part of the microflora of plants, in good conditions they can propagate on their surface. *Enterococcus faecalis*, *Enterococcus faecium*, *Enterococcus hirae*, *Enterococcus mundtii*, *Enterococcus casseliflavus*, and *Enterococcus sulfureus* were isolated from plants. They are generally isolated more often from flowers than from buds or leaves. This indicates that insects are probably involved in the dissemination of enterococci. Soil can be contaminated by enterococci from animals and/or plants thanks to wind or rain. It is not naturally inhabited by enterococci as shown by Medrek and Lidsky (1960) who isolated enterococci in only 8 out of 369 samples of soils that were unaffected by human activity. Occurrence of enterococci in waters is generally considered to be the result of fecal contamination. Typically, *Enterococcus faecalis*, *Enterococcus faecium*, *Enterococcus durans*, and *Enterococcus hirae* can be commonly found in contaminated waters.”

On species: “Isolated from human and veterinary clinical materials, from food, and the environment. Typically associated with intestines of humans and animals.”

Table 116, page 598 of Bergey Firmicutes says *E. faecalis* is non-motile.

Table 1 in ⁷³ says *E. faecalis* found in: Human, animal (multiple), plant, insect.

Paper also says about *Enterococcus* sp.: “these bacteria are widely distributed in a variety of environmental habitats, even when there is little or no input from human and/or animal fecal sources. These extraenteric habitats include soil and sediments, beach sand, aquatic and terrestrial vegetation, and ambient waters (rivers, streams, and creeks); they may also be considered heterothermic habitats, in which temperatures are variable, in contrast to the gastrointestinal tract of warm-blooded animals, where the temperature is relatively constant.”

Enterococcus faecium

Bergey on species: “Isolated from human and veterinary clinical materials, from food, and the environment.”

Table 116, page 598 of Bergey Firmicutes says *E. faecium* is non-motile.

Table 1 in ⁷³ says *E. faecium* found in: Human, animal (multiple), plant, insect

Escherichia coli

Bergey: “*E. coli* is a natural and essential part of the bacterial flora in the gut of humans and animals. Most *E. coli* strains are nonpathogenic and reside harmlessly in the colon; however, certain serotypes or clones play an important role in both intestinal and extraintestinal diseases.

The diverse pathogenesis of this bacterium in apparently healthy individuals is largely attributable to its possession of a variety of specific virulence factors. In hosts with compromised defenses, *E. coli* can also be an excellent opportunistic pathogen.”

On species: “Occurs naturally in the lower part of the intestine of warm- blooded animals, and as intestinal (some foodborne) and extraintestinal pathogens of humans and animals.”

“The commensal form of *E. coli*, exemplified by strain MG1655 (Touchon et al., 2009), is traditionally considered as a harmless bacterium that lives in the intestinal system of mammals and assists its host in the breakdown of particular carbon compounds.”⁷⁴

“The extent to which *E. coli*, in particular pathogenic strains, can survive in open environments and which factors affect this survival rate are crucial issues from a fundamental point of view. Next to their capabilities to acquire nutrients, some *E. coli* strains produce filamentous structures that extend from the cell surface and help cells to attach to surfaces (for example, plant surfaces). Owing to this ability, *E. coli* coming from soil, manure, irrigation water or contaminated seeds may colonize (and thus find a refuge niche in) plants such as radish and lettuce. Specifically, *E. coli* may access roots or leaf surfaces by splashing during rainfall or irrigation. Internal plant compartments can also be colonized, so that the *E. coli* cells cannot be easily washed from plant parts or be killed and removed by disinfectants and washing.”⁷⁴

“Provided resource availability and key abiotic conditions are propitious, *E. coli* populations can survive and even grow in open environments... *E. coli* can, to varying extents, survive in different open environments such as soil, manure and water. There are also possibilities for migration between these habitats. For instance, *E. coli* may reach the groundwater from top soil layers, as revealed in several studies. Soil factors such as porosity, surface area, bulk density and macropore structure have important roles in the leaching of invading bacteria by their influence on adsorption and gravitational movement with water...”⁷⁴ However, says population sizes often decline over time in soil, though can survive for almost a year. Still, seems like just there to get into next host.

“*Escherichia coli* is a non-spore-forming, Gram-negative bacterium, usually motile by peritrichous flagella.”⁷⁵

Wild birds are carriers of *E. coli*.⁷⁶

Francisella tularensis

Bergey on genus: Non-motile, no spores. “*F. tularensis* is a facultative intracellular bacterium ... cause the zoonotic disease tularemia in humans and animals, predominantly rodents and lagomorphs.”

“*F. tularensis* is widely distributed in nature and is found throughout the Northern Hemisphere. Large outbreaks, especially in parts of the continental USA, the southern part of the former USSR, and northern Scandinavia, have been reported. Four subspecies... are recognized. All have been associated with human disease, although they differ in virulence for humans and rabbits. Tularemia is transmitted by direct contact with infected animals, through contaminated water or food, or by vectors such as biting insects or ticks. Airborne transmission also occurs, especially during processing of agricultural products. The disease is often epidemic, both in humans and in animals, and clinical manifestations depend on the type of reservoir involved and the means of transmission. The bacterium has been isolated from approximately 250 wildlife species, many of which allow transmission of tularemia to man (Olsufjev, 1974).”

“The gram-negative bacterium *Francisella tularensis* survives in arthropods, fresh water amoeba, and mammals with both intracellular and extracellular phases and could reasonably be expected to express distinct phenotypes in these environments. The presence of a capsule on this bacterium has been controversial with some groups finding such a structure while other groups report that no capsule could be identified. Previously we reported in vitro culture conditions for this bacterium which, in contrast to typical methods, yielded a bacterial phenotype that mimics that of the bacterium's mammalian, extracellular phase.... *F. tularensis* also is a naturally-occurring zoonotic bacterium found in strikingly diverse environments including an array of warm- and cold-blooded hosts (mammals, insects, arthropods, fresh water protozoans... In addition to the broad range of hosts, it has recently become appreciated that within both mammalian and protozoan infection models, the bacterium has both intracellular (replicative) and extracellular (transmissive) phases”⁷⁷

“Tularaemia has been reported in more than 250 animal species including man, other mammals, birds, fish, amphibians, arthropods and protozoa.”⁷⁸

Fusobacterium nucleatum

Bergey on genus: Gram negative, don't form spores. “*Fusobacterium* are normal inhabitants of the mucous membrane of humans and animals. The habitat of *Fusobacterium nucleatum* and *Fusobacterium periodonticum* is the human oral cavity; the gingival crevice is the niche of both.”

On species: cells don't possess pilli or flagella. Five subspecies.

“*Fusobacterium nucleatum* is a gram-negative anaerobe, which belongs to the Bacteroidaceae family and is found naturally in the microflora of the mouth in healthy or diseased humans.... Many of the oral flora are commensals but a few are opportunistic pathogens. *F. nucleatum* can be isolated not only from the mouth but also from infections such as skin ulcers, peritonsillar abscesses, septic arthritis, and endocarditis”⁷⁹

Seems to live in humans only – can only find mice experiments where they purposely infected them with it.

“The majority of *F. nucleatum* isolates originate from oral sites, however the species is also a resident of the human gastrointestinal tract.”⁸⁰

Fusobacterium periodonticum

Bergey on species: “Obligately anaerobic, nonmotile, nonsporeforming, Gram-stain-negative rod”. In above section, says also lives in gums. Same habitat as *Fusobacterium nucleatum*.”

“*F. periodonticum* isolates are not restricted to the oral niche [found in gut]... Interestingly, 4 isolates recovered from gut biopsy specimens were typed as *F. periodonticum*, suggesting that this particular species, like *F. nucleatum*, is not restricted to the oral niche.”⁸⁰

Granulibacter bethesdensis

No info about it in Bergey – genus not listed in contents under Acetobacteraceae.

“*Granulibacter bethesdensis* is a recently described gram-negative bacterium in the family Acetobacteraceae; it has been isolated from 6 patients with CGD from North and Central America and Spain... Unlike most bacteria, *G. bethesdensis* can cause acute infections, relapses after apparently effective therapy, and reinfections, which indicate a lack of sterilizing and protective immunity in persons with CG... *G. bethesdensis* has thus far been isolated only from patients with CGD”⁸¹

“*Granulibacter bethesdensis*, an emergent Gram-negative pathogen, has been cultured from at least 9 CGD patients and has been associated with 2 fatalities in CGD patients in North America and Europe... *Granulibacter* shares with several other known intracellular pathogens a phenotype of causing delayed neutrophil apoptosis as well as decreased stimulation of host defenses compared to other microbes. Ongoing studies are examining the persistence of *Granulibacter*, particularly in macrophages, and its subcellular trafficking during infection, with the working hypothesis being that metabolic adaptations of this microbe to slow intracellular growth contribute to its apparent long-term survival in human patients with CGD without causing overt clinical disease.”⁸²

Non-motile.⁸³

Haemophilus influenzae

Bergey on genus: non-motile, gram-negative, “Occur as obligate parasites on the mucous membranes of humans and a variety of animal species.”

Pathogenicity: “Among the *Haemophilus* species that colonize man, *H. influenzae* is clearly the most important from a clinical point of view. Although not responsible for epidemic influenza, as was originally believed, it is involved in a variety of numerically important and severe infections (for review see Turk and May, 1967). These infections can be divided into two groups: (a) acute, pyogenic, and usually invasive infections in which *H. influenzae* (almost exclusively serovar b) is the primary pathogen, and (b) noninvasive infections (often chronic) in which primarily non-encapsulated *H. influenzae* play an important, though in many cases probably a secondary, part.”

Ecology: “Humans are the only natural host of the following species: *H. influenzae*. *H. influenzae* is found exclusively behind the palatine arches.” 25-84% of children carry *H. influenzae* in the pharynx.

On species: “*H. influenzae* is present in the nasopharynxes of a majority of healthy children. The carriage rate in adults is somewhat lower (Kilian and Frederiksen, 1981a). It is rarely encountered in the human oral cavity and has not been detected in any animal species apart from chimpanzees.”

Chimps are part of same family as humans (Hominidae) and also same subfamily (Hominae).

Helicobacter pylori

Synonym of *Campylobacter pylori*.

Bergey on genus: “Nonsporeforming. Gram negative. Motile with a rapid corkscrew- or slower wave-like motion due to flagellar activity... Many species are pathogenic for humans and animals. Found in the intestinal tract, oral cavity, and internal organs of man and animals.” *H. pylori* is leading microbiological cause of gastric ulcers in humans.

“The principal reservoir of *H. pylori* is the human, with infection occurring mainly in childhood (Mitchell et al., 1992). *H. pylori* has been found in rhesus monkeys, certain macaque species, and in a closed colony of barrier-maintained cats (Fox and Lee, 1997). The latter instance appears to be a unique report in domestic pets, and human-to-animal transmission has been suggested as the most likely explanation (El-Zaatari et al., 1997). Subsequent investigations have not detected *H. pylori* in either cats (Jalava et al., 1998) or dogs (Eaton et al., 1996a) from random sources.” Basically only naturally in humans, maybe in monkeys, but unclear if these are captive. Can live in animals in lab setting, but usually asymptomatic.

Table 1 of ⁸⁴ states human & primates are primary host, and paper describes range as ‘narrow’: “*H. pylori* has a narrow host range and is found almost exclusively in humans and some nonhuman primates”. Can be used as animal model in lab, but requires high number of cells etc. Also: “The only known niche of *H. pylori* is the human gastric mucosa”

Seems rhesus monkeys only infected when ‘socially housed’ i.e. kept in captivity.⁸⁵

Klebsiella aerogenes

Was called *Enterobacter aerogenes*.⁸⁶ Can cause opportunistic infections.⁸⁷

Also known as *Klebsiella mobilis*. Bergey’s notes on *K. mobilis*: “This is the only motile species in the genus. Strains producing extended-spectrum β -lactamase occur as nosocomial pathogens. Occur in water, sewage, soil, dairy products, and the feces of humans and animals. Also an opportunistic pathogen.”

“Its natural habitat is believed to be soil and water, but the organism is occasionally found in the feces and respiratory tract of humans.”⁸⁸

Isolated from cow⁸⁹ – suggests broad range.

Klebsiella oxytoca

Bergey on species: “Present in the intestinal tract of humans and animals. Can be isolated from various pathological processes and from botanical and aquatic environments.”

Bergey on genus ecology: “*Klebsiella* can frequently be isolated from the root surfaces of various plants. *K. pneumoniae*, *K. oxytoca*, or *K. planticola* are capable of fixing nitrogen and are classified as associative nitrogen fixers.”

“*Klebsiella* is a common opportunistic pathogen for humans and other animals, as well as being resident or transient flora (particularly in the gastrointestinal tract). Other habitats include sewage, drinking water, soils, surface waters, industrial effluents, and vegetation. Until recently, almost all these *Klebsiella* have been identified as one species, ie, *K. pneumoniae*. However, phenotypic and genotypic studies have shown that “*K. pneumoniae*” actually consists of at least four species, all with distinct characteristics and habitats. General habitat associations of *Klebsiella* species are as follows: *K. pneumoniae*--humans, animals, sewage, and polluted waters and soils; *K. oxytoca*--frequent association with most habitats; *K. terrigena*--unpolluted surface waters and soils, drinking water, and vegetation; *K. planticola*--sewage, polluted surface waters, soils, and vegetation; and *K. ozaenae*/*K. rhinoscleromatis*--infrequently detected (primarily with humans).”⁹⁰

K. oxytoca isolated from sweet potato plants, particularly in the stems.⁹¹ Considered an endophyte, i.e. promotes plant growth.

Klebsiella pneumoniae

From above ref, found in humans, animals, sewage, and polluted water and soils.

Bergey: “*K. pneumoniae* is normally found in the intestinal tract of humans and animals. It may be isolated in association with several pathological processes in humans... In animals, *K. pneumoniae* may be isolated from metritis in mares, bovine mastitis, or generalized infections in captive monkeys. Environmental strains generally utilize more carbon sources than clinical strains”

Lactobacillus brevis

L. brevis group now moved into own genus called *Levilactobacillus*. Description of species: “The species was described in [2, 289]. Strains of *L. brevis* widely occur in vegetable and cereal fermentations and as beer-spoilage organisms and were also isolated from insects. Niche adaptation is mediated by acquisition of plasmids [290]. The genome size of the type strain is 2.47 Mbp. The mol% G+C content of DNA is 46.0. Isolated from milk, cheese, sauerkraut and related vegetable fermentations, sourdough, silage, cow manure, faeces, and the mouth and intestinal tract of humans and rats.”⁹²

“Specifically, the *L. perolens*, *L. sakei*, *L. vaccinostercus*, *L. collinoides*, *L. brevis* and *L. buchneri* groups are almost completely composed of species that are rarely found in animals and are therefore likely free living.”

Bergey has Table 83 on page 478, where it says if in intestine of different animals. *L. brevis* in human, pig, birds and cattle. Bergey has same wording as above description for source of species.

Lactobacillus casei

L. casei group now called *Lacticaseibacillus*.

Bergey on *L. casei*: non-motile. Isolated from milk, cheese, and intestinal tract.

“Isolates were obtained from diverse sources including Chinese traditional pickle, infant faeces, corn liquor, oat silage, commercial dietary supplements, sputum, nasopharynx [47]. Information on the lifestyle of *L. casei* is confounded by the unclear taxonomy over the past decades; most genomes of designated as *L. casei* in the NCBI database should be classified as *L. paracasei* instead [10, 47].”⁹²

Table 1 of ⁹³: habitat is “Raw and fermented dairy; silage, fermented vegetables, vertebrate digestive tract”

Lactobacillus delbrueckii

Bergey: non-motile, 4 subspecies. Sources: fermented plant material, yogurt and cheese, compressed yeast, grain mash, dairy products.

Isolated also from intestine of piglets, veg, fermented turnip⁹²

Isolated from plants in Bulgaria, may be natural habitat. ⁹⁴

“Nevertheless, the three subspecies are also very interesting from an evolutionary point of view since they present different fermentation patterns and habitat: whereas the subspecies *bulgaricus* and *lactis* are almost exclusively present in milk, the subspecies *delbrueckii* colonizes vegetable sources.”⁹⁵ Also says non-motile & non-spore.

“The large and diverse *L. delbrueckii* group comprises a cluster of species adapted to insects and another adapted to vertebrates including pigs and hamsters and different species of birds.”⁹³

Lactobacillus fermentum

New genus: *Limosilactobacillus*.

Description in Zheng 2020: “*L. fermentum* is the only species in *Limosilactobacillus* that is not adapted to the intestine of vertebrates [17]. Occurs widely in spontaneously fermented cereals and other fermenting plant materials [58, 255], also in dairy products, manure and sewage, and the faeces and vagina of humans.”⁹²

“*L. fermentum* is the only species in the *L. reuteri* group which is rarely found in intestinal ecosystems but frequently isolated from plants and spontaneously fermented cereals (Mundt and Hammer 1968; Hammes and Hertel 2006; Gänzle and Ripari 2016). *L. fermentum* could be an example of a species undergoing reversion of the lifestyle from host adapted to free living, a process that has been documented for environmental species that cluster within phylogenetic clades dominated by symbionts.”⁹³

Bergey: “Isolated from yeast, milk products, sourdough, fermenting plant material, manure, sewage, and mouth and feces of humans.”

Lactobacillus helveticus

Zheng 2020: “Part of the core microbiome of chicken [92] but it was also isolated from sour milk, cheese starter cultures and cheese, particularly Emmental and Gruyère cheeses, and in tomato pomace and silage.”

“Interestingly, our analysis proposes the vertebrate gut as the real habitat of *L. helveticus* (Fig. 2 and Fig. S1) although the species is well recognized for its role in cheese production. Accordingly, several recent studies show that *L. helveticus* is a dominant member of the chicken microbiota”⁹³

Lactobacillus paracasei

Now in genus *Lacticaseibacillus*.

Zheng 2020: “Strains of this species have a nomadic lifestyle and were isolated from a variety of courses including the human oral cavity [169], fermented cereals, vegetables, meats, dairy products and in invertebrate hosts.”

Bergey: 2 subspecies, isolated from: “dairy products, sewage, silage, humans, and clinical sources” and “dairy products”.

Very similar to *L. casei*. Found in plants, dairy and mammalian origin: “*Lactobacillus paracasei* is a member of the normal human and animal gut microbiota and is used extensively in the food industry in starter cultures for dairy products or as probiotics”⁹⁶

“The most diverse genus of LAB is *Lactobacillus*, which encompasses species found mainly in dairy products (e.g., *Lactobacillus delbrueckii* ssp. *bulgaricus* and *L. helveticus*), species commonly found in human and animal gastrointestinal tracts (e.g., *Lactobacillus acidophilus* and *Lactobacillus gasseri*), and species with remarkable adaptability to diverse habitats (e.g., *Lactobacillus plantarum*, *L. pentosus*, *L. brevis* and *L. paracasei*)”⁹⁶

Lactobacillus plantarum

Now in genus: *Lactiplantibacillus*.

Zheng 2020: “*Lactiplantibacillus* species are isolated from many different fermented foods including fermented vegetables, meats, dairy products, and fermented cereals [58, 59], but they are also found in insect-associated habitats or as temporary residents of vertebrate intestinal microbiota and are characterized by a nomadic behaviour [230]. *L. plantarum* has been widely used as a model species for metabolic, ecological, and genetic studies in lactobacilli. *L. plantarum* is of commercial importance as starter culture for multiple food fermentations, and is applied as probiotic culture.”⁹²

Species: “The species has a nomadic lifestyle; it is a dominant member of the microbiota in spontaneous vegetable and olive fermentations and also occurs in sourdough, dairy fermentations, and fermented meats [58, 59]. *L. plantarum* contributes to spoilage of beer and wine. *L. plantarum* also is part of the microbiota of insects [232], and is isolated from the human intestinal tract, particularly the oral cavity.”⁹²

Subspecies *plantarum*: “Isolated from dairy products and dairy environments, silage, sauerkraut, pickled vegetables, sour- dough, cow dung, the human mouth, intestinal tract and stools, and from sewage.”⁹²

Subspecies *argentoratensis*: “Isolated from starchy food, fermenting food of plant origin, timothy, orchardgrass and elephant grass silage, fermented Uttapam batter, fermented idli batter.”⁹²

Table 1 of⁹³: habitat is: “Fruit flies; vertebrate digestive tract; plants and dairy products”

Lactobacillus rhamnosus

Now in genus *Lacticaseibacillus*.

Species in Zheng: “The species has a nomadic lifestyle and was isolated from a broad range of habitats including dairy products, fermented meat, fish, vegetables and cereals, sewage, humans (oral, vaginal and intestinal), invertebrate hosts and clinical sources.”⁹²

Table 1 of⁹³: habitat is: “Raw and fermented dairy, oral cavity, digestive tract of vertebrates, vagina”

Bergey on species: “Isolated from dairy products, sewage, humans, and clinical source.”

Lactobacillus sakei

Bergey: “Originally isolated from sake starter; regularly found in sauerkraut and other fermented plant material, meat products, prepacked finished dough, and human feces.”

Zheng 2020: “Isolated from sauerkraut, fermented plant material, fermented seafood, cold smoked salmon, fermented or refrigerated meat products, spontaneous sourdoughs, and prepacked finished dough. It is used commercially as starter culture for fermented meats.”⁹²

“Specifically, the *L. perolens*, *L. sakei*, *L. vaccinostercus*, *L. collinoides*, *L. brevis* and *L. buchneri* groups are almost completely composed of species that are rarely found in animals and are therefore likely free living.”⁹³

Lactococcus lactis

“Despite the common association of *Lactococcus lactis* with dairy products, the bacterium was originally isolated from plants where it was believed to be dormant, and only became active and multiplied in the gastrointestinal tract after being consumed by ruminants.” Passes through human gut but does not colonise it.⁹⁷

Bergey table 137, page 716 has isolated places for *Lactococcus* species. *L. lactis* isolated from: Cheese industry, raw milk, udder, cow saliva, grass, soil, silage.

More details from Bergey: “detected directly or following enrichment in plant material, including fresh and frozen corn, corn silks, navy beans, cabbage, lettuce, peas, wheat

middlings, grass, clover, potatoes, cucumbers, and cantaloupe. Lactococci are usually not found in fecal material or soil... only small numbers occur on the surface of the cow and in its saliva. This was ascertained by an environmental screening”

Bergey says also found in tonsils of cats, dogs and goats.

Some reports of it being opportunistic pathogen, but described as rare. On borderline but will put non-pathogen as pathogenicity is not key part of lifestyle.

Legionella pneumophila

Bergey on family/ genus: “Do not form endospores or microcysts. Not encapsulated. Not acid-fast in culture. Gram negative. Most species are motile by one, two, or more straight or curved polar or lateral flagella; nonmotile strains of motile species are occasionally seen and motility may be lost in vitro... Isolated from surface water, mud or moist soil, thermally polluted lakes and streams, and potable water systems. Associated with free-living environmental amoebae. There is no known soil or animal source. Pathogenic or potentially pathogenic for humans. Isolated from sputum, bronchoalveolar lavage washings, lung tissue, pleural fluid, pericardial fluid, blood, heart valves, abscesses, and prosthetic devices.” Usually isolated from human lung in clinical samples, causes legionellosis.

Bergey on species: “Isolated from human lung, bronchoalveolar lavage fluid, sputum, blood, intravascular devices, peritoneal fluid, abscess fluids, and potable and surface water.”

“*L. pneumophila* is an intracellular pathogen, and as part of its pathogenesis, the bacteria avoid phagolysosome fusion and replicate within alveolar macrophages and epithelial cells in a vacuole that exhibits many characteristics of the endoplasmic reticulum (ER)... *L. pneumophila* has been recovered from a wide range of both human-made and natural aquatic habitats, from lakes and streams to air-conditioning cooling towers, fountains, and spa baths...”⁹⁸

Legionella bacteria are not free-living aquatic bacteria; rather, they parasitize or form a commensal relationship with free-living, freshwater, and soil amoebae (288, 307, 319). *Legionella* species multiply intracellularly in many types of protozoa, and this relationship is central to the ecology of the organism in both aquatic and soil environments... Within human-made water systems, *Legionella* bacteria are found almost exclusively within complex biofilms (282). The characterization of *Legionella* within these ecosystems is difficult, but model biofilm systems have demonstrated that the replication of *L. pneumophila* within this niche depends on the presence of a protozoan host”⁹⁸

“Therefore, the evolution of virulence traits in *L. pneumophila* has resulted largely from the organism's need to replicate in an intracellular niche and also avoid predation by environmental protozoa.”⁹⁸

“This environmental bacterium inhabits fresh waters, where it parasitizes intracellularly within free living protozoa... Protozoa species, which can be distinctive in a variety of environmental settings, are essential for the growth of *Legionella* in natural and man-made environments [4]. Therefore, the presence of *Legionella* in these environments also appears to depend on the spectrum of host protozoa that can be utilized.”⁹⁹

“In the environment *Legionella* is able to enter a viable but non-culturable (VBNC) state. The bacteria persist in biofilms and grow within protozoa. B: Upon transmission by technical vectors (showers, air conditioning systems, cooling towers, etc.) *Legionella* colonizes the human respiratory tract. C: After uptake by macrophages *Legionella* replicates within a maturation-blocked vacuole. Finally the bacteria are released by host cell lysis.”

“Among those, the intracellular pathogen *Legionella pneumophila* thrives in fresh water environments, where it either spreads planktonically as free-living microbe or it is associated with biofilm communities (Steinert et al., 2002; Hilbi et al., 2011), but it never has been demonstrated to replicate in these environments. In the environment *Legionella* replicate within eukaryotic phagocytic cells like the environmental amoeba *Acanthamoeba castellanii*, as well as in human monocytes and alveolar macrophages.”¹⁰⁰

My summary: Requires protozoa or other eukaryotic cells to reproduce and grow, but exists in biofilms, so is described as facultative intracellular parasite. Unclear if it parasitises protozoa – no one knows how they exit cell, but they do lyse human macrophages – most authors assume its parasitic. Has a broad range, in that can technically use any eukaryotic cell, but actually only grows in protozoa in the ‘wild’. Pretty difficult to categorise. Will keep as ‘opportunistic pathogen’, as that is that case for humans (causes named disease). But its natural habitat is actually growing in protozoa, and is unclear what kind of symbiosis this is.

Leuconostoc mesenteroides

Bergey on genus: “The cells are Gram-stain-positive, nonmotile and asporogenous....

Reports on the association of leuconostocs and weissellas with humans and animals are relatively rare and mainly refer to the digestive tract. In the small and large intestines, the numbers of leuconostocs may range around 10⁵/g...”

Bergey on habitat: “*Leuconostoc mesenteroides* subsp. *mesenteroides* plays an important role in the fermentation of vegetables such as sauerkraut and cucumbers. Although not the dominant species, *Leuconostoc mesenteroides* subsp. *mesenteroides* initiates the fermentation of sauerkraut and is then succeeded by the more acid-tolerant lactobacilli.”

“*Leuconostoc mesenteroides* subsp. *mesenteroides* is present in high numbers in vegetable products such as Sayur-Asin prepared from mustard cabbage and in starchy products such as cassava or kocho, produced from *Ensete ventricose*. *Leuconostoc mesenteroides* subsp. *mesenteroides* is also involved in the submerged fermentation of coffee beans.

Leuconostoc mesenteroides subsp. *mesenteroides* is also found in sugarcane, where it produces polysaccharides and causes souring.” [does this count as plant pathogen or?]

Naturally found on plants, often commensally and can be in high numbers.¹⁰¹

Acts as an opportunistic pathogen in humans, and also causes degradation of plants in some instances.¹⁰²

Listeria monocytogenes

Bergey on *Listeria* genus: Do not form spores, all species motile with flagella when cultured <30. “All members of the genus *Listeria* are widely distributed in nature and have been isolated from soil, vegetation, sewage, water, animal feed, fresh and frozen poultry, slaughterhouse wastes, and the feces of healthy animals including humans. *Listeria monocytogenes* is pathogenic to man. *Listeria monocytogenes* and, to a lesser extent, *Listeria ivanovii* are pathogenic to a wide range of animals, especially sheep and goats. The disease in humans and livestock is predominantly transmitted by the consumption of food or feed contaminated by *Listeria monocytogenes*. The colonization by *Listeria monocytogenes* of specific sites within food manufacturing environments for long periods, together with its ability to grow in a wide range of foods.”

Bergey on species: “Widely distributed in nature, found in soil, mud, sewage, vegetation and in the feces of animals and man. Pathogenic for animals and man; most cases are food- or feed-borne. In humans causes systemic illness (most often septicemia and/ or meningitis) in immunocompromised adults or juveniles, as well as infecting the unborn infant.”

“*Listeria monocytogenes* has the ability to survive and multiply in diverse habitats and to cause infection in a variety of animal species and humans. We evaluated the literature on survival and multiplication within and transmission among multiple host populations and habitats, including man, sewage, general environment (soil, water, and vegetation), silage (fermented plant material), animals (including wild and domestic animals), and food processing plants.”¹⁰³

“*Listeria monocytogenes* is an opportunistic foodborne pathogen causing listeriosis, an often fatal infection leading to meningitis, sepsis, or infection of the fetus and abortion in susceptible individuals... *Listeria monocytogenes* is a Gram-positive foodborne pathogen that is ubiquitously found in diverse environments such as soil, water, various food products, animals, and humans.”¹⁰⁴

“The opportunistic intracellular pathogen *Listeria monocytogenes* has become a paradigm for the study of host–pathogen interactions and bacterial adaptation to mammalian hosts... ubiquitous pathogen that thrives in diverse environments such as soil, water, various food products, humans and animals. The disease caused by this bacterium, listeriosis, is acquired by

ingesting contaminated food products and mainly affects immunocompromised individuals, pregnant women and newborns... *L. monocytogenes* is a facultative intracellular pathogen that can live both inside and outside its host.”¹⁰⁵

Mannheimia haemolytica

Bergey on genus: Non-motile, gram neg, no endospores.

Species: “Isolated from pneumonia in cattle and sheep and from septicemia in lambs and mastitis in ewes. Some of the serotypes are probably part of the resident microflora of the upper respiratory tract of ruminants.”

“A commensal of the nasopharynx, *M. haemolytica* is an opportunist, gaining access to the lungs when host defenses are compromised by stress or infection with respiratory viruses or mycoplasma.”¹⁰⁶

“As an opportunistic pathogen, *M. haemolytica* is also frequently isolated from the respiratory tract of healthy cattle...”¹⁰⁷

“Here, we demonstrated that pathogenic serotype A1 *M. haemolytica*, but not commensal serotype A2 *M. haemolytica*, invades and replicates within bovine AECs.”¹⁰⁸

Mycobacterium avium

Bergey on genus: Most species difficult to stain, but considered gram-stain positive. Non-motile and asporogeneous, no capsules.

Bergey on species source: “isolated from tubercles in fowls. Widely distributed as the causal agent of tuberculosis in birds and less frequently found in lesions or lymph nodes of cattle, swine, or other animals. Rarely found in soil.”

For subspecies *silvaticum*: “isolated from birds, especially wood pigeons and cranes, and from mammals, especially deer. Strains have not been isolated from the environment and are obligate pathogens for animals, causing tuberculosis in birds and paratuberculosis in mammals.”

For subspecies *paratuberculosis*: “isolated from cattle with chronic hypertrophic enteritis or Johne’s disease by Johne and Frothingham (1895). The organism also causes enteritis in other ruminants and has been implicated in the pathogenesis of Crohn’s disease in humans (McFadden et al., 1992; McFadden et al., 1987). Strains have not been isolated from the environment.”

Taxonomy is unclear, given subspecies. Paper summarises this, and suggests that the ‘environmental’ strains of *M. avium* may be other species. The two subspecies above are

obligate pathogens.... “A possible explanation is that birds are infected by a specific subset of *M. avium* strains that are obligate pathogens of birds, in the same fashion that *M. avium* subsp. *paratuberculosis* is an obligate pathogen of ruminants.”¹⁰⁹

“The result is that *M. a. paratuberculosis* is essentially an obligate parasitic pathogen in that, except for laboratory culture conditions, virtually all replication of this organism occurs in the host, within macrophages. There it can ‘steal’ iron, essential for growth, from host iron-binding proteins such as transferrin. Once *M. a. paratuberculosis* leaves the host, it depends on its tenacious survival characteristics to persist in contaminated environments, such as soil, water, and animal feed, until it is ingested by another suitable host and establishes infection. *Mycobacterium a. avium* by contrast is commonly found in soil and water, particularly those with a lower pH, where it can multiply as a free-living organism. Hence, isolation of *M. a. paratuberculosis* from food, water, or soil indicates contamination of those substances with material, usually feces, from an infected animal.”¹¹⁰

One subspecies obligate, the other is unclear – but can divide outside, so likely facultative.

“The most clinically significant organism for human disease within MAC is *Mycobacterium avium*, with four distinct subspecies. This group of bacteria ranges from environmental bacteria that can cause opportunistic infections in immunosuppressed individuals to pathogens for birds and other animals. The characteristics of the four distinct species of *M. avium* are discussed. *M. avium* subsp. *Hominissus* (MAH) is an opportunistic environmental pathogen for humans, swine, and other animals that are found in soil and water. Drinking water and tap aerosols are considered the primary sources for infection in humans. MAH is clinically the most relevant organism within MAC for humans and a major pathogen for individuals with deficient T cell immunity.”¹¹¹

Mycobacterium bovis

Bergey on species: “Experimentally, highly pathogenic for calves, guinea pigs, and rabbits; at least moderately pathogenic for hamsters and mice; slightly pathogenic for cats, dogs, horses, and rats; not pathogenic for most fowl. Certain strains isolated from cases of lupus and scrofuloderma in man have low pathogenicity for animals. originally isolated from tubercles in cattle, and generally more pathogenic for animals than *Mycobacterium tuberculosis*. Causes tuberculosis in cattle, both domestic and wild ruminants; in man and other primates; in carnivores including cats and dogs; and in swine, parrots and possibly some birds of prey.

“Finally, an environmental source of infection cannot be excluded because while *M. bovis* is an obligate pathogen, it can survive in the environment for weeks depending on temperature, sunlight exposure, soil moisture and the presence of organic matter.”

Mycobacterium tuberculosis

Bergey on species: “isolated from cases of tuberculosis in man, other primates, dogs and some other animals which have contact with man.”

“Its etiological agent, *Mycobacterium tuberculosis*, is a primarily intracellular pathogen that is capable of successfully maintaining its viability within the host, involving a delicate interplay between the immune system and the pathogen's armaments, acquired and trained through thousands of years of host-pathogen interaction.”¹¹²

“Thus, while *Mtb* is considered an intracellular pathogen infecting live cells, its proliferation during active pulmonary tuberculosis occurs in an environment containing many dead cells and cell remnants.”¹¹³

Mycobacteroides abscessus

Was called *Mycobacterium abscessus*, now new genus.¹¹⁴

Bergey on species: “originally isolated from the synovium of the knee and associated gluteal abscesses of a patient. Causes wound and soft tissue infections, but is also found in soil.”

“Similar to most NTM species, these organisms are ubiquitous in the environment and have been found in soil, natural and drinking water sources, sewage water, as well as decaying vegetation [87]. They are typically non-pathogenic but are emerging as opportunistic pathogens that can cause a wide range of clinical diseases such as pulmonary lung disease, skin and soft tissue infections, including in the cartilage, tendons, and the layers of fat and muscle under the skin... Similar to other NTM, they are widely dispersed throughout the environment, with *M. abscessus* isolates found in freshwater rivers and lakes, seawater, and animal drinking troughs, as well as peat-rich potting soils.”¹¹¹

Cause disease in fish.¹¹⁵

Mycoplasma bovis

Now called *Mycoplasma mycoides* *bovis*.

Bergey on genus: “Cells lack a cell wall and are bounded by a single plasma membrane. Gram-stain-negative due to the absence of a cell wall... Usually nonmotile, but gliding motility has been demonstrated in some species... Commensals or pathogens in a wide range of vertebrate hosts.”

“*Mycoplasma bovis* is a wide- spread agent of otitis media, pneumonia, mastitis, polyarthriti- tis, and urogenital disease in cattle and buffaloes.

Table 137 (page 137) lists ‘representative host’ and whether its pathogen, commensal or opportunistic. *M.bovis* is a pathogen of cattle.

Bergey on species: “Pathogenic; causes mastitis, polyarthritis, keratoconjunctivitis, pneumonia, and otitis media, and is rarely associated with infertility, abortion, endometritis, salpingitis, and vesiculitis in cattle; pneumonia and polyarthritis in bison; and is rarely associated with pneumonia, mastitis, and arthritis in goats. Mode of transmission is via direct contact with infected animals or fomites, most commonly during feeding (suckling or trough), milking (cows), aerosol, or sexual contact.” Non-motile.

Also Bergey: “Source: isolated from the udders, expelled milk, synovial fluid, synovial membranes, conjunctivae, lungs, ear canals, tympanic membranes, aborted calves, uterus, cervix, vagina, and semen of cattle; from the lungs and synovial fluid of bison; and from the lungs and udders of goats.”

Most *Mycoplasma* remain attached to the cell, but some can enter.¹¹⁶

Mycoplasma gallisepticum

Table 137 says ‘Pathogen’, and representative host is ‘galliforms’ (order of birds)

Says motile via gliding.

Bergey on species pathogenicity: “Pathogenic. Causes a characteristic combination of pneumonia, tracheitis, and airsacculitis (collectively termed chronic respiratory disease); salpingitis and atrophy of the ovaries, isthmus, and cloaca resulting in poor egg quality and reduced hatchability; arthritis or synovitis; and keratoconjunctivitis in chickens; infectious sinusitis, coryza, airsacculitis, arthritis or synovitis, encephalitis, meningitis, ataxia, and torticollis in turkeys; conjunctivitis and coryza featuring a high mortality rate in finches and grosbeaks; and respiratory disease in additional game birds including quail, partridges, pheasants, and peafowl.”

Bergey on species: “Source: isolated from the trachea, lungs, air sacs, ovaries, oviducts, brain, arterial walls, synovial membranes, synovial fluid, conjunctivae, and eggs of chickens; from the infraorbital sinuses, air sacs, brain, meninges, conjunctivae, synovial membranes, and synovial fluid of turkeys; from the conjunctivae, infraorbital sinuses, and trachea of finches; and from the respiratory tract of quail, partridges, pheasants, peafowl, ducks, grosbeak, crows, robins, and blue jays”

Infects wide range of passerine birds.¹¹⁷ Will put host range as class, Aves.

Mycoplasma mycoides

Bergey says has capsule, but Rendueles study didn't?
Says cells are non-motile.

In Table 137, has two subspecies. Both pathogens, one host range is cattle and one is goats.

Bergey on subspecies capri: "isolated from the synovial fluid, synovial membranes, udders, expelled milk, conjunctivae, lungs, blood, and ear canals of goats; and the urogenital tract of sheep."

On subspecies mycoides: "isolated from the lungs, pleural fluid, lymph nodes, sinuses, kidneys, urine, synovial fluid, and synovial membranes of cattle and water buffalo."

Mycoplasma pneumoniae

Table 137 says pathogen, and host range is humans.

Cells are motile via gliding. "isolated from the upper and lower respiratory tract, cerebrospinal fluid, synovial fluid, and urogenital tract of humans."

Neisseria gonorrhoeae

Bergey on genus: "Swimming motility does not occur and flagella are absent."

Strictly human, obligate human pathogen.¹¹⁸ "Neisseria gonorrhoeae primarily colonizes the urogenital tract after sexual contact with an infected individual. The gonococcus can exist as both an extracellular and intracellular organism, with the bulk of its genes being devoted to colonization and survival, due to the fact that it cannot survive outside of a human host"

Neisseria meningitidis

Bergey: "Only N. meningitidis and N. gonorrhoeae are considered to be primary pathogens of humans... N. meningitidis strains are carried as normal flora in the oro- and nasopharynx of adults and children. The prevalence of N. meningitidis carriage varies geographically... From its commensal state in the pharyngeal mucous membrane, N. meningitidis may opportunistically disseminate to the bloodstream and, in the absence of bactericidal serum activity, cause sudden onset of disease."

Species: "Can be cultivated from petechiae, joints, nasopharynx, and conjunctiva, occasionally found in venereal discharges. Frequently found in a commensal state in the oro- or nasopharynx of asymptomatic carriers."

Paenibacillus polymyxa

Bergey on genus: “Gram-positive structure, but usually stain variable or negative in the laboratory. Oval endospores are formed that distend the sporangium. Motile by means of peritrichous flagella... Members of the genus *Paenibacillus* have not been associated with human or mammalian pathogenicity”

Ecology: “The normal habitat of the *paenibacilli* is the soil, particularly soils rich in humus and plant materials in which they presumably aid composting through the secretion of extracellular carbohydrases and other enzymes. Strains of several species, in particular the nitrogen-fixing species such as *Paenibacillus polymyxa*, have been associated with the rhizo- sphere of plants and important crop species. These bacteria enhance the growth of various plants by the production of phytohormones or by providing nutrients including nitrogen. *Paenibacillus polymyxa* is ubiquitous as a rhizosphere bacterium, particularly associated with grasses including crop plants such as wheat.

“Species source: Isolated from decomposing plants and soil. Associated with the rhizosphere where many strains provide protection to the plant and enhance plant growth.”

“*Paenibacillus polymyxa* (previously *Bacillus polymyxa*) is one of many plant growth-promoting rhizobacteria (PGPR) and is known to have a broad host plant range. It has been isolated from the rhizospheres of wheat and barley, white clover, perennial ryegrass, crested wheatgrass, lodgepole pine, Douglas fir, green bean, and garlic... Fluorescence microscopy and electron scanning microscopy indicated that the bacteria colonized predominantly the root tip, where they formed biofilms. Accumulation of bacteria was observed in the intercellular spaces outside the vascular cylinder.”¹¹⁹

Pasteurella multocida

Bergey on genus: Gram-neg, though can be variable staining. No endospores, non-motile. “Parasitic in verte- brates, particularly mammals and birds.”

Ecology: “With the exception of certain strains of *P. multocida*, organisms classified as *Pasteurella* are usually regarded as opportunistic pathogens that may colonize and form part of the indigenous flora of the mucous membranes of the upper respiratory and lower genital tracts... *P. multocida* is distributed worldwide among terrestrial as well as aquatic species of mammals and birds, and any species of these groups should be considered as a possible host. Although the host spectrum seems very large compared to other Pasteurellaceae, indications also exist that certain subtypes have developed, the disease potential of which seems to be limited to only a few species (see pathogenicity).”

Pathogenicity: “Among species classified as *Pasteurella*, *P. multocida* has been recognized as an important veterinary pathogen for more than a century. It causes a wide range of diseases in animals. Capsule type A serotypes 1, 3, and 4 are recognized as the primary cause of fowl cholera in poultry and wild birds (Rhodes and Rimler, 1989). Disease may appear as an acute

septicemia characterized by disseminated intravascular coagulation, petechial or ecchymotic hemorrhages, multifocal necroses, and fibrinous pneumonia. Chronic infections may involve a variety of local infections (Christensen and Bisgaard, 1997, 2000). *P. multocida* capsule types B and E are associated with hemorrhagic septicemia of cattle, water buffaloes, and occasionally other species, resulting in major economic losses, mainly in Southeast Asia... *P. multocida* and other species of the 16S rRNA cluster 12 are considered as zoonotic pathogens (Bisgaard et al., 1994). Most human infections associated with *Pasteurella* species result from animal bites.”

Species: “Strains of the species are isolated from most mammals, including humans and birds, in which a wide range of diseases are reported due to *P. multocida*.”

Considered opportunistic pathogen, can be commensal but can cause pathogenicity in wide range of animals.¹²⁰

“*P. multocida* is a common commensal or opportunistic pathogen found in the upper respiratory tracts of most livestock, domestic, and wild animals, including chickens, turkeys, and other wild birds, cattle and bison, swine, rabbits, dogs, cats (domestic house cats as well as large wild cats, such as tigers, leopards, cougars, and lions), goats, chimpanzees, marine mammals (seals, sea lions, and walruses), and even komodo dragons.”¹²⁰

Phaeobacter inhibens

“The roseobacter *Phaeobacter inhibens* exploits both symbiotic and pathogenic niches while interacting with its microalgal host *Emiliana huxleyi*... *P. inhibens* is a member of the roseobacter clade that is frequently identified within *E. huxleyi* blooms... *P. inhibens* to live a duplicitous lifestyle as both a beneficial symbiont and as a pathogen... Indeed, this bacterium is a symbiont of *Ulva australis*, chemically defending this ubiquitous seaweed from colonization”¹²¹

Not in Bergey, neither is genus – only named in 2006.

“One conspicuous genus of Roseobacter clade 1 is *Phaeobacter*, which is often found in microbial communities associated with a wide variety of marine eukaryotes including micro- and macroalgae, mesozooplankton, and larger animals such as bivalve molluscs.”¹²²

They are motile.¹²³

Piscirickettsia salmonis

Causes disease in salmonid fish, which is a family of fish.¹²⁴

Only grew in cell line, did not grow on media. “non-motile, highly fastidious, aerobic, and an intracellular pathogen of fish. The bacterium replicates within membrane-bound cytoplasmic

vacuoles in cells of infected hosts. *Piscirickettsia* can be grown only in vitro in fish cell cultures but not on any known artificial medium.”¹²⁴

Porphyromonas gingivalis

Bergey on genus: Gram-neg, non-motile and non-spore. “Isolated from oral infections and various other clinical specimens of human and animal origin.”

Pathogenicity: “Some species are considered true pathogens and are associated with human or animal infections. In particular, *Porphyromonas gingivalis* is a major causative agent in the initiation and progression of severe forms of periodontal disease.”

Ecology: “Several of the members of *Porphyromonas* are indigenous bacterial flora in the oral cavity of humans and mammals. Many species are also found in the urogenital and intestinal tracts. *Porphyromonas* species have been isolated from oral infections, and from many infections throughout the body, e.g., blood, amniotic fluid, umbilical cord, pleural empyema, peritoneal and pelvic abscesses, inflamed endometrium, and other infected tissues. *Porphyromonas* species of animal origin have been encountered in humans with animal bite infections.”

Bergey on species: “*Porphyromonas gingivalis* is considered a major periodontal pathogen and reported to be a cause of extraoral infections, such as lung abscesses and pulmonary infections. It has also been suggested that *Porphyromonas gingivalis* may contribute to the development of atheromas in cardiovascular disease. Source: infected dental root canals, periodontal pockets, and other oral sites.”

“*P. gingivalis* has been shown to invade into a variety of host cells in several in vitro studies (Andrian et al. 2006; Eick et al. 2002; Deshpande et al. 1998; Amornchat et al. 2003), and to persist and multiply within the epithelial cells (Madianos et al. 1996).”¹²⁵

Described as opportunistic pathogen. “In periodontal disease, the opportunistic pathogen, *Porphyromonas gingivalis*, manipulates the gingival epithelial cells (GECs) to promote microbial survival and persistence in the oral mucosa.”¹²⁶ and “Moreover, considering that *P. gingivalis* may not be equally pathogenic in all individuals, it could be considered as an opportunistic keystone pathogen.”¹²⁷

Prevotella intermedia

Bergey on genus: Nonspore, non-motile, gram-neg. “The primary site of isolation is the oral cavity, but more recently species have been reported from the intestinal tract of man and animals.”

Species: “Source: isolated initially from human dental plaque but has since been reported from head, neck and pleural infections, from blood, and from abdominal and pelvic sites.”

Causes periodontal disease, but is considered opportunistic.^{128,129}

Prochlorococcus marinus

Bergey on genus: Members of genus ubiquitous in water column 0-200m in most tropical and temperate oceans. Dominate lower (100-200m) in clear waters. Type strains of *P. marinus* isolated from 120m in Sargasso Sea.

“free-living autotroph”¹³⁰ and “Small, nonmotile bacteria such as *Pelagibacter ubique* and *Prochlorococcus marinus* numerically dominate in the surface of the open ocean, where sunlight dominates as an energy source and photosynthate provides a relatively steady if low nutrient supply to heterotrophs.”¹³¹

Propionibacterium freudenreichii

Bergey on genus: Gram positive, non-motile, non-spore forming. “The genus was first described as a result of the study of propionic acid-producing bacteria isolated from cheese by a number of authors in the early 1900s... isolated from fermenting food and plant materials such as silage and fermenting olives, and from soil, including soil from rice paddy fields. Related bacteria have been found associated with nematodes in the Zebra gut and anaerobic digestors.”

Bergey on species: “Source: raw milk, Swiss cheese and other dairy products; particularly associated with the ripening, flavor and aroma of Swiss cheese.”

Can be isolated from silage, soil, rumen and other habitats.¹³² Also isolated from barley grains.¹³³ “*Propionibacterium* spp. are present on the herbaceous plants and in the rumen of the bovine species, excrements of the herbivores, soil, sewage, sludge, milk, pickle, water after oil production, and in fermented orange juice.”¹³⁴

Proteus mirabilis

Bergey on genus: Gram neg, motile by peritrichous flagella, most strains swarm. “Human pathogens, causing urinary tract infections; also are secondary invaders, causing septic lesions at other sites of the body. Occurs in the intestines of humans and a wide variety of animals; also occurs in manure, soil, and polluted waters.”

Pathogenicity: “*P. mirabilis* is most frequently isolated from clinical specimens and is one of the most important pathogens of the urinary tract. Underlying conditions such as diabetes or structural abnormalities of the urinary tract are associated with infections acquired outside the

hospital... Incidences of 17–24% for *P. mirabilis* and 3–5% for *P. vulgaris* in healthy humans have been reported.”

Ecology: “Early studies have shown that *Proteus* strains are widely distributed in nature, occurring in manure, soil, and polluted water, where they are believed to have important roles in the decomposition of organic matter... also present in the intestinal contents of both wild and domesticated animals as well as in the intestines of patients and healthy humans.”

“*Proteus* spp. bacteria are mostly known as opportunistic human pathogens... It is postulated that human intestines are a reservoir of *Proteus* bacteria, especially those belonging to prevailing *P. mirabilis* species, and they are members of natural fecal microflora of several percent of human population... *Proteus* spp. are regarded to be an undesired element of intestinal microflora, as the bacteria may also become a causative agent of diarrhea... Drzewiecka et al. , by the use of serological and molecular methods as well as the Dienes test, proved that *P. mirabilis* strains isolated from feces and urine of patients were in fact the same spreading clone causing autoinfection and nosocomial infection.”¹³⁵

Table 3 of ¹³⁵ summarises relationships of 3 *Proteus* species and different hosts, and whether positive or negative! *P. mirabilis* is found in humans, gorillas, dogs, cats, pigs, flying fox, rats, birds, snakes, turtles, fish, oysters, shrimp, lobster, sponge, millipede, cockroaches, wild grass, *Candida albicans* (fungi). Some are positive, some commensal, and some negative. Table 4 of ¹³⁵ found in contaminated soil, coastal seawater, wild grass rhizosphere, wastewater.

Pseudomonas aeruginosa

Bergey on genus: motile by one or more flagella, rarely nonmotile. Gram-negative. No resting stages are known (i.e. no spores?). “Widely distributed in nature. Some species are pathogenic for humans, animals, or plants... In regard to opportunistic pathogens such as the fluorescent pseudomonads and, in particular, *P. aeruginosa*, the functions in this “gray area” can lead to different interpretations”

Pathogenicity: “*P. aeruginosa* seldom infects healthy individuals outside the hospital environment, and the condition of the host is essential in determining the clinical relevance of this opportunistic pathogen.”

Ecology: “Strains of many species are ubiquitous, and isolation data often throw little light on their ecology. When dealing with organisms of such versatility, ecological conclusions are particularly difficult to draw. Of the 57 strains of fluorescent *Pseudomonas* species isolated by den Dooren de Jong, 23 had their origin in soil, and with one exception all were classified as *P. putida*, while all other strains were gelatin-liquefiers isolated from water, and were assigned to *P. fluorescens* (den Dooren de Jong, 1926). However, the conclusion that *P. putida* is a soil organism whereas *P. fluorescens* predominates in water could not be supported by an analysis of isolation data of many other strains from the collections of the Department of Bacteriology

at Berkeley, California, and of the Serum Institut in Copenhagen (unpublished observations). Obviously, a decision on this point is not easy in view of the possibility of cross-contamination of materials from the two habitats... An early observation suggests that the pre-dominant fluorescent pseudomonads in wheat rhizosphere comprise one of the biotypes of *P. fluorescens*... Many other reports have confirmed the fact that these organisms proliferate in general in plant rhizospheres, where they seem to have a stimulating effect on plant growth... An additional beneficial effect is suggested by studies on the rhizosphere of plants growing in the Canadian Arctic, in which *P. putida* can grow and stimulate root elongation of plants during spring and winter at 5°C, as shown by using a strain that can survive exposure to -20°C to -50°C... A numerical taxonomic analysis of strains of fluorescent pseudomonads associated with the roots of tomato plants has been published... Many species of *Pseudomonas* have been isolated from *Lolium* leaves (Stout, 1960). *P. fluorescens* has been found frequently... Continued association with living hosts is important to the survival of members of the *P. syringae* group. The association does not necessarily have to involve lesions, since the pathogen may be able to survive as an epiphyte on hosts as well as on nonhosts. Survival of the *P. syringae* pathovars in soil may not be long, in contrast with other fluorescent species”

“Opportunistic pathogen”¹³⁶ and “*P. aeruginosa* was most commonly isolated from sediment, with solid-water interfaces also exhibiting high frequencies of isolation.”¹³⁷

Bergey on species: “*P. aeruginosa* is probably the most widespread of all bacterial species. It can be isolated from soil and water, particularly from enrichment cultures for denitrifying bacteria. Commonly isolated from clinical specimens (wound, burn, and urinary tract infections). Causative agent of “blue pus”, which accounts for the origin of the synonym *pyocyaneus*. Occasionally pathogenic for plants. Strains isolated from leaf spot of tobacco, identical with or similar to *P. aeruginosa* have been named “*P. polycolor*”

Some recent suggestion not so widespread? “As *P. Aeruginosa* has been considered a common soil inhabitant the largest number of collected and cultured environmental samples included in this study belonged to the category of ‘uncontaminated soil/rhizo-sphere’ (55 samples). However, we detected *P. aeruginosa* in only 7% of uncontaminated soil samples. Water is also generally regarded as a *P. aeruginosa* habitat [e.g., (14, 17, 36)], so we investigated 34 uncontaminated water samples. Surprisingly, none of these samples were *P. aeruginosa* culture positive. This is in line with the study of Pirnay et al.(2005) that found almost no *P. aeruginosa* at the source of a river and the most *P. aeruginosa* in the part of the river receiving wastewater.”¹³⁸

But take with a pinch of salt maybe? “In line with our culture analysis of environmental samples, the 16S rRNA database analysis showed that *P. aeruginosa* sequences were present with relatively low frequencies in soil and water, and the meta-analysis showed that the occurrence of *P. Aeruginosa* in uncontaminated soil is low(17.7%). In contrast, our meta-analysis indicated a higher occurrence rate of *P. Aeruginosa* in natural water without intense human contact (18.9%) compared to our screening of water samples (0%).”¹³⁸

Can infect dogs, cats, and cows, among other animals. Also infects ¹³⁹

“The versatile and ubiquitous bacterium *Pseudomonas aeruginosa* is the quintessential opportunistic pathogen, because it can infect a broad range of hosts, from amoeba to humans (1, 2), where it is found associated with severe burns, cystic fibrosis (CF), AIDS, and cancer (3).”

Pseudomonas fluorescens

Note: Put *fluorescens*, *putida* and *stutzeri* as host type: plant, and host range and ‘plantae’. Seems they only infect extremely ill people, and very rarely. Bergey describes *aeruginosa* as more virulent than the others.

Bergey on species: “Found in soil and water, from which it can be isolated after enrichment in media containing various carbon sources, incubated aerobically; strains of the denitrifying biovars can be enriched in similar media containing nitrate, incubated under anaerobic conditions. Commonly associated with spoilage of foods (eggs, cured meats, fish, and milk). Often isolated from clinical specimens. Some strains assigned to this species (biovar II) have been isolated from diseased plants (e.g., lettuce), and identified as *Pseudomonas marginalis* (Brown, 1918) Stevens 1925AL.”

Can be an opportunistic pathogen to humans, but usually lives in soil & rhizosphere. Can promote plant growth or cause disease.¹⁴⁰

Pseudomonas putida

From description of genus, widely found in soil, and sometimes rhizospheres.

Bergey on species: Isolated from soil and water.

“Strains of the species *Pseudomonas putida* are frequent rhizosphere and freshwater inhabitants and exhibit an amazing ability to metabolize a wide range of biogenic and xenobiotic compounds. Several strains of this species have been isolated from patients who have acquired infections in hospital environments. Infections caused by *P. putida* are rare and are mostly reported in immuno-compromised individuals, such as those with neutropenia, newborns, and cancer patients... Despite the fact that *P. putida* may cause healthcare-related infections, clinical data on *P. putida* infections is scarce; this likely due to the rarity, relatively lower virulence, and higher antimicrobial susceptibility of *P. putida* compared to *P. aeruginosa*.”¹⁴¹

Pseudomonas stutzeri

Bergey on species: Found in soil and water, many strains isolated from clinical specimens. “*P. stutzeri* and *P. mendocina* have attracted attention as denitrifiers, and, in recent years, an

infamous side of their reputation is their potential activities as opportunistic pathogens... *P. stutzeri* is frequently isolated from clinical materials but rarely causes disease. In the few cases in which it was the cause of infections, the patients often had another serious underlying disease, and they responded to antibiotics (aminoglycosides, some of the β -lactams, or cephalosporins)”

Pseudomonas syringae

In above genus description, host plant is described as required for *P. syringae*, and while can survive on own in soil, declines.

“*Pseudomonas syringae* is one of the best-studied plant pathogens and serves as a model for understanding bacterial pathogenicity, molecular mechanisms of plant-microbe interactions as well as microbial ecology and epidemiology. *P. syringae* was originally isolated from diseased plants and was largely studied with respect to its plant pathogenic potential... *P. syringae* bacteria have two interconnected phases of growth in or on plants: the epiphytic phase, when the bacteria live on the surface of plant tissues (usually the above-ground parts, such as leaves, stems and fruits, collectively known as the phyllosphere), and the endophytic phase, when bacteria enter the plant tissue and colonize the intercellular space called the apoplast... *Pseudomonas syringae* is one of the most common plant pathogens that infect the phyllosphere (i.e., the aboveground plant organs). *P. syringae* can live on the plant surface as an epiphyte. To cause disease it enters the plant, through wounds or natural openings such as stomata, and multiplies within the intercellular space called the apoplast.”¹⁴²

“Here, we report the abundance of *P. syringae* in rain, snow, alpine streams and lakes and in wild plants, in addition to the previously reported abundance in epilithic biofilms... This cycle opens the opportunity to evaluate the importance of non-agricultural habitats in the evolution of a plant pathogen and the emergence of virulence. The ice-nucleation activity of all strains from snow, unlike from other substrates, strongly suggests that *P. syringae* plays an active role in the water cycle as an ice nucleus in clouds.”¹⁴³

“We show that for each test, strains display a diversity of host ranges from very restricted to very broad regardless of the gamut of phylogroups used in the test. Overall, strains form an overlapping continuum of host range potential with equal representation of narrow, moderate and broad host ranges... The extent of host range was positively correlated with the capacity of strains to swarm on semi-solid agar medium and with the abundance of genes in biosynthetic clusters and was inversely correlated with the abundance of genes for proteins with transmembrane domains in their genomes.”¹⁴⁴

Ralstonia solanacearum

Bergey on genus: Gram-neg, no spores, motile and non-motile species (motile have flagella).

Pathogenicity: “*Ralstonia solanacearum* causes a lethal vascular wilt disease in more than 200 plant species in 50 botanical families in the tropics, subtropics, and warm temperate regions of the world. Its agronomically important hosts include tomato, potato, tobacco, peanut, and banana... Thus, *Ralstonia* organisms might be living in a variety of ecological niches. Furthermore, the long survival of *R. solanacearum* organisms in soil and rhizospheres might be possible by an asymptomatic infection of the roots of nonhost plants, such as bean, peas, soybean, corn, and rice”.

Bergey says species is non-motile. “Pathogenic for tomato, potato, tobacco, banana, and peanut by the production of a copious amount of extracellular polysaccharide, which occludes vessels in the stem, inhibits water flow, and causes death of plant by wilt disease. Recently the nucleotide full sequence of the chromosome and megaplasmid were determined in order to elucidate and manipulate genes related to the virulence of the organism. The type strain was isolated from tomato.”

“able to infect over 400 plant species worldwide, being a major threat to agriculture. These pathogens are soil and water borne, penetrate the host through the roots, and cause wilting by massively colonizing the xylem vessels and producing vascular dysfunction. The damage they cause has been related to the unusually high number of virulence and pathogenicity factors they synthesize.”¹⁴⁵

Rhizobium leguminosarum

Bergey on genus: No spores, gram-neg, motile by 1-6 flagella. “All known *Rhizobium* species include strains which induce hypertrophisms in plants as root nodules with or without symbiotic nitrogen fixation.”

“Some cells of symbiotic bacterial species enter root hair cells of leguminous plants (Family Leguminosae) via invagination or by wounds (“crack entry”) and elicit the production of root nodules wherein the bacteria engage as intracellular symbionts, usually fixing nitrogen. Many well-defined nodulation (nod) and nitrogen fixation (nif) genes are clustered on large plasmids or megaplasmids (pSyms). Plasmid transfer between species results in the expression and stable inheritance of the particular plant-interactive properties of the plasmid-donor species... In root nodules the bacteria occur as endophytes that exhibit pleomorphic forms, termed “bacteroids”, which reduce or fix gaseous atmospheric nitrogen into a combined form utilizable by the host plant.”

Ecology: “*rhizobium* occurs worldwide in soils and especially in the rhizosphere of plants. As many as 10⁶–10⁷ cells/g soil of symbiotic *Rhizobium* have been reported”

Bergey on species: has 2-6 peritrichous flagella. “*R. leguminosarum* nodulates with some, but not necessarily all, *Lathyrus* spp., *Lens* spp., temperate species of *Phaseolus* (*P. vulgaris*, *P. angustifolius*, *P. multiflorus*), *Pisum* spp., *Trifolium* spp., and *Vicia* spp.”

Rhizobium phaseoli

Was previously a biovar of *R. leguminosarum*.

Enhanced in rhizosphere and lives in soil, and forms root nodules.¹⁴⁶

Not too much about it, but very similar to *R. leguminosarum*.

Rickettsia japonica

Bergey on genus: Gram-neg, “obligately intracellular and reside free in the cytoplasm of the eukaryotic host cell... Rickettsiae are closely associated with arthropods (ticks, mites, fleas, lice, and other in- sects) for their maintenance in nature. Their natural cycle usually involves both a vertebrate and an invertebrate host... For some, the arthropod host is both a reservoir and a vector... Rickettsial cells are usually un- stable when separated from host components, except for highly stable forms found in the feces of arthropod hosts... *R. japonica* (Japanese spotted fever)... Rickettsiae are inoculated into the skin in the saliva during feeding by an infected tick, mite, or flea or by scratching of rickettsia-laden feces deposited by an infected louse or flea... A consistent characteristic of *Rickettsia* species is their residence in an arthropod host as at least a part of their ecological niche. Transovarian maintenance from one generation of tick, mite, or flea to the next via infected ova that hatch into infected larvae is a factor in the maintenance in nature of all SFG rickettsiae...”

On species: “The organisms are pathogenic for guinea pigs. Guinea pigs develop fever and scrotal swelling. C3H/He strain of inbred mice and conventional strain ddY mice are resistant to *R. japonica* infection. Chicken embryos die 5–7 days after yolk sac inoculation... *R. japonica* is presumably transmitted by ticks...”

“Although the details of host and reservoir association for *R. japonica* have not yet been analyzed, the bacterium has been isolated from or detected by PCR in the following eight tick species: *Dermacentor taiwanensis*, *Haemaphysalis hystericis*, *H. cornigera*, *H. flava*, *H. longicornis*, *H. fromosensis*, *H. megaspinosa*, and *Ixodes ovatus*... This frequent transmission may be related to the broad tick host range of *R. japonica*, which is somewhat unusual among SFG rickettsiae.”¹⁴⁷

All tick species are in family Ixodidae. Unclear if causes disease in ticks – other species described as harming insect host, so will keep as ‘pathogen’.

Rickettsia prowazekii

Type strain of genus. “*R. prowazekii* is the etiologic agent of epidemic typhus fever, which is acquired through contact with lice. It is also the etiologic agent of recrudescent typhus (called

Brill– Zinsser disease) and occasionally of sporadic typhus in individuals who have been in contact with flying squirrels or their ectoparasites... The most effective arthropod vector of epidemic typhus is the human body louse, *Pediculus humanus corporis*, possibly because it takes frequent large blood meals and because it tends to desert febrile hosts to seek new ones... The guinea pig is highly susceptible to infection, but develops a mild disease, usually manifested only as fever lasting approximately a week. The cotton rat, *Sigmodon hispidus*, is also highly susceptible to infection, but signs of disease and death are produced only by doses in excess of 3×10^5 viable rickettsiae... Strains isolated from various geographic locations are remarkably similar to each other in biological properties. Differences in % DNA–DNA hybridization were negligible among strains isolated in Poland, Spain, and Burundi from human sources and from flying squirrels from the United States. Minor differences in protein migration patterns obtained by isoelectric focusing in polyacrylamide gels can distinguish strains derived from Eastern Europe, Spain or Africa, or from American flying squirrels”

Rickettsia rickettsii

Bergey on species: “*R. rickettsii* is the most widely studied species of the spotted fever group... Because of the high virulence of *R. rickettsii* for chicken embryos, the embryos die before extensive growth has taken place, and the procedure for optimal harvest from yolk sacs differs considerably from the one employed for the typhus group... Strains of this species vary considerably in virulence for the guinea pig. Virulence for humans and guinea pigs appears to vary independently. The guinea pig is highly susceptible to infection... In humans, *R. rickettsii* is the causative agent of Rocky Mountain spotted fever, the most severe disease of the spotted fever group. The disease is characterized by high fever and widespread damage to the small blood vessels; this damage results in a skin rash, increased vascular permeability associated with non-cardiogenic pulmonary edema and vascular lesions in various organs including encephalitis... Although there is considerable serological evidence of widespread exposure to SFG rickettsiae among wild vertebrates and domestic dogs, demonstration of infection by recovery of the microorganisms has been difficult, presumably because sufficient numbers of rickettsiae are present in the vertebrate host only during brief periods...”

Riemerella anatipestifer

Bergey on genus: Non-motile, no spore, Gram-negative. “The illness caused by *Riemerella anatipestifer* is an exudative septicemia in ducks, pigeons, and other domestic and wild birds. *Riemerella anatipestifer* has been isolated from ducks, geese, turkeys, chickens, pheasants, quails, and wild free-living waterfowl. The organism is thought to be transmitted vertically by transovarian passage. Lateral transmission occurs through injuries, such as toenail scratches of the webbed foot in ducklings, or by entrance through the respiratory epithelium.”

Waterfowl (aka Anseriformes) is an order of 180 species. Turkeys, quails, pheasants etc. in order Galliformes. So host-range is Class: Aves.

Transmitted from bird to bird, so could be obligate – but can't find any mention.

“Members of the genus *Riemerella*, as with those of the *Pasteurella*, are facultative intracellular pathogens.”¹⁴⁸

Salmonella enterica

Bergey on genus: Gram-neg, usually motile via peritrichous flagella. “Pathogenic for humans, causing enteric fevers, gastroenteritis, and septicemia; may also infect many animal species besides humans. Some serovars are strictly host-adapted.”

Pathogenicity: “*Salmonella* serovars may be strictly adapted to one particular host (these serovars are auxotrophic), may be ubiquitous (found in a large number of animal species), or may be of still unknown pathogenicity.... Salmonellosis is transmitted from person to person, without an intermediate host, through fecal contamination of water and food.” Enters epithelial cells in gut. “In the second step of *Salmonella* pathogenesis, bacteria gain access to the mesenteric lymph nodes, drain through the lymphatics to the thoracic duct into the blood, and ultimately infect the liver and spleen.”

Ecology: “Although some *Salmonella* serovars are strictly host adapted, the majority have a wide host range (e.g., serovar Typhimurium). Some are localized in a particular region of the globe (e.g., serovar Sendai in the Far East, serovar Berta in North America), but others are ubiquitous (e.g., serovar Typhimurium). Strains belonging to *S. enterica* subsp. *salamae*, subsp. *arizonae*, and subsp. *diarizonae* are frequently isolated from the intestinal contents of cold-blooded animals and only rarely from human beings and warm-blooded animals. Strains of subsp. *houtenae* and *S. bongori* are isolated chiefly from the environment and are rarely pathogenic for humans.”

Bergey says some serovars infect warm blooded and some infect cold blooded.

“*Salmonella* bacteria have been isolated from a wide range of animals and their food products, including poultry, bovine, ovine, porcine, fish, seafood and lacertilians (cold-blooded animals) such as lizards and snakes... *Salmonella* bacteria are considered a major health problem in reptile meat as this genus is commensal in the intestine microbiota of these animals”¹⁴⁹

Serratia marcescens

Bergey on genus: Gram-neg, generally motile via peritrichous flagella. “The organisms occur in the natural environment (soil, water, plant surfaces) or as opportunistic human pathogens.”

Bergey: “prominent opportunistic pathogen for hospitalized human patients. At present, *S. marcescens* is the only known nosocomial species of *Serratia*. Clinically, *Serratia* infections do not differ from infections by other opportunistic pathogens (von Graevenitz, 1980). Other

Serratia species can be involved in respiratory tract infection or colonization and septicemia... *S. marcescens* and *S. liquefaciens* are known to infect and cause mortality in a variety of insects which can be serious pests of crops, ornamentals, and turf throughout the world.. *Serratia* species occur on plants, in the digestive tract of rodents (unpublished data), and in soil and water.”

Bergey on species: “biogroups A3 and A4 are ubiquitous. Nonpigmented biogroups A5/8 and TCT are almost confined to hospitalized patients. Pigmented biogroups A1 and A2/6 are found in the natural environment and occasionally in human patients.”

“*S. marcescens* was considered originally to be an innocuous, non-pathogenic saprophytic water organism and was often used as a biological marker because of its easily recognised red colonies... Today, *S. marcescens* has attained the status of a fully fledged pathogen that causes infections particularly in two disparate groups: heroin addicts and hospitalised patients... Environmental isolates of *S. marcescens* characteristically produce a red pigment, prodigiosin, and in early times such growth was often mistaken for fresh blood. The pigmented bacterium is found in various ecological niches, including soil, water, air, plants and animals. The ability to form prodigiosin is characteristic of *S. marcescens*, but the function of this red pigment remains unclear because clinical isolates are rarely pigmented.”¹⁵⁰

Also has been isolated from deep sea sediment. ¹⁵¹

Sinorhizobium meliloti

Now called *Ensifer meliloti*.

Similar to genus *Rhizobium*.

Bergey on species: Motile via 2-6 peritrichous flagella. “Forms nitrogen-fixing nodules on *Melilotus*, *Medicago*, and *Trigonella*. Gao and Yang (1995) reported a Chinese *Sinorhizobium meliloti* strain that nodulated and fixed nitrogen in association with both alfalfa and soybean. Well characterized genetically. Symbiosis-controlling genes are carried on megaplasmids. In addition, strains harbor various numbers (0–4) of large pRme plasmids”

Staphylococcus aureus

Bergey on genus: Gram-stain positive, non-motile, no ‘resting stages’. “Natural populations are mainly associated with skin, skin glands, and mucous membranes of warm-blooded animals. Host or niche range may be narrow or wide, depending upon the particular species or subspecies. Some organisms may be isolated from a variety of animal products (e.g., meat, milk, cheese) and environmental sources (e.g., fomites, soil, sand, dust, air, or natural waters). Some species are opportunistic pathogens of humans and/or animals.”

Pathogenicity: “In the late 1950s and early 1960s, *Staphylococcus aureus* caused considerable morbidity and mortality as a nosocomial pathogen of hospitalized patients... *Staphylococcus aureus* is also capable of producing infections in a variety of other mammals and birds. The more common natural infections include mastitis, synovitis, arthritis, endometritis, furuncles, suppurative dermatitis, pyemia, and septicemia.” Also MRSA problem since 1980s. *Staph epidermidis* is species ‘associated most commonly with disease’.

Ecology: “*Staphylococcus aureus* is a major species of primates, though specific ecovars or biotypes can occasionally be found living on different domestic animals or birds. This species is found infrequently on nonprimate wild animals. In humans, *Staphylococcus aureus* has a niche preference for the anterior nares, especially in the adult. Nasal carrier rates range from less than 10% to more than 40% in normal adult human populations residing outside of the hospital. *Staphylococcus epidermidis* (Schleifer and Kloos, 1975) is the most prevalent and persistent *Staphylococcus* species on human skin. It is found over much of the body surface and produces the largest populations where moisture content and nutrition are high, such as in the anterior nares, axillae, inguinal and perineal area, and toe webs. This species may be found occasionally on other hosts, such as domestic animals, but it is presumably transferred there from human sources.”

Berge on species: Subspecies *aureus*: “Isolated from nasal membranes (anterior nares, nasopharynx) and skin of warm-blooded animals. Can cause infection, food poisoning, and toxic shock syndrome.” Subspecies *anaerobius*: “Found in abscesses of sheep; also pathogenic for goats. Not pathogenic for mice, rabbits, or guinea pigs. Primary isolation requires a medium supplemented with serum, egg yolk, or blood.”

“*Staphylococcus aureus* is both a commensal bacterium and a human pathogen. Approximately 30% of the human population is colonized with *S. aureus*. Simultaneously, it is a leading cause of bacteremia and infective endocarditis (IE) as well as osteoarticular, skin and soft tissue, pleuropulmonary, and device-related infections”¹⁵²

Staphylococcus epidermidis

Above Bergey says is mostly on human skin, and only really on animals when transferred from humans.

Bergey on species: non-motile. “Isolated from human skin where it is a resident; occasionally found on the skin of other mammals, particularly those living in close association with humans. Opportunistic pathogen, may colonize various indwelling medical devices, postoperative infections, urinary tract, and wound infections.”

Stenotrophomonas maltophilia

Bergey on genus: Gram-negative. Motile by 2 or more polar flagella. “One of the species (*S. maltophilia*) is widely distributed in nature and is commonly isolated from clinical materials and nosocomial infections... A predominance of *S. maltophilia* over other pseudomonads has been observed in the rhizosphere of several cultivated plants, such as cabbage, rape, mustard, corn, and beet... In the rhizosphere of oilseed rape, *S. maltophilia* is able to interfere with growth of plant pathogenic fungi... *S. maltophilia* has been found on the surface of leaves of the grass *Lolium perenne*, although at a low frequency in comparison to other aerobic pseudomonads...”

Pathogenicity: “*S. maltophilia* has been implicated as a direct agent of disease and as the cause of secondary infections that aggravate various human pathological conditions... The literature records concern for the emergence of the species as a dangerous opportunistic pathogen...”

Ecology: [materials from which *S. maltophilia* isolated from includes] “raw and pasteurized milk; well, stagnant, and river waters; sewage; frozen fish; feces of snakes, lizards, frogs, and rabbits; rotten eggs; and soil in petroleum zones. It has been isolated from the internal tissue of banana pseudostem, decaying banana sucker, cotton seed, bean pod, and tobacco seedling. As mentioned above, strains of the species have been isolated from the rhizosphere and aerial parts of plants. These organisms may represent a very high proportion of the non-fermentative, Gram-negative bacteria in the soil microflora. The isolation of three strains similar to *S. maltophilia*, from gut and feces of the arthropod *Folsomia candida* was reported by Duckworth et al. (1996).”

On species: “*S. maltophilia* is the second most frequently isolated aerobic pseudomonad, after *P. aeruginosa*, in the clinical laboratory. Strains of the species appear to be opportunistic human pathogens. Also found in water, milk, frozen foods, and many other sources. De-bette and Blondeau (1980) were able to isolate more strains of *S. maltophilia* than of any other pseudomonad from the rhizosphere of some cultivated plants.”

Streptococcus agalactiae

Bergey on genus: Non-motile, no spores, gram positive. “Many species exist as commensals or parasites on man and animals, some are highly pathogenic.”

Ecology & pathogenicity: “Streptococci are associated with warm-blooded animals and birds. Most species can be regarded as commensal, being usually found on mucosal surfaces in the oral cavity, upper respiratory tract, and gastrointestinal tract, and under appropriate conditions can cause localized and systemic infections. The success of streptococci as commensals and potential pathogens has been attributed to i) “the ability to adhere to almost any surface present within their natural environment”, ii) “the ability to rapidly utilize available nutrients under fluctuating environmental conditions”, and iii) “the ability to tolerate, resist, or even destroy host immune defences”. The pyogenic streptococci together with *Streptococcus pneumoniae* are the major pathogens. *Streptococcus pyogenes* can colonize the throat or skin and can cause

a number of suppurative infections and non-suppurative sequelae. This species is the most common cause of bacterial pharyngitis, impetigo, and scarlet fever as well as erysipelas and other spreading infections. *Streptococcus pneumoniae* causes pneumonia, meningitis, otitis media, and septicemia and is a serious pathogen among patients with underlying illness and compromised immunity.”

Table 131 and 132 on page 673 lists hosts: *S. pyogenes* is ‘man’, *S. agalactiae* is ‘Man, animals’, *S. pneumoniae* is ‘Man’, *S. thermophilus* is ‘(Dairy products)’, *S. suis* is ‘Animals, man’.

Bergey on species: “Initially recognized as a cause of puerperal sepsis, in man group B streptococci can produce a variety of clinical conditions, some of them very serious. Disease can be invasive (normally defined as occurring when group B streptococci can be isolated from usually sterile site such as blood or cerebrospinal fluid) or non-invasive. Neonatal infections are divided into early and late-onset. In the former, occurrence is usually within the first 7 d of life where prolonged rupture of the membranes and chorioamnionitis features pneumonia, septicemia, or meningitis. The organism may be cultured from the mother’s genital tract. In animals these organisms represent a highly contagious, economically important, obligate pathogen of the mammary gland and are one of the main causes of bovine mastitis. Sources of infection in man are the vaginal mucosa, the upper respiratory tract, urine, stool, and, in animals, milk and udder tissue.

“Group B Streptococcus (GBS), also referred to as *Streptococcus agalactiae*, is a Gram-positive, opportunistic pathogen that colonizes the gastrointestinal and genitourinary tracts of up to 50% of healthy adults. This microorganism causes pneumonia, septicaemia and meningitis in neonates, is responsible for significant morbidity in pregnant women and the elderly, and is a serious cause of mortality in immunocompromised adults. GBS can also colonize the mammary glands of ruminants, where the organism can survive for extended periods, causing clinical and subclinical mastitis, which affects milk quality and quantity. GBS has been isolated from other animals including dogs, horses, guinea pigs and even fish.”¹⁵³

Streptococcus pneumoniae

Bergey on species: The clinical patterns of pneumococcal infections in man are numerous. They include pneumonia, meningitis, otitis media, and some less frequent conditions such as abscesses, conjunctivitis, pericarditis, and arthritis. In animals pneumococci may occasionally cause mastitis and septicemia in cows, sheep and goats, and respiratory tract infections in monkeys. Strains are isolated from the upper respiratory tract, inflammatory exudates and various body fluids of diseased humans and, rarely, domestic animals. The normal habitat in humans is the nasopharynx with an estimate of as high as 60% of the population colonized at any one time.” And “highly pathogenic species *Streptococcus pneumoniae* that may also be found resident in the upper respiratory tract of healthy humans.”

“It is generally accepted that *Streptococcus pneumoniae* is strictly a human pathogen despite there being animal models (mouse, rat) available to study pneumococcal infections. However, on the other hand, recent literature describing naturally occurring carriage of or infections by pneumococci in animal hosts is scarce.”¹⁵⁴

“*Streptococcus pneumoniae* (also known as pneumococcus) is a Gram-positive, extracellular, opportunistic pathogen that colonizes the mucosal surfaces of the human upper respiratory tract (URT).”¹⁵⁵

Streptococcus pyogenes

Bergey on species: “*Streptococcus pyogenes* (GAS) is an important human pathogen which colonizes the throat or skin, causing suppurative infections and non-suppurative sequelae.”

“*Streptococcus pyogenes*, or Group A *Streptococcus* (GAS) is an exclusively human opportunistic pathogen that causes a variety of diseases.”¹⁵⁶

Streptococcus suis

Bergey on genus: “*Streptococcus suis* is an important pig pathogen and has been isolated from cases of bacteremias and meningitis in piglets and from respiratory disease. Human infections are often associated with patients having worked in contact with pigs.”

“*S. suis* infection in pigs is reported worldwide, from North America (United States and Canada) to South America (Brazil), Europe (United Kingdom, The Netherlands, France, Denmark, Norway, Spain, and Germany), Asia (China, Thailand, Vietnam, and Japan), Australia, and New Zealand [7]. In addition to pigs, *S. suis* can be isolated from other animals, such as ruminants, cats, dogs, deer, and horses, and is believed to be a commensal in the intestinal flora [7]. Healthy pigs can carry multiple serotypes of *S. suis* in their nasal cavities, tonsils, and upper respiratory, genital, and alimentary tracts.”¹⁵⁷

Streptococcus thermophilus

Bergey on genus: “*Streptococcus thermophilus*, a species isolated from dairy sources but of unknown habitat... *Streptococcus thermophilus* is found in dairy sources including heated and pasteurized milk.”

“The genome of *S. thermophilus* LMD-9 is shaped by its domestication in the dairy environment, with gene features that conferred rapid growth in milk, stress response mechanisms and host defense systems that are relevant to its industrial applications... *Streptococcus thermophilus* is a low G + C, Gram-positive, nonmotile, non-spore-forming, catalase-negative, facultative anaerobic, homofermentative lactic acid bacterium that has restricted natural habitats in the bovine mammary mucosa and raw milk. Among the ninety-

three currently classified species from the genus *Streptococcus* [2], *S. thermophilus* represents the only species that is “Generally Regarded As Safe”.¹⁵⁸

“*Streptococcus thermophilus* is the only species of this genus to be widely used as a starter in the dairy industry and to have the “Generally Regarded As Safe” status. It belongs to the *salivarius* group of the *Viridians streptococci* [13], which includes two other species, *Streptococcus salivarius* and *Streptococcus vestibularis*. These two species are both commensal bacteria of the human gut, whereas the environmental reservoir of *S. thermophilus* has not been identified [14]. It grows spontaneously in traditional dairy products and is believed to persist in the farm environment [15–17]. Multilocus sequence typing and comparative genomic analysis have revealed that there is little polymorphism in the *S. thermophilus* population, and that this species displays significant allelic divergence from the other two species of the *salivarius* group [14, 18, 19]. *S. thermophilus* is a clonal species that emerged only recently on the evolutionary timescale (3,000–30,000 years ago), from a commensal ancestor of the *salivarius* group [20]. Its adaptation to a narrow and well defined niche (milk) has shaped its genome through loss-of-function events and horizontal gene transfer (HGT)”.¹⁵⁹

Vibrio alginolyticus

Bergey on genus: Motile by polar flagellar enclosed in a sheath. Gram-negative. No endospores or microcysts. “Primarily aquatic; species distribution is usually dependent on Na and nutrient content of the water as well as its temperature. Very common in marine and estuarine environments and on the surfaces and in the intestinal contents of marine animals. Species with a low Na⁺ requirement are also found in freshwater habitats. Twelve species occur in human clinical specimens; 11 of these are apparently pathogenic for humans, causing diarrhea or extraintestinal infections. Several species cause diseases of other vertebrates and invertebrates.”

Pathogenicity: “The two most frequently isolated enteric pathogens of this group are *V. cholerae*, the etiologic agent of pandemic cholera, and *V. parahaemolyticus*, a major cause of foodborne disease, particularly in Japan and South East Asia... *V. parahaemolyticus* has caused numerous cases of gastroenteritis, including many outbreaks. Cases are associated with the consumption of raw or undercooked shellfish such as oysters, shrimp, crabs, and lobster... *V. vulnificus* is an important pathogen that causes wound infections and septicemia (Hollis et al., 1976; Blake et al., 1979; Tacket et al., 1984; Klontz et al., 1988). Wound infections result from direct inoculation of the organism into traumatized cutaneous surfaces after contact with marine animals or the marine or estuary environment... Interestingly, the halophilic strains of *V. vulnificus* biogroup 3 were able to grow in the “fresh water” inland fishponds. However, these ponds had a high salt content because of the source of the water and evaporation during the hot dry summer months.”

TABLE BXII.c.158. Diseases in marine fish and invertebrates caused by or associated with *Vibrio* species: *V. alginolyticus* is 'Kuruma prawns, tiger prawns', *V. anguillarum* is 'Eels, ayu, rainbow trout, salmonids'.

Ecology: "Water (and its associated microorganisms, animals and plants) is the natural habitat of most of the nonhalophilic and marine vibrios... This wide range partially explains why *V. cholerae*, *V. mimicus*, *V. fluvialis*, and *V. furnissii* are recovered from freshwater rivers, streams, and ponds; other species such as *V. vulnificus* prefer moderate salinity and grow in shellfish beds in coastal areas... Although salinity is a critical parameter, it does not completely explain the environmental distribution of all vibrios because halophilic species such as *V. parahaemolyticus* can survive in suboptimal Naconcentrations..."

Bergey on species: "Occurs in human clinical specimens, particularly in soft tissue infections (Table BXII.c.157) and in the marine environment.

Many reports describe the isolation of *V. alginolyticus* from soft tissue infections (Rubin and Tilton, 1975; Pien et al., 1977). Wound infections and ear infections are usually mentioned, with eye infections mentioned much less frequently. There is a clear association of *V. alginolyticus* with infection at these sites; however, the etiological role of *V. alginolyticus* has rarely been shown conclusively and little has been reported about the pathogenesis of these infections. Most authors list *V. alginolyticus* as a pathogen, particularly of wound and ear infections. Antibiotic treatment has been used in most cases; surgical debridement has been used in some."

"One of the most pathogenic species is *Vibrio alginolyticus*, a gram-negative and curved halophilic bacterium... *V. alginolyticus* is mainly found in marine estuaries, coastal and aquatic environments with worldwide distribution. It may exist as free-living, a parasite or associated with surfaces of organisms such as marine vertebrates/invertebrates and flora, and even humans... *Vibrio alginolyticus* has been found to cause varied infections and inflammation in both humans and animals (such as otitis, ocular infections, intracranial infection, peritonitis and osteomyelitis among others)"¹⁶⁰

"*Vibrio alginolyticus* belongs to the *Vibrio* genus in the Vibrionaceae family. *V. alginolyticus* is a Gram-negative, rod-shaped, flagellar bacterium that has halophilic features and widely exists in ocean and estuarine areas. *V. alginolyticus* is considered one of the most harmful *Vibrio* species and is pathogenic to both humans and aquatic animals. In humans, soft tissues, the ear, and superficial wounds are easily invaded by *V. alginolyticus* when exposed to contaminated seawater... For aquatic animals, *V. alginolyticus* causes a variety of diseases, such as septicemia of *Sparus aurata*, exophthalmia and corneal opaqueness of *Epinephelus* spp., melanosis of *Rachycentron canadum*, white spot syndrome of *Penaeus vannamei*, necrosis of *Macrobrachium rosenbergii* larvae, and massive mortality of *Tapes decussatus*."¹⁶¹

Produces toxin in puffer fish: "Our findings suggest that some strains of *V. alginolyticus* are closely related to the toxification of the puffer, and probably of other species."¹⁶²

“*Vibrio alginolyticus* is a Gram-negative bacterium and an important opportunistic pathogen for marine organisms. This bacterium is associated with epidemic vibriosis, which results in high mortality for cultured marine animals, including fish, shellfish and shrimp. In addition, *V. alginolyticus* can be pathogenic in humans and can lead to otitis and wound infections after contact with *V. alginolyticus*-containing seawater”¹⁶³

Vibrio anguillarum

Bergey on species: “In some marine vibrios (e. g., *V. anguillarum*), the polar flagellum appears critical for disease production in estuarine fish... Many non-clinical *Vibrio* species cause serious infections in a variety of marine animals including shellfish and fish. The best studied of these pathogens is *V. anguillarum*, the causative agent of epizootic disease in marine fish and shellfish (Austin and Austin, 1993). The general term vibriosis is used to describe fulminant septicemic infections produced by a number of *vibrio* species in marine fish. The etiologic agent of the disease may be specific for a single type of aquatic life or may have a very broad host range.”

Range is: Eels, ayu, rainbow trout, salmonids for disease.

“*Vibrio anguillarum* is a marine bacterium inhabitant of the estuarine and marine coastal ecosystems worldwide. It is an important fish pathogen since it is the etiological agent of classical vibriosis in warm- and cold-water fish species leading to high mortalities and economic losses in aquaculture (Toranzo et al., 2017). Bivalve molluscs and crustaceans are also occasionally affected by this bacterium... The remaining serotypes are mostly environmental strains isolated from seawater, marine animals, or sediments.”

“*Vibrio anguillarum*, one of the most common bacteria in the marine environment, is widely distributed in coastal and estuarine seawater. *V. anguillarum* is a representative opportunistic bacterial pathogen that causes disease in marine fish... At least 50 species of fish can be infected with *V. anguillarum*, including pacific salmon, Atlantic salmon, rainbow trout, turbot, perch, sea carp, striped bass, codfish and Japanese flounder... During the initial stages of pathogenesis, *V. anguillarum* uses flagellum-mediated motility to infect and colonise the surface and gut of fish hosts.”¹⁶⁴

“*V. anguillarum* causes diseases in crustaceans and bivalves, and is the leading causative agent of vibriosis in finfish including salmon, rainbow trout, turbot, sea bass, sea bream, cod, eel, and ayu... *V. anguillarum* is an extracellular pathogen that invades its host fish through the intestine, skin or gills.”¹⁶⁵

Some suggestion it is also commensal in fish.¹⁶⁶

Vibrio cholerae

Bergey Ecology cont.: “*V. cholerae* can survive in a “free living state” in both fresh- water and saline environments. It is widely distributed in habitats such as sewage, brackish water, estuaries, and coastal inlets, as well as in polluted streams, rivers, ponds, and lakes. *V. cholerae* can also persist in an epibiotic form associated with various microscopic life (Hood et al., 1984). Common microorganisms associated with *V. cholerae* include cyanobacteria, phytoplankton (diatoms, fresh water algae), and zooplankton (Huq et al., 1995; Chakraborty et al., 1997). *V. cholerae* may attach to the tissues or chitinous exoskeleton of crustaceans and production of the enzyme chitinase might be important in this process. All these combined niches provide a continuous environmental source for the maintenance and dissemination of *V. cholerae* throughout the world... Strains of non-O1 *V. cholerae* greatly outnumber O1 strains in the environment. *V. cholerae* non-O1 has been recovered from birds, amphibians, herbivores, and freshwater fish”

“*V. cholerae* has been detected on many abiotic and biotic surfaces, including ship hulls (Shikuma and Hadfield, 2010), zooplankton (Tamplin et al., 1990; Epstein, 1993; Huq et al., 2005; Turner et al., 2009), macroalgae (Hood and Winter, 1997), and as floating aggregates (Alam et al., 2006).”¹⁶⁷

Vibrio parahaemolyticus

Bergey: “Occurs in human clinical specimens (Table BXII.c.157) and is an important cause of diarrhea. Also occurs in marine environments. ... Outbreaks of gastroenteritis caused by *V. parahaemolyticus* occur worldwide. In Japan, *V. parahaemolyticus* causes 50– 70% of foodborne enteritis cases (Sakazaki and Balows, 1981), which are invariably associated with seafood. In the early 1970s, several articles described *V. parahaemolyticus* as the cause of severe wound infections and bacteremia (Roland, 1970, 1971; Zide et al., 1974; Weaver and Ehrenkranz, 1975). It is now known that the infectious agent in the case was really *Vibrio vulnificus* (Weaver and Ehrenkranz, 1975), not *V. parahaemolyticus*. Similarly, many ecological and environmental reports about *V. parahaemolyticus* may be incorrect because of our limited understanding of the large number of *Vibrio* species that inhabit these environments.”

Bergey ecology cont. “*V. parahaemolyticus* inhabits inshore coastal areas and estuaries and has only rarely been recovered from pelagic (open ocean) regions (Joseph et al., 1982). It has been isolated from various parts of the water column, sediment, zooplankton, shellfish, and fish. Occasionally, *V. parahaemolyticus* has been recovered from fresh water in Indonesia and India (Chakraborty et al., 1997). It has also been recovered from inland bodies of water in the United States where the Na content is high... *V. parahaemolyticus* has been isolated from a variety of marine animals including clam, oyster, lobster, scallop, sardine, squid, eel, crab, and shrimp (Joseph et al., 1982). Most outbreaks of gastroenteritis caused by *V. parahaemolyticus* have been linked to the consumption of crabs, shrimp, oysters, and lobsters. In Japan, *V. parahaemolyticus* is a major cause of food poisoning and is associated with the ingestion of raw fish such as sashimi and sushi”

Vibrio vulnificus

Bergey: “Occurs in human clinical specimens (Table BXII.c.157) and the marine environment. Cause of wound infections, bacteremia, and septicemia. The type strain was isolated from human blood.”

Ecology cont. “*V. vulnificus* has been isolated from various locales around the world, including Europe and the Pacific and Atlantic coasts of the United States as far north as Washington and Maine, respectively. *V. vulnificus* is typically found in waters having intermediate salinities (5–25 ppt) and temperatures up to 26°C (Motes et al., 1998); it does not grow at temperatures less than 10°C. DePaola et al. (1994) isolated *V. vulnificus* from seawater, crustacea, and estuarine fish from U.S. waters in the Gulf of Mexico. The highest concentration of *V. vulnificus* in one study was found in the intestinal contents of bottom-feeding estuarine fish (sea catfish, sheepshead, Atlantic croaker) that consume mollusks and crustacea. It is rarely recovered from offshore fish... The presence of *V. vulnificus* in shellfish may result from the constant filtering by these organisms of seawater containing vibrios rather than the active multiplication of *V. vulnificus* in shellfish tissues (Kelly and Dinuzzo, 1985). However, *V. vulnificus* can survive in oysters for 10–14 d when they are held at 2–10°C (Kaysner et al., 1989). In humans, *V. vulnificus* septicemia is almost exclusively associated with the consumption of raw oysters”

Xanthomonas campestris

Bergey on genus: Gram-neg, motile by single polar flagella, no ‘resting stages’. “Species so far described are plant pathogens or are plant associated.”

Pathogenicity and ecology: “Species and pathovars of *Xanthomonas* have been reported to cause disease in at least 124 monocotyledons and 268 dicotyledons, but no gymnosperms, ferns, or lower plants. The symptoms produced also vary widely and are helpful, sometimes essential, for identification of the pathogen involved. Most species and pathovars produce leaf spots, at least initially. These may spread very little, or be limited by the leaf veins, causing angular leaf spots in dicotyledons. They may spread to include the veins and then may develop systemically. Systemic infections produce wilts, death of shoots, or cankers on twigs and branches, or combinations of these symptoms, and if severe the whole plant may be killed. The extent of symptom development and type of symptoms expressed may also depend on the environmental conditions and the cultivar of the host plant.”

Path & ecol cont.: “A critical point in the life cycle of xanthomonads, as with other plant pathogens, is transmission to a new host, particularly if a period of survival in the absence of the host is necessary. Such survival may be achieved in many ways, such as with seed, plant residues, perennial hosts, epiphytically, saprophytically in soil, and in insects. Many xanthomonads solve this problem by transmission with the seed of their host. The bacteria may

be carried in detritus with the seed, on or in the seed coat, or deeper inside the tissues of the seed itself. Epiphytic survival is becoming well known, e.g., *X. axonopodis* pathovar *manihotis* and *X. arboricola* pathovar *pruni* are both known to spend the interseasonal time epiphytically.”

Path & Ecol cont “In agriculture, many pathogens are most effectively carried by the activities of man, either in infected planting material, which may be symptomless, or on tools, wheeled vehicles, and even grazing animals, as for *X. axonopodis*. Survival in soil saprophytically is unusual, but has been suggested by the work of Goto et al. (1978) for *X. axonopodis* pathovar *citri*. Xanthomonads have occasionally been detected in run-off water and ditches around fields of infected plants. Results suggest that survival in this situation would be short.”

Path & Ecol cont.: “All organisms currently named in the genus *Xanthomonas* are plant pathogens, but isolates that are apparently non-pathogenic are also found (Vauterin et al. 1996a). Some have been identified as known pathogens growing epiphytically, e.g., *X. axonopodis* pathovar *phaseoli* isolated from symptomless weed.”

Bergey on species: “The original isolates of this species caused a vascular disease of *Brassica* spp. Currently, the species includes a number of pathovars that cause diseases predominantly within the family *Cruciferae*. In general, pathovars are not distinguishable by phenotypic characterization and identification is reliant on knowledge of their hosts.”

Brassicaceae or Cruciferae is same family of plants.

A few pathovars cause disease in other families like *Plantaginaceae* and *Solonaceae*, but pathovars themselves have narrow range on only a few hosts.

“Black rot is primarily a seed-borne disease (Cook et al., 1952). However, the disease can also be transmitted in infected transplants, infested soil, crop residues and carry-over in related weed species (Schaad and Alvarez, 1993; Walker, 1953). Schaad and White (1974) and Dane and Shaw (1996) showed that *Xcc* can survive in the soil, independent from the host, for approximately 40 days in winter and 20 days in summer. The results of Arias et al. (2000) showed that high soil matric potential (saturated soils) can reduce the survival of the pathogen. The pathogen can survive longer in soil within plant tissues than as free living cells... The bacteria can disperse over short distances via wind, insects, aerosols, irrigation water, rain, farm equipment and workers.”¹⁶⁸

Xanthomonas citri

Bergey has description while it was still a pathovar of *X. axonopodis*. Says host range is within family *Rutaceae*.

“*Xanthomonas citri* ssp. *citri* is the causative agent of citrus canker (CC) disease and, as a pathogen of a globally important fruit crop, Citrus, has been the subject of extensive study with

respect to epidemiology and disease management... Compatible hosts vary in their susceptibility to citrus canker (CC), with grapefruit, lime and lemon being the most susceptible, sweet orange being moderately susceptible, and kumquat and calamondin being amongst the least susceptible... Outside of lesions, Xcc survives only 1–3 days on inanimate surfaces, such as clothing and agricultural equipment, and no more than 2 months in soil as a result of competition with saprophytes (Graham et al., 1989; Schubert et al., 2001). In tropical environments with mild winter temperatures, viable Xcc cells persist in the margins of older lesions on leaves, fruit and twigs”¹⁶⁹

“*Xanthomonas citri* subsp. *citri* pathotypes cause bacterial citrus canker, being responsible for severe agricultural losses worldwide. The A pathotype has a broad host spectrum, while A* and Aw are more restricted both in hosts and in geography... Citrus canker is a bacterial disease affecting all commercial citrus varieties.”¹⁷⁰ And table 1 shows all isolates of this species are from family Rutaceae, of which citrus is a genus. Spread driven by ‘wind-driven rain’.

Xanthomonas oryzae

Bergey: “The isolates cause blight and leaf streak of *Oryza* spp. (fam. Gramineae).” Pathovar hosts: Hosts: *Isachne globosa*, *Leersia* spp., *Leptochloa* spp., *Oryza* spp., *O. sativa*, *Phalaris arundinacea*, *Phragmites communis*, *Zizania aquatica* (fam. Gramineae). And Hosts: *Oryza sativa*, *Oryza* spp., *Leersia hexandra* (weak) (fam. Gramineae).

“Severe rice diseases, such as bacterial leaf streak (BLS) caused by *Xanthomonas oryzae* pv. *oryzicola* (Xoc) and bacterial blight (BB), caused by *X. o.* pv. *oryzae* (Xoo), are increasing in prevalence in parts of Asia and sub-Saharan Africa and cause significant yield losses. In Asia, perennial weeds are considered an important source of primary pathogen inoculum for these two diseases.” Can infect weeds within same family, sometimes asymptotically, which can act as reservoir for rice infection.”¹⁷¹

Xylella fastidiosa

Bergey on genus: Non-motile (no flagella) [papers say has pili to move], gram-neg, no spores, found mainly in xylem of plant tissue.

Path & ecol: “Symptoms produced in host plants vary, the most characteristic being a necrosis of interveinal leaf tissue, producing a characteristic scorching effect in a wide range of woody plants. Of primary importance is Pierce’s disease of grapevine, which can cause 100% destruction in some vineyards, but which is normally not a serious pathogen... Pierce’s disease of grapevine can be transmitted by xylem-feeding leafhoppers... The significance of disease reservoirs, such as weeds, has also been studied. Weeds collected from plum orchards infested with leaf scald were examined, and the presence of the pathogen was confirmed by isolation... Populations of *X. fastidiosa* in the root xylem fluid of symptomatic peach trees were high and the distribution of these bacteria was uniform both along the length of roots and

among roots within the root ball; in contrast, distribution of the bacteria within asymptomatic trees was discontinuous.”

“*Xylella fastidiosa* is a Gram-negative, slow growing and strictly aerobic bacterium in the family Xanthomonadaceae. It is a widely distributed plant pathogen as it can colonize the xylem of many different species, causing a variety of diseases such as Pierce's disease (PD) in grape (*Vitis vinifera*) or citrus variegated chlorosis (CVC) (Purcell, 2013). *X. fastidiosa* can move upstream and downstream along plant xylem, thanks to the presence of long type IV pili [so motile?]. Many sap-feeding insects can function as vectors for the transmission of *X. fastidiosa* to host plants, especially sharpshooters and froghoppers or spittlebugs. After acquisition from the source plant, the bacterium is persistent in the vector (Severin, 1949) and can multiply in the foregut... The initial belief that *X. fastidiosa* was a generalist pathogen capable of infecting a very large range of host plants has gradually changed with the discovery and characterization of genetically different strains of *X. fastidiosa*, each capable of infecting distinct hosts. Also the infection characteristics can vary considerably in different species.”¹⁷² Paper has table showing very broad, at level of angiosperms.

Yersinia enterocolitica

Bergey on genus: *Y. pestis* always non-motile, but some species motile at 37C but non-motile below 30C. Widely distributed in nature with some species adapted to specific animal hosts and humans. Several species are pathogenic for humans and animals including *Y. pestis*, the causative agent of plague. A significant cause of food-borne and water-borne disease.

Bergey on species: “*Y. enterocolitica* has been isolated from a wide variety of sources (live and inanimate) in every country in which it has been sought and probably has a worldwide distribution. As shown in Table BXII.c.284, biovar IA strains are ubiquitous, having been found in a wide range of animal and environmental sources (including foods), whereas other biovars or serogroups are frequently associated with a specific host. The species has been isolated from a wide variety of sources in the environment (live and inanimate) including foods and from healthy humans and animals, especially the pig. *Y. enterocolitica* has been recognized as pathogenic for chinchillas, hares, monkeys, and humans. The pathogenicity for animals is similar to that of *Y. pseudotuberculosis*.”

“Indistinguishable genotypes have also been found between human strains and dog, cat, sheep and wild rodent strains, indicating that these animals are other possible infection sources for humans.”¹⁷³

“Stream and lake water from the Mammoth Lakes region of California was sampled for *Yersinia enterocolitica*. From 10 of the 34 sites examined, organisms were isolated that were biochemically identified as *Y. enterocolitica*. Only one of the ten strains could be serologically confirmed. This strain was identified as *Y. enterocolitica* serotype 16. Although an outbreak of

enteritis in the area prompted this study, no correlation with gastrointestinal disease could be established since the majority of the strains were untypeable.”¹⁷⁴

“Taken together, *Y. enterocolitica* BT 1A group represents opportunistic pathogens whose opportunity to establish infection seems to rely mainly on the state of the host defence system.”¹⁷⁵

“*Yersinia enterocolitica* is primarily a zoonotic pathogen frequently associated with human non-specific gastroenteritis... *Y. enterocolitica* evolved independently after divergence from *Y. pseudotuberculosis* within the last 200 million years, while *Y. pestis* originated from the closely related *Y. pseudotuberculosis* subspecies just 1500–20 000 years ago... . In contrast, *Y. enterocolitica* strains, especially serobiotype O:3/4, are reported worldwide and in some countries they have become one of the main causes of human enteritis... *Y. enterocolitica*, the *Yersinia* species most frequently associated with human infections. Mammals and birds are natural reservoirs of *Yersinia* species, with humans being mostly incidental hosts of these agents of zoonotic diseases... Phylogenetically, *Y. enterocolitica* strains are evidently clustered in three clades related to the highly virulent, low-virulence and non-virulent groups... *Y. enterocolitica* is primarily a zoonotic pathogen of mammals with humans being mostly accidental hosts... Pigs mostly do not develop clinical signs, but they do carry *Y. enterocolitica* in their oral cavity, on tongues, on tonsils, and in lymph nodes, and excrete bacteria in their faeces... Other animal species, such as cattle, sheep, poultry, fish, deer, small rodents, cats, dogs, as well as frogs, fleas and flies, can host pathogenic *Y. enterocolitica*. However, most environmental isolates from soil and water belong to the non-virulent biotype 1A”¹⁷⁶

Yersinia pestis

Bergey: always non-motile. “*Y. pestis* is the causative agent of plague. Plague is primarily a disease of wild rodents. *Y. pestis* is transmitted among wild rodents by flea bites or ingestion of contaminated animal tissues (Butler, 1983). In fleas, the bacterium multiplies and blocks the esophagus and the pharynx. The fleas regurgitate the bacteria when they take their next blood meal and transmit *Y. pestis* to humans if no other hosts are available. Infective flea bites produce the typical bubonic form of plague within 2–8 d. *Y. pestis* multiplies intracellularly in macrophages and extracellularly and proceeds through the lymphatic system. The lymph nodes near the flea bite are the first to become inflamed, enlarged, and painful, and constitute the bubo. As the evolution of the infection is usually rapid with massive growth of *Y. pestis* in the blood, characteristic lesions are not found in the spleen or liver at autopsy.”

Bergey:”” *Y. pestis* remains localized in enzootic or maintenance hosts (Butler, 1983) and has been isolated from more than a hundred different naturally infected species of rodents, but rarely from predatory animals (carnivores and birds, the latter being resistant to the infection). The spread of plague is usually accomplished by the epizootic cycle of rodents to fleas and fleas to rodents. The reservoir for *Y. pestis* is soil contaminated by infected dead fleas and rodents in which the microorganism survives for months in deep rodent burrows... *Y. pestis* is

the causative agent of plague. The disease can be reproduced experimentally in mice, rats, guinea pigs, and monkeys.”

Yersinia pseudotuberculosis

Bergey on species: “*Y. pseudotuberculosis* is responsible for epizootics in nearly all animal species, especially in rodents and birds. Animals are usually contaminated by the oral route and, after 1–2 weeks of incubation, the bacteria are found in mesenteric lymph nodes. The main symptoms are mesenteric adenitis and chronic diarrhea. Infection evolves either in self-cure, or in fatal septicemia. *Y. pseudotuberculosis* is an intracellular parasite and, like *Y. pestis*, reaches the lymphatic system. At autopsy, caseous lesions are found in Peyer’s patches, mesenteric lymph nodes, the spleen, and the liver. Humans orally contaminated by *Y. pseudotuberculosis* develop either a mesenteric adenitis, which simulates acute appendicitis, or, in the compromised host, a severe septicemia. *Y. pseudotuberculosis* is distributed worldwide. It has been found in numerous animal species, especially rodents and birds, in soil, and in humans (Wetzler, 1970). In Japan, cats and dogs have been associated with human cases (Fukushima et al., 1989). Wild animals, which are often asymptomatic carriers, are considered the reservoir of the bacteria. Humans and animals are contaminated orally either by direct contact with sick or asymptomatic animals or through food contaminated by the excretions of these animals.”

Bergey: “*Y. pseudotuberculosis* is a human and animal pathogen responsible for mesenteric lymphadenitis, diarrhea, and septicemia. The disease can be reproduced experimentally in guinea pigs challenged and in mice.”

“*Yersinia pseudotuberculosis* is a foodborne enteric pathogen that causes a mild self-limiting diarrhea in humans. *Yersinia pseudotuberculosis* is able to persist in soil and water and in association with fresh produce, but the mechanism by which it persists is unknown. It has been shown that *Y. pseudotuberculosis* co-occurs with protozoans in these environments. *Y. pseudotuberculosis* is able to resist the bacterivorous nature of FLA and instead exhibits an enhanced ability to replicate and persist in coculture with amoeba. This study sheds light on the potential role of FLA in the ecology of *Y. pseudotuberculosis* which may have implications for food safety. The ecology of *Y. pseudotuberculosis* with regard to its mode of transmission, survival in the environment and on food, and its source of contamination on farms, is not well known... FLA and *Y. pseudotuberculosis* co-occur in natural soil and water environments, on fresh produce, including lettuce and carrots.”¹⁷⁷

Zymomonas mobilis

Bergey on genus: Gram-neg. Usually non-motile, but if motile possess one to four polar flagella. “*Zymomonas* occurs as a spoiler in beers, ciders, and perries; as fermenting agents in Agave sap, palm sap, and sugarcane juice; and on honeybees and in ripening honey.”

Pathogenicity: “*Zymomonas* is not known to be pathogenic for humans, animals, or plants. Lindner (1929, 1931) recommended the use of *Zymomonas* in human nutrition as a kind of yogurt. Antagonistic effects of *Zymomonas* against bacteria and fungi in vitro, and the therapeutic use of *Zymomonas* in cases of chronic enteric and gynecological infections, have been reported.”

Ecology: Causes secondary fermentation in cider. “*Zymomonas* is also present in fermenting sugarcane juice in Brazil, and on bees and ripening honey in Spain... Genetically, phenotypically, and ecologically, *Zymomonas* is distantly related to the acetic acid bacteria: they both occur in acid, sugary, and alcoholized niches such as tropical plant juices and beer. They are ecologically complementary in that *Zymomonas* produces ethanol, which is further oxidized by the acetic acid and bacteria.”

Isolation: “Isolated from bees, from ripening honey in Spain, from the fermenting sap of *Agave americana* (atrovirens) in Mexico, from fermenting palm juice (*Arenga pinnata*) in Indonesia, from sugarcane juice in Queensland, Australia, and the Fiji Islands (Warr et al., 1984), from *Elaeis guineensis* and *Raphia uinifera* in Zaire and Nigeria, and from fermenting sugarcane juice or molasses in Brazil. It has also been isolated in England from beer, from the surface of brewery yards, and from the brushes of cask- washing machines.” And “Isolated in England from sick cider and from apple pulp.”

“*Zymomonas* has been isolated primarily from alcoholic liquids, but it has been reported from other habitats as well. It has been isolated as a spoilage organism from cider (Millis 1956), perry (Millis 1956), ale (Dadds et al. 1973), and beer... *Z. mobilis* has been repeatedly isolated from fermenting *Agave* juice, or pulque... These plant saps provide the conditions *Z. mobilis* requires; a nutrient solution with fermentable sugars... In addition to the above, *Zymomonas* has been reported in molasses (Panilla et al. 2011), apple pulp (Carr and Passmore 1971), rotting oranges (Rabah et al. 2011), banana wine (Obaedo and Ikenebomeh 2009), and water kefir (Marsh et al. 2013). It is unclear how common *Z. mobilis* is in most of these, and it would be helpful to replicate isolations from these sources.... It is often stated that *Zymomonas* can be found in ripening honey and on bees (Ruiz-Argueso and Rodriguez-Navarro 1975). The isolates identified in the original report, were found to use maltose, which is not currently diagnostic for *Z. mobilis*. Either these organisms were not actually *Zymomonas*, or they were strains that had increased substrate range. Thus, the connection between *Z. mobilis*, honey, and bees needs further examination... *Zymomonas* may contaminate a location such as a brewery, soil, or water, but its ultimate source must be from elsewhere. How long *Zymomonas* may survive in such locations has not been studied. Excluding the few anomalous reports, the normal habitat for *Zymomonas* is sugary plant materials... It has been shown that *Z. mobilis* doesn’t survive long in soil, but it does survive for long periods of time, up to several months, on some objects contaminated with it... *Z. mobilis* has not been shown to be part of the persistent resident microbiota of any plant.”¹⁷⁸

References:

1. Pallach, M. *et al.* Structure and inflammatory activity of the LPS isolated from *Acetobacter pasteurianus* CIP103108. *Int. J. Biol. Macromol.* **119**, 1027–1035 (2018).
2. Tanasupawat, S. *et al.* Identification of *Acetobacter*, *Gluconobacter*, and *Asaia* strains isolated in Thailand based on 16S-23S rRNA gene internal transcribed spacer restriction and 16S rRNA gene sequence analyses. *Microbes Environ.* **24**, 135–143 (2009).
3. Yeom, J., Shin, J.-H., Yang, J.-Y., Kim, J. & Hwang, G.-S. ¹H NMR-Based Metabolite Profiling of Planktonic and Biofilm Cells in *Acinetobacter baumannii* 1656-2. *PLoS ONE* **8**, e57730 (2013).
4. Antunes, L. C. S., Visca, P. & Towner, K. J. *Acinetobacter baumannii*: evolution of a global pathogen. *Pathog. Dis.* **71**, 292–301 (2014).
5. Eveillard, M., Kempf, M., Belmonte, O., Pailhoriès, H. & Joly-Guillou, M.-L. Reservoirs of *Acinetobacter baumannii* outside the hospital and potential involvement in emerging human community-acquired infections. *Int. J. Infect. Dis.* **17**, e802–e805 (2013).
6. Nemec, A. *et al.* Genotypic and phenotypic characterization of the *Acinetobacter calcoaceticus*–*Acinetobacter baumannii* complex with the proposal of *Acinetobacter pittii* sp. nov. (formerly *Acinetobacter* genomic species 3) and *Acinetobacter nosocomialis* sp. nov. (formerly *Acinetobacter* genomic species 13TU). *Res. Microbiol.* **162**, 393–404 (2011).
7. Pailhoriès, H., Tiry, C., Eveillard, M. & Kempf, M. *Acinetobacter pittii* isolated more frequently than *Acinetobacter baumannii* in blood cultures: the experience of a French hospital. *J. Hosp. Infect.* **99**, 360–363 (2018).
8. Larcher, R., Pantel, A., Arnaud, E., Sotto, A. & Lavigne, J.-P. First report of cavitory pneumonia due to community-acquired *Acinetobacter pittii*, study of virulence and overview of pathogenesis and treatment. *BMC Infect. Dis.* **17**, 477 (2017).
9. Harikrishnan, R. & Balasundaram, C. Modern Trends in *Aeromonas hydrophila* Disease Management with Fish. *Rev. Fish. Sci.* **13**, 281–320 (2005).
10. Saraceni, P. R., Romero, A., Figueras, A. & Novoa, B. Establishment of Infection Models in Zebrafish Larvae (*Danio rerio*) to Study the Pathogenesis of *Aeromonas hydrophila*. *Front. Microbiol.* **7**, (2016).
11. Ivanova, E. P. *et al.* Ecophysiological diversity of a novel member of the genus *Alteromonas*, and description of *Alteromonas mediterranea* sp. nov. *Antonie Van Leeuwenhoek* **107**, 119–132 (2015).
12. Fan, B., Blom, J., Klenk, H.-P. & Borriss, R. *Bacillus amyloliquefaciens*, *Bacillus velezensis*, and *Bacillus siamensis* Form an “Operational Group B. amyloliquefaciens” within the B. subtilis Species Complex. *Front. Microbiol.* **8**, (2017).
13. Reva, O. N., Dixelius, C., Meijer, J. & Priest, F. G. Taxonomic characterization and plant colonizing abilities of some bacteria related to *Bacillus amyloliquefaciens* and *Bacillus subtilis*. *FEMS Microbiol. Ecol.* **48**, 249–259 (2004).
14. Niu, D.-D. *et al.* The plant growth-promoting rhizobacterium *Bacillus cereus* AR156 induces systemic resistance in *Arabidopsis thaliana* by simultaneously activating salicylate- and jasmonate/ethylene-dependent signaling pathways. *Mol. Plant-Microbe Interact. MPMI* **24**, 533–542 (2011).
15. Whitaker, J. M., Cristol, D. A. & Forsyth, M. H. Prevalence and genetic diversity of *Bacillus licheniformis* in avian plumage. *J. Field Ornithol.* **76**, 264–270 (2005).
16. Hong, H. A. *et al.* *Bacillus subtilis* isolated from the human gastrointestinal tract. *Res. Microbiol.* **160**, 134–143 (2009).
17. Errington, J. & van der Aart, L. T. Microbe Profile: *Bacillus subtilis*: model organism for cellular development, and industrial workhorse. *Microbiology* **166**, 425–427 (2020).

18. Paul, S. I., Rahman, Md. M., Salam, M. A., Khan, Md. A. R. & Islam, Md. T. Identification of marine sponge-associated bacteria of the Saint Martin's island of the Bay of Bengal emphasizing on the prevention of motile *Aeromonas* septicemia in *Labeo rohita*. *Aquaculture* **545**, 737156 (2021).
19. Earl, A. M., Losick, R. & Kolter, R. Ecology and genomics of *Bacillus subtilis*. *Trends Microbiol.* **16**, 269 (2008).
20. Nielsen-LeRoux, C., Gaudriault, S., Ramarao, N., Lereclus, D. & Givaudan, A. How the insect pathogen bacteria *Bacillus thuringiensis* and *Xenorhabdus/Photorhabdus* occupy their hosts. *Curr. Opin. Microbiol.* **15**, 220–231 (2012).
21. Rabbee, M. F. *et al.* *Bacillus velezensis*: A Valuable Member of Bioactive Molecules within Plant Microbiomes. *Molecules* **24**, (2019).
22. Reva, O. N. *et al.* Genetic, Epigenetic and Phenotypic Diversity of Four *Bacillus velezensis* Strains Used for Plant Protection or as Probiotics. *Front. Microbiol.* **10**, (2019).
23. Bottacini, F. *et al.* Comparative genomics and genotype-phenotype associations in *Bifidobacterium breve*. *Sci. Rep.* **8**, 10633 (2018).
24. Rivera, I., Linz, B. & Harvill, E. T. Evolution and Conservation of *Bordetella* Intracellular Survival in Eukaryotic Host Cells. *Front. Microbiol.* **11**, (2020).
25. Pittet, L. F. & Posfay-Barbe, K. M. *Bordetella holmesii*: Still Emerging and Elusive 20 Years On. *Microbiol. Spectr.* **4**, 4.2.11 (2016).
26. van den Akker, W. M. R. Lipopolysaccharide expression within the genus *Bordetella*: influence of temperature and phase variation. *Microbiol. Read. Engl.* **144** (Pt 6), 1527–1535 (1998).
27. Hoffman, C. L. *et al.* *Bordetella pertussis* Can Be Motile and Express Flagellum-Like Structures. *mBio* **10**, e00787-19 (2019).
28. Ficht, T. *Brucella* taxonomy and evolution. *Future Microbiol.* **5**, 859–866 (2010).
29. El-Sayed, A. & Awad, W. Brucellosis: Evolution and expected comeback. *Int. J. Vet. Sci. Med.* **6**, S31–S35 (2018).
30. Moran, N. A. & Mira, A. The process of genome shrinkage in the obligate symbiont *Buchnera aphidicola*. *Genome Biol.* **2**, research0054.1 (2001).
31. Scoffone, V. C. *et al.* *Burkholderia cenocepacia* Infections in Cystic Fibrosis Patients: Drug Resistance and Therapeutic Approaches. *Front. Microbiol.* **8**, (2017).
32. Loutet, S. A. & Valvano, M. A. A Decade of *Burkholderia cenocepacia* Virulence Determinant Research. *Infect. Immun.* **78**, 4088–4100 (2010).
33. Lamothe, J., Huynh, K. K., Grinstein, S. & Valvano, M. A. Intracellular survival of *Burkholderia cenocepacia* in macrophages is associated with a delay in the maturation of bacteria-containing vacuoles. *Cell. Microbiol.* **9**, 40–53 (2007).
34. Nierman, W. C. *et al.* Structural flexibility in the *Burkholderia mallei* genome. *Proc. Natl. Acad. Sci. U. S. A.* **101**, 14246–14251 (2004).
35. Losada, L. *et al.* Continuing Evolution of *Burkholderia mallei* Through Genome Reduction and Large-Scale Rearrangements. *Genome Biol. Evol.* **2**, 102–116 (2010).
36. Chewapreecha, C. *et al.* Genetic variation associated with infection and the environment in the accidental pathogen *Burkholderia pseudomallei*. *Commun. Biol.* **2**, 1–11 (2019).
37. Lee, Y. H., Chen, Y., Ouyang, X. & Gan, Y.-H. Identification of tomato plant as a novel host model for *Burkholderia pseudomallei*. *BMC Microbiol.* **10**, 28 (2010).
38. Haraga, A., West, T. E., Brittnacher, M. J., Skerrett, S. J. & Miller, S. I. *Burkholderia thailandensis* as a Model System for the Study of the Virulence-Associated Type III Secretion System of *Burkholderia pseudomallei*. *Infect. Immun.* **76**, 5402–5411 (2008).
39. Gee, J. E. *et al.* *Burkholderia thailandensis* Isolated from Infected Wound, Arkansas, USA. *Emerg. Infect. Dis.* **24**, 2091–2094 (2018).

40. Sheppard, S. K. & Maiden, M. C. J. The Evolution of *Campylobacter jejuni* and *Campylobacter coli*. *Cold Spring Harb. Perspect. Biol.* **7**, (2015).
41. Iraola, G. *et al.* Distinct *Campylobacter* fetus lineages adapted as livestock pathogens and human pathobionts in the intestinal microbiota. *Nat. Commun.* **8**, 1367 (2017).
42. Kienesberger, S. *et al.* Comparative Genome Analysis of *Campylobacter* fetus Subspecies Revealed Horizontally Acquired Genetic Elements Important for Virulence and Niche Specificity. *PLOS ONE* **9**, e85491 (2014).
43. Andersen, A. A. Serotyping of US Isolates of *Chlamydophila Psittaci* from Domestic and Wild Birds. *J. Vet. Diagn. Invest.* **17**, 479–482 (2005).
44. Harkinezhad, T., Geens, T. & Vanrompay, D. *Chlamydophila psittaci* infections in birds: A review with emphasis on zoonotic consequences. *Vet. Microbiol.* **135**, 68–77 (2009).
45. Elwell, C., Mirrashidi, K. & Engel, J. *Chlamydia* cell biology and pathogenesis. *Nat. Rev. Microbiol.* **14**, 385–400 (2016).
46. Ranjan, K. P. & Ranjan, N. *Citrobacter*: An emerging health care associated urinary pathogen. *Urol. Ann.* **5**, 313–314 (2013).
47. Smith, L. D. *Clostridium botulinum*: characteristics and occurrence. *Rev. Infect. Dis.* **1**, 637–641 (1979).
48. Espelund, M. & Klaveness, D. Botulism outbreaks in natural environments – an update. *Front. Microbiol.* **5**, (2014).
49. Kurtböke, D. İ. Ecology and Habitat Distribution of Actinobacteria. in *Biology and Biotechnology of Actinobacteria* (eds. Wink, J., Mohammadipanah, F. & Hamed, J.) 123–149 (Springer International Publishing, 2017). doi:10.1007/978-3-319-60339-1_6.
50. Sing, A. *et al.* *Corynebacterium diphtheriae* in a free-roaming red fox: case report and historical review on diphtheria in animals. *Infection* **44**, 441–445 (2016).
51. Kliegman, R. *et al.* *Nelson textbook of pediatrics*. (2020).
52. Arisoy, E. S., Demmler, G. J. & Dunne, W. M. J. CORYNEBACTERIUM XEROSIS VENTRICULOPERITONEAL SHUNT INFECTION IN AN INFANT: REPORT OF A CASE AND REVIEW OF THE LITERATURE. *Pediatr. Infect. Dis. J.* **12**, 536–537 (1993).
53. Wang, L., Liu, Z., Dai, S., Yan, J. & Wise, M. J. The Sit-and-Wait Hypothesis in Bacterial Pathogens: A Theoretical Study of Durability and Virulence. *Front. Microbiol.* **8**, (2017).
54. Yang, J. & Yang, S. Comparative analysis of *Corynebacterium glutamicum* genomes: a new perspective for the industrial production of amino acids. *BMC Genomics* **18**, 940 (2017).
55. Sgobba, E., Blöbaum, L. & Wendisch, V. F. Production of Food and Feed Additives From Non-food-competing Feedstocks: Valorizing N-acetylmuramic Acid for Amino Acid and Carotenoid Fermentation With *Corynebacterium glutamicum*. *Front. Microbiol.* **0**, (2018).
56. Sabbadini, P. S. *et al.* *Corynebacterium diphtheriae* 67-72p hemagglutinin, characterized as the protein DIP0733, contributes to invasion and induction of apoptosis in HEP-2 cells. *Microb. Pathog.* **52**, 165–176 (2012).
57. Dorella, F. A., Pacheco, L. G. C., Oliveira, S. C., Miyoshi, A. & Azevedo, V. *Corynebacterium pseudotuberculosis*: microbiology, biochemical properties, pathogenesis and molecular studies of virulence. *Vet. Res.* **37**, 201–218 (2006).
58. Zendri, F. *et al.* Case Report: Toxigenic *Corynebacterium ulcerans* Diphtheria-Like Infection in a Horse in the United Kingdom. *Front. Vet. Sci.* **8**, (2021).
59. Moore, L. S. P. *et al.* *Corynebacterium ulcerans* cutaneous diphtheria. *Lancet Infect. Dis.* **15**, 1100–1107 (2015).
60. Shaw, E. I. & Voth, D. E. *Coxiella burnetii*: A Pathogenic Intracellular Acidophile. *Microbiology* **165**, 1–3 (2019).

61. Larson, C. L. *et al.* Right on Q: genetics begin to unravel *Coxiella burnetii* host cell interactions. *Future Microbiol.* **11**, 919–939 (2016).
62. Maurin, M. & Raoult, D. Q Fever. *Clin. Microbiol. Rev.* **12**, 518–553 (1999).
63. Löffler, F. E. *et al.* Dehalococcoides mccartyi gen. nov., sp. nov., obligately organohalide-respiring anaerobic bacteria relevant to halogen cycling and bioremediation, belong to a novel bacterial class, Dehalococcoidia classis nov., order Dehalococcoidales ord. nov. and family Dehalococcoidaceae fam. nov., within the phylum Chloroflexi. *Int. J. Syst. Evol. Microbiol.* **63**, 625–635 (2013).
64. Kämpfer, P. *et al.* Elizabethkingia anophelis sp. nov., isolated from the midgut of the mosquito *Anopheles gambiae*. *Int. J. Syst. Evol. Microbiol.* **61**, 2670–2675 (2011).
65. Chen, S., Bagdasarian, M. & Walker, E. D. Elizabethkingia anophelis: Molecular Manipulation and Interactions with Mosquito Hosts. *Appl. Environ. Microbiol.* **81**, 2233–2243 (2015).
66. Ngwa, C. J. *et al.* 16S rRNA gene-based identification of Elizabethkingia meningoseptica (Flavobacteriales: Flavobacteriaceae) as a dominant midgut bacterium of the Asian malaria vector *Anopheles stephensi* (Diptera: Culicidae) with antimicrobial activities. *J. Med. Entomol.* **50**, 404–414 (2013).
67. Teo, J. *et al.* Comparative Genomic Analysis of Malaria Mosquito Vector-Associated Novel Pathogen Elizabethkingia anophelis. *Genome Biol. Evol.* **6**, 1158–1165 (2014).
68. Keller, R., Pedroso, M. Z., Ritchmann, R. & Silva, R. M. Occurrence of Virulence-Associated Properties in *Enterobacter cloacae*. *Infect. Immun.* **66**, 645–649 (1998).
69. Mustafa, A. *et al.* Genome-wide Analysis of Four *Enterobacter cloacae* complex type strains: Insights into Virulence and Niche Adaptation. *Sci. Rep.* **10**, 8150 (2020).
70. Foti, M. *et al.* Antibiotic resistance assessment in bacteria isolated in migratory Passeriformes transiting through the Metaponto territory (Basilicata, Italy). *Avian Res.* **8**, 26 (2017).
71. Davin-Regli, A., Lavigne, J.-P. & Pagès, J.-M. *Enterobacter* spp.: Update on Taxonomy, Clinical Aspects, and Emerging Antimicrobial Resistance. *Clin. Microbiol. Rev.* **32**, e00002-19 (2019).
72. Wang, Z. *et al.* First report of *Enterobacter hormaechei* with respiratory disease in calves. *BMC Vet. Res.* **16**, 1 (2020).
73. Byappanahalli, M. N., Nevers, M. B., Korajkic, A., Staley, Z. R. & Harwood, V. J. Enterococci in the Environment. *Microbiol. Mol. Biol. Rev. MMBR* **76**, 685–706 (2012).
74. van Elsland, J. D., Semenov, A. V., Costa, R. & Trevors, J. T. Survival of *Escherichia coli* in the environment: fundamental and public health aspects. *ISME J.* **5**, 173–183 (2011).
75. Percival, S. L. & Williams, D. W. Chapter Six - *Escherichia coli*. in *Microbiology of Waterborne Diseases (Second Edition)* (eds. Percival, S. L., Yates, M. V., Williams, D. W., Chalmers, R. M. & Gray, N. F.) 89–117 (Academic Press, 2014). doi:10.1016/B978-0-12-415846-7.00006-8.
76. Borges, C. A. *et al.* Multidrug-resistant pathogenic *Escherichia coli* isolated from wild birds in a veterinary hospital. *Avian Pathol. J. WVPA* **46**, 76–83 (2017).
77. Zarrella, T. M. *et al.* Host-Adaptation of *Francisella tularensis* Alters the Bacterium's Surface-Carbohydrates to Hinder Effectors of Innate and Adaptive Immunity. *PLOS ONE* **6**, e22335 (2011).
78. Mörner, T. The ecology of tularaemia. *Rev. Sci. Tech. Int. Off. Epizoot.* **11**, 1123–1130 (1992).
79. Kapatral, V. *et al.* Genome Sequence and Analysis of the Oral Bacterium *Fusobacterium nucleatum* Strain ATCC 25586. *J. Bacteriol.* **184**, 2005–2018 (2002).

80. Strauss, J., White, A., Ambrose, C., McDonald, J. & Allen-Vercoe, E. Phenotypic and genotypic analyses of clinical *Fusobacterium nucleatum* and *Fusobacterium periodonticum* isolates from the human gut. *Anaerobe* **14**, 301–309 (2008).
81. Greenberg, D. E. *et al.* Recurrent *Granulibacter bethesdensis* Infections and Chronic Granulomatous Disease. *Emerg. Infect. Dis.* **16**, 1341–1348 (2010).
82. Greenberg, D. E. *et al.* Simultaneous Host-Pathogen Transcriptome Analysis during *Granulibacter bethesdensis* Infection of Neutrophils from Healthy Subjects and Patients with Chronic Granulomatous Disease. *Infect. Immun.* **83**, 4277–4292 (2015).
83. Greenberg, D. E. *et al.* *Granulibacter bethesdensis* gen. nov., sp. nov., a distinctive pathogenic acetic acid bacterium in the family Acetobacteraceae. *Int. J. Syst. Evol. Microbiol.* **56**, 2609–2616 (2006).
84. Kusters, J. G., van Vliet, A. H. M. & Kuipers, E. J. Pathogenesis of *Helicobacter pylori* Infection. *Clin. Microbiol. Rev.* **19**, 449–490 (2006).
85. Dubois, A. *et al.* Immunization against Natural *Helicobacter pylori* Infection in Nonhuman Primates. *Infect. Immun.* **66**, 4340–4346 (1998).
86. Wesevich, A. *et al.* Newly Named *Klebsiella aerogenes* (formerly *Enterobacter aerogenes*) Is Associated with Poor Clinical Outcomes Relative to Other *Enterobacter* Species in Patients with Bloodstream Infection. *J. Clin. Microbiol.* **58**, (2020).
87. Sanders, W. E. & Sanders, C. C. *Enterobacter* spp.: pathogens poised to flourish at the turn of the century. *Clin. Microbiol. Rev.* **10**, 220–241 (1997).
88. Ramachandran, S. G., Vikas Loiwal, Ajay Kumar, Piyush Gupta & Vg. *Enterobacter aerogenes* outbreak in a neonatal intensive care unit. *Pediatr. Int.* **41**, 157–161 (1999).
89. Szczerba, H. *et al.* Genome analysis of a wild rumen bacterium *Enterobacter aerogenes* LU2 - a novel bio-based succinic acid producer. *Sci. Rep.* **10**, 1986 (2020).
90. Bagley, S. T. Habitat association of *Klebsiella* species. *Infect. Control IC* **6**, 52–58 (1985).
91. Adachi, K., Nakatani, M. & Mochida, H. Isolation of an endophytic diazotroph, *Klebsiella oxytoca*, from sweet potato stems in Japan. *Soil Sci. Plant Nutr.* **48**, 889–895 (2002).
92. Zheng, J. *et al.* A taxonomic note on the genus *Lactobacillus*: Description of 23 novel genera, emended description of the genus *Lactobacillus* Beijerinck 1901, and union of *Lactobacillaceae* and *Leuconostocaceae*. *Int. J. Syst. Evol. Microbiol.* **70**, 2782–2858 (2020).
93. Duar, R. M. *et al.* Lifestyles in transition: evolution and natural history of the genus *Lactobacillus*. *FEMS Microbiol. Rev.* **41**, S27–S48 (2017).
94. Michaylova, M., Minkova, S., Kimura, K., Sasaki, T. & Isawa, K. Isolation and characterization of *Lactobacillus delbrueckii* ssp. *bulgaricus* and *Streptococcus thermophilus* from plants in Bulgaria. *FEMS Microbiol. Lett.* **269**, 160–169 (2007).
95. Germond, J.-E. *et al.* Evolution of the Bacterial Species *Lactobacillus delbrueckii*: A Partial Genomic Study with Reflections on Prokaryotic Species Concept. *Mol. Biol. Evol.* **20**, 93–104 (2003).
96. Smokvina, T. *et al.* *Lactobacillus paracasei* Comparative Genomics: Towards Species Pan-Genome Definition and Exploitation of Diversity. *PLoS ONE* **8**, e68731 (2013).
97. Song, A. A.-L., In, L. L. A., Lim, S. H. E. & Rahim, R. A. A review on *Lactococcus lactis*: from food to factory. *Microb. Cell Factories* **16**, 55 (2017).
98. Newton, H. J., Ang, D. K. Y., van Driel, I. R. & Hartland, E. L. Molecular pathogenesis of infections caused by *Legionella pneumophila*. *Clin. Microbiol. Rev.* **23**, 274–298 (2010).
99. Steinert, M., Hentschel, U. & Hacker, J. *Legionella pneumophila*: an aquatic microbe goes astray. *FEMS Microbiol. Rev.* **26**, 149–162 (2002).

100. Oliva, G., Sahr, T. & Buchrieser, C. The Life Cycle of *L. pneumophila*: Cellular Differentiation Is Linked to Virulence and Metabolism. *Front. Cell. Infect. Microbiol.* **8**, 3 (2018).
101. Mundt, J. O. LACTIC ACID BACTERIA ASSOCIATED WITH RAW PLANT FOOD MATERIAL1. *J. Milk Food Technol.* **33**, 550–553 (1970).
102. Bou, G. *et al.* Nosocomial Outbreaks Caused by *Leuconostoc mesenteroides* subsp. *mesenteroides*. *Emerg. Infect. Dis.* **14**, 968–971 (2008).
103. Ivanek, R., Gröhn, Y. T. & Wiedmann, M. *Listeria monocytogenes* in multiple habitats and host populations: review of available data for mathematical modeling. *Foodborne Pathog. Dis.* **3**, 319–336 (2006).
104. Schuppler, M. & Loessner, M. J. The Opportunistic Pathogen *Listeria monocytogenes*: Pathogenicity and Interaction with the Mucosal Immune System. *Int. J. Inflamm.* **2010**, e704321 (2010).
105. Hamon, M., Bierne, H. & Cossart, P. *Listeria monocytogenes*: a multifaceted model. *Nat. Rev. Microbiol.* **4**, 423–434 (2006).
106. Rice, J. A., Carrasco-Medina, L., Hodgins, D. C. & Shewen, P. E. Mannheimia haemolytica and bovine respiratory disease. *Anim. Health Res. Rev.* **8**, 117–128 (2007).
107. Klima, C. L., Alexander, T. W., Hendrick, S. & McAllister, T. A. Characterization of Mannheimia haemolytica isolated from feedlot cattle that were healthy or treated for bovine respiratory disease. *Can. J. Vet. Res.* **78**, 38–45 (2014).
108. Cozens, D. *et al.* Pathogenic Mannheimia haemolytica Invades Differentiated Bovine Airway Epithelial Cells. *Infect. Immun.* **87**, e00078-19 (2019).
109. Turenne, C. Y., Wallace, R. & Behr, M. A. Mycobacterium avium in the Postgenomic Era. *Clin. Microbiol. Rev.* **20**, 205–229 (2007).
110. Collins, M. T. & Stabel, J. R. Diseases of Dairy Animals | Infectious Diseases: Johne's Disease. in *Encyclopedia of Dairy Sciences (Second Edition)* (ed. Fuquay, J. W.) 174–180 (Academic Press, 2011). doi:10.1016/B978-0-12-374407-4.00134-5.
111. To, K., Cao, R., Yegiazaryan, A., Owens, J. & Venketaraman, V. General Overview of Nontuberculous Mycobacteria Opportunistic Pathogens: Mycobacterium avium and Mycobacterium abscessus. *J. Clin. Med.* **9**, 2541 (2020).
112. Madacki, J., Mas Fiol, G. & Brosch, R. Update on the virulence factors of the obligate pathogen Mycobacterium tuberculosis and related tuberculosis-causing mycobacteria. *Infect. Genet. Evol.* **72**, 67–77 (2019).
113. Mahamed, D. *et al.* Intracellular growth of Mycobacterium tuberculosis after macrophage cell death leads to serial killing of host cells. *eLife* **6**, e22028 (2017).
114. Gupta, R. S., Lo, B. & Son, J. Phylogenomics and Comparative Genomic Studies Robustly Support Division of the Genus Mycobacterium into an Emended Genus Mycobacterium and Four Novel Genera. *Front. Microbiol.* **9**, 67 (2018).
115. Teska, J. D., Twerdok, L. E., Beaman, J., Curry, M. & Finch, R. A. Isolation of Mycobacterium abscessus from Japanese Medaka. *J. Aquat. Anim. Health* **9**, 234–238 (1997).
116. Yavlovich, A., Tarshis, M. & Rottem, S. Internalization and intracellular survival of Mycoplasma pneumoniae by non-phagocytic cells. *FEMS Microbiol. Lett.* **233**, 241–246 (2004).
117. Dhondt, A. A., DeCoste, J. C., Ley, D. H. & Hochachka, W. M. Diverse Wild Bird Host Range of Mycoplasma gallisepticum in Eastern North America. *PLOS ONE* **9**, e103553 (2014).
118. Hill, S. A., Masters, T. L. & Wachter, J. Gonorrhea - an evolving disease of the new millennium. *Microb. Cell* **3**, 371–389.

119. Timmusk, S., Grantcharova, N. & Wagner, E. G. H. *Paenibacillus polymyxa* Invades Plant Roots and Forms Biofilms. *Appl. Environ. Microbiol.* **71**, 7292–7300 (2005).
120. Wilson, B. A. & Ho, M. *Pasteurella multocida*: from Zoonosis to Cellular Microbiology. *Clin. Microbiol. Rev.* **26**, 631–655 (2013).
121. Bramucci, A. R. *et al.* The Bacterial Symbiont *Phaeobacter inhibens* Shapes the Life History of Its Algal Host *Emiliania huxleyi*. *Front. Mar. Sci.* **5**, (2018).
122. Dittmann, K. K., Sonnenschein, E. C., Egan, S., Gram, L. & Bentzon-Tilia, M. Impact of *Phaeobacter inhibens* on marine eukaryote-associated microbial communities. *Environ. Microbiol. Rep.* **11**, 401–413 (2019).
123. Beyersmann, P. G. *et al.* Dual function of tropodithietic acid as antibiotic and signaling molecule in global gene regulation of the probiotic bacterium *Phaeobacter inhibens*. *Sci. Rep.* **7**, 730 (2017).
124. Fryer, J. L. & Hedrick, R. P. *Piscirickettsia salmonis*: a Gram-negative intracellular bacterial pathogen of fish. *J. Fish Dis.* **26**, 251–262 (2003).
125. Irshad, M., van der Reijden, W. A., Crielaard, W. & Laine, M. L. In Vitro Invasion and Survival of *Porphyromonas gingivalis* in Gingival Fibroblasts; Role of the Capsule. *Arch. Immunol. Ther. Exp. (Warsz.)* **60**, 469–476 (2012).
126. Roberts, J. S. *et al.* Opportunistic Pathogen *Porphyromonas gingivalis* Modulates Danger Signal ATP-Mediated Antibacterial NOX2 Pathways in Primary Epithelial Cells. *Front. Cell. Infect. Microbiol.* **7**, 291 (2017).
127. Olsen, I., Lambris, J. D. & Hajishengallis, G. *Porphyromonas gingivalis* disturbs host–commensal homeostasis by changing complement function. *J. Oral Microbiol.* **9**, 1340085 (2017).
128. Teanpa San, R., Douglas, C. W. I., Eley, A. R. & Walsh, T. F. Clonality of *Porphyromonas gingivalis*, *Prevotella intermedia* and *Prevotella nigrescens* isolated from periodontally diseased and healthy sites. *J. Periodontal Res.* **31**, 423–432 (1996).
129. Albertini, M. *et al.* Assessment of periodontal and opportunistic flora in patients with peri-implantitis. *Clin. Oral Implants Res.* **26**, 937–941 (2015).
130. Dufresne, A. *et al.* Genome sequence of the cyanobacterium *Prochlorococcus marinus* SS120, a nearly minimal oxyphototrophic genome. *Proc. Natl. Acad. Sci.* **100**, 10020–10025 (2003).
131. Lambert, B. S., Fernandez, V. I. & Stocker, R. Motility drives bacterial encounter with particles responsible for carbon export throughout the ocean. *Limnol. Oceanogr. Lett.* **4**, 113–118 (2019).
132. Frohnmeier, E. *et al.* Secretome profiling of *Propionibacterium freudenreichii* reveals highly variable responses even among the closely related strains. *Microb. Biotechnol.* **11**, 510–526 (2018).
133. Deptula, P. *et al.* De novo assembly of genomes from long sequence reads reveals uncharted territories of *Propionibacterium freudenreichii*. *BMC Genomics* **18**, 790 (2017).
134. Piwowarek, K., Lipińska, E., Hać-Szymańczuk, E., Kieliszek, M. & Ścibisz, I. *Propionibacterium* spp.—source of propionic acid, vitamin B12, and other metabolites important for the industry. *Appl. Microbiol. Biotechnol.* **102**, 515–538 (2018).
135. Drzewiecka, D. Significance and Roles of *Proteus* spp. Bacteria in Natural Environments. *Microb. Ecol.* **72**, 741–758 (2016).
136. Sandoz, K. M., Mitzimberg, S. M. & Schuster, M. Social cheating in *Pseudomonas aeruginosa* quorum sensing. *Proc. Natl. Acad. Sci.* **104**, 15876–15881 (2007).
137. Pellett, S., Bigley, D. V. & Grimes, D. J. Distribution of *Pseudomonas aeruginosa* in a riverine ecosystem. *Appl. Environ. Microbiol.* **45**, 328–332 (1983).
138. Crone, S. *et al.* The environmental occurrence of *Pseudomonas aeruginosa*. *APMIS* **128**, 220–231 (2020).

139. Haenni, M. *et al.* Resistance of Animal Strains of *Pseudomonas aeruginosa* to Carbapenems. *Front. Microbiol.* **8**, 1847 (2017).
140. Scales, B. S., Dickson, R. P., LiPuma, J. J. & Huffnagle, G. B. Microbiology, Genomics, and Clinical Significance of the *Pseudomonas fluorescens* Species Complex, an Unappreciated Colonizer of Humans. *Clin. Microbiol. Rev.* **27**, 927–948 (2014).
141. Fernández, M. *et al.* Analysis of the pathogenic potential of nosocomial *Pseudomonas putida* strains. *Front. Microbiol.* **6**, (2015).
142. Xin, X.-F., Kvitko, B. & He, S. Y. *Pseudomonas syringae*: what it takes to be a pathogen. *Nat. Rev. Microbiol.* **16**, 316–328 (2018).
143. Morris, C. E. *et al.* The life history of the plant pathogen *Pseudomonas syringae* is linked to the water cycle. *ISME J.* **2**, 321–334 (2008).
144. Morris, C. E., Lamichhane, J. R., Nikolić, I., Stanković, S. & Moury, B. The overlapping continuum of host range among strains in the *Pseudomonas syringae* complex. *Phytopathol. Res.* **1**, 4 (2019).
145. Álvarez, B., López, M. M. & Biosca, E. G. Biocontrol of the Major Plant Pathogen *Ralstonia solanacearum* in Irrigation Water and Host Plants by Novel Waterborne Lytic Bacteriophages. *Front. Microbiol.* **10**, (2019).
146. Lowendorf, H. S. & Alexander, M. Identification of *Rhizobium phaseoli* Strains That Are Tolerant or Sensitive to Soil Acidity. *Appl. Environ. Microbiol.* **45**, 737–742 (1983).
147. Akter, A. *et al.* Extremely Low Genomic Diversity of *Rickettsia japonica* Distributed in Japan. *Genome Biol. Evol.* **9**, 124–133 (2016).
148. Higgins, D. A., Henry, R. R. & Kounev, Z. V. Duck immune responses to *Riemerella anatipestifer* vaccines. *Dev. Comp. Immunol.* **24**, 153–167 (2000).
149. Lamas, A. *et al.* A comprehensive review of non-enterica subspecies of *Salmonella enterica*. *Microbiol. Res.* **206**, 60–73 (2018).
150. Hejazi, A. & Falkiner, F. R. *Serratia marcescens*. *J. Med. Microbiol.* **46**, 903–912 (1997).
151. Huang, G. *et al.* Isolation of a Novel Heterotrophic Nitrification–Aerobic Denitrification Bacterium *Serratia marcescens* CL1502 from Deep-Sea Sediment. *Environ. Eng. Sci.* **34**, 453–459 (2017).
152. Tong, S. Y. C., Davis, J. S., Eichenberger, E., Holland, T. L. & Fowler, V. G. *Staphylococcus aureus* infections: epidemiology, pathophysiology, clinical manifestations, and management. *Clin. Microbiol. Rev.* **28**, 603–661 (2015).
153. Johri, A. K. *et al.* Group B *Streptococcus*: global incidence and vaccine development. *Nat. Rev. Microbiol.* **4**, 932–942 (2006).
154. Linden, M. van der, Al-Lahham, A., Nicklas, W. & Reinert, R. R. Molecular Characterization of Pneumococcal Isolates from Pets and Laboratory Animals. *PLOS ONE* **4**, e8286 (2009).
155. Weiser, J. N., Ferreira, D. M. & Paton, J. C. *Streptococcus pneumoniae*: transmission, colonization and invasion. *Nat. Rev. Microbiol.* **16**, 355–367 (2018).
156. Chalmers, C. *et al.* *Streptococcus pyogenes* nuclease A (SpnA) mediated virulence does not exclusively depend on nuclease activity. *J. Microbiol. Immunol. Infect.* **53**, 42–48 (2020).
157. Hughes, J. M. *et al.* *Streptococcus suis*: An Emerging Human Pathogen. *Clin. Infect. Dis.* **48**, 617–625 (2009).
158. Goh, Y. J., Goin, C., O’Flaherty, S., Altermann, E. & Hutkins, R. Specialized adaptation of a lactic acid bacterium to the milk environment: the comparative genomics of *Streptococcus thermophilus* LMD-9. *Microb. Cell Factories* **10**, S22 (2011).
159. Couvigny, B. *et al.* *Streptococcus thermophilus* Biofilm Formation: A Remnant Trait of Ancestral Commensal Life? *PLoS ONE* **10**, e0128099 (2015).

160. Fu, K. *et al.* An Innovative Method for Rapid Identification and Detection of *Vibrio alginolyticus* in Different Infection Models. *Front. Microbiol.* **7**, (2016).
161. Dong, Y. *et al.* Fast, simple and highly specific molecular detection of *Vibrio alginolyticus* pathogenic strains using a visualized isothermal amplification method. *BMC Vet. Res.* **16**, 76 (2020).
162. Noguchi, T. *et al.* *Vibrio alginolyticus*, a tetrodotoxin-producing bacterium, in the intestines of the fish *Fugu vermicularis vermicularis*. *Mar. Biol.* **94**, 625–630 (1987).
163. Castillo, D. *et al.* Draft Genome Sequences of *Vibrio alginolyticus* Strains V1 and V2, Opportunistic Marine Pathogens. *Genome Announc.* **3**, e00729-15.
164. Lages, M. A., Balado, M. & Lemos, M. L. The Expression of Virulence Factors in *Vibrio anguillarum* Is Dually Regulated by Iron Levels and Temperature. *Front. Microbiol.* **10**, (2019).
165. Mou, X., Spinard, E. J., Hillman, S. L. & Nelson, D. R. Isocitrate dehydrogenase mutation in *Vibrio anguillarum* results in virulence attenuation and immunoprotection in rainbow trout (*Oncorhynchus mykiss*). *BMC Microbiol.* **17**, 217 (2017).
166. Pybus, V., Loutit, M. W., Lamont, I. L. & Tagg, J. R. Growth inhibition of the salmon pathogen *Vibrio ordalii* by a siderophore produced by *Vibrio anguillarum* strain VL4355. *J. Fish Dis.* **17**, 311–324 (1994).
167. Lutz, C., Erken, M., Noorian, P., Sun, S. & McDougald, D. Environmental reservoirs and mechanisms of persistence of *Vibrio cholerae*. *Front. Microbiol.* **4**, 375 (2013).
168. Vicente, J. G. & Holub, E. B. *Xanthomonas campestris* pv. *campestris* (cause of black rot of crucifers) in the genomic era is still a worldwide threat to brassica crops. *Mol. Plant Pathol.* **14**, 2–18 (2013).
169. Ference, C. M. *et al.* Recent advances in the understanding of *Xanthomonas citri* ssp. *citri* pathogenesis and citrus canker disease management. *Mol. Plant Pathol.* **19**, 1302–1318 (2018).
170. Patané, J. S. L. *et al.* Origin and diversification of *Xanthomonas citri* subsp. *citri* pathotypes revealed by inclusive phylogenomic, dating, and biogeographic analyses. *BMC Genomics* **20**, 700 (2019).
171. Lang, J. M. *et al.* A Pathovar of *Xanthomonas oryzae* Infecting Wild Grasses Provides Insight Into the Evolution of Pathogenicity in Rice Agroecosystems. *Front. Plant Sci.* **10**, (2019).
172. Baldi, P. & La Porta, N. *Xylella fastidiosa*: Host Range and Advance in Molecular Identification Techniques. *Front. Plant Sci.* **8**, (2017).
173. Fredriksson-Ahomaa, M., Stolle, A. & Korkeala, H. Molecular epidemiology of *Yersinia enterocolitica* infections. *FEMS Immunol. Med. Microbiol.* **47**, 315–329 (2006).
174. Harvey, S., Greenwood, J. R., Pickett, M. J. & Mah, R. A. Recovery of *Yersinia enterocolitica* from streams and lakes of California. *Appl. Environ. Microbiol.* **32**, 352–354 (1976).
175. Batzilla, J., Heesemann, J. & Rakin, A. The pathogenic potential of *Yersinia enterocolitica* 1A. *Int. J. Med. Microbiol.* **301**, 556–561 (2011).
176. Rakin, A., Garzetti, D., Bouabe, H. & Sprague, L. D. Chapter 73 - *Yersinia enterocolitica*. in *Molecular Medical Microbiology (Second Edition)* (eds. Tang, Y.-W., Sussman, M., Liu, D., Poxton, I. & Schwartzman, J.) 1319–1344 (Academic Press, 2015). doi:10.1016/B978-0-12-397169-2.00073-1.
177. Santos-Montañez, J., Benavides-Montaña, J. A., Hinz, A. K. & Vadyvaloo, V. *Yersinia pseudotuberculosis* IP32953 survives and replicates in trophozoites and persists in cysts of *Acanthamoeba castellanii*. *FEMS Microbiol. Lett.* **362**, (2015).
178. Weir, P. M. The ecology of *Zymomonas*: a review. *Folia Microbiol. (Praha)* **61**, 385–392 (2016).

