

	Causative Agent	Signs/Symptoms	Pathogenesis	Epidemiology	Other
Streptococcal Pharyngitis (Strep Throat)	<i>Streptococcus pyogenes</i> gram +, grows in chains, beta-hemolysis of blood agar	Sore throat, difficulty swallowing, fever, throat is red with patches of pus, tiny hemorrhages	-M protein important adhesion -protein F adherence -DNase, hyaluronidase, proteases degrade intracellular connections -streptokinase breaks blood clots -avoidance of host immune system	-Naturally only infects humans -spread by respiratory droplets	Post-Streptococcal Sequelae -acute rheumatic fever (3 wks) -acute post-streptococcal glomerulonephritis (10 days)
Diphtheria	<i>Corynebacterium diphtheriae</i> gram+, release diphtheria toxin (exo), gene carried by specific lysogenic bacteriophage, isolated on selective medium containing K-tellurite	Mild sore throat, slight fever, extreme fatigue, malaise, swelling of neck, formation of pseudomembrane on tonsils and throat, heart and kidney failure and paralysis may occur	-Results from potent diphtheria exotoxin -inhibits protein synthesis -bacterium is not very invasive	-Humans are primary reservoir -spread by air -acquired via inhalation or from fomites	Treatment and prevention: -injection of antiserum -delaying treatment can be fatal -antibiotics can clear <i>C. diphtheriae</i> , but toxins already absorbed unaffected -Immunization very effective with childhood DTaP
Pneumococcal Pneumonia	<i>Streptococcus pneumoniae</i> gram+ diplococcus known as pneumococcus, thick polysaccharide capsule necessary for virulence	1-2 days of runny nose, congestion, rise in temp.; cough, fever, chest pain, sputum production, rust colored sputum (from blood), severe chest pain, shallow rapid breathing	Encapsulated pneumococci inhaled into alveoli multiply rapidly, cause inflammatory response	-up to 30% of people carry encapsulated pneumococci in throat -mucociliary escalator effectively keeps from reaching lungs -risk of infection increases when this defense is impaired	
Pertussis (Whooping Cough)	<i>Bordetella pertussis</i> encapsulated aerobic gram- rod	Three stages 1.catarrhal stage (inflammation of mucous membranes) 1-2 weeks of upper respiratory signs 2.paroxyssmal stage (repeated sudden attacks)	1.cells are inhaled, attach to ciliated cells of respiratory epithelium 2.colonize nasopharynx, trachea, bronchi, and bronchioles 3.release 3 toxins -pertussis toxin (increased mucus,	-highly contagious -spread via air droplets -classically disease of infants; milder in older child/adults -2010 outbreak in CA killed 10 infants	-vaccination-preventable disease, but still endemic including US

		<p>violent uncontrollable coughing, dry but severe, small BV's may rupture, forceful inhalation of air="whoop" sound</p> <p>3.convalescent stage (not contagious, coughing decreases)</p>	<p>decreased killing ability of phagocytes)</p> <ul style="list-style-type: none"> -adenylate cyclase (lyses leukocytes) -tracheal cytotoxin (causes release of fever inducing interleukin-1; also toxic to ciliated epithelial cells 4.increased mucus production 5.some bronchioles obstructed, small areas of collapsed lung 6. cause of death is pneumonia or other secondary infection 		
Influenza	<p>Three major virus types bases on protein coat</p> <ol style="list-style-type: none"> 1.Type A - most serious disease 2.Type B - less extensive/severe 3.Type C - minor <p>-orthomyxovirus family</p>	<p>About 2 day incubation period</p> <ul style="list-style-type: none"> -headache, fever, sore throat, muscle pain, peaks in 6-12 hours, worsens over a few days -acute symptoms last about one week 	<ol style="list-style-type: none"> 1.inhalation of droplets or transfer to eyes, nose form fomites 2.mature virions bud from host cells, spread 3.infected cells die, slough off 4.individual susceptible to secondary respiratory infections 	<ul style="list-style-type: none"> -most deaths due to bacterial infection -epidemics annually; pandemics periodically -Antigenic drift - seasonal influenza -Antigenic shift - uncommon, causes pandemic influenza 	
<i>Helicobacter pylori</i> Gastritis	<p><i>Helicobacter pylori</i></p> <p>gram-microaerophile with multiple flagella</p>	<ul style="list-style-type: none"> -most asymptomatic -peptic ulcers of stomach, may produce localized abdominal pain, tenderness, bleed, stomach cancer may develop 	<ol style="list-style-type: none"> 1. can survive acidity of stomach -produces urease, make ammonia from urea 2.produce CagA, injects host cells, change shape/surface 3.VacA acts on mucosal cells to promote flow of urea 4.damage to epithelial cells and inflammatory response result in decreased mucus production 5.infectios persist for years 	<ul style="list-style-type: none"> -transmitted via fecal-oral route -bacteria found in well water -flies may transmit from feces 	<p>Treatment and Prevention:</p> <ul style="list-style-type: none"> -antibiotics plus medication to inhibit acid production

Mumps	Mumps virus; enveloped, single stranded RNA virus	-onset marked by fever, loss of appetite, headache -then painful swell of parotid glands -spasm of underlying muscle making talking, chewing hard -meningitis may occur	1.virus inhaled via saliva droplets 2.symptoms begin in 15-21 days 3.virus multiplies in parotid salivary glands, inflammatory response yields swelling, pain, may spread in blood	-humans only natural host -one type of virus, confers lifelong immunity -virus spreads 1-2 weeks after symptoms appear	-once common in US prior to routine vaccination -now rare but outbreaks occur due to waning immunity in college students and other young adults
Cholera	<i>Vibrio cholerae</i> ; curved gram- rod, halotolerant, can grow in alkaline conditions	-severe watery diarrhea -can amount to 20 liters/day -"rice water stool" appearance -severe muscle cramps result from loss of fluids and electrolytes. -severe dehydration can lead to organ failure/death	1.sensitive to acid; large #s must be ingested 2.adhere to epithelial cells of small intestine, establish infection, produce cholera toxin 3.toxin encoded by bacteriophage; lysogenic conversion 4.causes cells to secrete Cl ion; water follows the salts, yielding diarrhea	-common source = fecally contaminated water (aquatic food implicated) ->1 million <i>V. cholerae</i> cells in each ml of feces -relatively common worldwide, few cases in US -new <i>V. cholera</i> O139 strain has capsule	
Shigellosis	4 species of <i>Shigella</i> ; gram- rods; <i>S. dysenteriae</i> most virulent, <i>S. sonnei</i> least virulent	-usually dysentery (blood, pus in feces) -headache, vomiting, fever, stiff neck, convulsions, joint pain	1.takes advantage of antigen sampling of M cells 2.multiply inside macrophages, escapes 3.attach and induce uptake by epithelial cells, multiply, spread causing actin polymerization to propel 4.invasion results in death, sloughing of epithelium	-humans only -fecal-oral route -small infectious dose since not easily killed by stomach acid -spreads rapidly in populations with poor sanitation -fecally contaminated food/water cause outbreaks	- often fatal for infants in developing countries -some strains produce potent Shiga toxin, enters bloodstream, responsible for hemolytic uremic syndrome (HUS)