Micro Exam 2: Short Answers (my words)

BIOL 4640 · Exam Wed 10/28 · ★ = asked on a past Exam 2 · ★★ = asked both years · give 1 more point than asked

How I answer

- Term → how it works → result. Example: penicillin looks like D-Ala-D-Ala → binds PBPs → blocks the transpeptidase (crosslink) → holes in growing cells → water in → lysis.
- Short answers are 3-4 pts each, 7-8 per exam, and mostly come from B (external structures) and C (cell envelope).

A. Size and Shapes of Bacteria (multiple choice only)

Q1. Compare the size of eukaryotic microbes, bacteria and viruses, and name the basic shapes and arrangements.

- Size: eukaryotic microbes 10-100 µm > bacteria 0.4-10 µm (most 1-3) > viruses 0.01-0.1 µm (10-100 nm).
- Shapes: cocci (round), bacilli (rods), vibrio (curved rod), spiral (spirillum, spirochete).
- Arrangements: diplo (pairs), strepto (chains), tetrads (4), sarcinae (cubes of 8, 16, 32), staphylo (clusters), palisades.

B. External Structures

Q2. ★ Describe the number and arrangement of flagella observed in bacteria and how flagella move to cause motility. (4 pts, 2025)

- 0-many per cell, placed at one end (single or tuft), both ends, or all over the cell (peritrichous).
- In spirochetes the flagella are in the periplasm and wrap around the cell from each pole.
- Number and spot are set by the species.
- The motor rotates the flagellum at its base (H+ flows through Mot), so the helical filament spins like a propeller and pushes the cell (runs + tumbles).

Q3. Describe the parts of a bacterial flagellum and the role of each.

- 4 parts: basal body, hook, filament, motor.
- Basal body – rod + rings that anchor it (L = outer membrane, P = peptidoglycan, S/M = inner membrane).
- Hook – the joint between basal body and filament.
- Filament – hollow helical tube of flagellin with a cap at the tip.
- Motor – rotor (switches CW/CCW) + MotA/MotB, H+ flows through them to turn it.

Q4. ★ Describe the basic order in which the sections of a flagellum are assembled. (3 pts, 2024)

- M (Mcdonalds), S (sonic), R (resturants), P (powerrade), L (latte), H (Happy hour), F (fries), M (Matcha).
- Basal body first: M + S rings in the inner membrane, then the rod, then P and L rings.
- Hook added next.
- 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 and put in the membrane, H+ flows through them (proton motive force) to run the motor.

Q5. ★ List two forms of motility other than the typical flagella and describe how each occurs. (4 pts, 2025)

- Periplasmic (spirochetes) – flagella each pole in periplasm wrap around cell and rotate, to do the wiggle corkscrew burrow (mucus).

- Swarming – raft of cells together across a surface, needs slime layer.
- Gliding – smooth across a solid surface with no flagella (mechanism not fully known; Myxococcus).
- Twitching – Type IV pili extend, attach, shorten → pull the cell.

Q6. What is chemotaxis, and how does a bacterium move toward an attractant?

- Chemotaxis = moving toward attractants (nutrients) or away from repellents.
- Receptor in membrane senses the attractant → signals the flagella.
- Runs to attractant: CCW = run, CW = tumble (E. coli), so going up the attractant the runs get longer with fewer tumbles (tumbles never fully stop).

Q7. ★ List three tests a scientist could do to show or predict that a new organism is motile. (3 pts, 2025)

- Walking (purposeful walking, aka wet mount) – see real movement, not just vibrating.
- Feet stain (flagellar stain) or EM – see the flagella.
- Mobile agar (motility agar stab) – cloudy all through = motile.
- Swarm plate – spreads in a circle.
- Closest related species through phylogeny (motile?), genome (flagellar genes in the predicted genes).

Q8. How do pili differ from flagella in structure and assembly?

- Pili are thinner (3-10 nm vs 14-20 nm), shorter and way more of them (100s); made of pilin + an adhesin tip.
- Pili add subunits at the BASE, flagella add flagellin at the TIP.
- Type I: chaperone (PapD) → usher (PapC), adhesin tip first. Type IV: PilA pushed out through PilQ, shortens by taking pilins off at the base.

Q9. Describe the roles of common pili, sex pili and Type IV pili.

- Common pili (fimbriae) – attach to surfaces/host cells (adhesins = virulence factors), biofilms.
- Sex (F) pili – grab another cell and pull it close → conjugation (DNA transfer).
- Type IV pili – twitching, attaching, DNA uptake, biofilms.

C. Cell Envelope

Q10. ★★ Describe the layers and components found in the envelope of a Gram-NEGATIVE bacterium. (3 pts, 2024)

- S layer – proteins, capsule – sugars (some have these).
- Outer membrane – LPS (O-antigen (sugar), core polysaccharide (same within species), lipid A (inflammation)), porins, phospholipids.
- Cell wall – thin peptidoglycan (1-2 layers) in the periplasm (watery space).
- Cell (inner) membrane with phospholipids + proteins.

Q11. ★★ Describe the layers and components found in the envelope of a Gram-POSITIVE bacterium. (4 pts, 2025)

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

Q12. Describe the envelope of Mycobacteria, Archaea or Mollicutes and how it differs from typical bacteria.

- Mycobacteria (acid fast) – capsule (sugars + glycolipids), cell wall of mycolic acids → arabinogalactan → peptidoglycan, then cell membrane; waxy so it resists drying/drugs.
- Archaea – S layer (proteins), cell wall of pseudopeptidoglycan in some (NAG + NAT, β-1,3), cell membrane with isoprenoids (ether-linked, no phospholipids).
- Mollicutes – no cell wall, only a sugar layer, cell membrane stabilized by sterols; penicillin has nothing to act on.

Q13. ★ Describe two techniques to support that a purple-staining isolate has a Gram-positive envelope. (3 pts, 2024)

- TEM – one thick dark layer (thick peptidoglycan) and no outer membrane.
- Search genome for genes for teichoic acids (and no LPS / outer membrane genes).
- Isolate teichoic acid from the wall.
- Penicillin testing – sensitive = Gram + (works better on Gram +).
- MacConkey (grows Gram − only) – no growth = Gram +.

Q14. ★ Describe three ways the nature of the envelope affects the properties of a bacterium. (3 pts, 2025)

- Shape – the cell wall helps set the cell shape.
- Staining results – cell wall/peptidoglycan decides Gram (and acid-fast) color.
- Controls what molecules/chemicals can enter the cell – outer membrane + porins.
- Sensitivity to drugs and antibiotics, acids and bases – penicillins blocked by the Gram − outer membrane.
- Wall protects the cell from bursting in hypotonic water.

Q15. ★ What types of molecules are in the glycocalyx? Describe two roles. (3 pts, 2024)

- Sugars, peptides, DNA.
- Role in anti-phagocytosis, anti-desiccation (drying out), attachment to surfaces + biofilms.
- Two types: slime layer (loosely attached) and capsule (firmly attached).

Q16. What is the S-layer made of and where is it found?

- Crystalline layer of proteins/glycoproteins; on most archaea and many bacteria (often lost in lab strains).
- Sits on top of the peptidoglycan (Gram +), on top of the outer membrane (Gram −), right on the membrane in archaea (sometimes the only wall).

Q17. Describe the parts of LPS and how the outer membrane acts as a barrier.

- O-antigen (sugar, varies) + core polysaccharide (same within species) + lipid A (endotoxin → septic shock, inflammation) attach to each other.
- LPS packed tightly (only saturated fatty acids) so that hydrophobic molecules (dyes, bile salts, some drugs) cannot get in, and only small (<700 daltons) get in through the porins. Phospholipid bilayer blocks the hydrophilic molecules.

Q18. ★ Describe the basic structure of peptidoglycan in Gram-negative bacteria. (3 pts, 2025)

- NAG-NAM alternating sugar chain linked by β-1,4 bonds.
- Short peptides (4 amino acids) off NAM cross-link the chains.
- Gram − is much thinner (1-2 layers), sits in the periplasm, and DAP links directly to D-Ala.

Q19. How does peptidoglycan differ in Gram-negative vs Gram-positive bacteria?

- Gram − is thinner (1-2 layers) 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.

Q20. Describe the order of events in peptidoglycan synthesis, including the role of bactoprenol.

- (NAM + NAG, pentapeptide, bactoprenol, NAG, flip (MurJ), PBPs link + crosslink, bactoprenol recycle).
- UDP-NAM + NAG made in the cytoplasm → pentapeptide added to NAM → attached to bactoprenol → NAG added → MurJ flips it out → PBPs link the sugars (transglycosylation) + crosslink the peptides (transpeptidation, last D-Ala cut off) → bactoprenol recycled.
- Bactoprenol = the lipid carrier that moves units (also LPS, teichoic acids) across the membrane.

Q21. How does pseudopeptidoglycan differ 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. Found in some archaea.

Q22. What is the periplasm and what are its functions?

- Watery space between the two membranes (Gram −).
- Osmotic protection, binding proteins hand solutes to transporters, breaks down nutrients, stores H+ for the proton motive force.

Q23. ★ Describe two ways the components of membranes can differ. (3 pts, 2024)

- Archaea – isoprenoids (20 or 40 carbons) that are ether-linked to glycerol, vs bacteria with phospholipids that are ester-linked.
- Change the saturation or length of the fatty acids with temperature (cold → more unsaturated).
- Some bacteria have hopanoids (sterol-like, stabilize), others cardiolipin.
- Gram − outer membrane has LPS; types of phospholipids and proteins vary.

Q24. Compare passive and active transport and describe the three kinds of active transport.

- Passive – down the gradient, no energy (simple diffusion; facilitated through porins/carriers like GlpF).
- Ion-coupled – energy = ion gradient (H+/Na+); symport (together, lactose + H+), antiport (swap, Na+ out H+ in), uniport (solo, K+).
- ABC transport (ATP powered) – ATP-spender, uses a periplasm binding protein; also pumps drugs out.
- PTS (PEP) – sugar-tagging: sugar changes as it comes in (glucose → glucose-6-P).

Q25. How does the transport of ions differ from the transport of sugars?

- Ions move WITH their own gradient and power other transport (and ATP synthase, flagella).
- Sugars move AGAINST their gradient using an ion gradient, ATP or PEP.

Q26. ★ Describe how penicillin acts against bacteria: its target and its effect. (4 pts, 2025)

- 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.
- Holes form in the peptidoglycan of growing cells as the membrane expands through them (water comes in, osmosis) and the cell lyses.

Q27. ★★ Give two reasons why penicillin may not work (stream water 2024 / doctor 2025). (3 pts)

- Strain resistant to penicillin by making a β-lactamase or an altered PBP.
- Does not work for all kinds of bacteria (Gram − outer membrane keeps it out, Mycobacteria, 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), or not a high enough conc. of penicillin (stream water).

Q28. Describe the mechanism of action of cephalosporin, bacitracin, vancomycin, D-cycloserine or lysozyme.

- Cephalosporin – block crosslink for peptidoglycan synthesis → holes and lysed (same as penicillin).
- Bacitracin – blocks bactoprenol recycling → no new units carried out.
- Vancomycin – prevents crosslink by binding D-Ala-D-Ala (does not look like it).
- D-cycloserine – looks like D-Ala → blocks Ala racemase + ligase → no D-Ala-D-Ala made.
- Lysozyme – our enzyme, cuts the β-1,4 bond between NAG and NAM → lysis (or protoplast/spheroplast in isotonic).

Q29. Describe two ways bacteria resist antibiotics (for example penicillin).

- Destroy the drug – β-lactamase cuts the β-lactam ring.
- Change the target – altered PBP (mutation or acquired gene) no longer binds.
- Pump it out (efflux pump) or keep it out (lose a porin).
- Protect the ribosome (for ribosome drugs).

D. Internal Structures

Q30. How do bacterial ribosomes differ from eukaryotic ones, and why does that matter?

- Bacteria 70S (50S + 30S) vs eukaryotes 80S (60S + 40S); rRNA is about 65% of the weight.
- The difference lets antibiotics block bacterial translation only (tetracycline, streptomycin at 30S; chloramphenicol, erythromycin at 50S).

Q31. Describe the cytoskeleton proteins FtsZ, MreB and crescentin and their roles.

- Tubulin – FtsZ (center divider, Z ring; mutants make long filaments).
- Actin – MreB (define/maintain the rod for elongation).
- Intermediate filaments – CreS (curve, Caulobacter).

Q32. How is DNA packed in the nucleoid without histones?

- 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.
- Topoisomerases supercoil it into ~50 kilobase loops; nucleoid looks lighter in EM (no ribosomes).

Q33. ★ List two compartments found inside bacteria; describe their structure and role. (4 pts, 2024)

- 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.
- 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.
- Also count: magnetosome, membrane around DNA (Gemmata), forespore. NOT a compartment: nucleoid (no membrane).

Q34. Describe two inclusions and their roles.

- Gas vesicle (float), carboxysome (fix CO2), magnetosome (magnetite → magnetotaxis), PHA/glycogen (store energy + carbon), sulfur granules.
- NOT seen: clumps of lipids, ER/Golgi.

Q35. What is an endospore, what triggers its formation, and why is it resistant?

- Dormant spore made inside a cell (Bacillus, some Firmicutes).
- Trigger: starvation and high cell density.
- Cell divides but no septation of the wall → mother cell engulfs the forespore → cortex, coat, exosporium → released.
- Resists heat, drying, radiation (thick layers + dormant).

E. Cell Division

Q36. Describe the steps of bacterial cell division.

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

Q37. How does cell division differ in E. coli, Bacillus and Staphylococcus?

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

Q38. Describe the roles of FtsZ and the Min proteins.

- FtsZ – used to split cells by the Z ring (forms at mid-cell, shrinks = constriction); builds the divisome and brings PBP1/PBP3 for the septum.
- 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).

Q39. Describe asymmetrical division and cell polarity (Caulobacter).

- Makes a different cell type. Caulobacter swarmer cell loses its flagellum, grows a stalk, divides → new swarmer.
- Polarity: the two ends differ; TipN marks the new pole; each cell = 1 new + 1 old pole.

F. Viruses

Q40. Which components are found in all virus particles and which only in some? Compare naked and enveloped viruses.

- All: nucleic acid (DNA OR RNA) + protein capsid (= nucleocapsid).
- Some: envelope (membrane + spikes), enzymes (polymerase, reverse transcriptase).
- Naked – tougher, must lyse the cell to get out. Enveloped – must stay wet, can bud out.

Q41. What features are used to classify viruses?

- Genome type, how it replicates, envelope (also capsid shape, host); not cell wall chemicals as viruses have no cell wall.

Q42. Describe the steps of the virus multiplication cycle.

- Adsorption (spikes bind receptors; sets host range) → penetration (endocytosis or fusion) → uncoating → synthesis (depends on group) → assembly (from parts, no binary fission) → release (lysis, budding, cell-to-cell).

Q43. Describe synthesis for a (−) sense RNA virus or a retrovirus.

- + = ready, − = copy first.
- Positive ssRNA – is mRNA → translates right away; genome → (+ → − → +).
- Negative ssRNA – RdRP first makes a (+) copy (= mRNA + template) → new (−).
- Retrovirus – (+) RNA → DNA by reverse transcriptase → dsDNA → integrates → host makes new RNA.

Q44. Describe the possible outcomes of a viral infection of an animal cell.

- Acute – cell lyses and dies.
- Persistent – enveloped virus buds out slowly, cell survives.
- Latent – virus goes dormant (integrated/plasmid) and can reactivate (herpes).
- Transformation – cancer (oncogene). NOT an outcome: viruses dividing by binary fission.

G. Atypical Bacteria and Viruses

Q45. Describe how Rickettsia, Chlamydia or giant viruses are like bacteria and how they are like viruses.

- Like bacteria – DNA + RNA, ribosomes, enzymes, divide by binary fission (Rickettsia, Chlamydia).
- Like viruses – tiny, obligate intracellular, live inside hosts and steal from the host (ATP); first mistaken for viruses (poor Gram stain, pass filters).
- Rickettsia – energy parasite (steals ATP) → ancestor of mitochondria. Chlamydia – EB (infectious) → RB (divides) → EB.
- Giant viruses – bacterial size and genome, but no ribosomes and assembled, not divided.

Q46. How could you predict the structures and properties of a newly discovered bacterium?

- Phylogeny first (close relatives share structures) → search the genome (LPS, teichoic acid, mycolic acid, pseudopeptidoglycan, flagella genes) → test: stains, MacConkey, EM, motility tests.

Top Priority (write from memory before the exam)

- ★★ Q27 why penicillin may not work · ★★ Q10/Q11 Gram − and Gram + layers · ★ Q26 how penicillin works
- ★ Q2 flagella arrangement · ★ Q4 assembly order · ★ Q5 other motility · ★ Q7 motility tests
- ★ Q15 glycocalyx · ★ Q18 peptidoglycan structure · ★ Q14 envelope effects · ★ Q13 confirm Gram + · ★ Q23 membrane differences · ★ Q33 compartments