BIOL4640/8646

usesO each Study Guide for Exam 2 to be Given on Wednesday, 10/28/26

A. Size and shapes of bacteria

B. External Structures of Bacteria

1. Flagella and motility

Arrangements observed

0-many, with being placed at one end, all over the cell, or at both ends of cells, and in spirochetes they can be in the periplasm and wrap around the cell.

Parts and features of flagella

4 Parts of a flagellum are the basal body, hook, filament, motor, and they can move by the motor rotating the flagellum at its base CW/CCW.

Order of assembly of flagellum.

M (Mcdonalds), S (sonic), R (resturants), P (powerrade), L (latte), H (Happy hour), F (fries), M (Matcha)
Filaments are built last, growing at tip, new flagellins pass through the hollow tube of the filament and add at tip.
Mot proteins is last step of flagellum assembly and put in the membrane, H+ flows through them (proton motive force) to help with motor to control motility.

Types of motility observed in bacteria – know how each mediated

6 types (3 flagella-mediated: periplasmic (wiggle, corkscrew), swimming (run + tumble), swarming (together) and 3 non-flagella-mediated: twitching (pili IV), gliding (unknown), sliding (lube)).
How the 3 non-flagella ones work: twitching = Type IV pili extend, attach, shorten → pull the cell; gliding = smooth across a solid surface, no flagella; sliding = passive, pushed by dividing cells, needs surfactant or slime (lube).

Swimming motility mediated by flagella

Single: CW/CCW to run, stop or turn the other way to tumble; many does all at once with CCW to run (one bundle), CW to tumble (bundle comes apart).

Periplasmic flagella in spirochetes

Flagella each pole in periplasm that wrap around cell, to do the wiggle corkscrew burrow.

Swarming motility

Across surface, raft of cells together, needs slime layer, flagella may be lateral.

Gliding motility

Non-flagella-mediated, mechanism not fully known (Flavobacterium: membrane proteins act like feet), smooth across a solid surface.

Twitching motility

Uses extend, attach, shorten, pull cell - uses Type IV pili.

Chemotaxis – what is it

Stimulus, receptor in membrane that senses attractant, runs to attractant (away from repellent) with flagella, create longer runs with fewer tumbles.

How one could determine if a bacterium is motile and if has flagella

By tests such as wet mount (purposeful movement), flagellar stain/EM (see flagella), motility agar (motile by cloudy), swarm plate (spread in circle), chemotaxis (attractants) plates, genome (flagellar genes), and closest related species through phylogeny (motile?).
Memory: Walking (purposeful walking, aka wet mount), Feet stain (flagellar staining), EM (see the flagella), Mobile agar (cloudy all through = motile), Swarm plate (spread in circle).

2. Pili and Fimbriae

a. Structure, arrangement in cells and assembly

b. Roles of Common pili, Sex pili and Type IV pili

c. Assembly of Type I and Type IV, how differs from flagellar

3. Other external structures

C. Cell envelope (glycocalyx, outer membrane, cell wall, and cell membrane)

1. Know the layers and components of the envelope in each of the following types of bacteria? How similar and different from each other

a. Gram positive bacteria

Cancer (capsule), Serotonin (S layer), Collagen (cell wall), Cell membrane – this one lacks an outer membrane.
Capsule is sugars + proteins, S layer is proteins, cell wall is THICK peptidoglycan (3-20 layers) + teichoic acids, cell membrane is phospholipids + proteins.

b. Gram negative bacteria

S layer – proteins, capsule – sugars, outer membrane (LPS (O-antigen (sugar), core polysaccharide (same within species), lipid A (inflammation)), porins, phospholipids), cell wall (periplasm watery, thin peptidoglycan 1-2 layers, periplasm), cell membrane with phospholipids + proteins.

c. Mycobacteria

Capsule (sugars + glycolipids), cell wall of mycolic acids → arabinogalactan (arabinose + galactose) → peptidoglycan, cell membrane of phospholipids + proteins.

d. Archaea

S layer – proteins, cell wall made up of pseudopeptidoglycan (NAG + NAT, β-1,3 linkage, all L-amino acids), cell membrane with isoprenoids (ether-linked, no phospholipids).

e. Mollicutes (Mycoplasma)

Compared to others, no cell wall, uses sterols to stabilize the membrane (phospholipids + sterols) and only a sugar layer.
Because it has no cell wall (no peptidoglycan), penicillin has nothing to act on.

How do scientists determine the structure of the envelope of bacteria?

By testing such as TEM (one thick dark layer = Gram +, two dark bands = Gram −), Acid-Fast (red = mycolic acids), Staining (purple/pink). Genome (genes for teichoic acids = Gram +), Penicillin testing (sensitive = Gram +), MacConkey (no growth = Gram +), isolate teichoic acid from the wall.

How the nature of envelope affects bacterial cells?

Shape (cell wall), staining results (cell wall/peptidoglycan), protection (cell wall protects the cell from bursting in hypotonic water).
Controls molecules/chemicals that can enter the cell – porins (outer membrane), carriers/ion transport (uni/sym/antiports), ABC transport (ATP powered), PTS (sugar change).
Affects sensitivity to drugs and antibiotics – penicillins (blocked by the Gram − outer membrane), cephalosporins (block cross-link → holes and lysed), vancomycin (binds D-Ala-D-Ala).

2. Know the role, feature and basic composition of each of these

a Glycocalyx (capsule and slime layer)

Two types: slime layer (loosely attached) and capsule (firmly attached).

Role and composition

Sugars, peptides, DNA.
Role in anti-phagocytosis, anti-desiccation (drying out), attachment to surfaces + biofilms.

b. S-layer – composition and where located

c. Outer membrane

LPS (O-antigen, core polysaccharide, lipid A), phospholipids, proteins (porins); peptidoglycan is not found in the outer membrane but in the cell wall.
LPS packed tightly (only saturated fatty acids) so that hydrophobic molecules cannot get in, and only small (<700 daltons) can get in through the porins. Phospholipid bilayer blocks the hydrophilic molecules.

LPS

O-antigen (sugar, varies) + core polysaccharide (same within species) + lipid A (endotoxin, inflammation) attach to each other; LPS is only in the Gram − outer membrane.

d. Cell Wall - Role

Structural/shape/support, semi rigid, stops lysis (protects the cell from bursting in hypotonic water).

1) Peptidoglycan – structure – sugars and

NAG-NAM alternating sugar chain linked by β-1,4 bonds; short peptides off NAM cross-link the chains.

Know how differs in Gm neg. vs Gm pos

Gram − is much thinner (1-2 layers, sits in the periplasm) vs Gram + (3-20 layers).
Gram − (E. coli) has DAP that links directly, Gram + (Staphylococcus) uses L-Lys with a (Gly)5 bridge. Same in both: NAG-NAM sugars with β-1,4 links.

Basic order of events in Synthesis

(NAM + NAG, pentapeptide, bactoprenol, NAG, flip (MurJ), PBPs link + crosslink, bactoprenol recycle).

2) Pseudopeptidoglycan – how differs from peptidoglycan

β-1,3 sugar linkage instead of β-1,4, plus NAT instead of NAM, and also all L-amino acids, and lysozyme cannot cut.

3) Teichoic acids

Inside the Gram + cell wall together with the thick peptidoglycan (shape, division, identify).

4) Mycolic acids

Waxy lipids with 60-90 carbons, found in the cell wall of mycobacteria (acid fast); block diffusion plus resistant to acids, drugs, bases, drying.

e. Periplasm

f. Cell membrane

Role, function of hopanoids, isoprenoids,

Components (Types of lipids, etc) observed in bacteria

Components observed in Archaea and how different? Isoprenoids

Archaea – isoprenoids (20 or 40 carbons) that are ether-linked to glycerol, in the archaeal cell membrane, vs bacteria with phospholipids that are ester-linked.

How do types of fatty acids present change in response to temperature?

They change the saturation or length of the fatty acids (cold → more unsaturated).

3. Movement of Material across Cell membrane

Different types of transport:

Type of Transport proteins observed in bacteria

Porins (outer membrane), carriers/ion transport (uni/sym/antiports), ABC transport (ATP powered), PTS (sugar change proteins).

Difference in transport of ions verse sugars

4. How do different antibiotics interfere with peptidoglycan synthesis?

Target of antibacterial drugs/antibiotics:

Know specific mechanism of action of each of the following

Penicillin,

Blocks synthesis of new peptidoglycan and is structurally similar to the D-Ala-D-Ala end of the peptide in peptidoglycan.
It binds PBPs and inhibits the transpeptidase and transglycosylase so that peptide crosslinks are not made, and holes form in the peptidoglycan of growing cells as the membrane expands through them and the cell lyses.

Cephalosporin

Block cross-link for peptidoglycan synthesis in the cell wall → holes and lysed (same as penicillin).

Bacitracin

Blocks bactoprenol recycling.

Vancomycin

Prevents cross link by binding D-Ala-D-Ala.

D-cycloserine

Lysozyme

Cuts the β-1,4 bond between NAG and NAM.

How do bacteria resist the effects of antibiotics?

Strain resistant to penicillin by making a β-lactamase or an altered PBP.
Why penicillin may fail: doesn't work for all kinds of bacteria (Gram − outer membrane, no wall in Mycoplasma), some cells in the population are not growing (penicillin only kills growing cells), infection not bacterial (virus or eukaryotic → no peptidoglycan), not a high enough conc. of penicillin.

D. Bacterial Internal Structures

Know types of structures seen in bacteria

No Golgi/ER or membrane-enclosed organelles for protein processing, but do have ribosomes and cytoskeleton.

1. Cytoplasm and cytosol

2. Ribosomes

3. Cytoskeleton – types found and roles

Tubulin – FtsZ (center divider); actin – MreB (define/maintain the rod for elongation); intermediate filaments – CreS (curve).

4. Nucleoid – DNA and packing proteins

Compacted by nucleoid-associated proteins and supercoiling (does not use histones).
Harvard University (HU protein) is the number one wrap/package; International Healthy Friends (IHF) bends a lot.
Healthy-Neutral Structure (H-NS) – bridges DNA (structural support); Friendly Inclusive Supporter (FIS) – also helps pack the DNA.

5. Compartments – intracellular membrane structures

Know some Examples and their role, their possible arrangments

2 Trees (thylakoid, 2 layers) get lights – separate from each other and is cyanobacteria; 1 chromosome (chlorosome, 1 layer) wants another light, is connected to membrane – green.
2 invasive (invagination) surgeries connected with lights on – purple; a lot of stacked metro (methanotroph membrane) buses are breaking down (methane) and are either connected or separated – Methylococcus.

6. Inclusions and granules (know examples and their role)

Seen: gas vesicles, magnetosomes, sulfur granules; NOT seen: clumps of lipids.

Gas vesicles, Carboxysomes,

Gas vesicle – protein shell with gas inside that float (buoyancy); Carboxysome – many-sided protein shell that has Rubisco that fixes CO2 into sugar to feed (both count as compartments).

Energy/carbon storage polymers, Sulfur granules

7. Endospore: What they are

E Cell division and Differentiation

Features of cell division in bacteria

Different types of Cell division observed in bacteria

Bacterial Cell Division Basic Steps in process: Replication, cytokinesis, septation, separation

Replication, Segregation, Cytokinesis, Septation, Separation (no mitotic spindle / microtubules in bacteria).

How can the process differ?

E. coli (Gram −) – does cytokinesis, septation and separation all at once; Bacillus (Gram +) – does cytokinesis and septation at once then separation later.
Staphylococcus – does septation first, then cytokinesis and separation. So no, they do not always happen at the same time.

Asymmetrical cell divisions and cell polarity

Role of FtsZ and Min proteins

FtsZ – used to split cells by the Z ring (forms at mid-cell, shrinks = constriction).
Min proteins – make filaments on the membrane that move pole to pole; MinC blocks FtsZ at the poles so the Z ring forms only in the center (mutants → minicells).

Methods used to study cell division proteins

F. Viruses

1. Size and Shape observed

2. Structural features

Components of virus particles

Which ones are found in all viruses?

3. Features used to classify or group viruses

Genome type, how it replicates, envelope; not cell wall chemicals as viruses have no cell wall.

4. Reproduction

a. General features

b. Steps in multiplication cycle

c. Synthesis stage for 7 types of viruses

Positive ssRNA – is mRNA → translates and makes itself; genome → (+ → − → +).
Negative ssRNA – RdRP first makes a (+) copy (= mRNA + template) → new (−); this is the (−) sense RNA virus that must copy first.
Retrovirus – (+) RNA → DNA by reverse transcriptase → dsDNA → integrates → host makes new RNA (the RNA virus that makes dsDNA in the host).

5. Outcomes of Viral infections

Know different possible outcomes

Affects on infected cells and populations

G. Atypical Bacteria and Viruses (notes)

Know features of each and how differ from typical bacteria and viruses

Live inside hosts, reproduce by stealing or latching off the host (steal ATP); Rickettsia/Chlamydia are tiny but have ribosomes and divide by binary fission; giant viruses are bacterial size but no ribosomes and are assembled, not divided.

1. Rickettsia

2. Chlamydia

Obligate intracellular parasitism and similar in size; differ in that Chlamydia can do binary fission compared to viruses that cannot.

3. Endosymbiotic bacteria

4. Giant Viruses

5. Mitochondria and Chloroplasts

How can one predict the structures and properties of the cells of a newly discovered bacterium?