Microbiology and Immunology 2500 A/B: Bacteriology Tables

Introduction

Dear student,

This document contains content derived from the **bacteriology unit** of the Microbiology & Immunology 2500 course. Information has been condensed into organized tables to help students categorize the content presented in lectures. This resource has been created by the Education Team at Webstraw. The Education Team consists of students that have previously taken and/or students that are currently taking Microbiology 2500 A/B

Purpose

This resource focuses on key concepts that are important for students to understand to succeed within this course. This resource was created by students for other students. Our goal is to help students (1) further develop their understanding of course content and (2) achieve greater academic success. (3) Our resource is also open access meaning there are no financial or legal barriers to students who wish to access and use our resource.

Instructions

Before the exam, we recommend that you attempt to familiarize yourself with all the content covered in the bacteriology unit. This document is a supplementary resource used to help organize all the species of bacteria in the bacteriology unit, separated by lecture.

A table of contents is provided to show where each lecture topic is located on this document.

Disclaimer: This resource is supplementary to your course content and is not meant to (1) replace any of the resources provided to you by your instructor nor is it meant to (2) be used as a tool to learn the course material from scratch. We assume that students who use this resource will have a basic understanding of the course content. This resource does not contain everything you need to know for your evaluations. Please refer to the course material provided by your instructors if there are any discrepancies between our resource and your course content.

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We wish you the best of luck on your exams!

- The WebStraw Team

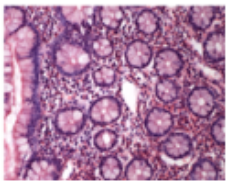
Note to Instructors:
If this resource has been created for your course and you would like to collaborate with us, please email us at team@webstraw.ca

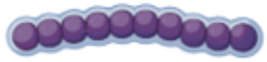
Table of Contents

Meningitis

From the "Meningitis" lecture, it covers all the bacteria species involved in this lecture

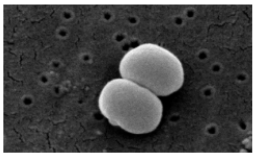
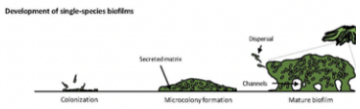
Bacteria Name	Type of Bacteria/Structure	Pathogenesis and Vectors	Disease and its Symptoms
Neisseria meningitidis	• Gram-negative, diplococcus  @BioRender • Produces a polysaccharide capsule	**Infection** 1. Colonizes the nose and throat 2. Enters the bloodstream and crosses the blood brain barrier 3. Endotoxin can lead to septic shock  @BioRender • Highly contagious • Can cause epidemics in boarding schools, military bases and dormitories • Only infects humans	**Meningococcal disease** • Asymptomatic carriage is common • Widespread blood clotting and ischemia • Clotting factors are used up → leading to small skin bleeds, resulting in purpuric rash • ***Serotype B*** is the most common invasive disease in Canada because the capsule is poorly immunogenic
Streptococcus pneumoniae	• Gram-positive • Cocci, grows in chains  @BioRender	• Commonly resides asymptomatically in the nasopharynx	**Pneumococcal disease** • Pneumonia • Ear infections • Sinusitis • It is the leading cause of bacterial meningitis in children >2 years old

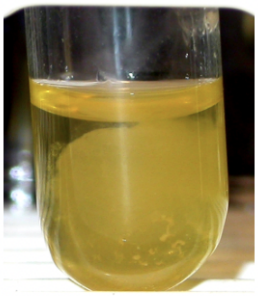
	• Produces a polysaccharide capsule		
Haemophilus Influenzae type B (Hib)	• Gram negative • Coccobacillus  @BioRender • Produces a polysaccharide capsule	• Meningitis condition usually following an upper respiratory infection, ear infection or sinusitis  @Wikimedia Commons	**Haemophilus B Meningitis** • Primarily causes meningitis in children under 5 years old • Before the Hib vaccine, *h. Influenzae* type B was the leading cause of bacterial meningitis
Listeria monocytogenes	• Gram positive rod  @BioRender	**Infection** 1. Listeria invades intestinal epithelial cells 2. Replicates within the cell's cytosol 3. Gastroenteritis 4. Blood infections 5. Meningitis • Can have growth at 4°C and is considered a ***food-borne pathogen*** • It is called a **psychrotroph** • Caused by eating contaminated foods	**Listeriosis** Range from 1. Meningitis 2. Bacteremia 3. Gastroenteritis • High rate of mortality in immunocompromised individuals including neonates and the fetus  @Wikimedia Commons

Streptococcus agalactiae	• Gram positive cocci • Grows in chains  @BioRender • Produces a capsule	**Infection** • Women can carry *S.agalactiae* within the urogenital tract • During baby delivery, *S.agalactiae* can get passed from the mother to the infant	**Group B Streptococcus** • Mothers tested positive for the bacteria will be treated with antibiotics to prevent the infection of the newborn

Staphylococci

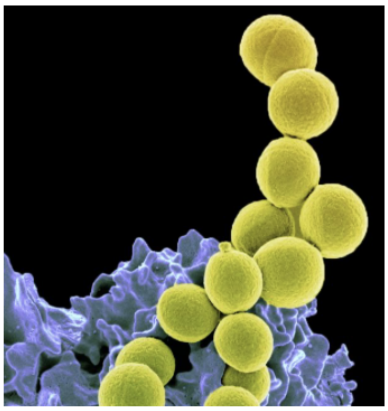
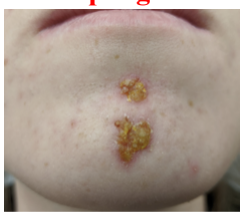
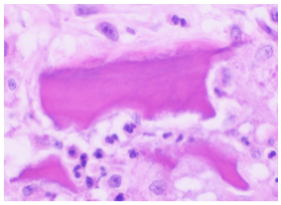
From the "Staphylococci" lecture, covering species other than S. aureus

Bacteria Name	Type of Bacteria/Structure	Pathogenesis / Mechanism of Action	Disease and its Symptoms
Staphylococcus epidermidis	• Gram positive • Cocci-shaped  @Wikipedia • Coagulase negative • Produces a polysaccharide capsule	**Biofilm Production** 1. Attaches to polymer surface 2. Bacteria adhere to each other, proliferates 3. Biofilm grows increasingly complex; resistant to antibiotics 4. Can detach once biofilm has maturated to repeat the cycle  @Wikipedia • Resistant to many antibiotics	**No associated disease** • Colonizes skin, but not as pathogenic as *S. aureus* • Biofilm production has consequences on implanted devices, such as catheters and medical prostheses
S. pseudintermedius	• Gram positive • Cocci-shaped • Coagulase positive	• Common colonizer of dogs **Causes canine pyoderma** (***pyo*** - pus, ***derma*** - skin) 1. Colonizes warm, moist areas of animal skin	**Canine pyoderma** • Transmission to humans can occur; but ***very rare***

	 @Wikipedia	2. Leads to pus-filled lesions • Resistant to many antibiotics • Infection may be a result of fleas	
S. lugdenesis	• Gram positive • Cocci shaped • 50/50 chance on coagulase positive; for the purposes of this course, they are ***coagulase positive*** (expresses clumping factors)	N/A	**N/A**
S. saprophyticus	• Gram positive • Cocci shaped • Coagulase negative	N/A	**N/A**

Staphylococcus aureus

From the "Staphylococci" lecture, covering the main features/diseases of S. aureus

Characteristics, Types, and Virulence Factors	Local Infections / Symptoms	Systemic Infections / Symptoms	Toxin-mediated / Symptoms
• Gram positive • Cocci-shaped • Coagulase positive • Gold-coloured  @Wikipedia • Generally an extracellular pathogen • 30% of population is persistently colonized; 20-30% transiently colonized • Colonizes skin and mucous membranes • Can spread via fomites or direct contact • Carriers of *S. aureus* tend to be asymptomatic **Virulence Factors** • Many factors = many diseases	**<u>Skin Lesions</u>** ***Abscess*** is the hallmark of *S. aureus* infection **Impetigo**  @Wikipedia **Non-bullous** (pimple-like lesions with pus; also caused by *Streptococcus pyogenes*) vs. **bullous** (painless, fluid-filled) **Ecthyma** (pus-filled sore, turns into a deep ulcer) **Stye**  @Wikipedia • Infection of the eye sebaceous glands (often drains on its own) **Furuncle (boil)**	These infections tend to be ***life-threatening*** • Bacteremia / sepsis • Pneumonia • Osteomyelitis • Endocarditis **Osteomyelitis**  @Wikipedia • Infection of bone or bone marrow • Sources of infection: hematogenous spread; fractures; joint replacement • May require open surgery and prolonged I.V. antibiotics • Remove dead bone	• Localized infection, systemic effects • Food poisoning • Toxic shock syndrome • Scalded skin syndrome **Staphylococcal food-borne illness** • Caused by enterotoxins; also act as superantigens • Toxin is ***pre-formed*** in food (does not require ingestion of actual *S. aureus*) • Symptoms: sudden onset nausea, projectile vomiting, stomach cramps, diarrhea • Tends to be self-limiting **Toxic shock syndrome (TSS)** • Acute onset of fever, hypotension, multi-organ dysfunction,

- Expression is regulated
- ***Surface*** virulence factors expressed during exponential growth
- ***Secreted*** virulence factors (aka **exotoxins**) expressed during stationary phase

Immune Evasion

- Has immune evasion mechanisms at every aspect of inflammatory response in the host

ex. **Staphylococcal protein A (SpA)**

- *S. aureus* surface protein
- Binds to Fc region of IgG immunoglobulins
- Antibodies bind to Fc receptor in the ***incorrect*** orientation
- Results in ***resistance to antibody-mediated phagocytosis***

Ex. **cytolytic toxins**

- Kill leukocytes (but also can lyse RBC → hence the name "**hemolysins**")
- Forms holes/pores in cell membrane to lyse
- Ex. **a-toxin, leukocidins**
- Helps protect *S. aureus* in abscesses and helps them spread

Can be **methicillin resistant (MRSA)**

HA-MRSA (hospital-associated)

@Wikipedia

- Infection of hair follicle

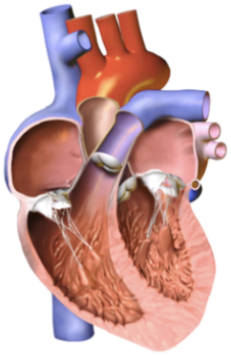
Carbuncles

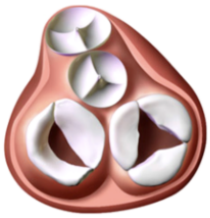
- infection of several hair follicles
- Coalescing furuncles

<u>Deep abscesses</u>

- More serious, but still localized
- Arise from wound or surgical infections
- Symptoms may not be obvious; more constitutional (whole body) → fever, chills, malaise
- Can become systemic

Infective Endocarditis





@Wikipedia

- Infection of heart valves
- Typically occurs on damaged or prosthetic heart valves
- Bacteria grow at large numbers → strokes, pulmonary embolisms
- Leads to fatigue, fever, heart murmurs, splinter hemorrhages

desquamation (sloughing of top of skin surface)

- Caused by both *S. aureus* and *Streptococcus pyogenes*
- Has menstrual and non-menstrual forms

Caused by **superantigens**

- Overactivates large numbers of T cells
- Leads to cytokine storm (systemic inflammatory response, overproduction)
- Results in vascular leakage → shock and organ failure
- **Toxin-1** causes **menstrual form**
- **Staph enterotoxins** are superantigens

Menstrual toxic shock syndrome

- Major cause: high absorbency tampons
- Affected healthy women, allowed bacteria to colonize and produce toxin-1
- Many affected women went through

- Found in health care settings, patients generally have co-morbidity

CA-MRSA (community-acquired)

- Hypervirulent, but still susceptible to some other antibiotics
- May cause necrotizing pneumonia
- Most common cause of skin and soft tissue infections (SSTI)

LA-MRSA (livestock-associated)

@Wikipedia

- Domestic livestock infected with *S. aureus* due to frequent contact with humans
- Ex. **Bovine mastitis** (infection of cow udder), **staphylococcosis** (in poultry)
- Exposure to antibiotics as growth factors for livestock leads to developed resistance
- Antibiotic-resistant strains can then be transmitted back to humans
- Due to increased antibiotic resistance, *S. aureus* are **superbugs**

amputation, disfigurement, etc.

Staphylococcal scalded skin syndrome

- Caused by **exfoliative toxin** (also causes bullous form), which are proteases that destroy host proteins that hold skin cells together in superficial layers
- Affects neonates
- Causes skin peeling
- **Self-limiting** → will heal in 1-2 weeks

Streptococcus pyogenes

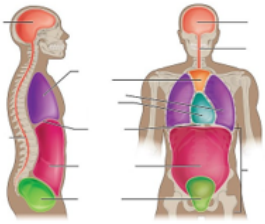
From the "Streptococci", covers all the bacterial species within this lecture

Characteristics, Types, and Virulence Factors	Virulence Factors	Local Infections / Symptoms	Systemic Infections / Symptoms
• Gram positive • **Human Specific →** many asymptomatic carriers • Grows in chains of cocci shaped bacteria  @BioRender • Non-Motile • Global burden of 500 000 deaths per year • Classification of all Strep based on blood hemolytic profile (**α, β, γ**) : • **α** - partial hemolysis (S. pneumoniae) • *β* - complete hemolysis (S.pyogenes) • **γ** - no hemolysis (Enterococcus species)	**Hyaluronic Acid** • Major polysaccharide for recognizing "self" • Makes bacteria look like human tissue → to hide • Blockage of opsonization **M-Protein** • Anti-phagocytic surface protein • Activates CD4-BP, usually to defend cells of the self • Serotypes based on N-terminal variance • Can be targeted for opsonization by designed antibodies **Hemolysins (S,O)** • Cleave blood cells 1. S is *β* hemolysis 2. O is O_2 labile (unstable in O2) **Superantigens (SPE)** • Triggers extreme immune response and T-cell activation (20% vs 0.01%)	**Impetigo** • **Non-bullous** Similar to the caused by *S. aureus* • **Ecthyma** (pus-filled sore, turns into a deep ulcer) • Most common in children **Pharyngitis** • ***"Strep Throat"*** • Common in teens and children • No cough • Swollen lymph nodes and skin rash • Viral infection can look similar, so take a rapid strep test to confirm	**Rheumatic Fever** • Post-infection "***sequelae***", 2-3 weeks after initial infection (pharyngitis) • **Autoimmune** - body attacks own M-proteins • Painful swollen joints and acute fever • Can lead to heart valve damage and endocarditis by other pathogens • Rare in developed world, endemic in developing world • 345 000 global deaths/year **Invasive Strep Disease** • Movement of bacteria to a normally sterile area • Into muscle = **necrotizing myositis** • Into soft tissue = **necrotizing fasciitis** • May lead to **Toxic shock syndrome** (toxin mediated) • Very serious and often

 @Wikipedia OR Lancefield classes (A, B, C)	• Triggers cytokine storm (toxic shock) • Effective only in humans, not mice (different MHC-II) • Functional T-cells are required for infection (counter intuitive to normal T-cell role) • No T-cell = not tropism • SpeA is the most crucial superantigen	Treat with: 1.***Beta-Lactams*** 2.***Erythomycin*** • Untreated leads to complications such as rheumatic fever and scarlet fever	deadly • Risk factors include: tissue injury, chickenpox, weakened immune system • 170 000 global deaths/year **Scarlet fever** • Often concurrent with strep throat • Children and teens are susceptible • Fever, strawberry tongue • Red rash across the chest that lasts 1 week • Mediated by scarlet fever exotoxins → same as streptococcal pyrogenic exotoxins • Can lead to rheumatic fever as a sequelae **Toxic Shock Syndrome** • Result of invasive strep disease • Overactivation of T-cells

Borrelia burgdorferi

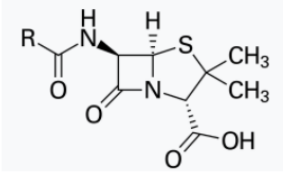
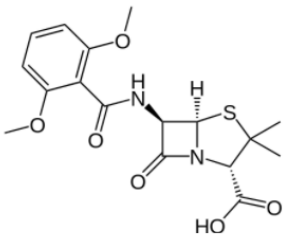
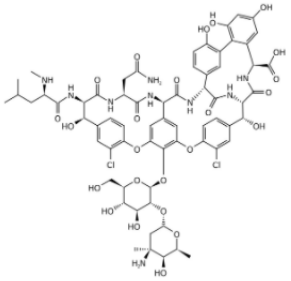
From the "Lyme Disease" lecture, covering the main features of B. burgdorferi and its associated disease

Bacteria Name	Type of Bacteria/Structure	Pathogenesis / Mechanism of Action	Disease and its Symptoms
Borrelia burgdorferi	• Gram-negative • Spirochete • Thin and long • Corkscrew shape  @BioRender • Periplasmic flagella (axial filaments) help for the movement required for invasion into matrix and vessels of hosts • No lipopolysaccharide • Contains lipoproteins to stick to the walls of the vessels • Linear chromosome with numerous plasmids	**Vector** • *Ixodes* species (ticks). Must acquire bacteria from reservoirs.  Kelly Summers et al, 2020 **Reservoir** • White-footed mice, squirrels, and other birds **Maturation cycle** • Infected, white-footed mouse transmits the bacteria to the larva of ticks • Further infect other white-footed mice when they become nymphs. • These nymphs that infect the mice then become adults. **Disease transmission** • Feeding tube inserts within the human (anesthetic release) → Blood sucked in and ticks become large and grey → Regurgitation of bacteria	**Lyme Disease** • Multisystemic  @Wikimedia Commons • Early-stage → Common bulls-eye rash around the area of infection  @Wikimedia Commons • Flu symptoms • Mid-stage → infections: swelling, meningitis, Bell's palsy • Long term → Arthritis, carditis, and nervous system problems • Was misdiagnosed early on as rheumatoid arthritis • Responds to antibiotics early on

		• No transmission in the first 24 hours, but after 24 hours transmission occurs • Tick saliva aids bacteria by decreasing many of the functions of dendritic cells **Infection** • Bacteria in skin → Spread around skin regions creating the bulls-eye rash → Entry into blood vessels → Entry into other tissues through flagella action and lipoprotein adhesion to vessel • Uses ***paracellular traversal*** mostly through the blood-brain barrier	• Potential for Chronic Lyme disease which does not respond to antibiotics

Antibiotics

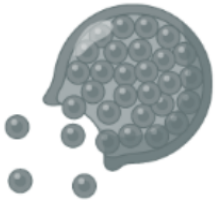
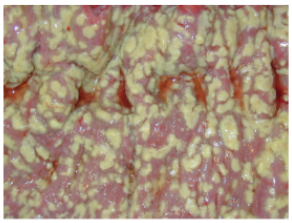
From the Antibiotics lecture, covering the types of antibiotics, Klebsiella pneumoniae and Clostridium difficile

Antibiotic Name/Class	Structure	Function	Resistance
Penicillin *B lactam*	 @Wikipedia • Contains a B lactam ring	• Inhibits cell wall synthesis • Binds **penicillin-binding proteins (PBPs)** → which disrupts cross-links and creates a weak cell wall leading to cell death	• Some bacteria can make a B lactamase, to cleave the B lactam ring
Methicillin *B lactam*	 @Wikipedia	• Inhibits cell wall synthesis • Cannot be cleaved by B lactamases	• Some bacteria can make a different PBP, which does not bind to B lactams
Vancomycin *Glycopeptide*	 @Wikipedia	• Inhibits synthesis of cell wall in Gram-positive bacteria • Binds D-Ala-D-Ala residues and inhibits transpeptidation	• Some bacteria have *van* genes that change residues to D-Ala-D-Lac so that vancomycin can no longer bind

Klebsiella and Clostridium

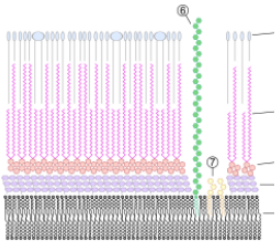
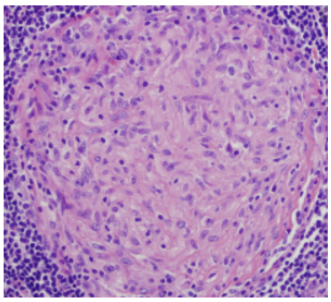
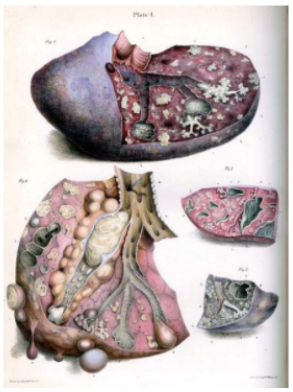
From the Antibiotics lecture

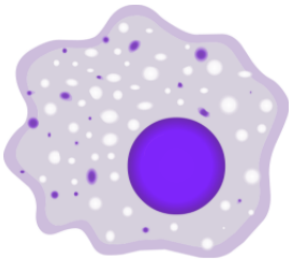
Bacteria Name	Type of Bacteria/Structure	Quick Facts	Disease and its Symptoms
Klebsiella pneumoniae	• Gram-negative, bacilli  @BioRender • Produces a capsule	• Resistant to carbapenem antibiotics  @BioRender • Produces a carbapenemase that destroys carbapenem antibiotics	**Nosocomial Pneumonia** • Also called hospital-acquired pneumonia • Cough with greenish phlegm • Fever and chills  @BioRender
Clostridium difficile	• Gram-positive  @BioRender • Bacilli, endospore-formers	• Asymptomatic carrier state in large intestine • Found in nursing homes and hospital environments • Hard to get rid of endospores from environment • Transmitted through spores: fecal-oral route • Produces A-B toxins	**Pseudomembranous colitis** • Inflammation of large intestine • Diarrhea, stomach pain, fever, nausea, dehydration • Yellow lesions on intestinal linings

	 @BioRender • Strict anaerobes → killed by O2	called **large clostridial cytotoxins** • A domain is active and inactivates important functions of host cell, B domain binds host cell to be taken up	 @Wikipedia

Mycobacteria

From the Tuberculosis and Leprosy Lecture, covers all the bacteria species within the lecture

Bacteria Name	Type of Bacteria/Structure	Pathogenesis and vectors	Disease and its Symptoms
Mycobacteria tuberculosis	• Gram-positive acid fast, rod-shaped • Intracellular pathogen (lives in macrophages) • Unusual cell envelope with lots of mycolic acids • Envelope is associated with resistance to antibiotics  @Wikipedia	1. Transmission via airborne droplets from an infected host 2. Phagocytosis of TB cells by alveolar macrophages and multiplication in macrophages 3. Infected macrophages form granulomas to stop spread → latent infection 4. Granuloma grows into airway allowing for transmission → active infection  @Wikipedia	**Extrapulmonary Tuberculosis** • Infection outside the lungs in another organ system • Often occurs in immunocompromised individuals **Pulmonary Tuberculosis** • Infection in lungs • Cough, fever, chills  @Wikipedia

Mycobacteria leprae	• Gram-positive acid fast, rod-shaped • Waxy cell envelope (mycolic acid) • Infects skin macrophages and Schwann cells  @Wikipedia	• Transmission not well understood, probably inhaled droplets • Treatable with antibiotics	**Tuberculoid Leprosy** • Macrophages contain the bacteria • Light-coloured lesions • Loss of hair and pigmentation **Lepromatous Leprosy** • Macrophages not activated • Nerve damage and loss of sensations • Loss of hair and pigmentation