Lecture 17:

	Bacteria	Distribution	Virulence Factors (VF)	Diseases	Notes
	Haemophilus (haemo: blood Philos: lover) (pleomorphic)	present in almost all individuals, primarily colonizing the mucosal membranes of the respiratory tract			*small, sometimes pleomorphic. *requires blood for growth on agar media. *Growth stimulating factors needed in media: **(1)** hemin (X factor for "unknown factor") & **(2)** nicotinamide adenine dinucleotide (NAD; also called V factor for "vitamin")
1	**H. influenzae** **(type b: the most significant pediatric pathogen in many countries of the world & was responsible for more than 95% of all invasive *Haemophilus* infections)**	*H. influenzae* (particularly serotype b [biotype I]) is uncommon in the upper respiratory tract	1.The surface of many, but not all, strains of H. influenzae is covered with a six antigenic serotypes (a through f) & **antiphagocytic polysaccharide capsule** (polyribitol phosphate [PRP], which contains ribose, ribitol, and phosphate >> antibodies against it develop because of natural infection, vaccination with purified PRP, or the passive transfer of maternal antibodies. (capsule is a major VF) 2. **Adhesins** mediate colonization of the oropharynx 3. **Cell wall components** of the bacteria damage the respiratory epithelium. Then translocation across both epithelial and endothelial cells occurs and the bacteria can enter the blood.	*A common cause of disease in unvaccinated children: **a. meningitis**: fever, sever headache, & systemic signs. **b. epiglottitis** [obstructive laryngitis]: initial pharyngitis, fever, and difficulty breathing and progressing to cellulitis and swelling of the supraglottic tissues, with obstruction of of the airways **c. cellulitis** ***Pneomonea** (in elderly with underlying chronic pulmonary disease): inflammation and consolidation of the lungstypically caused by nontypeable strains. *In the absence of specific opsonic antibodies directed against the polysaccharide capsule, **high-grade bacteremia** can develop. ***H. influenzae and S. pneumoniae are the two most common causes of acute and chronic otitis and sinusitis**	*After introduction of the vaccine, more than half of all invasive disease is now caused by nonencapsulated (nontypeable) strains which are opportunistic pathogens that can cause infections of the upper and lower airways. ***Treatment:** systemic *H. influenzae* infections require **prompt antimicrobial therapy** because the mortality rate in patients with untreated meningitis or epiglottitis approaches 100%. ***Identification & diagnosis:** -A **presumptive identification** of *H. influenzae* can be made by the **Gram stain morphology** and demonstration of a **requirement for both X and V factors**. -The **immunologic detection** of *H. influenzae* antigen, specifically the **PRP capsular antigen**, is a rapid and sensitive way to diagnose *H. influenzae* type b disease
2	**H. aegyptius** (the Koch-Weeks bacillus)			acute purulent **conjunctivitis**	
3	**H. ducreyi**			**Chancroids** (STD common in men): 5 to 7 days after exposure (occupation period) , a tender papule with an erythematous base develops on the genitalia or perianal area.	
4	**H. parainfluenzae**			**Bacteremia, endocarditis, opportunistic infections**	

the species most commonly associated with disease

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Aggregatibacter (A. actinomycetemcomitans & A. aphrophilus)		colonize the human mouth		can spread from the mouth into the blood and then stick to a previously damaged heart valve or artificial valve, leading to the development of **endocarditis**.	
1	**A. actinomycetemcomitans**			*often found in association with localized aggressive **periodontitis** ***endocarditis** ***bite wound infections**	*facultative anaerobe, non-motile bacterium *frequency: It's commonly found
Pasteurella (coccobacilli) **(P.multocida>> common P. canis>> uncommon)** Both are human pathogens		found as commensals in the oropharynx of healthy animals		**P. multocida** causes: Bite wound infections, chronic pulmonary disease, bacteremia, meningitis. **P. canis** causes: Bite wound infections	* genus of facultatively anaerobic, fermentative coccobacilli *Most human infections result from **animal contact (e.g., animal bites, scratches, shared food).**
Vibrio (Rods)			lipopolysaccharides consisting of lipid A (endotoxin), core polysaccharide, and an O polysaccharide side chain. ⬇ The O polysaccharide is used to subdivide *Vibrio* species into **serogroups** (*cholerae* O1 and O139 are vibrio serotypes)		*facultatively anaerobic, fermentative, oxidase positive, with polar flagella rods *Growth media description: can grow on a variety of simple media within a broad temperature range (from 14° C to 40° C). And tolerate a wide range of pH (e.g., pH of 6.5 to 9.0) but are **susceptible to stomach acids.** All species require **sodium chloride (NaCl).** *Most species are halophilic ("salt-loving"). *grow naturally in **estuarine and marine environments** worldwide & Pathogenic vibrios can also flourish in waters with chitinous **shellfish .**
1	**V. cholerae (cholerae O1 and O139** are the only to produce cholera toxin and are associated with epidemics of cholera)		***Cholera complex A-B toxin:** The active portion of the A subunit ↑ **adenylate cyclase activity >** ↑cAMP > hypersecretion of water and electrolytes. ***toxin co-regulated pilus (TCP):** surface receptor site for the bacteriophage CTXΦ permitting it to move into the bacterial cell, where it becomes integrated into the *V. cholerae* genome;	The majority of individuals exposed to toxigenic ***V. cholerae* O1** have asymptomatic infections or self-limited diarrhea; however, some individuals develop severe, **rapidly fatal diarrhea with rice water stools** (colorless and odourless, free of protein, and speckled with mucus) with the abrupt onset of watery diarrhea and **vomiting. Fever is rare.**	*Strains other than O1 & O139 of *V. cholerae* generally do not produce cholera toxin and do not cause epidemic disease. ***Transmission:** Cholera is spread by **contaminated water and food** rather than direct person-to-person spread, because a **high inoculum** (e.g., >10^8

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			mediates adherence to intestinal mucosal cells. (encoded on VPI-1) ***Chemotaxis protein** (adhesin factor) ***Accessory cholera enterotoxin**: ↑ fluid secretion. ***Zonula occludens toxin:** ↑ intestinal permeability. ***Neuraminidase:** ↑GM1 binding sites for cholera toxin. **How cholera strains gain toxicity? Virulence of *V. cholerae* involved acquisition of first a sequence of genes including the **toxin co-regulated pilus (TCP)** on what is termed the **vibrio pathogenicity island (VPI-1),** followed by infection with the **bacteriophage CTXΦ** that encodes the genes for the two subunits of **cholera toxin** (*ctxA* and *ctxB*).	*The resulting severe fluid and electrolyte loss can lead to **dehydration, painful muscle cramps, metabolic acidosis** (bicarbonate loss), and **hypokalemia** and **hypovolemic shock** (potassium loss), with **cardiac arrhythmia and renal failure**. ***V. cholerae* O1 does not produce a capsule, so infections with this organism do not spread beyond the confines of the intestine.	organisms is required to establish infection in a person with normal gastric acidity.). *usually seen in communities with **poor sanitation.** ***Identification & diagnosis:** **-Immunoassays** for the detection of cholera toxin or the O1 and O139 lipopolysaccharides are used for the diagnosis of cholera in endemic areas. * occupation period: 2 to 3 days (can be <12 hours) ***Treatment:** Patients with cholera must be promptly treated with **fluid and electrolyte replacement (**↓ mortality rate from 70% to 1%) before the resultant massive fluid loss leads to hypovolemic shock. *Some statistics: It is estimated that **3 to 5 million cases** of cholera and 120,000 deaths occur worldwide **each year**. Seven major pandemics of cholera have occurred since 1817, resulting in thousands of deaths and major socioeconomic changes.

*Note: Chocolate agar contains heated blood and named chocolate just for the similar brown colour.

*More info. about H. influenzae:

-Most of the *H. influenzae* type b infections now occur in children who are not immune (because of incomplete vaccination or a poor response to the vaccine) and in elderly adults with waning immunity.

-Antibodies directed against the capsule greatly stimulate bacterial phagocytosis and complement-mediated bactericidal activity

-When vaccines containing purified PRP antigens conjugated to protein carriers (i.e., diphtheria toxoid, tetanus toxoid, meningococcal outer membrane protein) were introduced in December 1987, a protective antibody response in infants aged 2 months and older was produced, and systemic disease in children younger than age 5 was virtually eliminated in the United States.

Satellite phenomenon caused by H. influenzae

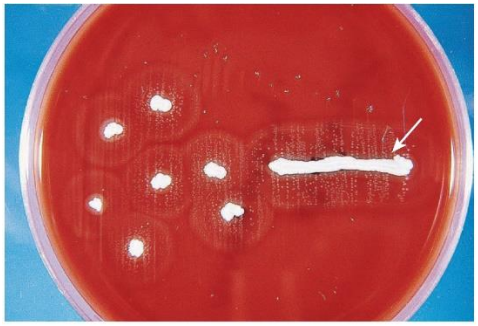

FIGURE 24-3 Satellite phenomenon. *Staphylococcus aureus* excretes nicotinamide adenine dinucleotide (NAD, or V factor) into the medium, providing a growth factor required for *Haemophilus influenzae* (small colonies surrounding *S. aureus* colonies *[arrow]*).

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Lecture 18: All are opportunistic pathogens

Bacteria	Description	Virulence Factors (VF)	Diseases	Notes
Pseudomonas (rods) **P. aeruginosa** (most important pathogen of the group which has intrinsically resistant to many antibiotics)	Motile, straight or slightly curved, aerobic, oxidase + rods typically arranged in pairs **P. aeruginosa** smells fruity, "grape-like" ,"fresh-tortilla" & Produces blue-green pigment pyocyanin. **Pyocyanin** catalyzes the production of superoxide and hydrogen peroxide, toxic forms of oxygen. This pigment also stimulates interleukin (IL)-8 release, leading to enhanced attraction of neutrophils	***Adhesins** : (1) flagella, (2) pili, (3) lipopolysaccharide (LPS), and (4) **alginate** (a mucoid exopolysaccharide that forms a prominent capsule) ***Toxins and Enzymes :** **1. Exotoxin A** (disrupts protein synthesis by blocking peptide chain elongation in eukaryotic cells). **2. Two elastases, LasA (serine protease) and LasB (zinc metalloprotease),** act synergistically to degrade elastin, related to lung parenchymal damage. **3. Phospholipase C** is a heat-labile hemolysin that breaks down lipids and lecithin, facilitating tissue destruction **4. Exoenzymes S** and **T** are extracellular toxins facilitating bacterial spread, tissue invasion, and necrosis.	***Pulmonary Infections**: asymptomatic colonization (in patients with Cystic Fibrosis and other chronic lung diseases) or benign inflammation of the bronchials (tracheobronchitis) to severe necrotizing bronchopneumonia. >> Previous therapy with broad-spectrum antibiotics and use of mechanical ventilation equipment predispose to infection. ***Skin and Soft-Tissue Infections:** burn wounds (most recognized) & Folliculitis (assosiated with immersion in contaminated water e.g. hot tubs) ***Urinary Tract Infections:** assosiated with long-term indwelling urinary catheters. ***Ear Infections:** range from external otitis ("swimmer's ear" so swimming is an important risk factor) to invasive destruction of cranial bones. ***Eye Infections :** Occur after initial trauma to the cornea (e.g., abrasion from contact lens). ***Bacteremia and Endocarditis:** characterized by ecthyma gangrenosum. -Mortality rate in affected patients is higher with P. aeruginosa.	*has **broad environmental distribution** because it can use many organic compounds as sources of carbon and nitrogen. *Members of the genus are found in soil, decaying organic matter, vegetation, and water. Also found throughout the hospital environment. And is resistant to many antibiotics and disinfectants. *infections are **primarily opportunistic.** *The underlying conditions required for most infections: (1) the presence of the organism in a **moist reservoir** and (2) **compromised host defenses** (e.g., cutaneous trauma, elimination of normal microbial flora as a result of antibiotic usage, neutropenia) ***Identification & diagnosis:** The presence of **cytochrome oxidase** (detected in a rapid 5-minute test) differentiate them from Enterobacteriaceae**.** ***Treatment: combination of antibiotics for serious infections.** **-antimicrobial therapy for *Pseudomonas* infections is frustrating** because the bacteria are typically resistant to most antibiotics and the infected patient has compromised immune defences ***Antibiotic resistance gained by** (the low rate of movement of antibiotics through the outer membrane pores into the bacterial cell, combined with the rapid efflux of antibiotics with efflux pumps . Also acquired and adaptive resistance)
Burkholderia **B. cepacia complex, B. gladioli, & B. pseudomallei**			**1. B. cepacia complex:** **a- Pulmonary infections:** Patients particularly susceptible to pulmonary infections with *B. cepacia* complex and *B. gladioli* are those with Cystic fibrosis, in whom infections can progress to significant destruction of pulmonary tissue. **b- Opportunistic infections:** UT infections (in catheterized patients, bacteremia (in	* colonize a variety of moist environmental surfaces and are **opportunistic pathogens** >> like *P. aeruginosa* ***pseudomallei** is of public health importance in **endemic areas**, particularly in northeast Thailand, Vietnam, and northern Australia, causing **melioidosis.**

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			immunocompromized patients with contaminated IV catheters) **2. B. pseudomallei:** asymptomatic colonization to abcess formation & Melioidosis	*__Transmission of Melioidosis:__ contracted through **direct contact** with contaminated soil and surface waters. ***Melioidosis Occupation period:** range from one day to many years; generally symptoms appear **two to four weeks after exposure**.
Acinetobacter **(coccobacilli)**	*strictly aerobic, oxidase-negative, plump coccobacilli.		**respiratory tract & pulmonary infections** (in patients receiving respiratory therapy), **UT infections**, & **wound infections** (traumatic;military conflicts, &nosocomial wounds) ; they also cause **septicemia** ** **A. baumannii** (frequent cause of hospital-acquired pneumonia, especially of late-onset, **ventilator-associated pneumonia)**	***opportunistic pathogens**
Moraxella **M. catarrhalis**	strictly aerobic, oxidase-positive, diplococci.		* Over the last 20 to 30 years, the bacterium has emerged as a genuine pathogen and is now considered an **important cause of upper respiratory tract infections** (in otherwise healthy children and elderly people) ***important cause of lower respiratory tract infections** (in adults with chronic obstructive pulmonary disease (COPD))	* The **peak rate of colonisation** by *M. catarrhalis* appears to occur around **2 years of age,** with a striking difference in colonization rates between children and adults (very high to very low). * ***M. catarrhalis*** is a nosocomial pathogen
Legionella **(Rods)** **L. pneumophila** (cause of 90% of all Legionella infections)	*Slender, pleomorphic rods *obligatively aerobic and nutritionally fastidious (They require media supplemented with **cysteine).**	Legionellae are **facultative intracellular bacteria (** infect and replicate in **macrophages and amoeba**). Cytokines released by the infected macrophages stimulate a **robust inflammatory response** that is characteristic of infections with Legionella	***L. pneumophila*** affects **the lungs** and present in one of two forms : (1) an influenza-like illness (referred to as **Pontiac fever** (self-limited, febrile illness) (2) a severe form of pneumonia (i.e., **legionnaires disease**). Legionnaires disease is characteristically more severe and, if untreated, promptly causes considerable morbidity.	***Legionella** acquired its name after an outbreak of a then-unknown "mystery disease" sickened 221 persons, causing 34 deaths, at a convention of the American **Legion**. *__Transmission:__ Human infections are most commonly associated with **exposure to contaminated aerosols** (e.g., air conditioning cooling towers, whirlpool spas, showerheads, water misters). *The organisms can survive in moist environments for a long time. *The medium most commonly used for the isolation of legionellae is **buffered charcoal yeast extract (BCYE) agar**

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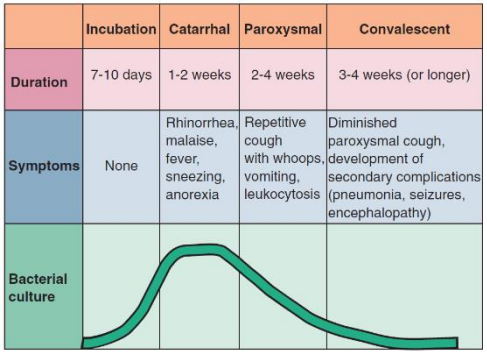
Bordetella **(Coccobacillus)** **B. pertussis**	extremely small (0.2 to 0.5 × 1 µm), fastidious, strictly aerobic, coccobacillus.	***Pertussis toxin** (A-B toxin) inactivates the protein that controls adenylate cyclase activity, ↑ cAMP levels and a subsequent ↑ in respiratory secretions and mucus production. *The bacteria attach to the **cilia of the respiratory epithelial cells**, produce toxins that paralyze the cilia, and cause inflammation of the respiratory tract, which interferes with the clearing of pulmonary secretions.	**pertussis** or whooping cough **Pertussis: a toxin-mediated human disease (human is the only reservoir).**  FIGURE 29-3 Clinical presentation of *Bordetella pertussis* disease.	***Transmition:** mainly by respiratory droplets. *Even under ideal conditions, recovery of *B. pertussis* in culture is difficult. ***vaccines** contain inactivated **pertussis toxin**, **filamentous hemagglutinin**, and **pertactin** -Since widespread use of the vaccine began, incidence has decreased more than 75% compared with the pre-vaccine era. ***incubation period: 7-10 days.** * In the summer of 1976, public attention was focused on an outbreak of severe pneumonia that caused many deaths and the clearance of mucus is impaired. This stage is characterized by the **classic whooping cough paroxysms**

**The oxidase test is used to identify bacteria that produce cytochrome c oxidase, an enzyme of the bacterial electron transport chain. (note: All bacteria that are oxidase positive are aerobic, and can use oxygen as a terminal electron acceptor in respiration. This does NOT mean that they are strict aerobes. Bacteria that are oxidase-negative may be anaerobic, aerobic, or facultative; the oxidase negative result just means that these organisms do not have the cytochrome c oxidase that oxidizes the test reagent. They may respire using other oxidases in electron transport.)

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Lecture 19:

Bacteria	Description	Virulence Factors (VF)	Diseases	Transmission	Notes
Campylobacter (Rods) (**C. jejuni** responsible for most infections**)**	Small (0.2 to 0.5 µm wide and 0.5 to 5.0 µm long), motile, curved, oxidase +, catalase + rods	**lipooligosaccharides** (LOSs lack O-antigen in LPS)	**the most common cause of bacterial gastroenteritis** *most commonly > **acute enteritis** with **diarrhea** (stools may be bloody on gross examination), **fever**, and **severe abdominal pain**. *Uncommon> **Guillain-Barré syndrome & reactive arthritis** are well-recognized complications. ((Probably through molecular mimicry)) ***C. jejuni*** GI disease characteristically produces histologic damage to the mucosal surfaces of the jejunum and other parts of the intestine.	*zoonotic infections with a variety of animals serving as reservoirs. *Contaminated poultry are responsible for more than half of the infections in developed countries. *Uncommon to be transmitted by food handlers.	***Microaerophilic:** grow best in an atmosphere of reduced O2 (5% to 7%) and increased CO2 (5% to 10%) *C. jejuni* grows better at 42°C than at 37°C. *The organisms are killed when exposed to gastric acids, (so conditions that decrease or neutralize gastric acid secretion favor disease. ***Identification & diagnosis:** A **presumptive identification** of isolates is based on **growth under selective conditions**, typical **microscopic morphology**, and positive oxidase and catalase tests. *Some statistics: Microbiological findings among US emergency department patients presenting with 549 episodes of bloody diarrhea at 11 *Emerg*ency ID NET sites.
Helicobacter (rods) **H. pylori is responsible for 85% of the gastric ulcers and 95% of the duodenal ulcers.**	spiral rods resembling campylobacters All gastric helicobacters, including *H. pylori,* are **highly motile** (corkscrew motility) and produce an abundance of **urease**	***urease** *urease byproducts that mediate localized tissue damage: **mucinase, phospholipases,** & **vacuolating cytotoxin A** (VacA> damages epithelial cells by producing vacuoles) ***cytotoxin-associated gene *(cagA)*** interferes with the normal cytoskeletal structure of the epithelial cells	*Colonization with *H. pylori* invariably leads to **gastritis** **- acute gastritis**: a feeling of fullness, nausea, vomiting, and hypochlorhydria. **- chronic gastritis:** disease confined to the gastric antrum or involve the entire stomach >>Chronic gastritis will progress to peptic **ulcers**. The ulcers develop at the sites of intense inflammation, (the junction between the corpus and antrum **(gastric ulcer)** or the proximal duodenum **(duodenal ulcer)**) *Chronic gastritis increases the risk of **gastric cancer and MALT lymphoma (mucosa-associated lymphoid tissue B-cell lymphomas)**	fecal-oral route. ((Humans are the primary reservoir for H. pylori, and colonization is believed to persist for life unless the host is specifically treated))	*Growth of H. pylori and other helicobacters requires a **complex medium** in **microaerophilic** conditions. *H. pylori adheres to gastric mucosa and is usually not recovered in stool or blood specimens *H. pylori adapt to the acidic conditions of the stomach by using their motility, chemotaxis, urease production, and other mechanisms. It also colonizes a narrow-protected niche near the surface of epithelial cells ***Identification & diagnosis:** *Since H. pylori* adheres to gastric mucosa , *H. pylori* can be detected by **histologic examination of gastric biopsy** specimens, but identification is usually done by non-invasive methods, A number of **polyclonal and monoclonal immunoassays for *H. pylori* antigens excreted in stool** have been developed and demonstrated to have sensitivities and specificities exceeding 95%.

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Bartonella (coccobacillary or bacillary rods) **B. henselae, B. bacilliformis, & B. Quintana**	coccobacillary or bacillary rods with fastidious growth requirements	**facultative intracellular bacteria**	**B. henselae** causes **cat-scratch disease**, 1–3 weeks after inoculation (acquired after exposure to cats (e.g., scratches, bites, contact with the contaminated feces of cat fleas)) Symptoms>> typically include a non-painful bump or blister at the site of injury & painful & swollen lymph nodes **B. bacilliformis** causes **Carrion's disease** **B. Quintana** causes **trench fever**	transmitted by vectors such as ticks, fleas, sand flies, and mosquitoes	Incubation period: prolonged (2 to 6 weeks).
Rickettsiaceae (Rods)	**aerobic rods**	**Obligate intracellular bacteria** (The primary clinical manifestations appear to result from the **replication of bacteria in endothelial cells**, with subsequent damage to the cells and leakage of the blood vessels)		transmitted by ectoparasites such as fleas, lice, mites, and ticks. So,, the distribution of rickettsial diseases is determined by the distribution of the arthropod host/vector.	*grow only in the cytoplasm of eukaryotic cells. *Seen best with Giemsa stain. *Rickettsia is subdivided into the **spotted fever group** and the **typhus group** *All age groups are at risk for rickettsial infections during travel to endemic areas.

Rickettsiaceae type	Disease	Reservoir	vector	distribution	Incubation period	Clinical presentation	Rash	Eschar	Mortality without treatment %
R. rickettsii	Rocky mountain spotted fever	Ticks, wild rodents	Hard ticks (dog tick, wood tick)	Western Canada, continental US, Mexico, Panama, Argentina, Brazil, Bolivia, Colombia, Costa Rica	7	Abrupt onset; fever headache, malaise, myalgias, nausea, vomiting, abdominal pain	>90%; macular; centripetal spread	No	10-25
R. prowazekii	Epidemic (louse-borne) typhus	Humans	Human body louse	Mountainous regions of Central & Eastern Africa (Burundi, Rwanda, Ethiopia), Central & South America, Asia.	8	Abrupt onset; fever headache, chills, myalgias, arthralgia	20-80%; macular; centrifugal spread	No	20

Tickborne spotted fever rickettsioses are the most frequently reported travel-associated rickettsial infections, while Epidemic typhus caused by *R. prowazekii* infection is rarely reported among tourists but can occur in impoverished communities and refugee populations where body lice are prevalent.

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Lecture 20: All are related to sexual transmitted disease (STD)

Bacteria	Description	Diseases	Transmission	Notes
Treponema **T. pallidum** (The most important treponemal species that causes human disease; the causative agent for Syphilis)	thin, helical (**0.1 to 0.5** × 5 to 20 µm),bacterial **Spirochetes** (**too thin** to be seen with light microscopy so traditional diagnostic tests such as gram stain and microscopy are of little value)	***Syphilis** (common STD found world wide) - Patients infected with syphilis are at **increased risk for transmitting and acquiring HIV** when genital lesions are present - Syphilis undergoes 3 phases: **1. primary phase** is characterized by skin lesions **(chancres)** at the site where the spirochete penetrated **2. secondary phase,** the clinical signs of disseminated disease appear, (e.g. **skin lesions** over the entire body, fever, headache). Symptoms resolve within weeks. **3. Late syphilis** severely damages organs involved (e.g., **neurosyphilis**, cardiovascular syphilis) leading to various symptoms (e.g. **dementia** or **blindness**) **If the patient is not treated, syphilis cause **systemic devastating damage**. ***Between 2000 and 2012, the incidence of newly acquired disease has **increased** each year.	*The most common route of spread is by **direct sexual contact.** *Syphilis **cannot be spread through contact with inanimate objects** such as toilet seats (since the bacteria is very labile to drying and disinfectants) . *Other routes include **congenitally** (from an infected mother**)** or by **transfusion** with contaminated blood.	***T. pallidum has not been cultured regularly in vitro because they are **dependent on host cells** for many metabolites (e.g. purines, pyrimidines, amino acids). Moreover, they're **extremely sensitive to oxygen** (microaerophilic or anaerobic) ***Identification & diagnosis:** -**darkfield microscopy** or **immuno-fluorescent stains** must be used for visualization and diagnosis. -**For diagnosis of syphilis**, serology is the most important tool, with tests like ***Treponema pallidum* particle agglutination (TP-PA) test** (Gelatin particles sensitized with *T. pallidum* antigens are mixed with dilutions of the patient's serum. If antibodies -against *T. pallidum*- are present, the particles agglutinate, indicating the patient has been infected and developed antibodies)**.** ***Treatment:** Syphilis be controlled only through the practice of **safe-sex** techniques and adequate **treatment with antibiotics**.
Borrelia	**Spirochetes**	**Lyme disease** and **relapsing fever** In untreated patients > hematogenous dissemination will occur within days to weeks of the primary infection. This stage is characterized by systemic signs of disease (e.g., severe fatigue, headache, fever, malaise). *60% of patients with untreated Lyme disease will develop **arthritis** *Tickborne diseases can have similar signs & symptoms: fever/chills, aches & pains, rash	*Hard ticks are the major vectors for lyme disease. *The ticks contaminate the bite wound with borreliae present in saliva or feces.	*stain well with dyes such as Giemsa and stain poorly with gram stain, but have an outer membrane similar to **gram-negative** bacteria ***Identification & diagnosis:** Because culture is generally unsuccessful, diagnosis of diseases caused by borreliae is by serology (**Lyme disease**) or microscopy (**relapsing fever**).
Chlamydia **most common bacterial STDs in humans & the leading**	**0.3 µm in diameter, with a unique life cycle**	***Trachoma** (**leading cause of preventable blindness):** chronic inflammatory granulomatous process of eye surface, leading to corneal ulceration, scarring, pannus formation and blindness. __Infections occur predominantly in children, who are the chief reservoir of *C. trachomatis* in endemic areas.	*Eye-to-eye transmission of trachoma is by **droplet, hands, contaminated clothing**, and flies that	*Unlike other bacteria, it has a unique developmental cycle, forming metabolically inactive **infectious** forms **(elementary bodies [EBs])** and metabolically active **noninfectious** forms **(reticulate bodies [RBs]).**

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cause of infectious blindness worldwide.			***Urogenital infections:** -Most genital tract infections in women are asymptomatic (80%) -Most *C. trachomatis* genital infections in men are symptomatic, 25% of the infections will be inapparent. -It can cause cervicitis in women and urethritis & proctitis in both men and women.	transmit ocular discharges from the eyes of infected children to the eyes of uninfected children. *Infects epithelial cells, which are found on the mucous membranes of the urethra, endocervix, endometrium, fallopian tubes, anorectum, respiratory tract, and conjunctivae.	**Virulence factor: Obligate intracellular parasites** **(**they use host cell ATP for their energy requirements & damage is thought to be caused by intracellular replication and destruction of infected cells upon release) ***Identification & diagnosis:** *C. trachomatis* infection can be diagnosed (1) on the basis of cytologic, serologic, or **culture** findings, (2) through the direct **detection of antigen** in clinical specimens, and (3) through the use of **nucleic acid–based** tests.
Neisseria **(Coccoid)**		*aerobic, coccoid shaped (0.6 to 1.0 µm in diameter) bacteria arranged in pairs (diplococci) *All are oxidase + & most are catalase + *Both strains are strictly human pathogens. *Gonococci attach to mucosal cells, penetrate into the cells and multiply, and then pass through the cells into the subepithelial space where infection is established.			***Identification & diagnosis:** properties that combined with the **Gram stain morphology** allow a **rapid, presumptive identification** of a clinical isolate .
1	**N. gonorrhoeae** **(Gonorrhea 2nd most commonly reported STD in the US after chlamydia)**	*fastidious (*only grows on enriched chocolate agar and other supplemented media)	***Gonorrhea** -Genital infection in men is primarily restricted to the urethra (purulent urethral discharge and dysuria) > all infected men have acute symptoms (symptomatic) -half of all infected women have mild or asymptomatic infections ***Gonococcemia** : Disseminated infections with septicaemia and infection of skin and joints (occur in 1% to 3% of infected women and in a much lower percentage of infected men)		*N. gonorrhoeae* in a clinical specimen is always considered significant. *incubation period: 2-5 days. ***Virulence factors:** **Pilin, Por protein, Opa protein, Rmp protein, transferrin- lactoferrin- & haemoglobin- binding proteins, LOS, IgA1 protease, β-lactamase**
2	**N. meningitidis**		***Meningitis:** begins abruptly with headache, meningeal signs, and fever; however, very young children may have only nonspecific signs such as fever and vomiting. ***Meningococcemia** *(*Septicaemia) with or without meningitis *Meningococcal disease occurs in patients who lack specific antibodies directed against the polysaccharide capsule and other expressed bacterial antigens: **(1)** children younger than 2 years (antibodies from the mother are disappearing) **(2)** Patients with deficiencies in C5, C6, C7, or C8 of the complement system (6000-fold greater risk) **(3)** Post-splenectomy patients		*N. meningitidis* can colonize the **nasopharynx** of healthy people without producing disease (not significant in clinical specimen) *ability to cause disease is determined by antigenic differences in the polysaccharide capsule *Meningitis mortality approaches 100% in untreated patients.

☺ All bacteria in this file are Gram Negative bacteria

Lecture 21: Mycoplasma

Mycoplasma ***M. pneumoniae* is a strict human pathogen.**	*Don't have a cell wall & their cell membrane contains sterols *pleomorphic shapes varying from 0.2 to 0.3 µm coccoid forms to rods 0.1 to 0.2 µm in width and 1 to 2 µm long.	***M. pneumoniae** infects respiratory tract; typically results in **asymptomatic carriage** but can cause: **Respiratory disease** occurs worldwide throughout the year.**:** -**tracheobronchitis** (most common) -**pneumonia**: referred to as primary **atypical pneumonia** can also develop, with a patchy bronchopneumonia seen on chest radiographs ***M. genitalium** infects genitourinary tract & can cause **nongonococcal urethritis** (NGU) and **pelvic inflammatory disease**.		**(Mycoplasma and Ureaplasma organisms are the smallest free-living bacteria).** They are unique among bacteria because they do not have a cell wall and their cell membrane contains sterols. **Absence of the cell wall renders the mycoplasmas resistant to penicillins, cephalosporins, vancomycin, and other antibiotics that interfere with synthesis of the cell wall. ***Identification & diagnosis:** -The most sensitive diagnostic tests are **PCR amplification** tests of species-specific gene targets. -(**Microscopy** is of **no diagnostic value** because mycoplasmas stain poorly with the Gram stain. Likewise, antigen tests have poor sensitivity and specificity and are not recommended.)

Table 23-2 Virulence Factors in *Neisseria gonorrhoeae*

Virulence Factor	Biological Effect
Pilin	Protein that mediates initial attachment to nonciliated human cells (e.g., epithelium of vagina, fallopian tube, and buccal cavity); interferes with neutrophil killing
Por protein	Porin protein: promotes intracellular survival by preventing phagolysosome fusion in neutrophils
Opa protein	Opacity protein: mediates firm attachment to eukaryotic cells
Rmp protein	Reduction-modifiable protein: protects other surface antigens (Por protein, lipooligosaccharide) from bactericidal antibodies
Transferrin-, lactoferrin-, and hemoglobin-binding proteins	Mediate acquisition of iron for bacterial metabolism
LOS	Lipooligosaccharide: has endotoxin activity
IgA1 protease	Destroys immunoglobulin A1 (role in virulence is unknown)
β-Lactamase	Hydrolyzes the β-lactam ring in penicillin

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