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CORE JOBS · INTERVIEW QUESTIONS

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General Biology Interview Questions

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Biomolecules and biological membranes

1. What is special about the peptide bond?

The peptide bond is an amide link between the carboxyl carbon of one amino acid and the amino nitrogen of the next. Resonance gives it partial double-bond character, so the peptide group is planar and rotation about the bond is restricted. The backbone can rotate only about the bonds to each alpha carbon (the phi and psi angles), and steric clashes restrict even these, which limits the folds a chain can adopt.

2. Why do cells keep unsaturated fatty acids and cholesterol in their membranes?

Both regulate fluidity. Cis double bonds put kinks in the fatty acid tails, so they pack loosely and the membrane stays fluid at lower temperatures. Cholesterol sits between the phospholipids: it restricts tail movement in a fluid membrane and prevents tight packing in a cold one, so it buffers fluidity against temperature change.

3. A neuron at 37 °C has 140 mM K+ inside and 5 mM K+ outside. Taking the membrane as permeable only to K+, what is the K+ equilibrium potential (inside relative to outside)? Use R = 8.314 J/(mol K) and F = 96485 C/mol.

The K+ equilibrium potential is about -89 mV, so the inside is negative. The Nernst equation gives $E_K = \frac{RT}{F}\ln\frac{[K^+]_{out}}{[K^+]_{in}}$, with $RT/F = 26.7$ mV at 310 K and $\ln(5/140) = -3.33$, so $E_K = 26.7 \times (-3.33) \approx -89$ mV.

WORKING

1. $E_K = \frac{RT}{zF}\ln\frac{[K^+]_{out}}{[K^+]_{in}}$, with $T = 310$ K and $z = +1$.
2. $RT/F = 8.314 \times 310/96485 = 0.0267$ V.
3. $\ln(5/140) = -3.33$.
4. $E_K = 0.0267 \times (-3.33) = -0.089$ V.

Metabolism

4. How much ATP does glycolysis make from one glucose, and where does it happen?

Glycolysis converts one glucose into two pyruvate with a net gain of 2 ATP and 2 NADH. It spends 2 ATP in the preparatory phase (hexokinase and phosphofructokinase-1) and makes 4 ATP by substrate-level phosphorylation in the payoff phase. It takes place in the cytosol and needs no oxygen.

5. What is the difference between substrate-level and oxidative phosphorylation?

In substrate-level phosphorylation an enzyme transfers a phosphate group from a high-energy substrate directly to ADP, as pyruvate kinase does. In oxidative phosphorylation, electrons from NADH and FADH2 pass down the electron transport chain, which pumps protons, and ATP synthase uses the proton gradient to make ATP. The second needs a membrane and a terminal electron acceptor (oxygen in aerobic respiration), and yields far more ATP.

6. Why is phosphofructokinase-1 the main control point of glycolysis?

It catalyses the first committed step. Fructose 6-phosphate to fructose 1,6-bisphosphate is essentially irreversible, and the product has no other fate than glycolysis. ATP and citrate inhibit the enzyme, while AMP and fructose 2,6-bisphosphate activate it, so the flux follows the energy state of the cell.

7. The NAD+/NADH couple has a standard reduction potential of -0.32 V at pH 7 and the 1/2 O2/H2O couple has +0.82 V. Find the standard free energy change for passing two electrons from NADH to oxygen. Use F = 96.485 kJ/(V mol).

The standard free energy change is -220 kJ/mol of NADH. The potential difference is $\Delta E^{\circ\prime} = 0.82 - (-0.32) = 1.14$ V, and $\Delta G^{\circ\prime} = -nF\,\Delta E^{\circ\prime} = -2 \times 96.485 \times 1.14 = -220$ kJ/mol. The large negative value shows the transfer is strongly exergonic.

WORKING

1. $\Delta E^{\circ\prime} = 0.82 - (-0.32) = 1.14$ V.
2. $\Delta G^{\circ\prime} = -nF\,\Delta E^{\circ\prime}$ with $n = 2$.
3. $\Delta G^{\circ\prime} = -2 \times 96.485 \times 1.14$.
4. $\Delta G^{\circ\prime} = -220$ kJ/mol.

Enzymes

8. How does an enzyme speed up a reaction?

An enzyme lowers the activation energy by binding and stabilising the transition state at its active site. It does not change the free energy difference between substrate and product, so the equilibrium position stays the same: the reaction only reaches it faster. The enzyme is unchanged at the end of each cycle.

9. What is the difference between competitive and non-competitive inhibition?

A competitive inhibitor binds the active site and competes with substrate, so the apparent Km rises while Vmax is unchanged; enough substrate overcomes it. A pure non-competitive inhibitor binds a different site, whether or not substrate is bound, so Vmax falls while Km is unchanged, and extra substrate cannot reverse it.

10. An enzyme has a Vmax of 100 µmol/min and a Km of 2 mM. What is the initial rate at a substrate concentration of 6 mM?

The initial rate is 75 µmol/min. The Michaelis-Menten equation gives $v = \frac{V_{max}[S]}{K_m + [S]} = \frac{100 \times 6}{2 + 6} = 75$ µmol/min. At a substrate concentration of three times Km, the enzyme runs at three quarters of Vmax.

WORKING

1. $v = \frac{V_{max}[S]}{K_m + [S]}$.
2. $v = \frac{100 \times 6}{2 + 6}$.
3. $v = 600/8 = 75$ µmol/min.

11. Why do allosteric enzymes give a sigmoidal curve of rate against substrate concentration?

They have several subunits that bind substrate cooperatively. Binding at one site shifts the enzyme from the low-affinity T state toward the high-affinity R state, so the remaining sites bind more easily. The rate therefore rises steeply over a narrow range of substrate, giving a sigmoidal curve instead of the hyperbola of Michaelis-Menten kinetics.

Bacterial classification, cell walls, growth and nutrition

12. What is the difference between the cell walls of Gram-positive and Gram-negative bacteria?

Gram-positive cells have a thick peptidoglycan layer with teichoic acids and no outer membrane. Gram-negative cells have a thin peptidoglycan layer under an outer membrane that carries lipopolysaccharide. In the Gram stain the thick wall retains crystal violet after the alcohol wash, so Gram-positive cells stay purple, while Gram-negative cells lose it and take up the pink counterstain.

13. Why is an autoclave run at 121 °C instead of boiling at 100 °C?

Boiling kills growing cells but not bacterial endospores. Steam under pressure (about 15 psi above atmospheric) raises the temperature to 121 °C, and moist heat at that temperature, held for 15 to 20 minutes, denatures the proteins of endospores and so sterilises the load.

14. A bacterial culture grows exponentially from 2 × 10^5 to 3.2 × 10^6 cells/mL in 3 hours. What is its generation time?

The generation time is 45 minutes. The population rose by a factor of $(3.2 \times 10^6)/(2 \times 10^5) = 16 = 2^4$, so the cells doubled 4 times in 180 minutes, and each doubling took $180/4 = 45$ minutes.

WORKING

1. $N = N_0\,2^n$, so $2^n = 3.2 \times 10^6 / 2 \times 10^5 = 16$.
2. $n = 4$ generations.
3. $g = t/n = 180\ \text{min}/4 = 45$ min.

Pathogens & antibiotics

15. How is the lac operon switched on?

When lactose is present, a little allolactose forms and binds the LacI repressor, which then leaves the operator, so RNA polymerase can transcribe lacZ, lacY and lacA. Strong expression also needs low glucose: cAMP then rises, and the cAMP-CAP complex helps RNA polymerase bind the promoter.

16. Why do legume root nodules contain leghemoglobin?

Nitrogenase is irreversibly damaged by oxygen, yet the nitrogen-fixing bacteroids need oxygen for respiration to make the ATP that fixation consumes. Leghemoglobin binds oxygen with high affinity, keeping free oxygen very low while still delivering it at the rate respiration needs.

17. Why do beta-lactam antibiotics kill growing bacteria but spare human cells?

Beta-lactams such as penicillin inhibit the transpeptidases that cross-link peptidoglycan strands. Growing cells keep building and remodelling their wall, so with cross-linking blocked the wall weakens and the cell bursts from internal osmotic pressure. Non-growing cells build little wall and are barely affected. Human cells have no peptidoglycan.

Immune system

18. What is the difference between innate and adaptive immunity?

Innate immunity acts within minutes to hours through germline-encoded receptors, such as Toll-like receptors, that recognise patterns shared by groups of microbes; it has no memory. Adaptive immunity takes days on first exposure, uses receptors made by gene rearrangement in each B and T cell and specific for one antigen, and leaves memory cells for a faster second response.

19. What is the structure of an IgG antibody and what does each part do?

IgG has two identical heavy chains and two identical light chains, joined by disulphide bonds into a Y shape. Each of the two Fab arms carries one antigen-binding site, formed by the variable domains of a heavy and a light chain. The Fc tail, made of heavy-chain constant domains, carries the effector functions, such as binding Fc receptors on phagocytes or activating complement.

20. What is the difference between the classical and alternative complement pathways?

The classical pathway starts when C1q binds IgG or IgM already attached to an antigen. The alternative pathway starts when C3 breaks down spontaneously at a low rate and the product C3b lands on a microbial surface, so it needs no antibody. Both produce a C3 convertase and then share the terminal steps that end in the membrane attack complex.

Adaptive immunity

21. What is the difference between MHC class I and MHC class II molecules?

MHC class I is on nearly all nucleated cells and presents peptides from proteins made inside the cell to CD8 T cells; loading uses the proteasome, TAP and the endoplasmic reticulum. MHC class II is on antigen-presenting cells such as dendritic cells, macrophages and B cells, and presents peptides from engulfed material to CD4 T cells after loading in endosomal compartments.

22. Why does the thymus delete some developing T cells?

A developing T cell is first kept only if its receptor recognises self MHC weakly (positive selection, in the cortex). Cells that then bind self peptide-MHC strongly are removed by apoptosis (negative selection, mainly in the medulla). This deletes cells that could attack the body's own tissues and leaves T cells that respond to foreign peptides on self MHC.

23. Why is the response to a second exposure to an antigen faster and stronger, and how do vaccine boosters use this?

The first exposure leaves memory B and T cells, which outnumber the naive cells that could recognise the antigen. Memory B cells have also undergone affinity maturation and class switching. On re-exposure they activate quickly and make high-affinity IgG within days. A booster dose triggers this memory response and strengthens it.

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