

# MCAT BONUS - PRACTICE TEST 1

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## Chemical And Physical Foundations of Biological Systems

Time	Questions
95 minutes	59

### **PASSAGE 1 (Questions 1-5): Thermodynamics of Protein Folding**

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Protein folding is a spontaneous process driven by thermodynamic principles. Native protein structures represent the lowest free energy conformation under physiological conditions. The Gibbs free energy change ( $\Delta G$ ) determines spontaneity:  $\Delta G = \Delta H - T\Delta S$ , where  $\Delta H$  is enthalpy change,  $T$  is temperature in Kelvin, and  $\Delta S$  is entropy change.

During folding, hydrophobic amino acids cluster in the protein interior, minimizing unfavorable interactions with water—the hydrophobic effect. This increases water entropy as structured water molecules around hydrophobic groups are released to bulk solvent. Simultaneously, the protein chain loses conformational entropy as it adopts a specific structure.

Hydrogen bonds, van der Waals forces, and electrostatic interactions stabilize the folded state enthalpically. Disulfide bonds between cysteine residues provide additional covalent stabilization in extracellular proteins. The balance between favorable enthalpy (stabilizing interactions) and unfavorable entropy (loss of conformational freedom) determines the folding equilibrium.

Levinthal's paradox notes that random sampling of all possible conformations would take longer than the age of the universe, yet proteins fold in milliseconds to seconds. This suggests folding follows specific pathways through intermediate states rather than random search, guided by local structure formation and progressive stabilization.

Chaperone proteins assist folding for some proteins, preventing aggregation and misfolding. Heat shock proteins (HSPs) bind to hydrophobic regions of unfolded proteins, preventing premature aggregation while allowing proper folding. Without chaperones, exposed hydrophobic surfaces can lead to aggregation, forming insoluble complexes implicated in diseases like Alzheimer's and Parkinson's.

**Researchers studied protein folding thermodynamics using a model protein at 25°C (298 K):**

**Unfolded → Folded**

- $\Delta H = -240 \text{ kJ/mol}$  (favorable)
- $\Delta S = -600 \text{ J/(mol}\cdot\text{K)}$  (unfavorable)
- Folding completed in 100 milliseconds

**At 75°C (348 K):**

- Protein became denatured (unfolded)
- Process was reversible upon cooling

**In the presence of 8 M urea (denaturant):**

- Protein remained unfolded at 25°C
- $\Delta G$  became positive

1. Based on the thermodynamic data at 25°C, what is the Gibbs free energy change ( $\Delta G$ ) for protein folding?

- A. -61 kJ/mol
- B. +179 kJ/mol
- C. -61 kJ/mol
- D. +419 kJ/mol

2. The hydrophobic effect contributes to protein folding primarily by:

- A. Increasing the entropy of the system (water + protein) as water molecules are released
- B. Forming covalent bonds between hydrophobic amino acids
- C. Decreasing the enthalpy of the protein through ionic interactions
- D. Creating disulfide bonds in the protein core

3. At what approximate temperature would  $\Delta G = 0$  for this protein folding reaction (folding-unfolding equilibrium)?

- A. 127°C (400 K)
- B. 227°C (500 K)
- C. 325°C (598 K)
- D. Cannot be determined from given information

4. The observation that protein denaturation at 75°C is reversible upon cooling demonstrates that:

- A. Covalent bonds in the backbone are broken at high temperature
- B. The primary structure determines the final three-dimensional conformation
- C. Chaperone proteins are required for all protein folding
- D. Hydrophobic amino acids become hydrophilic at high temperature

5. Urea causes protein denaturation at 25°C by:

- A. Breaking peptide bonds in the protein backbone
- B. Oxidizing disulfide bonds
- C. Disrupting hydrogen bonds and hydrophobic interactions, making  $\Delta G$  positive
- D. Decreasing the temperature of the solution

### **PASSAGE 2 (Questions 6-9): Electrochemical Cells and Bioenergetics**

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Electrochemistry principles govern many biological processes, including cellular respiration and nerve impulse transmission. The standard reduction potential ( $E^\circ$ ) measures a half-reaction's tendency to gain electrons, with more positive values indicating stronger oxidizing agents.

**The Nernst equation relates cell potential to concentrations:**

$$E = E^\circ - (RT/nF) \ln(Q)$$

Where  $R = 8.314 \text{ J}/(\text{mol}\cdot\text{K})$ ,  $T$  is temperature (K),  $n$  is electrons transferred,  $F = 96,485 \text{ C}/\text{mol}$  (Faraday constant), and  $Q$  is the reaction quotient.

**At 25°C (298 K), this simplifies to:**

$$E = E^\circ - (0.0592/n) \log(Q)$$

The relationship between cell potential and Gibbs free energy is:

$$\Delta G = -nFE$$

Cellular respiration uses electron transfer from NADH and  $\text{FADH}_2$  to oxygen through the electron transport chain. The  $\text{NAD}^+/\text{NADH}$  half-reaction has  $E^\circ = -0.32 \text{ V}$ , while the  $\text{O}_2/\text{H}_2\text{O}$  half-reaction has  $E^\circ = +0.82 \text{ V}$ .

Galvanic (voltaic) cells produce electrical energy from spontaneous redox reactions (positive cell potential, negative  $\Delta G$ ). Electrolytic cells use electrical energy to drive non-spontaneous reactions (negative cell potential, positive  $\Delta G$ ).

**Researchers constructed an electrochemical cell:**

Anode (oxidation):  $\text{Zn(s)} \rightarrow \text{Zn}^{2+}(\text{aq}) + 2\text{e}^-$ ,  $E^\circ = -0.76 \text{ V}$

Cathode (reduction):  $\text{Cu}^{2+}(\text{aq}) + 2\text{e}^- \rightarrow \text{Cu(s)}$ ,  $E^\circ = +0.34 \text{ V}$

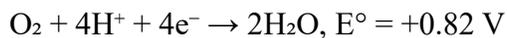
**Standard conditions:**

- $E^\circ_{\text{cell}} = +1.10 \text{ V}$
- Spontaneous electron flow from Zn to Cu

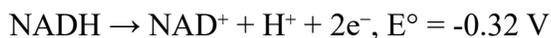
**Modified conditions:**

- $[\text{Zn}^{2+}] = 0.01 \text{ M}$
- $[\text{Cu}^{2+}] = 1.0 \text{ M}$
- Temperature = 25°C

**They then examined the biological reduction of oxygen:**



**Combined with NADH oxidation:**



6. What is the standard cell potential ( $E^\circ_{\text{cell}}$ ) for the biological oxidation of NADH by oxygen?

- A. +1.14 V
- B. +0.50 V
- C. -0.50 V
- D. -1.14 V

7. Under the modified conditions ( $[\text{Zn}^{2+}] = 0.01 \text{ M}$ ,  $[\text{Cu}^{2+}] = 1.0 \text{ M}$ ), the actual cell potential will be:

- A. Lower than  $E^\circ_{\text{cell}}$
- B. Higher than  $E^\circ_{\text{cell}}$  due to decreased product concentration
- C. Exactly equal to  $E^\circ_{\text{cell}}$
- D. Negative, making the reaction non-spontaneous

8. How much free energy is released when 2 moles of electrons are transferred at standard conditions in the Zn-Cu cell?

- A. -53 kJ
- B. -106 kJ
- C. -159 kJ
- D. -212 kJ

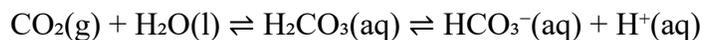
9. If the cell in the modified conditions ( $[\text{Zn}^{2+}] = 0.01 \text{ M}$ ,  $[\text{Cu}^{2+}] = 1.0 \text{ M}$ ) operates until equilibrium, which statement is correct?

- A.  $[\text{Zn}^{2+}]$  will increase and  $[\text{Cu}^{2+}]$  will decrease
- B.  $[\text{Zn}^{2+}]$  will decrease and  $[\text{Cu}^{2+}]$  will increase
- C. Both concentrations will remain constant
- D. The cell potential will increase over time

### **PASSAGE 3 (Questions 10-14): Acid-Base Equilibria and Buffer Systems**

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Blood pH must be maintained within a narrow range (7.35-7.45) for proper physiological function. Buffer systems resist pH changes when acids or bases are added. The bicarbonate buffer system is the primary blood buffer:



**The Henderson-Hasselbalch equation describes buffer pH:**

$$\text{pH} = \text{pK}_a + \log\left(\frac{[\text{A}^-]}{[\text{HA}]}\right)$$

For the bicarbonate system,  $\text{pK}_a = 6.1$ , and normal blood concentrations are  $[\text{HCO}_3^-] = 24 \text{ mM}$  and  $[\text{H}_2\text{CO}_3] = 1.2 \text{ mM}$ .

Respiratory acidosis occurs when  $\text{CO}_2$  accumulates (hypoventilation), shifting equilibrium right and decreasing pH. Metabolic acidosis results from excess metabolic acids (like lactic acid during intense exercise) consuming  $\text{HCO}_3^-$ . The body compensates through respiratory rate changes and renal  $\text{HCO}_3^-$  reabsorption.

Buffer capacity depends on concentration and pH proximity to  $\text{pK}_a$ . Buffers work best within  $\pm 1$  pH unit of  $\text{pK}_a$ , where the ratio  $[\text{A}^-]/[\text{HA}]$  ranges from 10:1 to 1:10.

**Researchers studied buffer systems:**

**Experiment 1: Bicarbonate buffer**

- Initial:  $[\text{HCO}_3^-] = 24 \text{ mM}$ ,  $[\text{H}_2\text{CO}_3] = 1.2 \text{ mM}$
- $\text{pH} = 7.4$
- Added strong acid to reduce  $[\text{HCO}_3^-]$  to  $12 \text{ mM}$
- $[\text{H}_2\text{CO}_3]$  increased to  $2.4 \text{ mM}$

**Experiment 2: Phosphate buffer ( $\text{pK}_a = 7.2$ )**

- $[\text{HPO}_4^{2-}] = [\text{H}_2\text{PO}_4^-] = 50 \text{ mM}$
- $\text{pH} = 7.2$

**Experiment 3: Hyperventilation (decreased  $\text{CO}_2$ )**

- $\text{CO}_2$  removal shifted equilibrium left
- $[\text{H}_2\text{CO}_3]$  decreased to  $0.6 \text{ mM}$
- $[\text{HCO}_3^-]$  remained initially at  $24 \text{ mM}$

10. What is the normal blood pH based on the bicarbonate buffer system with  $[\text{HCO}_3^-] = 24 \text{ mM}$  and  $[\text{H}_2\text{CO}_3] = 1.2 \text{ mM}$ ?

- A. 6.1
- B. 7.4
- C. 7.9
- D. 8.4

11. After adding strong acid in Experiment 1, what is the new pH?

- A. 6.1
- B. 6.4
- C. 6.8

D. 7.1

12. Why is the phosphate buffer ( $pK_a = 7.2$ ) more effective at maintaining pH 7.2 than pH 8.2?

- A. Phosphate becomes toxic at high pH
- B. The buffer capacity is greatest when  $pH = pK_a$
- C. Phosphate cannot accept protons at pH 8.2
- D. The Henderson-Hasselbalch equation doesn't apply at pH 8.2

13. Hyperventilation causing decreased  $[H_2CO_3]$  will initially result in:

- A. Respiratory alkalosis with increased blood pH
- B. Respiratory acidosis with decreased blood pH
- C. No pH change because  $[HCO_3^-]$  is unchanged
- D. Metabolic acidosis

14. Which buffer system would be most effective for maintaining pH around 7.8?

- A. Acetic acid/acetate ( $pK_a = 4.76$ )
- B. Tris buffer ( $pK_a = 8.1$ )
- C. Bicarbonate ( $pK_a = 6.1$ )
- D. Citric acid ( $pK_{a1} = 3.1$ )

#### **PASSAGE 4 (Questions 15-18): Enzyme Kinetics and Inhibition Mechanisms**

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Enzyme kinetics describe the rates of enzyme-catalyzed reactions. The Michaelis-Menten equation relates reaction velocity ( $v$ ) to substrate concentration  $[S]$ :

$$v = (V_{max}[S]) / (K_m + [S])$$

Where  $V_{max}$  is maximum velocity (all enzyme saturated) and  $K_m$  is the Michaelis constant (substrate concentration at half-maximal velocity). A lower  $K_m$  indicates higher enzyme-substrate affinity.

**The Lineweaver-Burk plot (double-reciprocal plot) linearizes Michaelis-Menten kinetics:**

$$1/v = (K_m/V_{max})(1/[S]) + 1/V_{max}$$

This plot aids in identifying inhibition types. Competitive inhibitors resemble substrate and compete for the active site, increasing apparent  $K_m$  without changing  $V_{max}$ . Noncompetitive inhibitors bind elsewhere, decreasing  $V_{max}$  without affecting  $K_m$ . Uncompetitive inhibitors bind only to enzyme-substrate complex, decreasing both  $K_m$  and  $V_{max}$  proportionally.

Allosteric enzymes don't follow Michaelis-Menten kinetics. They exhibit sigmoidal (S-shaped) velocity curves due to cooperative substrate binding. Allosteric activators increase enzyme activity; inhibitors decrease it.

**Researchers studied an enzyme that catalyzes:  $A \rightarrow B$**

**No inhibitor:**

- $V_{max} = 100 \mu\text{mol}/\text{min}$
- $K_m = 2 \text{ mM}$

**With Inhibitor X (10  $\mu\text{M}$ ):**

- $V_{max} = 100 \mu\text{mol}/\text{min}$
- Apparent  $K_m = 8 \text{ mM}$
- High substrate concentrations overcame inhibition

**With Inhibitor Y (5  $\mu\text{M}$ ):**

- $V_{max} = 50 \mu\text{mol}/\text{min}$
- $K_m = 2 \text{ mM}$
- High substrate concentrations couldn't overcome inhibition

**With Inhibitor Z (20  $\mu\text{M}$ ):**

- $V_{max} = 60 \mu\text{mol}/\text{min}$
- Apparent  $K_m = 1.2 \text{ mM}$
- Inhibition increased with substrate concentration

15. Inhibitor X functions as a:

- A. Noncompetitive inhibitor
- B. Uncompetitive inhibitor
- C. Allosteric activator
- D. Competitive inhibitor

16. Based on the kinetic data, Inhibitor Y most likely:

- A. Competes with substrate for the active site
- B. Binds to an allosteric site
- C. Binds reversibly to the enzyme-substrate complex
- D. Permanently modifies the active site through covalent bonding

17. Inhibitor Z is best classified as:

- A. Competitive inhibitor
- B. Uncompetitive inhibitor
- C. Mixed inhibitor
- D. Irreversible inhibitor

18. At  $[S] = 2 \text{ mM}$  (equal to  $K_m$ ) with no inhibitor present, the reaction velocity is:

- A.  $50 \text{ } \mu\text{mol/min}$
- B.  $25 \text{ } \mu\text{mol/min}$
- C.  $100 \text{ } \mu\text{mol/min}$
- D.  $33 \text{ } \mu\text{mol/min}$

## **PASSAGE 5 (Questions 19-23): Calorimetry and Thermochemistry**

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Calorimetry measures heat transfer during physical and chemical processes. Heat ( $q$ ) transferred is related to temperature change ( $\Delta T$ ) by:

$$q = mc\Delta T$$

Where  $m$  is mass,  $c$  is specific heat capacity, and  $\Delta T$  is temperature change. For water,  $c = 4.18 \text{ J}/(\text{g}\cdot^\circ\text{C})$ .

A bomb calorimeter measures combustion heat at constant volume. The heat released raises the calorimeter and contents' temperature. If the calorimeter's heat capacity ( $C_{\text{cal}}$ ) is known:

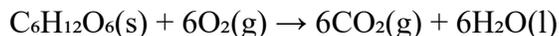
$$q_{\text{rxn}} = -C_{\text{cal}}\Delta T$$

The negative sign indicates that exothermic reactions release heat, warming the calorimeter.

Enthalpy of formation ( $\Delta H^\circ_f$ ) is the enthalpy change when one mole of a compound forms from elements in standard states. Hess's Law states that enthalpy change depends only on initial and final states, allowing calculation of reaction enthalpies from formation enthalpies:

$$\Delta H^\circ_{\text{rxn}} = \Sigma(\Delta H^\circ_f \text{ products}) - \Sigma(\Delta H^\circ_f \text{ reactants})$$

### **Cellular metabolism involves combustion of glucose:**



$$\Delta H^\circ_{\text{rxn}} = -2803 \text{ kJ/mol}$$

This energy is captured in ATP rather than released as heat.

### **Researchers conducted calorimetry experiments:**

#### **Experiment 1: Coffee cup calorimeter**

- 100 g of water at  $25.0^\circ\text{C}$

- Added 5.0 g of  $\text{NH}_4\text{NO}_3(\text{s})$
- Final temperature:  $20.0^\circ\text{C}$
- Assume solution has properties of water

### Experiment 2: Bomb calorimeter

- Burned 1.00 g glucose ( $\text{C}_6\text{H}_{12}\text{O}_6$ , MW = 180 g/mol)
- Calorimeter heat capacity:  $10.0 \text{ kJ}/^\circ\text{C}$
- Temperature increased from  $25.0^\circ\text{C}$  to  $26.6^\circ\text{C}$

### Experiment 3: Neutralization reaction

- Mixed 50.0 mL of 1.0 M HCl with 50.0 mL of 1.0 M NaOH
- Initial temperature:  $25.0^\circ\text{C}$
- Final temperature:  $31.4^\circ\text{C}$
- Assume solution density = 1.0 g/mL and  $c = 4.18 \text{ J}/(\text{g}\cdot^\circ\text{C})$

19. In Experiment 1, what is the heat of dissolution ( $\Delta H$ ) for  $\text{NH}_4\text{NO}_3$  per mole? (MW of  $\text{NH}_4\text{NO}_3 = 80 \text{ g/mol}$ )

- A.  $+26.7 \text{ kJ/mol}$  (endothermic)
- B.  $-26.7 \text{ kJ/mol}$  (exothermic)
- C.  $+33.4 \text{ kJ/mol}$  (endothermic)
- D.  $-33.4 \text{ kJ/mol}$  (exothermic)

20. The heat released during glucose combustion in Experiment 2 was:

- A. 10.0 kJ
- B. 15.6 kJ
- C. 16.0 kJ
- D. 26.0 kJ

21. Does the experimental value from Experiment 2 match the theoretical  $\Delta H^\circ_{\text{rxn}}$  for glucose combustion?

- A. Yes, it's very close to -2803 kJ/mol
- B. No, the experimental value is much higher than expected
- C. No, the experimental value is much lower than expected
- D. Cannot compare because units are different

22. In Experiment 3, what is the heat released by the neutralization reaction?

- A. 1.34 kJ
- B. 2.67 kJ
- C. 5.34 kJ
- D. 10.7 kJ

23. Based on Experiment 3, what is the molar enthalpy of neutralization ( $\Delta H$  per mole of water formed)?

- A. -53.4 kJ/mol
- B. -26.7 kJ/mol
- C. +53.4 kJ/mol
- D. +26.7 kJ/mol

### **PASSAGE 6 (Questions 24-28): Optics and Vision**

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The human eye functions as a complex optical system involving refraction through multiple surfaces. Light enters through the cornea, passes through the aqueous humor, lens, and vitreous humor before reaching the retina. The cornea and lens together provide approximately 60 diopters (D) of refractive power.

**Lens power in diopters relates to focal length:**

$$P = 1/f \text{ (f in meters)}$$

**The thin lens equation is:**

$$1/f = 1/d_o + 1/d_i$$

Where  $d_o$  is object distance and  $d_i$  is image distance.

For a converging (convex) lens, focal length is positive. Diverging (concave) lenses have negative focal lengths. When light passes through multiple lenses, total power equals the sum of individual powers.

Accommodation allows the eye to focus on objects at varying distances. The ciliary muscles change lens curvature, altering focal length. Maximum accommodation ability decreases with age (presbyopia).

Myopia (nearsightedness) occurs when the eye's optical power is too strong or the eyeball is too long. Distant objects focus in front of the retina. Diverging lenses correct myopia. Hyperopia (farsightedness) occurs when optical power is too weak or the eyeball is too short. Converging lenses correct hyperopia.

**A researcher studied vision correction:**

**Patient A (Myopia):**

- Far point: 50 cm (farthest clear vision distance)
- Can see clearly up to 50 cm without correction
- Cannot focus on distant objects

**Patient B (Hyperopia):**

- Near point: 75 cm (closest clear vision)
- Can see distant objects clearly
- Cannot focus on objects closer than 75 cm

**Normal vision:**

- Near point: ~25 cm (standard reading distance)
- Far point: infinity

**Corrective lenses:**

- Patient A prescribed  $-2.0$  D lenses
- Patient B prescribed  $+2.7$  D lenses

24. To correct Patient A's myopia for viewing distant objects (at infinity), the corrective lens must create a virtual image at:

- A. Infinity
- B. 25 cm
- C. 50 cm (the patient's far point)
- D. The retina

25. What is the focal length of Patient A's corrective lens ( $-2.0$  D)?

- A. +50 cm
- B. -50 cm
- C. +200 cm
- D. -200 cm

26. For Patient B to read at the normal near point (25 cm), the  $+2.7$  D lens creates a virtual image at approximately:

- A. 37 cm
- B. 50 cm
- C. 75 cm
- D. 100 cm

27. A compound microscope uses an objective lens with focal length 4 mm and an eyepiece with focal length 25 mm. What is the total magnification if the objective forms an image 160 mm from its lens?

- A.  $-40\times$
- B.  $-160\times$
- C.  $-400\times$
- D.  $-640\times$

28. If Patient A wears the prescribed glasses and looks at an object 2.0 m away, where does the corrective lens form the image?

- A. 50 cm in front of the lens (virtual image)
- B. 50 cm behind the lens (real image)
- C. 40 cm in front of the lens (virtual image)
- D. 67 cm behind the lens (real image)

### **PASSAGE 7 (Questions 29-32): Kinetic Molecular Theory and Gas Laws**

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The kinetic molecular theory (KMT) describes gas behavior at the molecular level. Key assumptions include: gas molecules are point particles in constant random motion, collisions are perfectly elastic, intermolecular forces are negligible, and average kinetic energy depends only on temperature.

**The ideal gas law combines Boyle's, Charles's, and Avogadro's laws:**

$$PV = nRT$$

Where P is pressure, V is volume, n is moles,  $R = 0.0821 \text{ L}\cdot\text{atm}/(\text{mol}\cdot\text{K})$  or  $8.314 \text{ J}/(\text{mol}\cdot\text{K})$ , and T is temperature in Kelvin.

**Average kinetic energy per molecule is:**

$$KE_{\text{avg}} = (3/2)k_B T$$

Where  $k_B = 1.38 \times 10^{-23} \text{ J/K}$  is Boltzmann's constant.

**Root mean square (rms) velocity is:**

$$v_{\text{rms}} = \sqrt{3RT/M}$$

Where M is molar mass in kg/mol. Lighter molecules move faster at the same temperature.

Real gases deviate from ideal behavior at high pressures (molecules occupy significant volume) and low temperatures (intermolecular forces become significant). The van der Waals equation corrects for these deviations:

$$(P + a(n/V)^2)(V - nb) = nRT$$

Where a corrects for intermolecular attractions and b corrects for molecular volume.

**Graham's law of effusion states that effusion rate is inversely proportional to the square root of molar mass:**

$$\text{Rate}_1/\text{Rate}_2 = \sqrt{M_2/M_1}$$

**Researchers studied gas behavior:****Experiment 1: Ideal gas**

- 2.0 moles of gas at 27°C
- Pressure: 4.0 atm
- Calculate volume

**Experiment 2: Gas mixtures**

- Container with He (4 g/mol), N<sub>2</sub> (28 g/mol), and O<sub>2</sub> (32 g/mol)
- Equal moles of each gas

- Total pressure: 9.0 atm

**Experiment 3: Effusion**

- Unknown gas effuses 2.0 times slower than He (4 g/mol)

**Experiment 4: Real gas behavior**

- CO<sub>2</sub> at high pressure deviates from ideal
- Measured pressure lower than predicted

29. In Experiment 1, what is the volume of the gas?

- A. 6.2 L
- B. 12.3 L
- C. 18.5 L
- D. 24.6 L

30. In Experiment 2, what is the partial pressure of  $N_2$ ?

- A. 1.5 atm
- B. 3.0 atm
- C. 4.5 atm
- D. 9.0 atm

31. What is the approximate molar mass of the unknown gas in Experiment 3?

- A. 1 g/mol
- B. 8 g/mol
- C. 16 g/mol
- D. 32 g/mol

32. The observation that  $CO_2$ 's measured pressure is lower than predicted by ideal gas law at high pressure is best explained by:

- A. Intermolecular attractive forces reducing pressure
- B. Molecules moving too slowly
- C. The gas becoming a liquid
- D. Measurement error in the apparatus

## **PASSAGE 8 (Questions 33-37): Electrochemistry of Concentration Cells**

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Concentration cells are galvanic cells where both electrodes consist of the same material but are immersed in solutions of different concentrations. The cell potential arises solely from the concentration difference, not from different electrode materials.

**For a concentration cell, the Nernst equation becomes:**

$$E_{\text{cell}} = (RT/nF) \ln([\text{concentrated}]/[\text{dilute}])$$

At 25°C (298 K):

$$E_{\text{cell}} = (0.0592/n) \log([\text{concentrated}]/[\text{dilute}])$$

The electrode in the dilute solution undergoes oxidation (anode), while the concentrated solution electrode undergoes reduction (cathode). Electrons flow from low concentration to high concentration compartment through the external circuit, while ions flow through the salt bridge to maintain electrical neutrality.

Concentration cells are important in biological systems. Nerve cells maintain  $\text{Na}^+$  and  $\text{K}^+$  concentration gradients across membranes, creating electrical potentials used for signaling. The Nernst equation predicts equilibrium potentials for ions across membranes.

**The relationship between cell potential and maximum work is:**

$$w_{\text{max}} = -nFE_{\text{cell}}$$

Where work is maximized when the cell operates reversibly.

**Researchers constructed concentration cells:**

### **Cell 1: Silver concentration cell**

- Anode:  $\text{Ag(s)} \mid \text{Ag}^+(0.010 \text{ M})$
- Cathode:  $\text{Ag(s)} \mid \text{Ag}^+(1.0 \text{ M})$
- Overall:  $\text{Ag}^+(1.0 \text{ M}) + \text{e}^- \rightarrow \text{Ag(s)} \rightarrow \text{Ag}^+(0.010 \text{ M}) + \text{e}^-$
- Temperature: 25°C

### **Cell 2: Copper concentration cell**

- Anode:  $\text{Cu(s)} \mid \text{Cu}^{2+}(0.001 \text{ M})$
- Cathode:  $\text{Cu(s)} \mid \text{Cu}^{2+}(0.100 \text{ M})$
- Temperature: 25°C

### Cell 3: Biological membrane

- Inside cell:  $[K^+] = 140 \text{ mM}$
- Outside cell:  $[K^+] = 4 \text{ mM}$
- $K^+$  flows through ion channels
- Temperature:  $37^\circ\text{C}$  (310 K)

### Cell 4: Operation over time

- Initial  $\text{Cu}^{2+}$  concentration gradient: 1.0 M to 0.01 M
- Cell allowed to operate until equilibrium

33. What is the cell potential for Cell 1 (silver concentration cell)?

- A. 0.000 V
- B. 0.030 V
- C. 0.059 V
- D. 0.118 V

34. What is the cell potential for Cell 2 (copper concentration cell)?

- A. 0.059 V
- B. 0.030 V
- C. 0.089 V
- D. 0.118 V

35. In Cell 1, which electrode is the anode (undergoes oxidation)?

- A. Neither, because both electrodes are silver
- B. The electrode in 0.010 M  $\text{Ag}^+$  solution
- C. The electrode in 1.0 M  $\text{Ag}^+$  solution
- D. Both electrodes equally

36. The equilibrium potential for  $K^+$  across the biological membrane (Cell 3) is approximately: (Use  $R = 8.314 \text{ J}/(\text{mol}\cdot\text{K})$ ,  $F = 96,485 \text{ C}/\text{mol}$ )

- A. -47 mV
- B. -71 mV
- C. -94 mV
- D. -118 mV

37. As Cell 4 operates over time and approaches equilibrium:

- A. The cell potential increases
- B. The concentration gradient increases
- C. The cell potential increases until work output is maximized
- D. The cell potential decreases as concentrations equalize

### **PASSAGE 9 (Questions 38-42): Fluids and Circulation**

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Fluid dynamics govern blood flow through the circulatory system. Pressure, flow rate, and resistance are related by:

$$\Delta P = Q \times R$$

Where  $\Delta P$  is pressure difference,  $Q$  is volumetric flow rate, and  $R$  is resistance.

**For laminar flow through cylindrical vessels, Poiseuille's equation describes flow rate:**

$$Q = (\pi r^4 \Delta P) / (8 \eta L)$$

Where  $r$  is vessel radius,  $\eta$  is fluid viscosity, and  $L$  is vessel length. Flow rate is extremely sensitive to radius (fourth power relationship).

**Blood pressure is measured as systolic/diastolic (e.g., 120/80 mmHg). Mean arterial pressure (MAP) approximates:**

$$\text{MAP} \approx \text{diastolic} + (1/3)(\text{systolic} - \text{diastolic})$$

**The continuity equation for incompressible fluids states that flow rate is constant throughout a system:**

$$A_1v_1 = A_2v_2$$

Where  $A$  is cross-sectional area and  $v$  is velocity. In narrow vessels, velocity increases; in wide vessels, velocity decreases.

**Bernoulli's equation relates pressure, velocity, and height:**

$$P_1 + (1/2)\rho v_1^2 + \rho gh_1 = P_2 + (1/2)\rho v_2^2 + \rho gh_2$$

Where  $\rho$  is fluid density and  $g$  is gravitational acceleration.

**Researchers studied cardiovascular hemodynamics:**

**Normal aorta:**

- Radius: 1.0 cm
- Blood flow rate: 5.0 L/min = 83 cm<sup>3</sup>/s
- Blood viscosity: 0.004 Pa·s
- Pressure at heart: 100 mmHg (13,300 Pa)

**Atherosclerotic narrowing:**

- Plaque reduces radius to 0.5 cm in affected region
- Length of narrowed region: 2 cm
- Same flow rate maintained

**Measurements:**

- Velocity in normal aorta: 26.5 cm/s
- Velocity in narrowed region: 106 cm/s
- Blood density: 1060 kg/m<sup>3</sup>

**Capillaries:**

- Individual capillary radius: 4 μm
- Total cross-sectional area of all capillaries: 2500 cm<sup>2</sup>
- Same total flow rate: 83 cm<sup>3</sup>/s

38. Using Bernoulli's equation (ignoring height changes), what is the approximate pressure in the narrowed atherosclerotic region compared to the normal aorta?

- A. Higher due to increased velocity
- B. Lower due to conservation of mass

- C. The same due to constant flow rate
- D. Lower due to increased kinetic energy of faster-moving blood

39. What is the average velocity of blood in the capillaries?

- A. 0.0033 cm/s
- B. 0.033 cm/s
- C. 0.33 cm/s
- D. 3.3 cm/s

40. If a patient's blood pressure is 120/80 mmHg, what is their approximate mean arterial pressure?

- A. 93 mmHg
- B. 100 mmHg
- C. 107 mmHg
- D. 120 mmHg

41. The relationship  $Q = (\pi r^4 \Delta P) / (8 \eta L)$  explains why small increases in blood vessel diameter have large effects on blood flow because:

- A. Flow rate depends linearly on radius
- B. Flow rate depends on the square of radius
- C. Flow rate depends on the fourth power of radius
- D. Viscosity decreases with increasing radius

42. If atherosclerosis reduces the radius of an artery from 1.0 cm to 0.8 cm, by what factor does the resistance to flow increase?

- A. 1.25× (25% increase)

- B.  $1.56\times$  (56% increase)
- C.  $2.44\times$  (144% increase)
- D.  $3.81\times$  (281% increase)

### **PASSAGE 10 (Questions 43-46): Quantum Mechanics and Atomic Structure**

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Quantum mechanics revolutionized understanding of atomic structure. The Bohr model, though limited, successfully explained hydrogen's emission spectrum. Electrons occupy discrete energy levels, and photon emission occurs when electrons transition between levels:

$$\Delta E = E_{\text{final}} - E_{\text{initial}} = h\nu = hc/\lambda$$

Where  $h = 6.626 \times 10^{-34} \text{ J}\cdot\text{s}$  is Planck's constant,  $\nu$  is frequency,  $c = 3.00 \times 10^8 \text{ m/s}$  is light speed, and  $\lambda$  is wavelength.

**For hydrogen, energy levels are:**

$$E_n = -13.6 \text{ eV} / n^2$$

Where  $n$  is the principal quantum number ( $n = 1, 2, 3\dots$ ). Ground state is  $n = 1$  ( $E_1 = -13.6 \text{ eV}$ ).

**The de Broglie wavelength relates particle momentum to wavelength:**

$$\lambda = h/p = h/(m\nu)$$

This wave-particle duality applies to all matter, though wavelength is negligible for macroscopic objects.

**The Heisenberg uncertainty principle states:**

$$\Delta x \cdot \Delta p \geq h/(4\pi)$$

Position and momentum cannot both be precisely known simultaneously.

**Quantum numbers describe electron states:**

- $n$  (principal): energy and distance from nucleus
- $l$  (angular momentum): 0 to  $n-1$  (s, p, d, f orbitals)
- $m_l$  (magnetic):  $-l$  to  $+l$  (orbital orientation)
- $m_s$  (spin):  $+1/2$  or  $-1/2$

The Pauli exclusion principle states no two electrons can have identical quantum numbers.

**Researchers studied atomic transitions:****Hydrogen emission:**

- Electron transitions from  $n = 3$  to  $n = 2$
- Photon emitted in visible spectrum (Balmer series)

**Electron acceleration:**

- Electron accelerated through 100 V potential difference
- Kinetic energy gained: 100 eV
- Calculate de Broglie wavelength

**Photoelectric effect:**

- Light with  $\lambda = 400$  nm strikes metal surface
- Work function: 3.0 eV
- Electrons ejected

**Uncertainty measurement:**

- Electron position known within  $\Delta x = 0.1$  nm
- Calculate minimum uncertainty in momentum

43. What is the energy of the photon emitted when an electron transitions from  $n = 3$  to  $n = 2$  in hydrogen?

A. 1.89 eV

- B. 3.40 eV
- C. 10.2 eV
- D. 12.1 eV

44. What is the wavelength of the photon emitted in the  $n = 3$  to  $n = 2$  transition?

- A. 364 nm (UV)
- B. 486 nm (blue-green)
- C. 656 nm (red)
- D. 820 nm (infrared)

45. An electron accelerated through 100 V gains kinetic energy of 100 eV ( $1.60 \times 10^{-17}$  J). What is its de Broglie wavelength? (electron mass =  $9.11 \times 10^{-31}$  kg)

- A. 0.123 nm
- B. 1.23 nm
- C. 12.3 nm
- D. 123 nm

46. For light with  $\lambda = 400$  nm striking a metal with work function 3.0 eV, what is the maximum kinetic energy of ejected electrons? ( $1 \text{ eV} = 1.602 \times 10^{-19}$  J)

- A. 0.1 eV
- B. 3.0 eV
- C. 3.1 eV
- D. 6.1 eV

## DISCRETE QUESTIONS (47-59)

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47. Which of the following has the highest boiling point?

- A. CH<sub>4</sub> (methane)
- B. CH<sub>3</sub>CH<sub>2</sub>CH<sub>2</sub>CH<sub>3</sub> (butane)
- C. CH<sub>3</sub>OH (methanol)
- D. Ne (neon)

48. In the reaction:  $\text{N}_2(\text{g}) + 3\text{H}_2(\text{g}) \rightleftharpoons 2\text{NH}_3(\text{g})$ ,  $\Delta H = -92 \text{ kJ/mol}$ , which change would increase the equilibrium yield of NH<sub>3</sub>?

- A. Increasing temperature and decreasing pressure
- B. Decreasing temperature and increasing pressure
- C. Increasing both temperature and pressure
- D. Decreasing both temperature and pressure

49. What is the oxidation state of sulfur in H<sub>2</sub>SO<sub>4</sub>?

- A. +6
- B. +4
- C. -2
- D. +2

50. A 1.0 M solution of a weak acid ( $K_a = 1.0 \times 10^{-5}$ ) has approximately what pH?

- A. 2.0
- B. 2.5
- C. 3.0

D. 5.0

51. Which statement correctly describes the first law of thermodynamics?

- A. Entropy of the universe is always increasing
- B. Energy cannot be created or destroyed, only converted between forms
- C. Heat flows spontaneously from cold to hot objects
- D. All processes are reversible given enough energy

52. The hybridization of carbon in  $\text{CO}_2$  is:

- A. sp
- B.  $\text{sp}^2$
- C.  $\text{sp}^3$
- D.  $\text{sp}^3\text{d}$

53. How many atoms are in a face-centered cubic (FCC) unit cell?

- A. 4
- B. 2
- C. 1
- D. 8

54. Which of the following molecules is nonpolar despite containing polar bonds?

- A.  $\text{H}_2\text{O}$
- B.  $\text{NH}_3$
- C.  $\text{CO}_2$

D.  $\text{CH}_3\text{Cl}$

55. In the reaction:  $2\text{A} + \text{B} \rightarrow \text{C}$ , if the concentration of A is doubled while B is held constant, the rate quadruples. The rate law is:

A.  $\text{Rate} = k[\text{A}][\text{B}]$

B.  $\text{Rate} = k[\text{A}]^2[\text{B}]$

C.  $\text{Rate} = k[\text{A}]^3[\text{B}]$

D.  $\text{Rate} = k[\text{A}]^2[\text{B}]^2$

56. The half-life of a first-order reaction is 20 minutes. After 60 minutes, what fraction of the original reactant remains?

A.  $1/2$

B.  $1/4$

C.  $1/6$

D.  $1/8$

57. Which particle has approximately the same mass as a neutron?

A. Proton

B. Electron

C. Alpha particle

D. Positron

58. A sound wave with frequency 1000 Hz travels through air at 340 m/s. What is its wavelength?

A. 0.17 m

B. 0.29 m

- C. 0.34 m
- D. 2.94 m

59. Two point charges (+2Q and -Q) are separated by distance d. Where along the line connecting them is the electric field zero?

- A. At distance  $d/3$  from +2Q
- B. At distance  $d/2$  from +2Q (midpoint)
- C. At distance  $2d/3$  from +2Q
- D. The electric field is never zero between opposite charges

## Critical Analysis and Reasoning Skills (CARS)

Time	Questions
90 minutes	53

### **PASSAGE 1 (Questions 1-6): Ethics of Artificial Intelligence**

The rapid advancement of artificial intelligence raises profound ethical questions that philosophy has struggled to address adequately. Traditional ethical frameworks—whether Kantian deontology, utilitarian consequentialism, or virtue ethics—were developed for human moral agents operating within human social contexts. Their application to artificial systems that may achieve or exceed human cognitive capabilities presents conceptual challenges that exceed mere technical extrapolation.

Consider the trolley problem, philosophy's favorite thought experiment. When transplanted to autonomous vehicles, this hypothetical scenario transforms into an engineering specification requiring precise algorithmic implementation. The vehicle's decision-making system must encode moral principles into executable code, crystallizing what were previously abstract philosophical debates into concrete design choices with real-world consequences. Yet the reification of ethical principles into algorithms reveals how philosophically underdetermined our moral intuitions truly are.

The challenge deepens when we consider artificial general intelligence (AGI)—systems with human-level cognitive abilities across diverse domains. If such systems achieve genuine understanding and perhaps

even consciousness, do they deserve moral consideration? The traditional criteria for moral status—sentience, rationality, autonomy—may apply, yet we lack consensus on whether these properties emerge from particular physical substrates or could manifest in silicon as readily as carbon-based neural networks.

More troubling is the "alignment problem": ensuring that increasingly capable AI systems pursue goals aligned with human values. The difficulty isn't merely technical but conceptual. Human values are inconsistent, context-dependent, and culturally variable. Whose values should AI systems encode? The utilitarian's aggregate welfare? The deontologist's categorical imperatives? Moreover, values evolve historically; any static encoding risks ossifying contemporary moral prejudices that future generations may reject.

Some philosophers argue for a pluralistic approach, programming AI systems with multiple ethical frameworks that can be weighted contextually. But this merely displaces the problem: who determines the appropriate weights, and by what criteria? The governance of AI development becomes itself an ethical challenge, involving questions of democracy, expertise, and representation that transcend traditional applied ethics.

The stakes are unprecedented. Unlike past technological revolutions, AI development may produce entities that surpass human intelligence—what philosopher Nick Bostrom calls "superintelligence." Such systems could recursively improve themselves, rapidly achieving capabilities that exceed human understanding. The margin for error vanishes; a superintelligent system pursuing misaligned goals could pose existential risks that preclude correction. The temporal paradox is acute: we must solve philosophical problems of profound difficulty before technological capabilities outpace our ability to control them, yet our philosophical methods—dialogue, reflection, gradual consensus-building—operate on timescales mismatched to rapid technical progress.

Perhaps the deepest challenge is epistemological. How can we specify human values formally when we ourselves lack complete self-knowledge? Our moral judgments often reflect unconscious biases, evolutionary adaptations to ancestral environments, and cultural conditioning rather than rational principles. Attempting to encode human values into AI systems may require confronting uncomfortable truths about the incoherence and contingency of our moral psychology. The project of AI ethics thus becomes unexpectedly reflexive, forcing us to understand ourselves more deeply precisely when we seek to create artificial minds.

1. The author's primary purpose in this passage is to:

A. Advocate for halting AI development until ethical problems are resolved

- B. Demonstrate that traditional ethical frameworks are inadequate for addressing AI challenges
- C. Argue that AI systems will inevitably develop independent moral status
- D. Propose a specific ethical framework for AI development

2. According to the passage, the trolley problem becomes more complex when applied to autonomous vehicles because:

- A. Vehicles operate at higher speeds than pedestrians
- B. Engineers lack philosophical training
- C. Multiple passengers may be affected simultaneously
- D. Abstract principles must be converted into concrete algorithmic decisions

3. The author's attitude toward a pluralistic approach to AI ethics (programming multiple ethical frameworks) can best be described as:

- A. Enthusiastic endorsement
- B. Cautious optimism
- C. Mild skepticism
- D. Fundamental rejection

4. Based on the passage, which of the following would the author most likely consider the greatest difficulty in solving the alignment problem?

- A. Insufficient computing power
- B. Lack of international cooperation
- C. The inconsistency and cultural variability of human values
- D. The resistance of AI companies to regulation

5. The author uses the term "reification" (paragraph 2) to suggest that:

- A. Ethical principles become easier to understand when coded
- B. Converting philosophical abstractions into algorithms reveals their underlying indeterminacy
- C. Computer programs are superior to philosophical arguments
- D. Engineers are better moral reasoners than philosophers

6. The passage suggests that the "temporal paradox" in AI development refers to:

- A. The possibility of AI systems existing across multiple timeframes
- B. Philosophical work requiring more time than AI capabilities advance
- D. The difficulty of predicting future human values
- C. Time travel becoming possible through superintelligence

## **PASSAGE 2 (Questions 7-12): Behavioral Economics and Rational Choice**

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Classical economic theory posits homo economicus—the rational economic actor who maximizes utility through consistent preferences and perfect information processing. This idealized agent served economics well as a simplifying assumption, enabling elegant mathematical models of market behavior. Yet decades of experimental research have revealed systematic deviations from rational choice theory so pervasive that they challenge the model's descriptive validity.

The pioneering work of Daniel Kahneman and Amos Tversky demonstrated that human decision-making violates fundamental axioms of rational choice. Consider the framing effect: people exhibit risk aversion when choices are framed in terms of gains but risk-seeking behavior when equivalent choices are framed as losses. A medical treatment with "90% survival rate" is preferred over one with "10% mortality rate," despite their logical equivalence. Rational actors should be invariant to such superficial reformulations, yet empirical evidence overwhelmingly confirms framing's influence.

Loss aversion—the principle that losses loom larger than equivalent gains—undermines another pillar of rational choice theory. People demand roughly twice as much to relinquish an object they possess than they would pay to acquire it (the endowment effect). This asymmetry between buying and selling prices contradicts the assumption of stable preferences independent of reference points. The implication is profound: value is not an intrinsic property of goods but depends on whether we frame transactions as acquisitions or losses.

These anomalies might seem mere cognitive errors—"bugs" in an otherwise rational system that better education could correct. But prospect theory, Kahneman and Tversky's alternative model, suggests these patterns reflect deep features of human psychology shaped by evolutionary pressures. Organisms facing uncertain environments may have benefited more from avoiding catastrophic losses than from maximizing expected value. Our ancestors who carefully protected existing resources likely survived better than those who optimized according to expected utility calculations.

The existence of such systematic biases poses challenges beyond academic interest. Financial markets, supposedly paradigmatic examples of rational choice, exhibit pronounced anomalies: momentum effects, where past returns predict future returns (contradicting the efficient market hypothesis), and equity premium puzzles, where stocks provide returns inexplicably higher than bonds relative to their risk. If professional investors in high-stakes environments display irrational patterns, what hope exists for rational choice theory's descriptive accuracy?

Some economists defend rational choice as a normative ideal even while conceding its descriptive failures. Markets might aggregate individual irrationalities into collective rationality through arbitrage and competition. Irrational traders lose money and exit markets, leaving rational actors to dominate. This evolutionary argument, however, requires timescales and competitive pressures that may not obtain in actual markets. Moreover, if systematic biases are hardwired through evolution, they may persist despite their suboptimality in modern contexts.

The behavioral economics revolution has spawned policy interventions called "nudges"—choice architecture designed to steer behavior toward welfare-improving outcomes without restricting options. Default options, salience manipulations, and commitment devices can dramatically alter behavior while preserving formal freedom of choice. Yet this approach raises normative questions: Who determines which outcomes improve welfare? When does nudging become manipulation? The economist's traditional role as neutral analyst transforms into paternalistic designer of choice contexts.

Perhaps most troubling is what behavioral economics reveals about human autonomy. If our preferences are unstable, context-dependent, and predictably manipulated, in what sense do we possess the rational agency that justifies market freedom? The foundations of liberal economics—consumer sovereignty, voluntary exchange, preference satisfaction—rest on assumptions about human rationality that behavioral research has called into question. The science meant to illuminate human decision-making may ultimately undermine the ethical premises of market societies.

7. The main argument of this passage is that:

- A. Behavioral economics proves humans are irrational
- B. Markets function efficiently despite individual irrationality
- C. Behavioral economics challenges both the descriptive and normative foundations of rational choice theory
- D. Nudges represent an unjustified expansion of government power

8. According to the passage, the framing effect demonstrates that:

- A. People prefer survival to mortality
- B. Medical decisions are especially difficult
- C. Logically equivalent presentations of choices produce different decisions
- D. Mathematical training improves decision-making

9. The author introduces evolutionary psychology (paragraph 4) primarily to:

- A. Prove that humans are irrational
- B. Suggest that behavioral patterns reflect adaptive responses rather than mere errors
- C. Argue for biological determinism in economics
- D. Undermine behavioral economics research

10. Which of the following scenarios would best illustrate the endowment effect described in the passage?

- A. Preferring to keep a coffee mug one owns rather than trading it for an identical mug
- B. Buying more of a product when its price decreases
- C. Valuing a coffee mug at \$5 and being willing to pay exactly \$5 to buy it
- D. Refusing to sell a coffee mug one values at \$8 for any price below \$16

11. The passage suggests that "nudges" are ethically problematic because:

- A. They restrict freedom of choice
- B. They always reduce welfare
- C. They involve determining welfare without clear justification for who decides
- D. They contradict evolutionary psychology

12. The author's tone toward rational choice theory can best be characterized as:

- A. Critical but acknowledging its historical utility and ongoing normative questions
- B. Dismissive and advocating complete rejection
- C. Defensive and protecting its empirical validity
- D. Neutral and presenting both sides equally

### **PASSAGE 3 (Questions 13-18): The Avant-Garde and Artistic Innovation**

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The concept of the avant-garde—literally "advance guard" in military terminology—emerged in the nineteenth century to describe artists who positioned themselves at the forefront of aesthetic innovation, challenging established conventions and anticipating future developments. Yet this martial metaphor contains assumptions about artistic progress that warrant scrutiny. Does art actually advance like an army toward victory, or does the avant-garde's self-conception reflect modernist ideology more than aesthetic reality?

Avant-garde movements defined themselves oppositionally. The Impressionists rejected academic salon painting's precise draftsmanship and historical subjects. The Cubists shattered perspective conventions that had governed Western art since the Renaissance. The Dadaists repudiated the very notion of art as aesthetic object, presenting urinals as sculpture and nonsense as poetry. Each movement proclaimed its rupture with tradition as necessary progress, implying that previous art forms had become exhausted or obsolete.

This progressivist narrative served the avant-garde's institutional needs. By positioning themselves as discoverers of new aesthetic territories, avant-garde artists could claim historical significance despite market rejection and critical incomprehension. The hostile reception from traditional critics became evidence of revolutionary importance rather than artistic failure. Those who didn't understand were simply behind history's march—a convenient way to transform marginalization into validation.

Yet the military metaphor misleads. Armies advance toward concrete objectives in zero-sum conflicts where one side's gain is another's loss. Artistic innovation operates differently. Impressionism didn't make Renaissance painting obsolete; both coexist as valid aesthetic approaches. The emergence of atonal music didn't invalidate tonal composition. Photography didn't eliminate painting. Rather than linear progress toward superior forms, art history exhibits proliferating diversity—an expanding repertoire of possibilities rather than displacement of the past by the future.

Moreover, the avant-garde's obsession with novelty may reflect market pressures more than aesthetic imperatives. In an art world increasingly commodified, where success requires distinguishing oneself in crowded markets, innovation becomes strategic necessity. The "shock of the new" generates publicity, critical attention, and ultimately commercial value. What appears as aesthetic radicalism may function as brand differentiation.

The postmodern turn challenged avant-garde assumptions directly. Appropriation artists like Sherrie Levine re-photographed Walker Evans's Depression-era photographs, presenting them as original works. This gesture simultaneously honored tradition and mocked originality fetishism. The avant-garde dream of perpetual revolution gave way to playful recombination of historical styles, acknowledging that aesthetic innovation had reached diminishing returns.

Some critics defend the avant-garde's legacy by distinguishing genuine innovation from mere novelty. True avant-garde work, they argue, doesn't just break rules but establishes new artistic possibilities—new ways of seeing, thinking, or feeling that expand human experience. Duchamp's readymades asked genuine philosophical questions about art's ontology. Schoenberg's atonality explored new emotional territories. By this account, not all innovation qualifies as avant-garde; only transformative breakthroughs merit the term.

Yet even this defense preserves problematic assumptions. It suggests that art progresses through revolutionary breakthroughs achieved by visionary individuals—a romantic myth of genius that obscures art's fundamentally social and collaborative nature. Artistic change typically occurs through gradual accumulation of small modifications across communities of practitioners, not through the isolated genius's dramatic ruptures. The avant-garde narrative, while historically influential, may ultimately distort more than it illuminates about how artistic practices actually evolve.

13. The author's main purpose in this passage is to:

A. Celebrate the achievements of avant-garde artists

- B. Provide a historical overview of avant-garde movements
- C. Explain why the avant-garde eventually failed
- D. Question the assumptions underlying the concept of the avant-garde

14. According to the passage, the avant-garde's "oppositional" self-definition served which function?

- A. Improved their artistic techniques
- B. Allowed them to reinterpret hostile reception as validation
- C. Helped them sell more artwork
- D. Made their work more accessible to the public

15. The author uses the phrase "zero-sum conflicts" (paragraph 4) to emphasize that:

- A. Avant-garde movements often fought among themselves
- B. Art galleries had limited space for exhibitions
- C. Military victories involve winners and losers unlike artistic innovation
- D. Some artistic styles are objectively better than others

16. Which of the following would most weaken the author's argument about the avant-garde?

- A. Evidence that Impressionist paintings now sell for high prices
- B. A demonstration that certain artistic techniques genuinely became obsolete
- C. Research showing that avant-garde artists were financially successful
- D. Discovery of previously unknown avant-garde movements

17. The author suggests that Sherrie Levine's re-photographing of Walker Evans's work represents:

- A. A lack of original artistic vision

- B. Copyright infringement
- C. Proof that photography is not art
- D. A postmodern critique of originality as artistic value

18. Based on the passage, the author would most likely agree that:

- A. All artistic innovation is motivated by commercial concerns
- B. The avant-garde contributed nothing of value to art history
- C. Artistic change occurs more gradually and collaboratively than the avant-garde narrative suggests
- D. Contemporary art is superior to traditional art forms

#### **PASSAGE 4 (Questions 19-24): Democracy and Expertise**

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Modern democracies face a persistent tension between popular sovereignty and expert knowledge. Democratic theory holds that political legitimacy derives from the consent of the governed, expressed through majoritarian decision-making. Yet contemporary policy challenges—climate change, pandemic response, financial regulation—involve technical complexities that exceed most citizens' expertise. Should democracies defer to expert judgment even when it conflicts with popular opinion, or does such deference undermine democracy's foundational commitment to political equality?

The technocratic position argues that good policy requires specialized knowledge that ordinary citizens lack. Medical experts understand epidemiology better than laypeople; climate scientists grasp atmospheric physics more thoroughly than the general public. Effective governance requires translating technical knowledge into policy, and this translation is itself a technical task best left to experts. Democracy should constrain its ambitions to selecting competent leaders while leaving substantive policy decisions to those with relevant expertise.

This view faces powerful objections. First, many policy questions involve value judgments that expertise cannot resolve. Climate policy requires balancing economic costs against environmental protection—a normative choice that technical knowledge informs but cannot determine. Experts can describe consequences of different policies but cannot dictate which values should guide policy selection. The technocratic position threatens to smuggle value judgments into ostensibly technical analysis, shielding normative choices from democratic scrutiny by presenting them as scientific necessities.

Second, experts disagree among themselves. Economists dispute optimal monetary policy; epidemiologists debate public health interventions. When experts conflict, political decisions become unavoidable. Selecting which experts to trust or how to adjudicate expert disagreement requires democratic judgment that cannot be delegated to expertise itself. The image of unified expert consensus guiding enlightened policy proves largely mythical; real expertise is contested and pluralistic.

Third, expertise creates power asymmetries. Those possessing specialized knowledge gain influence over policy that ordinary citizens cannot match. If democracy means political equality—equal voice in collective self-governance—then the concentration of effective power among credentialed experts violates democratic principles. The problem intensifies when expert communities exhibit demographic homogeneity or ideological conformity, raising questions about whose perspectives their "objective" analysis reflects.

Some democratic theorists propose that expertise and democracy can be reconciled through deliberative mechanisms. Rather than deferring to experts or ignoring them, citizens should engage in informed deliberation where expert testimony informs but doesn't determine collective decisions. Deliberative forums can educate citizens about technical matters while subjecting expert claims to critical public scrutiny. This approach preserves democratic equality while acknowledging expertise's legitimate role.

Yet deliberation's promise remains largely unfulfilled. Genuine deliberation requires time, cognitive effort, and good faith that contemporary political culture rarely provides. The proliferation of misinformation, partisan polarization, and attention scarcity undermines conditions for productive deliberation. Moreover, deliberative mechanisms risk becoming mere legitimization rituals—creating the appearance of democratic participation while expert agendas prevail.

Perhaps the deepest problem is epistemological. Democracy presumes that ordinary citizens possess sufficient competence to participate meaningfully in collective self-governance. But if modern governance requires expertise that citizens cannot acquire, this presumption becomes questionable. The democratic ideal of an informed citizenry capable of rational collective decision-making may be increasingly untenable in complex technological societies.

This doesn't imply abandoning democracy, but it does suggest recognizing its limitations. Democracy's value may lie less in generating optimal policy than in other considerations: respecting human dignity through self-governance, preventing tyranny through dispersed power, or maintaining political legitimacy through consent. If we value democracy for these reasons rather than its policy wisdom, we can acknowledge expertise's importance without abandoning democratic commitments. The challenge is institutional design that balances these competing values rather than pretending they automatically harmonize.

19. The main thesis of this passage is that:

- A. Democracy and expertise are fundamentally compatible through deliberation
- B. Modern democracies face difficult tensions between popular sovereignty and expert knowledge that lack easy resolution
- C. Technocratic governance is superior to democratic decision-making
- D. Citizens are too uninformed to participate meaningfully in governance

20. According to the passage, which of the following represents a normative rather than technical question?

- A. The atmospheric concentration of carbon dioxide
- B. The economic costs of reducing emissions
- C. Whether economic or environmental values should take priority
- D. The effectiveness of different emission reduction technologies

21. The author suggests that expert disagreement poses a problem for technocracy because:

- A. It proves experts are no better than ordinary citizens
- B. It reveals that all expertise is socially constructed
- C. Adjudicating between conflicting experts requires democratic judgment rather than expertise
- D. It makes policy-making impossible

22. Based on the passage, the author would most likely view deliberative democracy as:

- A. An ideal solution to democracy's problems
- B. Theoretically promising but practically difficult to implement
- C. A disguised form of technocracy
- D. A complete failure in all circumstances

23. The passage suggests that democracy might be valuable even if it produces suboptimal policy because:

- A. Optimal policy is impossible to determine
- B. Experts are always wrong
- C. It serves other important values like human dignity and preventing tyranny
- D. Policy outcomes don't matter

24. Which of the following scenarios best illustrates the "power asymmetries" concern raised in paragraph 5?

- A. A scientist conducting unbiased research
- B. Economic policymakers from similar elite educational backgrounds making decisions affecting diverse populations
- C. Doctors treating patients with specialized medical knowledge
- D. Engineers designing safer automobiles

### **PASSAGE 5 (Questions 25-30): Narrative Perspective in Modern Fiction**

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The technical innovation of stream-of-consciousness narration in modernist fiction represented more than stylistic experimentation; it embodied a philosophical shift in understanding human subjectivity. Traditional third-person omniscient narration presumes a transparent, stable self that can be observed and described from an external vantage point. Stream-of-consciousness technique, by contrast, suggests that consciousness is fluid, fragmented, and resistant to objective representation—accessible only through immersion in its temporal flow.

Virginia Woolf's *To the Lighthouse* exemplifies this narrative revolution. Rather than describing Mrs. Ramsay from outside, Woolf plunges readers into her consciousness, rendering thought as it occurs—associative, meandering, interrupted. The narrative voice dissolves into multiple perspectives that blend imperceptibly, questioning whether stable narrative authority exists at all. The novel's famous "Time Passes" section dispenses with human consciousness entirely, describing the house's decay in bracketed asides that relegate human events to parenthetical interruptions in nature's indifferent process.

This dissolution of narrative authority parallels developments in philosophy and psychology. William James's description of consciousness as a "stream" rather than discrete mental states influenced modernist

writers. Freud's theory of the unconscious suggested that much mental life occurs beyond awareness, undermining the notion of transparent self-knowledge. These intellectual currents converged in modernist fiction's rejection of omniscient narration as philosophically naive—a pretense that consciousness could be objectively described from nowhere.

Yet stream-of-consciousness narration faces its own limitations. The technique claims to represent consciousness as it actually occurs, but this claim is paradoxical. Consciousness is pre-linguistic; the stream of thought includes images, sensations, and inchoate feelings that resist verbal articulation. Any written representation necessarily imposes linguistic structure onto experiences that exceed language. The modernist claim to authentic psychological realism may substitute one artifice for another rather than achieving direct access to consciousness.

Moreover, stream-of-consciousness tends toward solipsism. By immersing readers in individual consciousness, it risks isolating that consciousness from social context. Mrs. Ramsay's thoughts interest us not merely as psychological phenomena but as responses to social situations—her role as wife and mother, her class position, her historical moment. Pure interiority without social embedding becomes paradoxically impoverished, severing individual consciousness from the intersubjective world that constitutes its meaning.

Some critics defend stream-of-consciousness against these charges by emphasizing its stylistic rather than representational ambitions. Joyce's *Ulysses* doesn't pretend to capture consciousness accurately; rather, it creates verbal artifacts that evoke consciousness's qualities through literary devices—free indirect discourse, interior monologue, temporal fragmentation. The technique's value lies in aesthetic achievement rather than psychological verisimilitude. By this account, the question of whether stream-of-consciousness "really" represents consciousness becomes as misguided as asking whether cubist painting "really" depicts faces.

Contemporary fiction has largely abandoned pure stream-of-consciousness while inheriting its insights. Writers employ multiple perspectives, unreliable narrators, and temporal fragmentation without committing to radical subjectivism. The return to more accessible narrative forms doesn't repudiate modernism's discoveries but integrates them into more capacious fictional structures. If omniscient narration proved philosophically untenable, pure stream-of-consciousness proved aesthetically limiting. The synthesis preserves self-consciousness about narrative perspective while recovering storytelling's traditional pleasures.

This historical trajectory suggests that literary techniques embody philosophical commitments that can be affirmed, questioned, or superseded. Narrative choices aren't merely formal decisions but implicit theories about consciousness, identity, and reality. The evolution from realist omniscience to modernist interiority

to contemporary pluralism maps philosophical transformations in understanding human subjectivity. Literary form and philosophical content prove inseparable; how we tell stories reflects and shapes how we conceive ourselves.

25. The author's primary argument is that:

- A. Stream-of-consciousness is superior to traditional narration
- B. Virginia Woolf was the greatest modernist writer
- C. Narrative techniques embody philosophical assumptions about consciousness and subjectivity
- D. Contemporary fiction has rejected modernism entirely

26. According to the passage, traditional third-person omniscient narration assumes that:

- A. Authors are more intelligent than readers
- B. Consciousness is stable and can be objectively observed
- C. Fiction should avoid psychological complexity
- D. Multiple perspectives confuse readers

27. The passage suggests that the "Time Passes" section of *To the Lighthouse* is significant because it:

- A. Describes the house's architectural features
- B. Provides relief from the novel's complexity
- C. Relegates human consciousness to peripheral status
- D. Demonstrates Woolf's knowledge of architecture

28. The author identifies which paradox in stream-of-consciousness narration?

- A. It claims to be realistic while using unrealistic techniques
- B. It's both popular and unpopular with readers

- C. It attempts to represent pre-linguistic consciousness using language
- D. It was developed by multiple writers independently

29. The comparison to cubist painting (paragraph 6) suggests that:

- A. Joyce was also a painter
- B. Stream-of-consciousness should be evaluated aesthetically rather than as accurate psychological representation
- C. Modernist literature and art are identical
- D. Representation is impossible in any medium

30. Based on the passage, contemporary fiction's relationship to modernism can best be described as:

- A. Complete rejection
- B. Uncritical acceptance
- C. Hostile opposition
- D. Selective integration of insights while recovering accessibility

### **PASSAGE 6 (Questions 31-36): Cultural Relativism and Universal Values**

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Anthropology's encounter with cultural diversity generated ethical relativism as a methodological necessity. To understand societies on their own terms rather than judging them by external standards, anthropologists suspended their own cultural assumptions, practicing what Clifford Geertz called "thick description"—interpreting practices within indigenous frameworks of meaning. This methodological relativism, however, readily collapsed into moral relativism: the claim that no culture's values can legitimately judge another's, since moral standards are culturally constructed rather than universal.

Cultural relativism offered important correctives to ethnocentrism. Victorian anthropologists notoriously interpreted non-Western practices through colonial prejudices, dismissing entire societies as "primitive" or "savage." Relativism demanded respect for cultural difference and recognition that Western modernity represented one cultural form among many rather than civilization's pinnacle. It undermined the ideological justifications for colonialism by denying European culture's superiority.

Yet moral relativism proves difficult to sustain consistently. Consider practices like female genital cutting, honor killings, or systematic gender subordination. Can anthropologists maintain that these practices are beyond cross-cultural judgment merely because they're culturally embedded? The relativist position seems to prohibit criticism of any cultural practice, however harmful, so long as it enjoys indigenous legitimacy. This threatens to abandon the victims of harmful practices to cultural traditions that they may themselves oppose.

The philosophical problems run deeper. Moral relativism is typically supported by descriptive cultural variation: different societies hold different moral beliefs. But descriptive diversity doesn't entail normative relativity—the inference from "is" to "ought" that philosophers since Hume have recognized as fallacious. That cultures disagree about slavery's morality doesn't imply slavery's moral status is culturally relative; it might mean some cultures are simply wrong.

Moreover, relativism generates pragmatic dilemmas. When cultures conflict, how should disputes be adjudicated? If each culture's values are valid within its own context, what principle resolves intercultural conflicts? The relativist might appeal to tolerance, but tolerance is itself a culture-specific Western liberal value that relativism cannot privilege without inconsistency. Thoroughgoing relativism undermines its own foundation by asserting universal respect for cultural difference while denying universal values.

Some anthropologists defend a moderate position: recognizing genuine cultural variation while maintaining certain universal moral minimum—basic human rights, prohibitions against torture, protections for the vulnerable. This compromise acknowledges that cultural practices warrant presumptive respect while maintaining that some actions violate standards applicable across cultures. The challenge lies in specifying which values are universal and justifying that universality without imposing Western cultural frameworks.

One approach grounds universal values in human capabilities and needs. Philosopher Martha Nussbaum argues that certain capabilities—health, bodily integrity, affiliation, practical reason—are universally required for human flourishing regardless of cultural context. Practices that systematically deny these capabilities can be legitimately criticized cross-culturally. This "capabilities approach" provides substantive normative content while remaining sensitive to how capabilities might be realized differently across cultures.

Critics worry that any specification of universal values inevitably reflects particular cultural perspectives masquerading as universal reason. The capabilities Nussbaum identifies look suspiciously Western-liberal: individual autonomy, freedom of conscience, political participation. Non-Western societies might prioritize different values—communal harmony, filial piety, spiritual transcendence—that the capabilities

approach marginalizes. The search for universal values may reproduce the ethnocentrism that relativism sought to overcome.

Perhaps the debate's terms require reconsideration. Rather than choosing between relativism and universalism, we might recognize both cultural specificity and overlapping consensus. Different cultures articulate values through distinct conceptual frameworks, yet these frameworks may converge on core moral prohibitions and aspirations. The task becomes identifying genuine agreement while respecting diversity in moral reasoning. This hermeneutic approach—interpretive rather than foundational—acknowledges both universal and particular elements in moral life without claiming either exhausts moral truth.

31. The main argument of this passage is that:

- A. Cultural relativism is completely correct
- B. Western values are universal
- C. Neither pure relativism nor simple universalism is adequate; a nuanced position is needed
- D. Anthropology should abandon ethical concerns entirely

32. According to the passage, "thick description" involves:

- A. Using many words to describe simple practices
- B. Interpreting practices within indigenous frameworks of meaning
- C. Judging other cultures by Western standards
- D. Collecting extensive quantitative data

33. The author suggests that the inference from cultural diversity to moral relativism is problematic because:

- A. Cultural diversity doesn't actually exist
- B. All cultures secretly share the same values
- C. Descriptive variation doesn't logically entail normative relativity

D. Anthropologists are biased

34. Which of the following scenarios would best illustrate the "pragmatic dilemma" for relativism described in paragraph 5?

- A. An anthropologist learning a new language
- B. Two cultures with conflicting practices needing to resolve a dispute
- C. A researcher choosing which culture to study
- D. A government funding anthropological research

35. The author presents Nussbaum's capabilities approach as:

- A. A perfect solution to the relativism debate
- B. An attempt to specify universal values while remaining culturally sensitive
- C. A disguised form of cultural imperialism
- D. Identical to traditional relativism

36. Based on the passage, the author would most likely agree that:

- A. All cultural practices deserve equal respect
- B. Western culture is superior to all others
- C. Some practices can be legitimately criticized across cultures while respecting cultural variation
- D. Anthropologists should not engage with ethical questions

### **PASSAGE 7 (Questions 37-42): The Evolution of Jazz**

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Jazz's historical trajectory defies simplistic narratives of artistic progress. Unlike classical music's evolution through increasingly complex harmonic and formal structures, jazz developed through cyclical returns to foundational elements, each return generating new possibilities rather than merely repeating the past. This spiral pattern—simultaneously conservative and revolutionary—reveals something essential about tradition's role in innovation.

Early jazz emerged from confluence: African rhythmic traditions, European harmonic frameworks, blues tonality, brass band instrumentation. New Orleans jazz of the 1920s established core vocabulary—improvisation over chord changes, collective polyphony, swing rhythm—that remained foundational despite subsequent transformations. Louis Armstrong's virtuosic solos demonstrated improvisation's expressive potential, shifting focus from collective improvisation to individual voice.

The swing era (1930s-1940s) represented jazz's commercial apex and artistic consolidation. Big bands like Duke Ellington's achieved sophisticated arrangements that balanced composition and improvisation. The music became more accessible, losing some of the earlier style's rough vitality but gaining orchestral possibility. Critics debate whether swing's popularity represented artistic compromise or jazz's maturation into a fully developed art form.

Bebop reacted against swing's commercialism through deliberate inaccessibility. Charlie Parker and Dizzy Gillespie accelerated tempos, employed complex harmonies, and privileged virtuosic improvisation over danceable rhythms. Bebop musicians rejected entertainment in favor of art, positioning jazz as modernist project parallel to abstract expressionism or serial composition. The music's difficulty became a badge of authenticity, separating "serious" artists from commercial sellouts.

Yet bebop's revolutionary rhetoric obscured its conservative foundation. The music retained standard song forms and harmonic progressions, simply elaborating them with greater complexity. What appeared as radical break actually demonstrated tradition's persistence; bebop musicians needed mainstream standards as frameworks for their improvisatory flights. Innovation occurred within tradition rather than replacing it.

Modal jazz (exemplified by Miles Davis's *Kind of Blue*) simplified bebop's complexity, using static modes rather than changing chord progressions. This minimalism paradoxically opened new improvisatory possibilities. Without harmonic motion dictating melodic choices, soloists achieved greater freedom. The return to simplicity—echoing early jazz's more spacious approach—generated innovations that complexity had foreclosed.

Free jazz pushed further, abandoning preset structures entirely. Ornette Coleman's free improvisation dispensed with predetermined harmonies, meters, and forms. The music approached pure sonic exploration, limited only by musicians' real-time interaction. Critics divided sharply: was this jazz's liberation from arbitrary constraints or its dissolution into incoherent noise?

Contemporary jazz exhibits stylistic pluralism. Musicians draw eclectically on the tradition's entire history, combining elements across supposedly distinct periods. Wynton Marsalis champions hard bop's

legacy; others incorporate hip-hop, electronic music, or global traditions. The search for the next revolutionary movement has given way to recognition that jazz's richness lies in its accumulated possibilities rather than linear progress toward some ultimate form.

This historical pattern suggests that artistic traditions function differently than scientific paradigms. Science (in Kuhn's account) advances through revolutionary paradigm shifts that render previous frameworks obsolete. Jazz demonstrates alternative developmental logic: each innovation adds to an expanding vocabulary without invalidating previous styles. The tradition becomes richer precisely through retaining its history rather than superseding it. Perhaps this model applies beyond jazz—artistic traditions as accumulations rather than progressions, where innovation means expanding possibility rather than displacing the past.

37. The author's main point about jazz history is that:

- A. Jazz has steadily improved over time
- B. Early jazz was superior to later developments
- C. Jazz has declined since the swing era
- D. Jazz develops through cyclical returns to foundational elements that generate new possibilities

38. According to the passage, bebop musicians:

- A. Rejected all connection to jazz tradition
- B. Simplified jazz to make it more accessible
- C. Positioned jazz as serious art while retaining traditional song forms
- D. Invented completely new musical structures

39. The passage suggests that modal jazz's use of static modes (fewer chord changes) demonstrates that:

- A. Simpler approaches can enable greater freedom
- B. Jazz was returning to primitive forms
- C. Bebop was completely misguided

D. Complexity is always superior to simplicity

40. The author uses the comparison to scientific paradigms (final paragraph) to argue that:

A. Jazz is scientific

B. Artistic traditions accumulate rather than supersede previous forms

C. Science is superior to art

D. Thomas Kuhn's theory is wrong

41. Based on the passage, the author would most likely view contemporary jazz's stylistic pluralism as:

A. A sign of jazz's death

B. Inferior to earlier unified styles

C. Proof that jazz has lost its direction

D. A natural result of jazz's accumulated historical richness

42. Which of the following best describes the author's attitude toward the various jazz styles discussed?

A. Clear preference for bebop over other styles

B. Belief that early jazz was the most authentic

C. Appreciation for how each style contributed to jazz's evolving vocabulary

D. Dismissal of free jazz as incoherent

### **PASSAGE 8 (Questions 43-47): Memory and Personal Identity**

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The persistence of personal identity over time poses a philosophical puzzle that psychology's discoveries about memory have deepened rather than resolved. Locke's classic criterion held that psychological continuity—especially memory—constitutes personal identity: you are the same person today as yesterday because you remember yesterday's experiences. Yet memory's unreliability threatens this criterion. If memory alone grounds identity, false memories would create false identities.

Psychological research reveals memory's reconstructive nature. Rather than retrieving stored records like files from a cabinet, remembering involves reconstructing past experiences from fragmentary traces, filling gaps with plausible inferences. This reconstructive process introduces systematic distortions. Elizabeth Loftus's research on false memories demonstrated that entirely fictitious events can be implanted through suggestion, producing vivid "memories" of experiences that never occurred.

These findings challenge identity's memorial foundations. If memories are constructed rather than retrieved, and if suggestion can create false memories indistinguishable from genuine ones, what grounds the distinction between authentic and inauthentic memories? Without that distinction, memory cannot provide identity's criterion—false memories would count equally with true ones in constituting who we are.

Philosophers have proposed refinements. Derek Parfit distinguishes between psychological connectedness (direct memory links between adjacent stages) and psychological continuity (overlapping chains of connections). Perhaps identity requires continuity rather than perfect memory. I am the same person as the child I was not because I remember all my childhood experiences, but because there exists a chain of overlapping connections linking present to past.

Yet this solution faces problems. If identity is constituted by psychological continuity, and continuity is compatible with complete memory loss between distant temporal stages, then identity becomes uncomfortably thin. The person I am today might share no memories with the child I was, connected only through intermediate stages I've also forgotten. At what point does continuity's attenuation make sameness questionable? The gradualist solution—identity as matter of degree—conflicts with our intuition that identity is all-or-nothing.

Neuroscience adds complexity. Neurological conditions like severe amnesia or dissociative identity disorder challenge identity's unity. Patients with anterograde amnesia cannot form new memories, living in perpetual present. Are they the same person they were before the condition's onset? Patients with dissociative identity disorder exhibit multiple personality states with different memories, preferences, and behavioral patterns. If distinct memory systems constitute distinct persons, one body might contain several identities.

Some philosophers argue that identity doesn't require strict psychological continuity. Perhaps biological continuity grounds identity: you are the same person because you're the same living organism, regardless of psychological changes. This animalist view avoids memory's problems but creates others. The view implies that irreversible coma patients remain fully persons (they're still living organisms), while it's unclear whether gradual biological replacement (cell turnover) preserves identity.

Perhaps the search for identity's criterion reflects a mistake. Rather than discovering what constitutes personal identity, we might be imposing conceptual order on phenomena that resist neat categorization. Personal identity might be a useful fiction—pragmatically indispensable but metaphysically suspect. We treat ourselves and others as persisting persons because social life requires it, not because reality contains the sort of entity our identity concepts presume.

This deflationary conclusion may seem unsatisfying, but it might better acknowledge human existence's actual complexity. Rather than forcing experience into philosophical categories, we might recognize that personhood involves multiple, sometimes conflicting aspects—biological continuity, psychological connections, social roles, narrative self-interpretation—that don't reduce to a single criterion. Identity's appearance of simplicity conceals underlying multiplicity that philosophical analysis reveals.

43. The main thesis of this passage is that:

- A. Locke's memory criterion is completely adequate
- B. Personal identity is grounded in biological continuity
- C. Memory's unreliability and psychological research complicate rather than resolve questions about personal identity
- D. Personal identity is an illusion with no basis in reality

44. According to the passage, memory is "reconstructive" in the sense that:

- A. It involves rebuilding past events from fragmentary traces rather than retrieving complete records
- B. It only works for recent events
- C. It requires conscious effort
- D. It cannot be studied scientifically

45. The passage suggests that Parfit's distinction between connectedness and continuity aims to:

- A. Prove that memory is perfectly reliable
- B. Allow for personal identity despite incomplete memory between distant life stages

- C. Demonstrate that identity doesn't exist
- D. Support biological theories of identity

46. Which of the following cases would best illustrate the problem of "identity attenuation" discussed in paragraph 5?

- A. A person remembering yesterday's breakfast
- B. An elderly person retaining vivid childhood memories
- C. A middle-aged person sharing no memories with their infant self, connected only through forgotten intermediate stages
- D. A person forming new memories normally

47. The "deflationary conclusion" mentioned near the end suggests that:

- A. Personal identity is simple and easily defined
- B. Identity might be a pragmatically useful concept rather than a discovered metaphysical fact
- C. All philosophical questions are meaningless
- D. Biology determines everything about identity

### **PASSAGE 9 (Questions 48-53): Aesthetic Experience and Beauty**

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The eighteenth-century invention of aesthetics as philosophical discipline presumed beauty's universality. Kant argued that aesthetic judgments, while not derived from concepts, nevertheless claim universal validity. When I judge a sunset beautiful, I'm not merely reporting my subjective reaction but implicitly asserting that others should share my response. This "universal subjective validity" distinguished aesthetic from merely personal preferences. I might prefer vanilla to chocolate ice cream without expecting others to share my preference, but aesthetic judgments carry normative force—they claim validity beyond individual taste.

Yet aesthetic disagreement is pervasive. People dispute artworks' merit endlessly without approaching consensus. If aesthetic judgments claim universality, why do they so frequently conflict? Kant's solution invoked an ideal aesthetic judge—someone with requisite sensibility, attention, and freedom from

prejudice. Actual disagreements reflect deviations from this ideal rather than genuine aesthetic relativity. But this solution risks being ad hoc: anyone disagreeing with me simply lacks proper aesthetic sensibility.

Twentieth-century theorists increasingly rejected beauty's universality. Aesthetic value, they argued, is culturally constructed. What counts as beautiful depends on historical and social context—artistic conventions, class formations, cultural capital. Bourdieu's sociology of taste demonstrated how aesthetic preferences correlate with social position, suggesting that disputes about beauty encode struggles over cultural authority rather than disagreements about objective aesthetic properties.

This sociological reduction of aesthetics faces objections. While cultural context certainly influences aesthetic experience, it doesn't follow that aesthetic value is merely conventional. Some artworks achieve cross-cultural recognition; others prove ephemeral despite contemporary acclaim. If aesthetic value were purely conventional, such patterns would be inexplicable. Moreover, the sociological approach struggles to account for aesthetic experience's phenomenology—the felt sense that beauty inheres in objects rather than being projected onto them by social forces.

Contemporary aesthetics has largely abandoned the search for beauty's universal criteria, embracing aesthetic pluralism. Different cultures and individuals pursue different aesthetic values—beauty, sublimity, elegance, rawness, authenticity. No single value exhausts aesthetic possibility. This pluralism avoids both naive universalism and reductive relativism. It acknowledges genuine diversity in aesthetic response while maintaining that aesthetic judgments can be rationally defended within particular traditions.

Yet pluralism creates new problems. If multiple aesthetic values exist without hierarchical ordering, how do we adjudicate conflicts? When indigenous land-use practices clash with preservationist aesthetics valuing "wilderness," which value should prevail? Pluralism without some principle for resolving conflicts threatens to collapse into relativism—the view that all aesthetic judgments are equally valid, which is functionally equivalent to saying none are valid.

Perhaps aesthetic disagreement's persistence suggests that aesthetics addresses questions without determinate answers. Mathematical disputes can be resolved through proof; empirical disputes through evidence. Aesthetic questions might lack such resolution procedures not because we haven't discovered them but because none exist. Beauty might be essentially contestable—subject to reasoned argument without admitting final demonstration.

This doesn't reduce aesthetics to arbitrary opinion. Aesthetic judgments can be better or worse reasoned, more or less sensitive to relevant features, supported by stronger or weaker arguments. What's impossible

is compelling agreement through pure reason or evidence. Aesthetic discourse becomes persuasion rather than proof, aimed at sharing experience rather than demonstrating truth.

If this characterization is apt, aesthetic disagreement's intractability reflects not our failure to discover beauty's true nature but rather the character of aesthetic questions themselves. We might then approach aesthetic disputes differently—not seeking to resolve them definitively but to articulate our perspectives more fully, understand others' more deeply, and refine our sensibilities through dialogue. The goal becomes aesthetic cultivation rather than aesthetic knowledge, participation in ongoing conversation rather than arriving at final answers.

48. The author's main argument is that:

- A. Kant's theory is entirely correct
- B. Beauty is purely conventional
- C. Aesthetic pluralism offers the best approach but faces challenges
- D. Aesthetic judgments are meaningless

49. According to the passage, Kant distinguished aesthetic judgments from mere preferences because aesthetic judgments:

- A. Are always correct
- B. Involve complex reasoning
- C. Claim universal validity even though they're subjective
- D. Are based on scientific evidence

50. The passage suggests that Bourdieu's sociology of taste demonstrates:

- A. Beauty is objective
- B. Aesthetic preferences correlate with social position
- C. All aesthetic judgments are wrong
- D. Culture has no influence on aesthetics

51. Which of the following scenarios would best illustrate an "essentially contestable" concept as described in the passage?

- A. A mathematical equation with one correct answer
- B. An empirical question resolved by experiments
- C. A value judgment subject to reasoned argument without final resolution
- D. A factual claim about history

52. The author suggests that if aesthetic questions lack resolution procedures, this indicates:

- A. Aesthetics is not worth studying
- B. All aesthetic judgments are equally invalid
- C. The questions themselves are essentially contestable rather than that we lack knowledge
- D. Beauty doesn't exist

53. Based on the passage, the author would most likely view aesthetic disagreement as:

- A. Evidence that aesthetics is worthless
- B. A problem that will eventually be solved
- C. An opportunity for dialogue and cultivation rather than a defect requiring resolution
- D. Proof that beauty is objective

# Biological and Biochemical Foundations of Living Systems

Time	Questions
95 minutes	59

## **PASSAGE 1 (Questions 1-5): Photosynthesis and Carbon Fixation**

Photosynthesis converts light energy into chemical energy through two interconnected stages: the light reactions and the Calvin cycle. The light reactions occur in thylakoid membranes where chlorophyll and accessory pigments absorb photons, exciting electrons that flow through photosystem II (PSII) and photosystem I (PSI). This electron transport generates a proton gradient driving ATP synthesis and produces NADPH.

The Calvin cycle occurs in the stroma, fixing atmospheric CO<sub>2</sub> into organic molecules. RuBisCO (ribulose-1,5-bisphosphate carboxylase/oxygenase) catalyzes the rate-limiting step: CO<sub>2</sub> combines with ribulose-1,5-bisphosphate (RuBP), forming two 3-phosphoglycerate molecules. ATP and NADPH from light reactions reduce 3-phosphoglycerate to glyceraldehyde-3-phosphate (G3P), which can form glucose or regenerate RuBP.

RuBisCO has a critical flaw: it can also catalyze photorespiration, where O<sub>2</sub> instead of CO<sub>2</sub> binds to RuBP. This produces phosphoglycolate, which must be recycled through an energy-consuming pathway in mitochondria and peroxisomes, yielding no sugar. Photorespiration wastes energy and carbon, reducing photosynthetic efficiency especially under hot, dry conditions when stomata close and O<sub>2</sub>/CO<sub>2</sub> ratio increases.

C<sub>4</sub> plants evolved a mechanism concentrating CO<sub>2</sub> around RuBisCO, minimizing photorespiration. In mesophyll cells, phosphoenolpyruvate (PEP) carboxylase fixes CO<sub>2</sub> into 4-carbon oxaloacetate, which converts to malate. Malate moves to bundle-sheath cells where it's decarboxylated, releasing CO<sub>2</sub> that RuBisCO fixes. This spatial separation maintains high CO<sub>2</sub> concentration around RuBisCO despite closed stomata.

CAM plants use temporal rather than spatial separation. At night with open stomata, PEP carboxylase fixes CO<sub>2</sub> into malate stored in vacuoles. During the day with closed stomata, malate is decarboxylated,

providing CO<sub>2</sub> for the Calvin cycle. This allows photosynthesis in arid environments with minimal water loss.

**Study data from comparing plant types:**

**C3 plants (wheat) at 25°C:**

- Optimal photosynthesis at 380 ppm CO<sub>2</sub>
- Photorespiration = 20% of gross photosynthesis
- At 40°C: photorespiration = 40%, net photosynthesis decreased 50%

**C4 plants (corn) at 25°C:**

- Continued photosynthesis even at low CO<sub>2</sub> levels
- Photorespiration <5% at all temperatures
- At 40°C: net photosynthesis only decreased 15%

**CAM plants (pineapple):**

- Stomata open at night, closed during day
- Water use efficiency 6× higher than C3 plants
- Growth rate 30% slower than C3 or C4 plants

1. RuBisCO's oxygenase activity increases at high temperatures primarily because:

- A. O<sub>2</sub> becomes more soluble relative to CO<sub>2</sub> at high temperatures
- B. The enzyme denatures
- C. Chlorophyll stops working
- D. Water evaporates from leaves

2. C4 plants minimize photorespiration by:

- A. Using a different form of RuBisCO that only binds CO<sub>2</sub>
- B. Concentrating CO<sub>2</sub> in bundle-sheath cells where RuBisCO operates
- C. Preventing O<sub>2</sub> production during light reactions
- D. Conducting photosynthesis only at night

3. CAM plants have lower growth rates than C<sub>3</sub>/C<sub>4</sub> plants because:
- A. They cannot perform photosynthesis during the day
  - B. Storing malate at night and processing it during day creates temporal separation that limits total CO<sub>2</sub> fixation
  - C. They lack chlorophyll
  - D. Their stomata never open
4. If a C<sub>3</sub> plant were placed in an atmosphere with 5% CO<sub>2</sub> and normal O<sub>2</sub>:
- A. Photorespiration would increase dramatically
  - B. Photorespiration would be virtually eliminated
  - C. The plant would die immediately
  - D. No change would occur
5. The fact that C<sub>4</sub> photosynthesis evolved independently in multiple plant families suggests:
- A. It provides no selective advantage
  - B. Strong selective pressure in hot, dry environments favored this adaptation
  - C. All plants will eventually become C<sub>4</sub>
  - D. C<sub>3</sub> photosynthesis is superior

**PASSAGE 2 (Questions 6-10): Cell Cycle and Cancer Biology**

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The eukaryotic cell cycle consists of interphase (G<sub>1</sub>, S, G<sub>2</sub>) and M phase (mitosis and cytokinesis). Progression through the cycle depends on cyclin-dependent kinases (CDKs) whose activity is regulated by cyclins—proteins that accumulate and degrade at specific cycle stages. Different cyclin-CDK complexes drive transitions: G<sub>1</sub>/S-Cdk initiates DNA replication, S-Cdk completes replication, and M-Cdk triggers mitosis.

Checkpoints ensure proper cycle progression. The G1 checkpoint (restriction point) assesses whether conditions favor division—adequate nutrients, growth signals, DNA integrity. Once past this point, cells commit to division. The G2/M checkpoint verifies DNA replication completion and checks for damage. The metaphase checkpoint (spindle assembly checkpoint) prevents anaphase until all chromosomes attach properly to spindle microtubules.

p53, the "guardian of the genome," functions as a tumor suppressor. DNA damage activates p53, which halts the cycle at G1 checkpoint, allowing repair. If damage is severe, p53 triggers apoptosis. p53 activates p21, a CDK inhibitor that blocks G1/S-Cdk. Over 50% of human cancers have p53 mutations, eliminating this critical checkpoint and allowing damaged cells to proliferate.

Rb (retinoblastoma protein) controls the G1/S transition. Hypophosphorylated Rb binds E2F transcription factors, preventing expression of S-phase genes. When G1/S-Cdk phosphorylates Rb, it releases E2F, permitting S-phase entry. Many cancers have defective Rb pathway—either Rb mutations, CDK overexpression, or cyclin amplification—causing uncontrolled proliferation.

Oncogenes promote cell division when overactive. Ras, a GTPase in growth factor signaling pathways, is mutated in 30% of cancers. Mutant Ras remains constitutively active, continuously signaling division even without growth factors. Proto-oncogenes become oncogenes through gain-of-function mutations—only one mutated allele needed (dominant). Tumor suppressors like p53 and Rb require loss of both alleles (recessive at cellular level) following Knudson's two-hit hypothesis.

### **Experimental data:**

#### **Cell line comparisons:**

- Normal cells: Stop dividing when confluent (contact inhibition)
- p53<sup>-/-</sup> cells: Continue dividing when confluent, form multilayers
- Rb<sup>-/-</sup> cells: Bypass G1 checkpoint, enter S phase without growth signals
- Ras mutant cells: Grow without added growth factors

#### **Drug treatments:**

- CDK inhibitor on normal cells: Arrested at G1
- CDK inhibitor on p53<sup>-/-</sup> cells: Arrested at G1 (CDK still required)
- MDM2 inhibitor (blocks p53 degradation): Increased p53 levels, cell cycle arrest

**Radiation experiment:**

- Normal cells: p53 accumulation, G1 arrest for 8 hrs, then resumed cycling
- p53<sup>-/-</sup> cells: No arrest, continued cycling, accumulated mutations

6. Cancer cells with mutant p53 continue dividing after DNA damage because:

- A. They cannot detect DNA damage
- B. They lack the checkpoint mechanism to halt division and allow repair
- C. Mutant p53 actively promotes division
- D. DNA damage doesn't affect cancer cells

7. Why does Ras mutation cause constitutive growth signaling?

- A. Mutant Ras cannot bind GTP
- B. Mutant Ras has impaired GTPase activity, remaining GTP-bound and active
- C. Ras overexpression causes excessive ATP production
- D. Mutant Ras blocks growth factor receptors

8. Rb phosphorylation by G1/S-Cdk leads to S-phase entry because:

- A. Phosphorylated Rb activates DNA polymerase
- B. Phosphorylated Rb releases E2F, allowing transcription of S-phase genes
- C. Phosphorylation destroys Rb permanently
- D. Phosphorylated Rb inhibits p53

9. MDM2 inhibitors cause cell cycle arrest by:

- A. Directly blocking CDKs

- B. Stabilizing p53 by preventing its degradation
- C. Damaging DNA
- D. Activating oncogenes

10. According to Knudson's two-hit hypothesis, a person who inherits one mutant Rb allele:

- A. Will definitely develop cancer
- B. Has increased cancer risk because only one more hit needed in a cell
- C. Is protected from cancer
- D. Cannot survive embryonic development

### **PASSAGE 3 (Questions 11-15): Cardiovascular Hemodynamics**

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The cardiovascular system delivers oxygen and nutrients while removing waste products through precisely regulated blood flow. Cardiac output (CO), the volume pumped per minute, equals stroke volume (SV) times heart rate (HR):  $CO = SV \times HR$ . Stroke volume depends on preload (venous return/end-diastolic volume), contractility (intrinsic force of contraction), and afterload (resistance against ejection).

The Frank-Starling mechanism describes how increased venous return enhances stroke volume. Greater end-diastolic volume stretches cardiac muscle fibers, increasing their contractile force—within physiological limits. This intrinsic regulation matches cardiac output to venous return without external signals. However, excessive stretch (as in heart failure) moves beyond optimal sarcomere length, reducing contractility.

Blood pressure regulation involves short-term and long-term mechanisms. Baroreceptors in carotid sinus and aortic arch detect pressure changes, sending signals to medullary cardiovascular centers. Decreased pressure triggers sympathetic activation: increased heart rate and contractility ( $\beta_1$  receptors), vasoconstriction ( $\alpha_1$  receptors), raising blood pressure within seconds. Increased pressure activates parasympathetic outflow (vagus nerve), decreasing heart rate.

Long-term pressure regulation involves the renin-angiotensin-aldosterone system (RAAS). Decreased renal perfusion stimulates renin secretion from juxtaglomerular cells. Renin cleaves angiotensinogen to angiotensin I, which ACE (angiotensin-converting enzyme) converts to angiotensin II. Angiotensin II causes vasoconstriction and stimulates aldosterone release, increasing  $\text{Na}^+$  reabsorption and blood volume.

Mean arterial pressure (MAP) equals cardiac output times total peripheral resistance (TPR):  $MAP = CO \times TPR$ . Exercise demonstrates coordinated cardiovascular adjustments. Skeletal muscle vasodilation decreases TPR, but sympathetic activation increases heart rate and contractility, raising CO sufficiently that MAP remains stable or slightly elevated despite decreased resistance.

### **Experimental observations:**

#### **Exercise response:**

- Rest: HR = 70 bpm, SV = 70 mL, CO = 4.9 L/min, MAP = 90 mmHg
- Moderate exercise: HR = 140 bpm, SV = 110 mL, CO = 15.4 L/min, MAP = 95 mmHg
- Muscle blood flow increased 10-fold
- Skin blood flow increased (thermoregulation)

#### **Heart failure patient:**

- End-diastolic volume: 200 mL (normal = 120 mL)
- Stroke volume: 40 mL (normal = 70 mL)
- Ejection fraction: 20% (normal = 58%)
- Reduced contractility despite increased preload

#### **Hemorrhage (blood loss):**

- Immediate: HR increased from 70 to 110 bpm
- Blood pressure dropped from 120/80 to 90/60 mmHg
- After 30 min: Sympathetic activation, vasoconstriction
- After 6 hrs: RAAS activation, Na<sup>+</sup> retention beginning

#### **ACE inhibitor treatment (blocks angiotensin II formation):**

- Blood pressure decreased
- Aldosterone decreased
- Reduced afterload improved heart failure symptoms

11. During exercise, MAP remains relatively stable despite muscle vasodilation because:

A. Vasoconstriction in other organs compensates

- B. Increased cardiac output compensates for decreased TPR
- C. Blood pressure doesn't depend on peripheral resistance
- D. Muscle vasodilation doesn't affect blood pressure

12. The Frank-Starling mechanism fails in severe heart failure because:

- A. The heart doesn't receive venous return
- B. Excessive fiber stretch moves beyond optimal sarcomere length for force generation
- C. The mechanism only works in healthy hearts
- D. Baroreceptors are damaged

13. Baroreceptor-mediated reflexes control blood pressure more rapidly than RAAS because:

- A. Neural reflexes work within seconds while RAAS requires hours to days
- B. RAAS doesn't affect blood pressure
- C. Baroreceptors are more sensitive
- D. RAAS only works during exercise

14. ACE inhibitors benefit heart failure patients by:

- A. Increasing heart rate
- B. Reducing afterload through decreased vasoconstriction and reducing preload through decreased blood volume
- C. Directly strengthening heart muscle
- D. Blocking all sympathetic activity

15. The low ejection fraction in heart failure indicates:

- A. The heart isn't filling adequately

- B. Despite large end-diastolic volume, the heart ejects only a small fraction due to poor contractility
- C. Normal heart function
- D. Excessive force of contraction

#### **PASSAGE 4 (Questions 16-20): Digestive Enzymes and Nutrient Absorption**

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Chemical digestion begins in the mouth where salivary amylase hydrolyzes starch into maltose and oligosaccharides. The stomach contributes pepsin, secreted as inactive pepsinogen and activated by HCl, which denatures proteins and creates optimal pH (1.5-2.0) for pepsin activity. Pepsin cleaves proteins into polypeptides.

The small intestine is the primary digestion and absorption site. Pancreatic secretions contain: (1) proteases (trypsin, chymotrypsin, carboxypeptidase) that cleave proteins to oligopeptides and amino acids, (2) pancreatic amylase completing starch digestion, (3) lipase hydrolyzing triglycerides to monoglycerides and fatty acids, and (4) nucleases digesting DNA/RNA. Bicarbonate in pancreatic juice neutralizes gastric acid, creating optimal pH (7-8) for pancreatic enzymes.

Brush border enzymes on intestinal epithelial microvilli complete digestion. Disaccharidases (maltase, sucrase, lactase) cleave disaccharides to monosaccharides. Peptidases hydrolyze oligopeptides to amino acids and dipeptides. Lactose intolerance results from lactase deficiency—undigested lactose remains in intestinal lumen, creating osmotic diarrhea as water follows the solute gradient, and colonic bacteria ferment lactose, producing gas.

Lipid digestion requires emulsification by bile salts, amphipathic molecules that disperse large lipid droplets into small micelles, increasing surface area for lipase action. Micelles ferry lipophilic products (fatty acids, monoglycerides, fat-soluble vitamins) to the brush border where they diffuse into enterocytes. Inside cells, fatty acids and monoglycerides reform triglycerides, package into chylomicrons, and enter lymphatic vessels (not directly into blood) because they're too large for intestinal capillaries.

Absorption mechanisms vary by nutrient type. Monosaccharides (glucose, galactose) use Na<sup>+</sup>-glucose symporters (secondary active transport), while fructose uses facilitated diffusion (GLUT5). Amino acids also use Na<sup>+</sup>-dependent cotransport. Water absorption is osmotic, following solute reabsorption—approximately 9 liters absorbed daily (2 from diet, 7 from secretions). Iron absorption is tightly regulated because the body cannot excrete excess iron; intestinal absorption adjusts to body stores.

**Clinical observations:**

**Lactase-deficient patient after 50g lactose:**

- Bloating, cramping, diarrhea within 30-60 min
- Breath hydrogen test: positive (bacterial fermentation)
- Stool: high osmolarity, low pH

**Pancreatic insufficiency patient (chronic pancreatitis):**

- Steatorrhea (fatty, oily stools)
- Weight loss despite adequate caloric intake
- Fat-soluble vitamin deficiencies (A, D, E, K)

- Normal disaccharide digestion

**Cholestatic disease (blocked bile ducts):**

- Steatorrhea
- Fat-soluble vitamin deficiencies
- Normal protein and carbohydrate absorption
- Elevated conjugated bilirubin

**Celiac disease (intestinal damage):**

- Reduced brush border enzyme activity
- Decreased absorption surface area
- Carbohydrate and protein malabsorption

16. Pancreatic insufficiency causes steatorrhea because:

- A. The stomach cannot digest fats
- B. Absence of pancreatic lipase prevents triglyceride hydrolysis
- C. Bile production stops
- D. Fatty acids cannot be absorbed across membranes

17. Why do patients with blocked bile ducts develop fat-soluble vitamin deficiencies?

- A. Bile contains these vitamins
- B. Without bile salt emulsification, lipid digestion and micelle formation are impaired
- C. Pancreatic enzymes cannot work without bile
- D. The liver stops producing these vitamins

18. Lactose intolerance causes diarrhea through:

- A. Direct stimulation of intestinal motility
- B. Unabsorbed lactose creating osmotic gradient that retains water in lumen
- C. Lactose toxicity to intestinal cells
- D. Immune reaction to lactose

19. Glucose absorption requires  $\text{Na}^+$  gradient because:

- A. Glucose and  $\text{Na}^+$  chemically bind
- B. The  $\text{Na}^+$ -glucose symporter uses  $\text{Na}^+$  gradient (established by  $\text{Na}^+/\text{K}^+$ -ATPase) to drive glucose uptake
- C.  $\text{Na}^+$  activates glucose
- D. Glucose creates the  $\text{Na}^+$  gradient

20. Chylomicrons enter lymphatic vessels rather than blood capillaries because:

- A. They are too large to enter intestinal capillaries
- B. Blood cannot transport lipids
- C. Lymphatic vessels have digestive enzymes
- D. Capillaries actively reject chylomicrons

### **PASSAGE 5 (Questions 21-25): Hormonal Regulation of Metabolism**

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Insulin and glucagon maintain blood glucose homeostasis through opposing actions. Insulin, secreted by pancreatic  $\beta$ -cells in response to elevated blood glucose, promotes glucose uptake (especially in muscle and adipose tissue), glycogen synthesis, fat storage, and protein synthesis. At the molecular level, insulin activates the PI3K-Akt pathway: insulin binds receptor tyrosine kinase, triggering phosphorylation cascades that translocate GLUT4 transporters to cell membranes and activate enzymes like glycogen synthase.

Glucagon, from pancreatic  $\alpha$ -cells, responds to low blood glucose by mobilizing stored energy. It activates G-protein coupled receptors, increasing cAMP, which activates protein kinase A (PKA). PKA phosphorylates and inactivates glycogen synthase while activating phosphorylase kinase, promoting

glycogen breakdown. Glucagon also stimulates gluconeogenesis (glucose synthesis from non-carbohydrate sources) and inhibits glycolysis in liver.

Type 1 diabetes results from autoimmune destruction of  $\beta$ -cells, eliminating insulin production. Without insulin, cells cannot take up glucose despite hyperglycemia; cells catabolize fats and proteins for energy, producing ketone bodies that cause metabolic acidosis (diabetic ketoacidosis). Type 2 diabetes involves insulin resistance—target cells don't respond adequately to insulin—often associated with obesity. Pancreas initially compensates by secreting more insulin (hyperinsulinemia), but  $\beta$ -cells eventually fail.

Thyroid hormones (T3, T4) regulate metabolic rate. They increase cellular respiration, protein synthesis, and sensitivity to catecholamines. Hyperthyroidism causes increased BMR, weight loss despite increased appetite, heat intolerance, and tachycardia. Hypothyroidism causes decreased BMR, weight gain, cold intolerance, and bradycardia. Thyroid hormones act through nuclear receptors, altering gene transcription—effects appear slowly (hours to days).

Cortisol, the primary glucocorticoid, promotes gluconeogenesis, protein catabolism, and fat redistribution. Chronic cortisol elevation (Cushing's syndrome) causes hyperglycemia, muscle wasting, central obesity, and immunosuppression. Cortisol follows diurnal rhythm, peaking in morning and declining through the day.

**Patient studies:**

**Untreated Type 1 diabetic:**

- Blood glucose: 350 mg/dL (normal = 90 mg/dL)
- Blood insulin: undetectable
- Urinary glucose: present (exceeded renal threshold)
- Ketones: present in blood and urine
- Weight loss: 20 lbs in 2 months

**Type 2 diabetic (early stage):**

- Fasting glucose: 160 mg/dL
- Insulin: 3× normal levels (hyperinsulinemia)

- Oral glucose tolerance test: glucose remained elevated for 3 hours
- BMI: 35 (obese)

**Hyperthyroid patient:**

- TSH: <0.1  $\mu$ IU/mL (suppressed)
- Free T4: 4× normal
- BMR: increased 40%
- Weight: decreased 15 lbs in 6 weeks
- Resting heart rate: 110 bpm

**Glucagon response test:**

- Healthy subject given glucagon
- Within 30 min: blood glucose increased from 90 to 120 mg/dL

- Glycogen phosphorylase activity: increased 5-fold
- Liver showed decreased glycogen content

21. Type 1 diabetics develop ketoacidosis because:

- A. Excessive glucose is converted to ketones
- B. Without insulin, cells metabolize fats for energy, producing ketone bodies
- C. Glucagon directly produces ketones
- D. The kidneys fail to excrete ketones

22. Why does Type 2 diabetes initially feature hyperinsulinemia?

- A. Insulin resistance requires more insulin to achieve the same glucose-lowering effect
- B.  $\beta$ -cells become overactive
- C. Glucagon inhibits insulin degradation
- D. Dietary sugar stimulates excessive insulin

23. Hyperthyroid patients lose weight despite increased appetite because:

- A. Food isn't digested properly
- B. Thyroid hormones increase metabolic rate, burning more calories
- C. They develop diabetes
- D. Appetite increase is imaginary

24. Glucagon increases blood glucose by:

- A. Stimulating glucose absorption from intestines

- B. Activating glycogen breakdown and gluconeogenesis in liver
- C. Blocking insulin receptors
- D. Converting proteins directly to glucose in blood

25. In Type 1 diabetes, urinary glucose appears because:

- A. The kidneys are damaged
- B. Blood glucose exceeds renal reabsorption capacity
- C. Glucose is produced in the kidneys
- D. Insulin directs glucose to urine

### **PASSAGE 6 (Questions 26-30): Mendelian Genetics and Pedigree Analysis**

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Mendel's laws describe inheritance patterns. The law of segregation states that allele pairs separate during gamete formation, each gamete receiving one allele. The law of independent assortment states that alleles for different genes segregate independently (applies to genes on different chromosomes or far apart on the same chromosome).

Dominant alleles mask recessive alleles in heterozygotes. Complete dominance produces identical phenotypes in AA and Aa individuals. Incomplete dominance yields intermediate heterozygote phenotypes—red and white flowers producing pink heterozygotes. Codominance shows both alleles' products simultaneously, as in ABO blood types where IA and IB alleles both express in IAIB individuals (Type AB blood).

Sex-linked traits involve genes on sex chromosomes. X-linked recessive disorders (hemophilia, color blindness) affect males predominantly because males have one X chromosome; a single recessive allele produces the phenotype. Females require two recessive alleles. Carrier females (heterozygous) don't express the phenotype but can transmit it. Male-to-male transmission never occurs for X-linked traits because fathers give Y chromosomes to sons.

Pedigree analysis reveals inheritance patterns. Autosomal recessive traits skip generations, require two affected parents to produce affected offspring (usually), and affect males and females equally. Autosomal dominant traits appear in every generation with at least one affected parent, affect both sexes equally, and

affected individuals often heterozygous. X-linked recessive traits affect mostly males, who receive them from carrier mothers, and show no male-to-male transmission.

Probability calculations use the product rule (independent events multiply) and sum rule (mutually exclusive events add). For dihybrid crosses ( $AaBb \times AaBb$ ), offspring ratios follow 9:3:3:1 for complete dominance, assuming independent assortment.

### **Pedigree and genetic scenarios:**

#### **Family A pedigree (trait appears to be X-linked recessive):**

- Affected males in generations I and III
- No male-to-male transmission
- Generation II has carrier females
- Affected male (III-2) has unaffected parents but affected grandfather

#### **Family B (autosomal recessive trait):**

- Two unaffected parents produce affected child
- 1/4 of offspring affected
- Males and females affected equally

#### **Blood type problem:**

- Mother: Type A (could be  $I^A I^A$  or  $I^A i$ )
- Father: Type B (could be  $I^B I^B$  or  $I^B i$ )
- Children: One Type O (must be  $ii$ )
- Question: What are parent genotypes?

#### **Dihybrid cross:**

- P: Round Yellow ( $RRYY$ )  $\times$  Wrinkled Green ( $rryy$ )
- F1: All Round Yellow ( $RrYy$ )
- F2: 9 Round Yellow : 3 Round Green : 3 Wrinkled Yellow : 1 Wrinkled Green

#### **Test cross:**

- Individual with dominant phenotype (could be  $AA$  or  $Aa$ )
- Crossed with homozygous recessive ( $aa$ )
- If any offspring show recessive phenotype, individual is heterozygous

26. In Family A, the trait is most likely X-linked recessive because:

- A. Only males are affected and there's no male-to-male transmission
- B. The trait affects everyone

- C. Females cannot carry the trait
- D. It appears in every generation

27. For the blood type problem, if a child is Type O, the parent genotypes must be:

- A.  $I^A I^A$  and  $I^B I^B$
- B.  $I^A i$  and  $I^B i$  (both heterozygous)
- C.  $I^A I^B$  and  $ii$
- D. Cannot be determined

28. The 9:3:3:1 ratio in the F<sub>2</sub> generation of a dihybrid cross indicates:

- A. The genes are linked
- B. The genes assort independently
- C. Incomplete dominance
- D. Sex linkage

29. If two heterozygous parents ( $Aa \times Aa$ ) have three unaffected children, what is the probability their fourth child will be affected ( $aa$ )?

- A. 0 (they've already had three unaffected)
- B. 1/4 (each child is independent event)
- C. 3/4
- D. 1/2

30. A test cross with an individual of unknown genotype ( $A\_$ ) produces 50% dominant and 50% recessive offspring. The unknown individual is:

- A. Homozygous dominant ( $AA$ )

- B. Heterozygous (Aa)
- C. Homozygous recessive (aa)
- D. Cannot be determined

### **PASSAGE 7 (Questions 31-35): Viral Replication and Host Interactions**

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Viruses are obligate intracellular parasites requiring host cellular machinery for replication. A viral particle (virion) consists of genetic material (DNA or RNA, single or double-stranded) enclosed in a protein coat (capsid), sometimes surrounded by a lipid envelope derived from host membranes. Viral replication follows stages: attachment to host receptors, entry, uncoating, replication, assembly, and release.

Bacteriophages (bacterial viruses) demonstrate two reproductive cycles. Lytic cycles produce progeny viruses that lyse the host cell. Phage DNA directs synthesis of viral components using host ribosomes and enzymes; new phages assemble and lyse the cell, releasing hundreds of virions. Lysogenic cycles integrate phage DNA (prophage) into the bacterial chromosome, replicating with the host genome. Environmental stress can induce prophage excision, initiating the lytic cycle.

Animal viruses show greater diversity. Enveloped viruses (influenza, HIV) acquire membranes by budding from host cells, incorporating viral glycoproteins. Non-enveloped viruses (polio, adenovirus) release via cell lysis. Retroviruses (HIV) use reverse transcriptase to synthesize DNA from RNA genomes, integrating into host chromosomes as proviruses. This integration makes retroviruses difficult to eliminate.

Viral specificity depends on attachment proteins recognizing host receptors. HIV's gp120 binds CD4 and coreceptors (CCR5 or CXCR4) on T cells. Influenza hemagglutinin binds sialic acid residues on respiratory epithelial cells. Host range—species and cell types infected—is determined by receptor compatibility.

Antiviral strategies target unique viral processes. Nucleoside analogs (acyclovir, AZT) inhibit viral polymerases more than host polymerases. Protease inhibitors block viral protein processing in HIV. Neuraminidase inhibitors prevent influenza virion release. Vaccines stimulate immunity before infection; most effective vaccines target viruses with stable surface proteins.

#### **Experimental observations:**

**Bacteriophage infection study:**

- T4 phage + E. coli: Cell lysis at 30 min, 200 phages released per cell
- Lambda phage + E. coli (optimal conditions): Lysogenic pathway, no lysis
- Lambda phage + E. coli (UV exposure): Prophage induced, lytic cycle initiated

**HIV infection:**

- Primary infection: CD4+ T cells
- Viral budding observed (no cell lysis)
- Reverse transcriptase inhibitor (AZT): Reduced viral load by 90%
- Protease inhibitor: Prevented production of mature viral particles

**Influenza experiment:**

- Neuraminidase inhibitor treatment: Virions produced but unable to release from infected cells
- Accumulated at cell surface

**Receptor specificity:**

- HIV with mutated gp120: Cannot infect CD4+ cells
- HIV can infect cells expressing CD4 but not CCR5/CXCR4: No infection

**Vaccine development:**

- Influenza: Annual vaccines needed (hemagglutinin and neuraminidase drift)
- Measles: Single vaccination provides lifelong immunity (stable proteins)

31. Lambda phage enters lysogenic cycle under optimal conditions because:

- A. The host cell is too strong for lytic cycle
- B. Lysogenic cycle allows viral genome persistence when conditions favor host survival
- C. Lambda phage cannot perform lytic cycle
- D. It lacks the genes for lysis

32. Reverse transcriptase inhibitors like AZT specifically target retroviruses because:

- A. Only retroviruses use reverse transcriptase to synthesize DNA from RNA
- B. All viruses require reverse transcriptase
- C. Host cells don't use DNA polymerase
- D. Retroviruses lack DNA entirely

33. HIV requires both CD4 and a coreceptor (CCR5/CXCR4) for infection because:

- A. Both are needed for complete viral entry
- B. CD4 alone is sufficient
- C. The coreceptor is optional
- D. HIV doesn't actually bind these molecules

34. Neuraminidase inhibitors prevent influenza spread by:

- A. Blocking viral attachment to cells
- B. Preventing viral release; virions remain attached to infected cell surface
- C. Destroying viral RNA
- D. Blocking host immune response

35. Annual influenza vaccines are needed while measles vaccine provides lifelong immunity because:

- A. Influenza viruses undergo antigenic drift (mutations in surface proteins)
- B. Measles is less common
- C. Influenza doesn't stimulate immunity
- D. Measles vaccine contains antibiotics

## **PASSAGE 8 (Questions 36-40): Population Genetics and Hardy-Weinberg Equilibrium**

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Population genetics studies allele frequency changes in populations. The Hardy-Weinberg principle states that allele and genotype frequencies remain constant across generations in the absence of evolutionary forces. For a gene with two alleles (A and a) with frequencies  $p$  and  $q$  (where  $p + q = 1$ ), genotype frequencies in Hardy-Weinberg equilibrium are:  $p^2$  (AA),  $2pq$  (Aa), and  $q^2$  (aa).

Five conditions must be met for Hardy-Weinberg equilibrium: (1) no mutations, (2) random mating, (3) no gene flow (migration), (4) infinite population size (no genetic drift), and (5) no natural selection. Violation of any condition causes evolutionary change. Mutations introduce new alleles. Non-random mating (inbreeding, assortative mating) alters genotype frequencies without changing allele frequencies. Gene flow introduces alleles from other populations. Genetic drift causes random frequency changes, especially in small populations. Natural selection changes frequencies based on fitness differences.

Natural selection acts on phenotypes. Directional selection favors one extreme, shifting allele frequencies. Stabilizing selection favors intermediates, reducing variation. Disruptive selection favors both extremes, potentially leading to polymorphism. Selection strength depends on fitness differences and allele dominance. Recessive lethal alleles persist at low frequencies because heterozygotes don't express deleterious phenotypes.

Heterozygote advantage maintains polymorphism. Sickle cell allele (HbS) persists in malaria-endemic regions because HbA/HbS heterozygotes resist malaria better than HbA/HbA homozygotes while avoiding sickle cell disease (HbS/HbS). Both alleles persist at intermediate frequencies.

Genetic drift has stronger effects in small populations. Founder effects occur when small groups establish new populations, carrying only subset of original genetic variation. Bottleneck effects reduce genetic diversity when populations shrink dramatically then recover. Both can lead to fixation of alleles that weren't necessarily advantageous.

### **Population genetics scenarios:**

#### **Population A (10,000 individuals):**

- Allele frequencies:  $A = 0.7$ ,  $a = 0.3$
- Genotype frequencies:  $AA = 0.49$ ,  $Aa = 0.42$ ,  $aa = 0.09$
- Next generation: Same frequencies (Hardy-Weinberg equilibrium maintained)

**Population B (PKU disease, autosomal recessive):**

- Disease frequency ( $aa$ ) =  $1/10,000 = 0.0001$
- Calculate carrier frequency ( $2pq$ )
- $q^2 = 0.0001$ , so  $q = 0.01$
- $p = 0.99$
- Carrier frequency:  $2(0.99)(0.01) = 0.0198 \approx 2\%$

**Sickle cell in West Africa:**

- HbA/HbA: Susceptible to malaria
- HbA/HbS: Malaria resistant, healthy
- HbS/HbS: Sickle cell disease
- HbS frequency maintained at 10-15% in malaria regions

- HbS frequency  $<1\%$  in non-malaria regions

**Founder effect—island population:**

- Mainland: Allele B frequency = 0.05
- Island founded by 20 individuals
- Island: Allele B frequency = 0.30 (one founder carried multiple B alleles)

**Selection against recessive lethal:**

- Initial  $q = 0.10$
- After one generation of selection:  $q$  decreased to 0.09
- Slow decline because most alleles hidden in heterozygotes

36. In Population B (PKU example), carriers are much more frequent than affected individuals because:

- A. PKU is not really genetic
- B. Carriers ( $Aa$ ) represent  $2pq$  while affected ( $aa$ ) represent  $q^2$ , and  $q^2 \ll 2pq$  when  $q$  is small
- C. Affected individuals die before birth
- D. Carriers have symptoms too

37. The high frequency of HbS allele in malaria-endemic regions demonstrates:

- A. Random genetic drift
- B. Heterozygote advantage maintaining balanced polymorphism
- C. Mutation pressure
- D. Founder effect

38. The island founder effect caused allele B frequency to be much higher than mainland because:

- A. The small founding group didn't represent the mainland genetic diversity
- B. Natural selection favored B on islands
- C. Mutation rates are higher on islands
- D. Large populations always have lower allele frequencies

39. In a population in Hardy-Weinberg equilibrium with allele frequencies  $p = 0.6$  and  $q = 0.4$ , what proportion of the population is heterozygous?

- A. 0.16
- B. 0.36
- C. 0.48
- D. 0.64

40. Recessive lethal alleles decline slowly under selection because:

- A. Mutations keep producing them
- B. Most copies are hidden in heterozygotes who have normal phenotypes
- C. They provide advantages
- D. Selection doesn't affect recessive alleles

### **PASSAGE 9 (Questions 41-45): Protein Synthesis and Post-Translational Modification**

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Translation converts mRNA sequences into polypeptides through ribosomal machinery. Ribosomes contain large and small subunits, each composed of rRNA and proteins. The small subunit binds mRNA and mediates codon-anticodon interactions; the large subunit catalyzes peptide bond formation through its peptidyl transferase activity (catalyzed by rRNA, not protein—ribozymes).

Translation proceeds in three stages. Initiation begins when the small ribosomal subunit binds mRNA's 5' cap, scans for the start codon (AUG), and initiates at the first AUG. Initiator tRNA (carrying methionine) pairs with AUG. The large subunit then joins, forming the complete initiation complex. Elongation involves aminoacyl-tRNA entering the A site, peptide bond formation transferring the growing chain to

the new amino acid, and translocation shifting the ribosome three nucleotides. Termination occurs when stop codons (UAA, UAG, UGA) enter the A site; release factors bind, hydrolyzing the polypeptide from tRNA.

The genetic code is degenerate—multiple codons encode the same amino acid—but non-ambiguous (each codon specifies only one amino acid). The third codon position shows wobble; non-standard base pairing allows single tRNAs to recognize multiple codons. Codon usage bias affects translation efficiency; rare codons slow translation when cognate tRNAs are scarce.

Post-translational modifications expand protein diversity. Phosphorylation regulates protein activity by adding phosphate groups to serine, threonine, or tyrosine residues. Glycosylation adds carbohydrate groups in the ER and Golgi. Ubiquitination tags proteins for degradation via proteasomes. Proteolytic cleavage activates precursors—insulin forms from proinsulin cleavage.

Protein folding, often assisted by chaperones, determines function. Misfolded proteins accumulate in diseases like Alzheimer's (amyloid plaques) and Parkinson's (Lewy bodies containing  $\alpha$ -synuclein). The unfolded protein response (UPR) activates when ER accumulates misfolded proteins, temporarily halting translation and increasing chaperone expression.

### **Experimental data:**

#### **In vitro translation:**

- Synthetic mRNA: 5'-CAP-AUG-CAU-GGC-UAG-3'
- Protein product: Met-His-Gly (3 amino acids)
- Without 5' cap: Translation efficiency decreased 90%

#### **Wobble base pairing:**

- tRNA anticodon: 3'-IAU-5' (I = inosine)
- Can recognize codons: AUA, AUC, AUU (all coding Ile)

#### **Cycloheximide treatment (blocks eEF2 elongation factor):**

- Translation stopped mid-chain
- Completed proteins released normally

- Nascent chains remained attached to ribosomes

**Proteasome inhibition:**

- Ubiquitinated proteins accumulated
- Cell cycle arrest (cyclins not degraded)
- ER stress increased

**Nonsense mutation experiment:**

- Normal: AUG-UAC-CAG → Met-Tyr-Gln
- Mutant: AUG-UAG-CAG → Met-STOP (truncated protein)
- Mutant protein: Nonfunctional

41. The 5' cap is essential for efficient translation because:

- A. It contains the start codon
- B. Ribosomes recognize and bind the cap to initiate translation
- C. It encodes the first amino acid
- D. Without it, mRNA degrades immediately

42. Cycloheximide stops translation by:

- A. Blocking elongation, preventing ribosome translocation
- B. Removing the 5' cap
- C. Degrading mRNA
- D. Blocking termination

43. Inosine in the wobble position allows one tRNA to recognize multiple codons because:

- A. Inosine can base pair flexibly with different nucleotides

- B. Inosine is identical to adenine
- C. The ribosome changes the mRNA sequence
- D. Wobble position doesn't affect codon recognition

44. Proteasome inhibition caused cell cycle arrest because:

- A. Proteasomes normally synthesize cyclin proteins
- B. Degradation of regulatory proteins like cyclins is required for cycle progression
- C. Proteasomes produce ATP for division
- D. DNA replication requires proteasomes

45. A nonsense mutation introduces a stop codon that:

- A. Adds extra amino acids
- B. Causes premature termination, producing truncated protein
- C. Doesn't affect the protein
- D. Changes one amino acid to another

### **PASSAGE 10 (Questions 46-50): Sensory Systems and Signal Transduction**

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Sensory systems convert environmental stimuli into neural signals through transduction. Receptor cells contain specialized proteins that respond to specific stimuli—photoreceptors to light, mechanoreceptors to pressure, chemoreceptors to chemicals. Stimulus energy opens or closes ion channels, altering membrane potential and neurotransmitter release.

Vision begins when photons strike rhodopsin in rod cells. Rhodopsin consists of opsin protein bound to retinal (vitamin A derivative). Light isomerizes 11-cis-retinal to all-trans-retinal, activating rhodopsin. Active rhodopsin activates transducin (G-protein), which activates phosphodiesterase (PDE). PDE hydrolyzes cGMP, causing cGMP-gated Na<sup>+</sup> channels to close. Na<sup>+</sup> influx stops, hyperpolarizing the cell and decreasing glutamate release—ironic that light causes hyperpolarization (other sensory cells depolarize with stimulation).

Signal amplification occurs at each step: one activated rhodopsin activates hundreds of transducin molecules; each PDE hydrolyzes thousands of cGMP molecules. This allows detection of single photons. Rods are extremely sensitive but don't provide color vision. Cones, containing different opsins sensitive to different wavelengths, enable color vision but require brighter light.

Hearing involves mechanotransduction in cochlear hair cells. Sound waves vibrate the basilar membrane; hair cell stereocilia bend, pulling on tip links connecting adjacent stereocilia. Tension opens mechanically-gated  $K^+$  channels.  $K^+$  influx depolarizes hair cells (unusually,  $K^+$  influx depolarizes because endolymph has high  $K^+$  concentration). Depolarization opens voltage-gated  $Ca^{2+}$  channels, triggering glutamate release onto auditory neurons.

Olfaction uses G-protein coupled receptors. Odorant binding to receptors activates G-proteins, increasing cAMP, which opens cAMP-gated cation channels. The resulting depolarization generates action potentials. Humans have ~400 different olfactory receptor genes; each olfactory neuron expresses only one receptor type, achieving specificity. Odor discrimination arises from combinatorial coding—each odorant activates multiple receptor types in unique patterns.

### **Sensory physiology experiments:**

#### **Rod cell adaptation:**

- Dark-adapted rods: High cGMP,  $Na^+$  channels open, depolarized (-40 mV), releasing glutamate
- Light exposure: cGMP decreased, channels closed, hyperpolarized (-70 mV), reduced glutamate
- Bright continuous light: Adaptation occurred,  $Ca^{2+}$  feedback restored sensitivity

#### **Color blindness test:**

- Red-green color blind individual lacks functional red or green cone opsins
- Could not distinguish red/green hues
- Blue cone function normal

#### **Hearing experiment:**

- High-frequency sounds: Basilar membrane vibration at cochlear base
- Low-frequency sounds: Vibration at apex
- Hair cell damage at base: High-frequency hearing loss

**Olfactory coding:**

- Receptor A activated by octanol and hexanol
- Receptor B activated by hexanol and geraniol
- Receptor C activated by geraniol and citronellal
- Brain distinguishes odors by comparing which receptors activate

46. In rod cells, light causes hyperpolarization rather than depolarization because:

- A. Photons destroy  $\text{Na}^+$  channels
- B. Light-activated cascade closes cGMP-gated  $\text{Na}^+$  channels, stopping depolarizing current
- C. Rhodopsin pumps  $\text{Na}^+$  out
- D. Light directly hyperpolarizes all cells

47. Signal amplification in phototransduction allows:

- A. Detection of single photons
- B. Color vision
- C. Focusing of light
- D. Pupil constriction

48.  $\text{K}^+$  influx depolarizes cochlear hair cells because:

- A.  $\text{K}^+$  is always depolarizing
- B. Endolymph has unusually high  $[\text{K}^+]$ , making  $\text{K}^+$  equilibrium potential positive
- C.  $\text{K}^+$  channels are actually  $\text{Na}^+$  channels
- D. Hair cells have reversed membrane polarity

49. The combinatorial coding of olfaction means:

- A. Each receptor detects one specific odorant
- B. Each odorant activates a unique combination of receptors
- C. Receptors don't discriminate between odorants
- D. Only one receptor type exists

50. High-frequency hearing loss from basilar membrane base damage occurs because:

- A. The base responds preferentially to high frequencies
- B. High frequencies only reach the apex
- C. The base cannot detect any sounds
- D. Frequency doesn't relate to location

### **DISCRETE QUESTIONS (51-59)**

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51. During DNA replication, the lagging strand is synthesized discontinuously as Okazaki fragments because:

- A. DNA polymerase can only synthesize 5' to 3'
- B. The lagging strand template runs 5' to 3'
- C. Primase cannot work continuously
- D. The leading strand prevents continuous synthesis

52. Which of the following best describes an exergonic reaction?

- A. Requires energy input ( $\Delta G > 0$ )
- B. Releases free energy ( $\Delta G < 0$ ) and can occur spontaneously
- C. Has reached equilibrium
- D. Requires ATP

53. In the alveoli, O<sub>2</sub> diffuses from air into blood because:

- A. Active transport pumps O<sub>2</sub> across membranes
- B. Partial pressure of O<sub>2</sub> is higher in alveolar air than in deoxygenated blood
- C. Hemoglobin actively pulls O<sub>2</sub> from air
- D. Blood pressure forces O<sub>2</sub> into blood

54. A point mutation that changes a codon from one amino acid to another is called:

- A. Silent mutation
- B. Nonsense mutation
- C. Missense mutation
- D. Frameshift mutation

55. The primary structure of a protein refers to:

- A. The overall 3D shape
- B. The amino acid sequence
- C. The  $\alpha$ -helices and  $\beta$ -sheets
- D. The assembly of multiple subunits

56. Antibiotics like penicillin work against bacteria but not human cells because they:

- A. Cannot cross human cell membranes
- B. Target bacterial cell walls, which human cells lack
- C. Only work at low temperatures
- D. Are immediately destroyed in human cells

57. Which molecule stores the most energy per gram?

- A. Carbohydrates
- B. Proteins
- C. Fats (lipids)
- D. Nucleic acids

58. The corpus luteum is maintained during early pregnancy by:

- A. Follicle-stimulating hormone (FSH)
- B. Luteinizing hormone (LH)
- C. Human chorionic gonadotropin (hCG)
- D. Progesterone alone

59. In a food web, approximately what percentage of energy transfers from one trophic level to the next?

- A. 90%
- B. 50%
- C. 10%
- D. 1%

# Psychological, Social, and Biological Foundations of Behavior

Time	Questions
95 minutes	59

## **PASSAGE 1 (Questions 1-5): Memory Consolidation and Sleep**

Memory consolidation is the process by which newly acquired information is transformed from labile short-term memories into stable long-term storage. This process occurs over hours to days and involves both synaptic and systems-level changes. Research increasingly demonstrates that sleep plays a critical role in memory consolidation, with different sleep stages contributing to different types of memory.

Sleep consists of alternating cycles of REM (rapid eye movement) and non-REM sleep. Non-REM includes stages N1 (light sleep), N2 (deeper sleep with sleep spindles and K-complexes), and N3 (slow-wave sleep characterized by high-amplitude delta waves). REM sleep features cortical activation similar to waking, rapid eye movements, muscle atonia, and vivid dreaming. A typical night progresses through 4-6 sleep cycles, with slow-wave sleep predominating early in the night and REM sleep increasing toward morning.

The dual-process hypothesis proposes that declarative memories (facts and events) consolidate primarily during slow-wave sleep, while procedural memories (motor skills, perceptual learning) consolidate during REM sleep. During slow-wave sleep, hippocampal-neocortical dialogue occurs: the hippocampus replays neural patterns from recent experiences, strengthening cortical representations and gradually making memories independent of hippocampal retrieval. Sleep spindles—bursts of 12-15 Hz oscillations—facilitate this information transfer.

REM sleep consolidates procedural memories through cortical reactivation without hippocampal involvement. Additionally, REM sleep may facilitate emotional memory processing and creative problem-solving through novel neural connections formed during cortical activation. Studies show individuals solving insight problems better after REM-rich sleep compared to slow-wave-rich sleep or waking periods.

Sleep deprivation impairs consolidation for both memory types. Total sleep deprivation prevents consolidation entirely, while selective REM or slow-wave sleep deprivation impairs specific memory

types. Neuroimaging shows that sleep-deprived individuals show reduced hippocampal activation during encoding, predicting poor subsequent memory. Chronic sleep restriction accumulates deficits, affecting academic performance, workplace productivity, and safety.

## **Experimental studies:**

### **Declarative memory experiment:**

- Participants learned 40 word pairs at 9 PM
- Group A: Normal sleep (8 hours), tested at 9 AM
- Group B: Sleep deprived, tested at 9 AM after staying awake
- Group C: Daytime learning at 9 AM, tested 9 PM (same time awake, no sleep)
- Results: Group A recalled 85%, Group B recalled 55%, Group C recalled 75%

### **Procedural memory experiment:**

- Participants trained on mirror-tracing task (visual-motor skill)
- Group 1: Training, immediate test, sleep 8 hours, retest
- Group 2: Training, immediate test, sleep deprived 8 hours, retest
- Group 1: 40% improvement after sleep
- Group 2: 15% improvement after sleep deprivation

### **Sleep stage manipulation:**

- Participants learned declarative material, then slept
- Group X: Selectively awakened during REM (preserved slow-wave sleep)
- Group Y: Selectively awakened during slow-wave sleep (preserved REM)
- Group X: Normal declarative memory consolidation
- Group Y: Impaired declarative memory consolidation

### **Insight problem study:**

- Participants given problem requiring creative insight
- Sleep group: 60% solved after full night's sleep
- Wake control: 25% solved after equivalent time awake
- Morning nap group (REM-rich): 45% solved
- Afternoon nap group (slow-wave-rich): 20% solved

1. Group A's superior recall compared to Group C demonstrates:

- A. Sleep provides time for passive memory decay
- B. Sleep actively consolidates memories beyond mere passage of time
- C. Word pairs are impossible to remember without sleep

D. Circadian rhythms alone determine memory performance

2. The finding that Group Y (slow-wave sleep deprivation) showed impaired declarative memory while Group X (REM deprivation) did not supports:

- A. REM sleep is unnecessary for any memory type
- B. Slow-wave sleep is specifically important for declarative memory consolidation
- C. All sleep stages contribute equally to all memory types
- D. Sleep deprivation improves memory

3. The superior insight problem solving after REM-rich morning naps compared to slow-wave-rich afternoon naps suggests:

- A. REM sleep facilitates creative problem-solving and novel associations
- B. Slow-wave sleep is the only stage that matters for cognition
- C. Napping always impairs problem-solving
- D. Time of day has no effect on cognitive function

4. Hippocampal-neocortical dialogue during slow-wave sleep primarily functions to:

- A. Create new memories from imagination
- B. Transfer hippocampal-dependent memories to neocortex for long-term storage
- C. Delete all memories from the hippocampus
- D. Prevent dreaming

5. Chronic sleep restriction likely affects academic performance because:

- A. Students become too relaxed to study
- B. Cumulative consolidation deficits impair both encoding and retrieval of learned material

- C. Sleep has no relationship to learning
- D. Caffeine completely compensates for sleep loss

## **PASSAGE 2 (Questions 6-10): Social Identity Theory and Intergroup Behavior**

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Social identity theory, developed by Henri Tajfel and John Turner, proposes that individuals derive part of their self-concept from membership in social groups. This theory explains intergroup behavior, prejudice, and discrimination through cognitive and motivational processes related to group membership. Social identity differs from personal identity—the former emphasizes group characteristics and boundaries, while the latter emphasizes individual uniqueness.

The theory posits three cognitive processes: social categorization (dividing the social world into groups), social identification (adopting the group's identity), and social comparison (comparing one's ingroup favorably to outgroups to maintain positive self-esteem). When social identity is salient, individuals perceive themselves as interchangeable group members rather than unique individuals, leading to depersonalization—seeing oneself through the lens of group prototypes.

Minimal group paradigm experiments demonstrate that even arbitrary group assignment causes ingroup favoritism. Participants randomly assigned to "over-estimators" or "under-estimators" based on trivial criteria (like dot estimation) subsequently allocate more resources to their ingroup and evaluate ingroup members more positively. This suggests that mere categorization, without competition or prior hostility, is sufficient for intergroup bias.

Realistic conflict theory complements social identity theory, proposing that intergroup conflict arises from competition for scarce resources. The Robbers Cave experiment illustrated this: boys at summer camp developed intense hostility after being divided into groups competing for prizes, but conflict reduced when groups worked toward superordinate goals requiring cooperation. However, social identity theory explains bias even without realistic conflict—people favor ingroups for self-esteem reasons independent of tangible benefits.

Self-categorization theory extends social identity theory, proposing that identity shifts along a continuum from personal to social depending on context. When social identity is salient, group norms strongly influence behavior through processes like depersonalization and conformity to prototypical group members. This explains how group membership can override individual preferences and explain collective behavior like protests, sports fan behavior, and workplace culture adherence.

## **Research findings:**

### **Minimal group experiment replication:**

- Participants randomly assigned to "Klee preference" or "Kandinsky preference" groups
- Asked to distribute monetary rewards between anonymous ingroup/outgroup members
- Results: 72% allocated more to ingroup members
- When choices pitted maximum joint profit against ingroup advantage, 58% chose ingroup advantage even at cost to total reward

### **Self-esteem and discrimination:**

- Participants completed bogus test, received negative feedback
- Group A: Then participated in intergroup task allowing ingroup favoritism
- Group B: Then completed individual task
- Measured self-esteem after tasks
- Group A: Self-esteem recovered to baseline
- Group B: Self-esteem remained depressed

### **Superordinate goals intervention:**

- Two rival high school groups with history of conflict
- Condition 1: Contact without shared goals (ate lunch in same space)
- Condition 2: Contact with cooperative task toward shared goal
- Condition 1: Prejudice unchanged or slightly increased
- Condition 2: Prejudice reduced by 40%, increased cross-group friendships

### **Identity salience manipulation:**

- University students (also women/men)
- Task: Rate competence of student in ambiguous performance scenario
- Condition A: Gender identity made salient before rating
- Condition B: University identity made salient
- Condition A: Gender biases emerged in ratings
- Condition B: No gender bias, but university affiliation bias emerged

6. The minimal group paradigm demonstrates that intergroup bias:

A. Only occurs when groups have historical conflicts

- B. Requires competition for scarce resources
  - C. Can emerge from mere categorization into groups
  - D. Never occurs in laboratory settings
7. When participants chose ingroup advantage over maximum joint profit, this demonstrates:
- A. Rational economic decision-making
  - B. Motivation to enhance relative ingroup status even at absolute cost
  - C. Inability to understand monetary value
  - D. Preference for equal outcomes
8. Group A's self-esteem recovery after displaying ingroup favoritism supports the idea that:
- A. Discrimination has no psychological function
  - B. Ingroup favoritism serves to maintain or restore positive social identity and self-esteem
  - C. Self-esteem is unrelated to group membership
  - D. Negative feedback always permanently damages self-esteem
9. The finding that superordinate goals reduced prejudice while mere contact did not supports:
- A. Contact alone is insufficient; cooperation toward shared goals is necessary
  - B. Contact always increases prejudice
  - C. Prejudice cannot be reduced through any intervention
  - D. Competition is necessary for positive intergroup relations
10. Identity salience manipulation showing different biases based on which identity was activated demonstrates:
- A. People have only one social identity

- B. Social identities are fixed and unchanging
- C. The context determines which identity is salient and influences behavior accordingly
- D. Social identities have no effect on judgment

### **PASSAGE 3 (Questions 11-15): The HPA Axis and Stress Response**

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The hypothalamic-pituitary-adrenal (HPA) axis is the body's primary stress response system, coordinating physiological adaptations to challenges. When the brain perceives a stressor, the paraventricular nucleus of the hypothalamus releases corticotropin-releasing hormone (CRH), which stimulates the anterior pituitary to secrete adrenocorticotropic hormone (ACTH). ACTH travels through the bloodstream to the adrenal cortex, stimulating cortisol release. This cascade occurs over minutes, complementing the rapid sympathetic nervous system response (fight-or-flight).

Cortisol has widespread effects: it mobilizes energy by promoting gluconeogenesis and lipolysis, suppresses non-essential functions like reproduction and growth, and modulates immune function. Acutely, cortisol enhances immunity, but chronic elevation causes immunosuppression. Cortisol also affects the brain, influencing memory consolidation, emotional processing, and the stress response itself through negative feedback loops—cortisol binds receptors in the hippocampus and hypothalamus, inhibiting further HPA activation once the stressor passes.

Chronic stress causes HPA axis dysregulation. Prolonged cortisol exposure can damage hippocampal neurons, impairing negative feedback and creating a vicious cycle of overactivation. Hippocampal atrophy observed in chronic stress and depression may result from this cortisol neurotoxicity combined with reduced neurogenesis. Additionally, chronic stress causes allostatic load—the cumulative wear and tear from repeated stress activation, contributing to cardiovascular disease, metabolic syndrome, and accelerated aging.

Individual differences in stress reactivity partly reflect early life experiences. The fetal programming hypothesis proposes that prenatal stress exposure alters HPA axis set points. Animal studies show that maternal stress during pregnancy leads to offspring with hyperreactive HPA axes and increased anxiety-like behavior. In humans, childhood adversity predicts exaggerated cortisol responses and increased vulnerability to stress-related disorders. Conversely, secure attachment and supportive environments buffer stress reactivity.

Stress appraisal—how individuals interpret situations—profoundly affects HPA activation. When people appraise situations as challenges (controllable, with potential for growth) rather than threats (uncontrollable, overwhelming), they show smaller cortisol responses and better performance. Cognitive

reappraisal techniques that reframe stressors can reduce physiological stress responses, demonstrating the powerful interaction between cognitive and biological stress systems.

### **Research data:**

#### **Acute stress study:**

- Participants gave public speech (social-evaluative stressor)
- Measured salivary cortisol at baseline, +20 min, +40 min, +60 min
- Baseline: 8 ng/mL
- +20 min: 22 ng/mL (peak)
- +40 min: 16 ng/mL
- +60 min: 10 ng/mL (returning to baseline)

#### **Chronic stress effects:**

- Caregivers of Alzheimer's patients (chronic stress group) vs. matched controls
- Chronic stress: Higher baseline cortisol, flatter diurnal rhythm
- Chronic stress: Hippocampal volume 8% smaller on MRI
- Chronic stress: 35% higher rates of depression and anxiety disorders

#### **Early adversity study:**

- Adults with documented childhood abuse vs. controls
- Laboratory stress test (Trier Social Stress Test)
- Abuse history group: Cortisol peak 50% higher than controls
- Abuse history group: Prolonged return to baseline (90 min vs. 60 min)
- Abuse history group: 3× higher lifetime risk for depression

#### **Reappraisal intervention:**

- Participants told upcoming speech would be stressful (threat frame) or opportunity to showcase skills (challenge frame)
- Threat frame: Peak cortisol 25 ng/mL, performance rating 6.2/10
- Challenge frame: Peak cortisol 18 ng/mL, performance rating 7.8/10
- Challenge frame: Participants reported less anxiety, more excitement

11. The peak in cortisol 20 minutes after the stressor rather than immediately demonstrates:

- A. Cortisol is unrelated to stress
- B. The HPA axis involves a hormonal cascade requiring time for synthesis and secretion
- C. Cortisol peaks before stressors occur
- D. The measurement was incorrect

12. Chronic stress caregivers' smaller hippocampal volume and flatter diurnal cortisol rhythm suggest:

- A. Caregiving has no biological effects
- B. Prolonged cortisol exposure damages hippocampus, impairing HPA regulation
- C. Hippocampal size is unrelated to stress
- D. Cortisol production stops with chronic stress

13. Adults with childhood abuse history showing exaggerated and prolonged cortisol responses demonstrates:

- A. Early adversity programs HPA axis sensitivity, creating lasting vulnerability
- B. Childhood experiences have no lasting effects
- C. All adults respond identically to stress
- D. Cortisol responses are entirely genetic

14. The challenge frame reducing cortisol and improving performance supports:

- A. Cognitive appraisal influences physiological stress responses
- B. Framing has no effect on biology
- C. Higher cortisol always improves performance
- D. Appraisal and physiology are completely separate

15. Cortisol's negative feedback through hippocampal and hypothalamic receptors functions to:

- A. Amplify the stress response indefinitely
- B. Terminate the stress response after the threat has passed
- C. Prevent any future stress responses
- D. Increase cortisol production

#### **PASSAGE 4 (Questions 16-20): Operant Conditioning and Behavior Modification**

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Operant conditioning, developed by B.F. Skinner, describes how consequences shape voluntary behavior. Unlike classical conditioning (which involves involuntary responses to stimuli), operant conditioning explains how organisms learn to increase behaviors followed by favorable consequences and decrease behaviors followed by unfavorable consequences. This learning process operates through four main contingencies: positive reinforcement, negative reinforcement, positive punishment, and negative punishment.

Positive reinforcement increases behavior by adding a pleasant stimulus (reward); negative reinforcement increases behavior by removing an aversive stimulus. Both increase behavior frequency but through different mechanisms. Positive punishment decreases behavior by adding an aversive stimulus; negative punishment decreases behavior by removing a pleasant stimulus. The terms "positive" and "negative" refer to whether something is added or removed, not whether the experience is good or bad.

Reinforcement schedules profoundly affect learning and extinction resistance. Continuous reinforcement (reinforcing every response) produces rapid learning but quick extinction when reinforcement stops. Partial reinforcement schedules produce slower learning but greater extinction resistance. Fixed-ratio schedules (reinforcement after set number of responses) produce high, steady response rates with brief pauses after reinforcement. Variable-ratio schedules (reinforcement after unpredictable number of responses) produce the highest response rates with greatest extinction resistance—explaining gambling persistence. Interval schedules reinforce the first response after time periods: fixed-interval creates scalloped response patterns, while variable-interval produces steady responding.

Shaping uses successive approximations to train complex behaviors by reinforcing progressively closer approximations to the target behavior. This explains how organisms learn behaviors that would never occur spontaneously. Primary reinforcers (food, water, sex) are innately rewarding; secondary reinforcers (money, praise, grades) acquire value through association with primary reinforcers via classical conditioning. Token economies in institutional settings use secondary reinforcers systematically to modify behavior.

Behavior modification applies operant principles to practical problems. Applied behavior analysis (ABA) treats autism spectrum disorder by breaking skills into small steps, using prompting and fading, and providing immediate reinforcement. Studies show intensive early ABA significantly improves outcomes. However, critics argue that exclusive reliance on external reinforcement may undermine intrinsic motivation—the overjustification effect occurs when external rewards decrease intrinsic interest in inherently enjoyable activities.

### **Research and applications:**

#### **Reinforcement schedule comparison:**

- Pigeons trained to peck key for food
- Group 1: Continuous reinforcement (every peck rewarded)
- Group 2: Fixed-ratio 5 (every 5th peck rewarded)
- Group 3: Variable-ratio 5 (average every 5th peck rewarded)
- Learning trials to criterion: Group 1 = 20, Group 2 = 35, Group 3 = 40
- Extinction (pecks after reinforcement stopped): Group 1 = 15, Group 2 = 80, Group 3 = 200

#### **Token economy study:**

- Psychiatric ward implemented token economy for 6 months
- Patients earned tokens for self-care, social interaction, attending therapy
- Tokens exchangeable for privileges, snacks, activities
- Target behaviors increased 65% from baseline
- Medication compliance increased 45%
- After removal: Behaviors decreased but remained 30% above original baseline

#### **ABA intervention:**

- Children with autism (ages 2-4) received intensive ABA (40 hrs/week) vs. control (standard intervention 10 hrs/week)
- After 2 years:
- ABA group: Average IQ increase of 25 points, 47% achieved normal educational placement
- Control group: Average IQ increase of 5 points, 10% achieved normal educational placement

**Overjustification effect:**

- Children who enjoyed drawing given art supplies
- Group A: Received reward for drawing
- Group B: No reward, just drew for enjoyment
- After 2 weeks, rewards removed
- Group A: Drawing time decreased 50% from baseline
- Group B: Drawing time remained stable

16. Variable-ratio schedules producing the greatest extinction resistance explains:

- A. Why continuous reinforcement is most effective
- B. Why gambling persists despite losses—unpredictable rewards create persistent behavior
- C. Why learning on variable-ratio is fastest
- D. Why punishment is more effective than reinforcement

17. The token economy increasing behaviors even after removal (maintained 30% above baseline) suggests:

- A. Token economies have no lasting effects
- B. Behaviors generalized and maintained through secondary reinforcement mechanisms
- C. All improvements were temporary
- D. Tokens are primary reinforcers

18. The ABA intervention's superior outcomes for autism demonstrate:

- A. Operant principles cannot be applied to developmental disorders
- B. Systematic application of reinforcement and shaping effectively teaches complex skills
- C. All children respond identically regardless of intervention
- D. Intensity of intervention is irrelevant

19. The overjustification effect (Group A's decreased drawing after rewards removed) illustrates:

- A. External rewards always increase motivation
- B. External rewards can undermine intrinsic motivation when applied to inherently enjoyable activities
- C. Children don't enjoy drawing
- D. Rewards have no effect on behavior

20. Negative reinforcement differs from positive reinforcement in that:

- A. It decreases behavior while positive reinforcement increases it
- B. It involves removing an aversive stimulus rather than adding a pleasant one, but both increase behavior
- C. It's less effective than positive reinforcement
- D. It only works with punishment present

### **PASSAGE 5 (Questions 21-25): Attention and Selective Processing**

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Attention is the cognitive process of selectively concentrating on relevant information while filtering out irrelevant stimuli. Given the vast amount of sensory input, attention acts as a limited-capacity filter determining what reaches conscious awareness and deeper processing. Attention research employs dichotic listening tasks (different auditory messages to each ear), visual search paradigms, and dual-task interference to understand selection mechanisms and capacity limits.

Early selection models propose that attention filters information based on physical characteristics before semantic processing. Broadbent's filter theory suggests unattended information is blocked at a perceptual stage. However, the cocktail party effect—hearing one's name in unattended conversation—challenges pure early selection, suggesting some semantic processing occurs for unattended stimuli. Late selection models propose that all information receives semantic processing, with attention selecting what enters awareness and memory.

Selective attention can be spatial (attending to locations), feature-based (attending to colors or shapes), or object-based (attending to whole objects). The spotlight metaphor describes spatial attention as illuminating a region of space, enhancing processing within that region. Feature-based attention enhances processing of attended features across the visual field—attending to red objects enhances red everywhere, not just in one location.

Divided attention—performing multiple tasks simultaneously—succeeds when tasks are well-practiced (automaticity) or use different processing resources. The multiple resource theory proposes separate pools for verbal versus spatial processing, visual versus auditory modalities, and perception versus response. Dual-task interference occurs when tasks compete for the same limited resource. Cell phone conversations impair driving because both require verbal-cognitive resources, whereas listening to music interferes less because it requires fewer overlapping resources.

Attention networks involve brain regions including: the frontal eye fields and intraparietal sulcus (spatial orienting), anterior cingulate (conflict monitoring and cognitive control), and ascending arousal systems from the brainstem and thalamus (alertness). Damage to these regions causes attention deficits: right parietal damage causes left-sided neglect (inability to attend to left space), and frontal damage impairs sustained attention and task-switching.

### **Experimental findings:**

#### **Dichotic listening study:**

- Participants attended to message in right ear, ignored left ear
- Right ear: "On sunny days, people enjoy walking outside"
- Left ear: "The cat jumped over the fence"
- After: Asked about left ear content
- 95% couldn't report any left ear content
- However, when "David" (participant's name) inserted in left ear, 33% noticed
- When left ear switched to German mid-task, only 5% noticed

#### **Visual search experiment:**

- Find target T among distractor Ls
- Condition A: T differs by color only (red T, green Ls)—pop-out search
- Condition B: T differs by feature combination (vertical/horizontal lines)—conjunction search
- Condition A: Search time ~500ms regardless of number of distractors (parallel processing)
- Condition B: Search time increased ~40ms per distractor (serial processing)

#### **Inattentional blindness:**

- Participants counted basketball passes in video
- Unexpected event: Person in gorilla suit walked through scene
- Only 50% noticed gorilla

- When told to just watch video (not count), 95% noticed gorilla

**Dual-task study:**

- Participants drove in simulator while performing secondary task
- Condition 1: Conversation on cell phone
- Condition 2: Listen to podcast
- Condition 3: No secondary task
- Reaction time to brake lights: Condition 1 = 640ms (+150ms), Condition 2 = 510ms (+20ms), Condition 3 = 490ms
- Miss rate for brake lights: Condition 1 = 18%, Condition 2 = 4%, Condition 3 = 2%

21. The finding that 33% noticed their name in the unattended ear despite not reporting other content supports:

- A. Pure early selection—no unattended information is processed
- B. Some semantic processing of unattended information occurs, especially for personally relevant stimuli
- C. Participants were not paying attention
- D. Names are physical, not semantic, features

22. Pop-out search (Condition A) being independent of distractor number demonstrates:

- A. Serial, attention-demanding processing
- B. Parallel, pre-attentive processing for simple feature differences
- C. Inability to detect color
- D. Attention is unnecessary for visual processing

23. Inattention blindness (50% missing gorilla when counting passes) illustrates:

- A. Attention is unlimited
- B. Without attention directed to an event, even obvious stimuli may not reach awareness

- C. People cannot see gorillas
- D. Counting impairs all visual perception

24. Cell phone conversations impairing driving more than podcast listening suggests:

- A. All auditory stimuli impair driving equally
- B. Conversation requires verbal-cognitive resources that compete with driving's attentional demands
- C. Driving requires no attention
- D. Music and speech are processed identically

25. Right parietal damage causing left-sided neglect demonstrates:

- A. The right parietal cortex is critical for spatial attention, especially to contralateral space
- B. Attention has no neural basis
- C. Vision is controlled only by the occipital lobe
- D. Neglect affects motor function, not attention

### **PASSAGE 6 (Questions 26-30): Health Disparities and Social Determinants**

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Health disparities—differences in health outcomes between population groups—persist across race, ethnicity, socioeconomic status, and geography. These disparities are not primarily genetic but reflect social determinants of health: economic stability, education, social support, physical environment, and healthcare access. Understanding these social determinants is essential for addressing health inequalities and improving population health.

Socioeconomic status (SES) shows robust relationships with health across societies. The SES-health gradient is not merely a poverty threshold effect—health improves continuously across the entire SES spectrum, from poorest to wealthiest. Multiple mechanisms link SES to health: material resources (nutrition, housing, healthcare access), psychosocial factors (chronic stress, sense of control, social support), health behaviors (smoking, exercise, diet), and early life conditions affecting development.

The fundamental cause theory proposes that SES is a "fundamental cause" of health disparities because it involves access to resources (knowledge, money, power, social connections) that can be used to avoid disease and death regardless of specific disease mechanisms. When new disease prevention or treatment emerges, higher SES individuals adopt it first, maintaining health advantages even as specific disease causes change. This explains why SES-health gradients persist across time and place.

Racial and ethnic health disparities reflect complex interactions of socioeconomic factors, discrimination, residential segregation, and cultural factors. African Americans experience higher rates of hypertension, diabetes, infant mortality, and lower life expectancy compared to White Americans, even after controlling for SES. These disparities partly reflect chronic stress from discrimination—the "weathering hypothesis" proposes that accumulated stress from racism accelerates biological aging (allostatic load). Additionally, residential segregation concentrates poverty and limits access to quality healthcare, healthy foods, and safe environments.

Healthcare access and quality contribute substantially to disparities. The uninsured receive less preventive care and delay treatment, leading to worse outcomes. Even with insurance, implicit bias affects provider-patient interactions and treatment decisions. Studies show racial minorities receive less aggressive pain management, fewer cardiac procedures, and lower quality care even within the same healthcare systems, suggesting bias affects clinical decision-making beyond access issues.

### **Epidemiological data:**

#### **SES and mortality:**

- U.S. adults tracked for 10 years by income quintile
- Lowest quintile (income <\$25k): Mortality rate 12.3 per 1000
- Second quintile: 9.8 per 1000
- Third quintile: 7.6 per 1000
- Fourth quintile: 6.1 per 1000
- Highest quintile (income >\$100k): 4.2 per 1000
- Pattern holds after controlling for health behaviors

#### **Life expectancy by race and location:**

- White Americans: 78.9 years
- Black Americans: 75.0 years (3.9 year gap)
- But: Black Americans in high-SES neighborhoods: 77.8 years
- Black Americans in low-SES neighborhoods: 72.6 years (5.2 year gap by neighborhood)

**Discrimination and health:**

- Survey: Self-reported discrimination experiences
- High discrimination group: 2.1× higher hypertension rates
- High discrimination group: 1.8× higher depression rates
- Effect remained after controlling for SES, health behaviors

**Healthcare disparities study:**

- Emergency department patients presenting with fracture pain
- White patients: 74% received opioid analgesia
- Black patients: 57% received opioid analgesia
- Hispanic patients: 55% received opioid analgesia
- Similar pain severity ratings across groups

**Intervention program:**

- Low-income neighborhoods received: Community health workers, improved transit to clinics, food assistance
- After 3 years:
- Intervention neighborhoods: Diabetes control improved 28%, ER visits decreased 35%
- Control neighborhoods: Minimal changes

26. The continuous SES-health gradient across all income levels (not just poverty) suggests:

- A. Only poverty affects health
- B. Resources and stressors associated with SES influence health across the entire spectrum
- C. Wealthy people have worse health than poor people
- D. SES has no relationship to health

27. The persistent Black-White mortality gap even after controlling for SES suggests:

- A. Genetics fully explain racial health disparities
- B. Factors beyond SES, including discrimination and residential segregation, contribute to racial health disparities

- C. SES is unrelated to race
- D. Health disparities don't exist

28. Higher discrimination associated with higher hypertension and depression rates supports:

- A. Discrimination has no health effects
- B. Chronic stress from discrimination contributes to poor health through psychophysiological pathways
- C. Self-reports are always inaccurate
- D. Hypertension is purely genetic

29. Differential opioid analgesia rates for fracture pain by race (74% vs. 57% vs. 55%) with similar pain severity demonstrates:

- A. Different groups experience pain differently
- B. Implicit bias affects clinical treatment decisions, creating disparities in care quality
- C. Pain medication is equally distributed
- D. Healthcare providers are perfectly objective

30. The intervention program's success in improving diabetes control and reducing ER visits illustrates:

- A. Health behaviors are entirely individual choices
- B. Addressing social determinants through community-level interventions can reduce health disparities
- C. Medical interventions are unnecessary
- D. Neighborhoods have no effect on health

### **PASSAGE 7 (Questions 31-35): Emotion and the Limbic System**

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Emotion involves coordinated changes across subjective experience, physiological arousal, expressive behavior, and cognitive appraisal. Multiple brain structures, collectively termed the limbic system, coordinate emotional processing and responses. Key structures include the amygdala, hippocampus,

hypothalamus, anterior cingulate cortex, and prefrontal cortex, each contributing distinct functions to emotional experience and regulation.

The amygdala, particularly its basolateral complex, plays a central role in emotional learning and fear processing. It receives input from sensory cortex and thalamus, evaluates emotional significance, and coordinates responses through projections to hypothalamus (autonomic responses), periaqueductal gray (behavioral responses), and hippocampus (memory consolidation). The amygdala enables rapid "low road" emotional responses: thalamic input reaches the amygdala before cortical processing, allowing quick responses to potential threats before conscious recognition.

Fear conditioning—pairing neutral stimuli with aversive outcomes—demonstrates amygdala function. After pairing a tone with shock, the tone alone elicits fear responses (freezing, increased heart rate, cortisol release). This learning requires the amygdala; lesions prevent fear conditioning. The amygdala also modulates memory consolidation for emotional events—arousal-enhanced memory. Emotionally arousing events activate amygdala, which modulates hippocampal consolidation, explaining why emotional memories are vivid and persistent.

The prefrontal cortex, particularly ventromedial and orbitofrontal regions, integrates emotional information with goals and context for emotional regulation and decision-making. The prefrontal cortex exerts top-down control over the amygdala through inhibitory projections—the neural basis of emotion regulation. Damage to ventromedial prefrontal cortex causes impaired decision-making despite intact intellect, demonstrating emotion's role in rational choice (Damasio's somatic marker hypothesis).

The anterior cingulate cortex detects conflict, monitors errors, and signals when increased cognitive control is needed—emotional aspects of executive function. It activates during social exclusion (Cyberball paradigm), physical pain, and cognitive conflict (Stroop task), suggesting common neural substrates for social and physical pain. The insula processes interoceptive awareness and disgust, both primary disgust (contamination) and moral disgust (norm violations).

### **Research evidence:**

#### **Amygdala lesion study:**

- Patient S.M. with bilateral amygdala damage (rare genetic condition)
- Cannot recognize fear in facial expressions (recognizes other emotions normally)
- Fails to show fear conditioning (no conditioned response to tone paired with loud noise)
- Approaches dangerous situations without appropriate caution

- Shows normal happiness, sadness, disgust recognition

**Emotional memory enhancement:**

- Participants viewed neutral and emotional pictures while undergoing fMRI
- Memory tested 1 week later
- Neutral pictures: 42% recalled, minimal amygdala activation
- Emotional pictures: 78% recalled, strong amygdala activation during encoding
- Amygdala activation during encoding predicted later memory

**Prefrontal regulation:**

- Participants viewed negative images while using cognitive reappraisal or passive viewing
- Reappraisal: "Imagine less negative interpretation"
- During reappraisal: Increased prefrontal activation, decreased amygdala activation
- Reappraisal reduced subjective negative emotion by 45%
- Reappraisal reduced physiological arousal (skin conductance)

**Social pain study (Cyberball):**

- Participants played virtual ball-toss game via computer
- Inclusion condition: Other "players" (actually computer) toss ball to participant
- Exclusion condition: Other "players" stop tossing ball to participant
- Exclusion: Increased anterior cingulate and insula activation
- Activation in these regions correlated with self-reported distress
- Pattern similar to physical pain activation

31. Patient S.M.'s inability to recognize fear or show fear conditioning after amygdala damage demonstrates:

- A. The amygdala is necessary for all emotions equally
- B. The amygdala is specifically critical for fear processing and fear learning
- C. Emotion recognition requires no neural structures
- D. All brain regions contribute equally to all emotions

32. Amygdala activation during emotional event encoding predicting later memory illustrates:

- A. Emotion impairs memory
- B. The amygdala modulates memory consolidation for emotional events, enhancing their retention
- C. Memory and emotion are completely separate
- D. Neutral events are remembered better than emotional ones

33. Cognitive reappraisal increasing prefrontal activity while decreasing amygdala activity suggests:

- A. The prefrontal cortex has no role in emotion
- B. Top-down prefrontal control can regulate amygdala-based emotional responses
- C. Emotions cannot be regulated
- D. The amygdala and prefrontal cortex never interact

34. Similar anterior cingulate and insula activation for social exclusion and physical pain supports:

- A. Social and physical pain have no relationship
- B. Social pain recruits neural systems also involved in physical pain processing
- C. Social exclusion is imaginary
- D. Physical pain is purely psychological

35. The "low road" pathway (thalamus → amygdala) allows:

- A. Only conscious emotional responses
- B. Rapid emotional responses to potential threats before detailed cortical processing
- C. Complete emotional analysis before any response
- D. Only negative emotions

## **PASSAGE 8 (Questions 36-40): Attribution Theory and Cognitive Biases**

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Attribution theory examines how people explain behavior—their own and others'. Attributions influence emotions, expectations, and subsequent behavior, making them central to social cognition. Fritz Heider proposed that people act as "naïve scientists," seeking to understand causality in social behavior. Harold Kelley's covariance model describes how people use consensus (do others act this way?), distinctiveness (does the person act this way in other situations?), and consistency (does the person always act this way?) information to make internal (dispositional) versus external (situational) attributions.

The fundamental attribution error (correspondence bias) is the tendency to overestimate dispositional factors and underestimate situational factors when explaining others' behavior. When a student fails an exam, observers attribute failure to laziness (disposition) while underweighting situational factors (exam difficulty, personal problems). This bias is culturally variable—more pronounced in individualistic than collectivistic cultures—suggesting it partly reflects cultural emphasis on personal agency versus situational context.

The actor-observer bias describes asymmetry in self versus other attributions: people explain their own behavior situationally but others' behavior dispositionally. This occurs because actors have more information about situational constraints on their own behavior and different attentional focus (actors attend to situation, observers attend to actor). Additionally, temporal perspective matters—people explain their past behavior more dispositionally than their current behavior.

Self-serving bias involves taking credit for success (internal attribution) while blaming failure on external factors. This bias protects self-esteem and maintains sense of control. Students attribute good grades to ability and effort but bad grades to unfair tests or poor teaching. However, depressed individuals show reversed pattern—attributing success externally and failure internally—contributing to depressive cognitive style. Attributional retraining programs teach adaptive attribution patterns to improve motivation and performance.

Just-world hypothesis reflects the belief that people get what they deserve. This belief provides sense of predictability and control but leads to victim-blaming. When bad things happen to people, observers may attribute it to victims' actions or character ("they must have done something wrong") to maintain belief in a just world. This bias affects judgments of crime victims, people in poverty, and those with illnesses, potentially reducing empathy and support.

## **Study findings:**

### **Fundamental attribution error:**

- Participants read essays favoring or opposing capital punishment
- Told: "Writer was randomly assigned position and required to argue it"
- Despite this situational information, 78% rated writers' true attitudes as consistent with essay position
- Control group without situational information: 85% made dispositional inference
- Minimal effect of providing clear situational explanation

### **Actor-observer study:**

- Participants explained their own and roommate's behavior: "Why did you/your roommate choose your major?"
- Self: 65% situational attributions ("interesting courses," "good professors")
- Roommate: 62% dispositional attributions ("interested in that subject," "that type of person")
- Pattern reversed when explaining own behavior from 10 years ago (more dispositional for own past)

### **Self-serving bias:**

- Students received bogus feedback on "social sensitivity test"
- Success condition: Told scored in 88th percentile
- Failure condition: Told scored in 32nd percentile
- Success: 82% attributed to ability, effort
- Failure: 74% attributed to test unreliability, bad luck

### **Depressive attribution style:**

- Depressed vs. non-depressed participants performed task with success/failure manipulation
- Non-depressed success: Internal attribution (85%)
- Non-depressed failure: External attribution (71%)
- Depressed success: External attribution (63%)
- Depressed failure: Internal attribution (78%)—reversed pattern

### **Just-world bias:**

- Participants watched video of woman receiving electric shocks
- Condition A: Told she was being shocked for study, would be compensated
- Condition B: Told she volunteered to be shocked to help researchers

- Measured attractiveness ratings and sympathy
- Condition A: Rated her as less attractive, reported less sympathy
- Condition B: Rated her as more attractive, reported more sympathy
- Derogation of innocent victim to maintain just-world belief

36. The fundamental attribution error persisting even when told essay position was randomly assigned demonstrates:

- A. People accurately consider situational information
- B. Dispositional attributions are strongly preferred even when situational explanations are obvious
- C. Essay writing has no situational constraints
- D. Participants couldn't understand the instructions

37. The actor-observer bias (65% situational for self vs. 62% dispositional for roommate) suggests:

- A. Everyone makes identical attributions
- B. Perspective influences attribution—people have different information and attention for own versus others' behavior
- C. The bias doesn't exist
- D. Roommates are always wrong

38. Self-serving bias (82% internal for success vs. 74% external for failure) functions to:

- A. Impair accurate self-assessment
- B. Protect self-esteem and maintain sense of control
- C. Blame others for everything
- D. Prevent learning from experience

39. Depressed individuals' reversed attribution pattern (external for success, internal for failure) suggests:

- A. Depression doesn't affect cognition
- B. Maladaptive attribution patterns contribute to maintaining depressive symptoms
- C. Depressed people are more accurate than non-depressed people
- D. Attribution has no relationship to mood

40. Derogating the innocent victim (Condition A) to maintain just-world belief illustrates:

- A. Observers accurately assess victim characteristics
- B. People distort perceptions of victims to preserve belief that the world is fair
- C. Victims are always responsible for their outcomes
- D. Sympathy is unrelated to circumstances

### **PASSAGE 9 (Questions 41-45): Language Development**

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Language acquisition is one of childhood's remarkable achievements. Children progress from babbling at 6 months to producing complex sentences by age 3-4, seemingly effortlessly acquiring grammar rules they're never explicitly taught. Several theoretical perspectives explain this development: behaviorist (Skinner's imitation and reinforcement), nativist (Chomsky's innate universal grammar), and interactionist (social-cognitive integration).

Chomsky's nativist theory proposes that humans possess an innate Language Acquisition Device (LAD) containing universal grammar—principles common to all languages. Evidence includes: children acquire language rapidly during a critical period despite "poverty of the stimulus" (input doesn't contain explicit grammatical instruction), children create grammatical utterances never heard before, and all children progress through similar stages regardless of language. Creole languages—fully grammatical languages created by children from pidgin input—demonstrate children's innate grammatical capacity.

The critical period hypothesis proposes that language acquisition must occur before puberty for native-like proficiency. Evidence includes second-language learners: those learning before age 7 achieve native-like proficiency, while proficiency decreases with age of acquisition after puberty. Feral children discovered after the critical period (Genie, age 13) fail to achieve normal grammatical competence despite intensive instruction. However, critical period effects are stronger for phonology and syntax than vocabulary.

Social-interactionist perspectives emphasize caregiver input and social context. Child-directed speech (motherese) features exaggerated intonation, simple grammar, and high pitch, facilitating attention and learning. Joint attention—caregiver and child focusing on the same object—scaffolds word learning. Statistical learning—sensitivity to distributional patterns in input—helps infants segment words and extract grammatical patterns. This suggests language learning uses general cognitive mechanisms, not entirely domain-specific innate knowledge.

Language and thought relationships remain debated. Linguistic relativity (Sapir-Whorf hypothesis) proposes that language influences thought. Strong version (linguistic determinism)—language determines thought—is largely rejected. Weak version—language influences some thought domains—finds support: Russian speakers (who have separate words for light blue and dark blue) show faster color discrimination at the linguistic boundary than English speakers. However, prelinguistic infants show similar core cognitive capacities across cultures, suggesting language doesn't determine all thought.

### **Developmental and comparative data:**

#### **Language milestone norms:**

- 6 months: Canonical babbling (ba-ba, da-da)
- 12 months: First words (mama, dada, ball)
- 18 months: Vocabulary spurt begins (~50 words)
- 24 months: Two-word combinations (telegraphic speech: "want cookie," "daddy go")
- 36 months: Grammatical morphemes emerge (-ing, plural -s, past -ed)
- 48 months: Complex sentences with embeddings

#### **Critical period evidence:**

- Korean and Chinese adoptees learning English in U.S.
- Adopted before age 3: Adult grammaticality judgment scores = native speakers (98% correct)
- Adopted ages 3-7: 95% correct
- Adopted ages 8-12: 87% correct
- Adopted ages 13-16: 78% correct
- Linear decline with age of acquisition after early childhood

#### **Poverty of stimulus:**

- Analysis of caregiver speech to 2-year-olds
- Grammatical errors in child speech: 8%

- Explicit corrections of grammar: 3% of errors
- Most corrections focus on truth, not form ("No, that's a dog, not a cat" vs. "Say 'I went,' not 'I goed'")
- Despite minimal explicit instruction, children acquire correct grammar by age 5

**Joint attention and word learning:**

- 18-month-olds taught novel words for novel objects
- Condition A: Adult looked at object while saying word
- Condition B: Adult looked away from object while saying word
- Condition A: Learned word after 3 exposures (mapped word to object adult was attending to)
- Condition B: Failed to learn word even after 12 exposures

**Linguistic relativity (color):**

- Russian speakers: Separate lexical items for light blue (goluboy) and dark blue (siniy)
- English speakers: Both called "blue"
- Color discrimination task: React when two blue shades differ
- Russian speakers: 124ms faster when shades cross linguistic boundary
- English speakers: No difference based on linguistic categories
- Verbal interference task eliminated Russian speakers' advantage (suggests linguistic mediation)

41. Children acquiring correct grammar despite minimal explicit correction (3% of errors) supports:

- A. Behaviorist theory—reinforcement shapes grammar
- B. Nativist theory—innate mechanisms guide acquisition rather than explicit instruction
- C. Children never make grammatical errors
- D. Parents provide explicit grammar lessons

42. Declining proficiency with later age of acquisition (98% → 78% from early to adolescent adoption) supports:

- A. Age has no effect on language learning
- B. Critical period hypothesis—language acquisition is constrained by developmental timing
- C. Adults learn language better than children

D. Korean and Chinese are harder than English

43. Joint attention's importance (Condition A learned vs. Condition B didn't learn) demonstrates:

- A. Words are learned through pure association
- B. Social-cognitive factors like shared attention facilitate word learning
- C. Children cannot learn words
- D. Adults should never look at objects

44. Russian speakers' faster discrimination at linguistic boundaries (eliminated under verbal interference) suggests:

- A. Language has no effect on perception
- B. Language categories can influence perceptual discrimination through linguistic mediation
- C. Russian speakers have better vision
- D. Color perception is entirely innate

45. Creole languages emerging from pidgin input with children creating full grammar demonstrates:

- A. Children passively imitate adult speech
- B. Children actively impose grammatical structure, supporting innate grammatical capacity
- C. Pidgins are grammatically complete
- D. Language learning is impossible without full input

### **PASSAGE 10 (Questions 46-50): Social Stratification and Inequality**

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Social stratification—the hierarchical arrangement of individuals in society—exists across all complex societies, though its form and rigidity vary. Stratification systems organize around multiple dimensions: economic (class), social (status), and political (power). Understanding stratification requires examining both individual mobility and structural barriers that maintain inequality across generations.

Karl Marx emphasized class stratification based on relationship to means of production: bourgeoisie (owners) versus proletariat (workers). Marx argued that class conflict drives historical change and that economic base (mode of production) shapes superstructure (culture, institutions, ideology). Max Weber added complexity with three-dimensional stratification: class (economic position), status (social prestige), and party (political power). These dimensions can vary independently—religious figures may have high status but low economic class.

Social mobility—movement between stratification positions—can be intragenerational (within a lifetime) or intergenerational (across generations). Structural mobility results from economic changes creating new positions (industrialization creating white-collar jobs), while exchange mobility involves individuals trading places. The American Dream emphasizes individual mobility through meritocracy, but research shows substantial mobility barriers. Income mobility in the U.S. is actually lower than many European countries, and parental income strongly predicts children's adult income.

Cultural capital (Bourdieu) refers to non-financial social assets like education, speech patterns, cultural knowledge, and social skills that facilitate upward mobility. Upper-class children inherit cultural capital through family socialization, providing advantages in education and employment beyond mere financial resources. This helps explain why class persists across generations even in seemingly meritocratic systems—advantages compound while disadvantages accumulate.

Structural functionalism (Davis-Moore thesis) argues stratification is inevitable and functional—unequal rewards motivate people to fill important positions requiring training and talent. Critics counter that stratification reflects power differences more than functional necessity, inherited privilege limits equal opportunity, and many crucial positions (teachers, nurses) are undervalued despite societal importance. Conflict theory views stratification as benefiting powerful groups who use ideological systems (meritocracy myth, just-world beliefs) to legitimize inequality.

### **Sociological data:**

#### **Intergenerational mobility study (U.S.):**

- Adult income tracked relative to parents' income quintile
- Children from lowest quintile parents:
  - 43% remain in lowest quintile (stay poor)
  - 27% reach second quintile
  - 17% reach middle quintile

- 9% reach fourth quintile
- 4% reach highest quintile
- Children from highest quintile parents:
  - 40% remain in highest quintile
  - 23% drop to fourth quintile
  - 18% reach middle quintile
  - 12% drop to second quintile
  - 7% drop to lowest quintile

### **International mobility comparison:**

- Correlation between father's and son's earnings (higher = less mobility):
- United Kingdom: 0.50
- United States: 0.47
- France: 0.41
- Germany: 0.32
- Canada: 0.19
- Denmark: 0.15
- U.S. has lower mobility than most comparison countries

### **Cultural capital study:**

- Students' cultural activities (museum visits, classical music, reading) predicted by parental education/occupation
- High cultural capital students: 2.1× more likely to attend college controlling for academic achievement
- High cultural capital students: Received better grades for equivalent work quality (teacher bias)
- Cultural capital mediated SES effects on educational outcomes

### **Occupational prestige ranking:**

- Physician: 86/100
- Lawyer: 75/100
- Teacher: 64/100
- Police officer: 60/100
- Retail worker: 28/100
- Pattern consistent across most societies despite different economic systems

**Wealth inequality:**

- Top 1% owns 32% of total wealth
- Top 10% owns 70% of total wealth
- Bottom 50% owns 2% of total wealth
- Wealth inequality increased substantially over 40 years

46. The finding that 43% of children born in the lowest quintile remain there as adults demonstrates:

- A. Perfect mobility exists
- B. Substantial intergenerational persistence—poverty tends to reproduce across generations
- C. Economic position is purely random
- D. All children have equal opportunities

47. The U.S. having higher father-son earnings correlation (0.47) than Denmark (0.15) indicates:

- A. Higher mobility in the U.S.
- B. Lower mobility in the U.S.—parental income more strongly predicts child's income
- C. Identical mobility across countries
- D. Mobility is impossible to measure

48. Cultural capital predicting college attendance beyond academic achievement illustrates:

- A. Academic achievement is the only factor in educational success
- B. Non-economic social assets transmitted through families create advantages independent of merit
- C. Cultural activities have no value
- D. All students have equal cultural resources

49. High-status positions (physician, lawyer) receiving greater prestige across societies suggests:

- A. Prestige is entirely arbitrary
- B. Cross-cultural consensus exists about occupational value, though stratification systems differ
- C. All occupations have equal prestige
- D. Prestige has no relationship to stratification

50. Wealth inequality with top 10% owning 70% while bottom 50% owns 2% demonstrates:

- A. Wealth is equally distributed
- B. Extreme concentration of resources at the top of the stratification system
- C. Everyone has the same wealth
- D. Wealth distribution doesn't affect society

### **DISCRETE QUESTIONS (51-59)**

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51. According to Piaget's theory, a child who understands that quantity remains constant despite changes in appearance has achieved:

- A. Object permanence
- B. Conservation
- C. Animism
- D. Centration

52. The bystander effect—decreased likelihood of helping when more people are present—is best explained by:

- A. Diffusion of responsibility
- B. Increased empathy with group size
- C. Physical inability to help in crowds
- D. Groupthink

53. Which neurotransmitter is primarily involved in reward and motivation, and is implicated in addiction?

- A. Serotonin
- B. GABA
- C. Dopamine
- D. Acetylcholine

54. Social facilitation refers to:

- A. Improved performance on simple/well-learned tasks in the presence of others
- B. Impaired performance on all tasks with others present
- C. Always helping others in social situations
- D. Social loafing in groups

55. A person who experiences recurrent, intrusive thoughts (obsessions) and feels compelled to perform repetitive behaviors (compulsions) most likely has:

- A. Generalized anxiety disorder
- B. Obsessive-compulsive disorder (OCD)
- C. Schizophrenia
- D. Major depressive disorder

56. According to Maslow's hierarchy of needs, which needs must be satisfied before self-actualization can be pursued?

- A. Only physiological needs
- B. Physiological, safety, love/belonging, and esteem needs
- C. No prerequisites exist
- D. Only safety needs

57. Ingroups and outgroups are defined as:

- A. Groups one belongs to (ingroup) versus groups one doesn't belong to (outgroup)
- B. Groups inside versus outside buildings
- C. Majority versus minority groups always
- D. Friends versus enemies exclusively

58. The prefrontal cortex, which continues developing into the mid-20s, is primarily responsible for:

- A. Basic reflexes
- B. Vision and hearing
- C. Executive functions including planning, impulse control, and decision-making
- D. Heart rate regulation

59. Medicalization refers to:

- A. Providing medical treatment to everyone
- B. Defining and treating conditions as medical problems that were not previously considered medical
- C. Building more hospitals
- D. Training more physicians

# SECTION 1: ANSWER EXPLANATIONS

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## 1. C - -61 kJ/mol

Using the Gibbs free energy equation:  $\Delta G = \Delta H - T\Delta S$ . Given:  $\Delta H = -240$  kJ/mol,  $\Delta S = -600$  J/(mol·K) =  $-0.600$  kJ/(mol·K), and  $T = 298$  K. Substituting:  $\Delta G = -240 - (298)(-0.600) = -240 + 178.8 = -61.2$  kJ/mol  $\approx -61$  kJ/mol. The negative value indicates the folding process is spontaneous at 25°C, with favorable enthalpy outweighing unfavorable entropy. Answer A would have a calculation error. Answer B incorrectly processes the signs. Answer D doesn't properly convert units. Remember to convert J to kJ when mixing units in thermodynamic calculations.

## 2. A - Increasing the entropy of the system (water + protein) as water molecules are released

The hydrophobic effect drives protein folding primarily through entropy increase of the overall system. When hydrophobic amino acids are exposed to water, surrounding water molecules become highly ordered in cage-like structures (decreased water entropy). When these residues cluster in the protein core away from water, the structured water molecules are released to bulk solvent, dramatically increasing water entropy. This entropy gain outweighs the conformational entropy loss of the protein chain. The passage explicitly states this "increases water entropy as structured water molecules around hydrophobic groups are released to bulk solvent." Answer B is wrong (hydrophobic interactions are noncovalent, not covalent bonds). Answer C describes electrostatic interactions but not the hydrophobic effect mechanism. Answer D describes disulfide bonds, which provide covalent stabilization but aren't the primary driver of the hydrophobic effect.

## 3. A - 127°C (400 K)

At equilibrium,  $\Delta G = 0$ , so the equation  $\Delta G = \Delta H - T\Delta S$  becomes  $0 = \Delta H - T\Delta S$ , which rearranges to  $T = \Delta H/\Delta S$ . Substituting the given values:  $T = (-240 \text{ kJ/mol}) / (-0.600 \text{ kJ/(mol·K)}) = 400$  K. Converting to Celsius:  $400 \text{ K} - 273 = 127^\circ\text{C}$ . At this temperature, the folding and unfolding reactions are equally favored, making  $\Delta G = 0$ . Above this temperature, the  $-T\Delta S$  term becomes increasingly negative (since  $\Delta S$  is negative,  $-T\Delta S$  becomes more negative as  $T$  increases), making  $\Delta G$  positive and favoring the unfolded state. This explains why the protein denatures at 75°C—we're approaching the equilibrium temperature where unfolding becomes favorable. Answer B would require different thermodynamic parameters. Answer C is far too high. Answer D is incorrect because the calculation is straightforward with the given data.

## 4. B - The primary structure determines the final three-dimensional conformation

The reversibility of heat denaturation demonstrates Anfinsen's principle: the amino acid sequence (primary structure) contains all information necessary to specify the three-dimensional structure. When heated to 75°C, the protein unfolds as noncovalent interactions (hydrogen bonds, hydrophobic interactions, van der Waals forces) are disrupted by thermal energy. Upon cooling, these interactions reform spontaneously, and the protein refolds to its native structure without external assistance. This proves that no additional information beyond the amino acid sequence is required for proper folding.

Answer A is wrong (peptide bonds are covalent with high bond energy and remain intact at 75°C—only noncovalent interactions are disrupted). Answer C is wrong (the passage and this experiment demonstrate that many proteins fold spontaneously without chaperones, though chaperones assist some proteins). Answer D is chemically nonsensical (hydrophobicity is an intrinsic property of amino acids that doesn't change with temperature).

### 5. C - Disrupting hydrogen bonds and hydrophobic interactions, making $\Delta G$ positive

Urea is a chaotropic (disorder-inducing) agent that disrupts the noncovalent interactions stabilizing protein structure through multiple mechanisms. It forms hydrogen bonds with both the protein backbone and polar side chains, competing with intramolecular hydrogen bonds. It also disrupts the hydrophobic effect by reducing the entropic penalty for exposing hydrophobic residues to solvent—essentially making water a "better" solvent for hydrophobic groups. These disruptions destabilize the folded state relative to the unfolded state, making  $\Delta G$  positive and shifting the equilibrium toward unfolding. The passage explicitly states that in 8 M urea, " $\Delta G$  became positive." Answer A is wrong (urea doesn't hydrolyze peptide bonds—these covalent bonds are chemically stable in aqueous urea). Answer B is wrong (urea is not a reducing agent and doesn't affect disulfide bonds; reducing agents like  $\beta$ -mercaptoethanol or DTT break disulfide bonds). Answer D is nonsensical (urea doesn't affect solution temperature).

### 6. A - +1.14 V

To find the cell potential for NADH oxidation by oxygen, we combine the two half-reactions. The passage gives  $\text{NAD}^+/\text{NADH}$  reduction potential as  $E^\circ = -0.32 \text{ V}$ , and  $\text{O}_2/\text{H}_2\text{O}$  reduction potential as  $E^\circ = +0.82 \text{ V}$ . For the overall reaction where NADH is oxidized (loses electrons) and  $\text{O}_2$  is reduced (gains electrons), the standard cell potential is:  $E^\circ_{\text{cell}} = E^\circ_{\text{cathode}} - E^\circ_{\text{anode}} = E^\circ(\text{O}_2/\text{H}_2\text{O}) - E^\circ(\text{NAD}^+/\text{NADH}) = 0.82 - (-0.32) = 1.14 \text{ V}$ . The large positive value indicates this is a highly spontaneous reaction, which is why it provides substantial free energy for ATP synthesis in cellular respiration. Answer B would result from incorrect arithmetic or using wrong half-reactions. Answers C and D have incorrect signs—a positive cell potential indicates a spontaneous, energy-releasing reaction.

### 7. B - Higher than $E^\circ_{\text{cell}}$ due to decreased product concentration

Using the Nernst equation:  $E = E^\circ - (0.0592/n)\log(Q)$ , where  $Q$  is the reaction quotient. For the cell reaction  $\text{Zn}(s) + \text{Cu}^{2+}(\text{aq}) \rightarrow \text{Zn}^{2+}(\text{aq}) + \text{Cu}(s)$ ,  $Q = [\text{Zn}^{2+}]/[\text{Cu}^{2+}] = 0.01/1.0 = 0.01$ . Since  $Q < 1$ ,  $\log(Q)$  is negative ( $\log(0.01) = -2$ ). The term  $-(0.0592/2)(-2)$  becomes positive, adding to  $E^\circ_{\text{cell}}$ . Therefore:  $E = 1.10 - (0.0592/2)(-2) = 1.10 + 0.0592 = 1.16 \text{ V}$ , which is higher than  $E^\circ_{\text{cell}}$ . Lower product concentration and higher reactant concentration both favor the forward reaction, increasing cell potential above the standard value. Answer A is opposite of what occurs. Answer C is wrong (concentration changes always affect cell potential according to the Nernst equation). Answer D is wrong (the positive  $E^\circ$  and favorable  $Q$  ensure the reaction remains spontaneous with positive potential).

### 8. D - -212 kJ

Using the relationship between free energy and cell potential:  $\Delta G = -nFE$ , where  $n = 2$  moles of electrons transferred,  $F = 96,485 \text{ C/mol}$  (Faraday constant), and  $E = 1.10 \text{ V}$ . Substituting:  $\Delta G = -(2 \text{ mol } e^-)(96,485 \text{ C/mol})(1.10 \text{ V}) = -212,267 \text{ J} \approx -212 \text{ kJ}$ . The negative value indicates energy is released (exergonic).

process), which can be harnessed to do work. Each mole of electrons transferred through the potential difference releases energy proportional to the voltage. Answer A would be for  $n = 1$  (only one mole of electrons). Answer B would be for a different voltage or electron count. Answer C doesn't match the calculation. Remember that  $\text{voltage} \times \text{charge} = \text{energy}$ , or in SI units:  $\text{volts} \times \text{coulombs} = \text{joules}$ .

### 9. A - $[\text{Zn}^{2+}]$ will increase and $[\text{Cu}^{2+}]$ will decrease

As the galvanic cell operates, the spontaneous redox reaction proceeds:  $\text{Zn(s)} + \text{Cu}^{2+}(\text{aq}) \rightarrow \text{Zn}^{2+}(\text{aq}) + \text{Cu(s)}$ . At the anode (negative terminal), zinc metal is oxidized:  $\text{Zn(s)} \rightarrow \text{Zn}^{2+}(\text{aq}) + 2\text{e}^-$ , producing  $\text{Zn}^{2+}$  ions and increasing their concentration. At the cathode (positive terminal), copper ions are reduced:  $\text{Cu}^{2+}(\text{aq}) + 2\text{e}^- \rightarrow \text{Cu(s)}$ , consuming  $\text{Cu}^{2+}$  ions from solution and decreasing their concentration. Solid copper plates out on the cathode surface. This process continues until the system reaches equilibrium, at which point the concentration ratio equals the equilibrium constant and the cell potential drops to zero. Answer B is opposite of what actually occurs. Answer C describes the final equilibrium state after the cell has stopped operating, not what happens during operation. Answer D is wrong (cell potential decreases over time as the concentration gradient diminishes and the system approaches equilibrium).

### 10. B - 7.4

Using the Henderson-Hasselbalch equation:  $\text{pH} = \text{pK}_a + \log\left(\frac{[\text{A}^-]}{[\text{HA}]}\right) = \text{pK}_a + \log\left(\frac{[\text{HCO}_3^-]}{[\text{H}_2\text{CO}_3]}\right)$ . Substituting the given values:  $\text{pH} = 6.1 + \log(24/1.2) = 6.1 + \log(20) = 6.1 + 1.30 = 7.40$ . This perfectly matches normal blood pH and demonstrates why the bicarbonate buffer system is so effective at maintaining physiological pH. The 20:1 ratio of bicarbonate to carbonic acid is carefully regulated in healthy individuals through respiratory control ( $\text{CO}_2$  levels) and renal function (bicarbonate reabsorption). Answer A (6.1) is just the  $\text{pK}_a$  value without accounting for the concentration ratio. Answer C (7.9) would require a ratio of approximately 63:1. Answer D (8.4) would require an even higher ratio of about 200:1, which would represent severe alkalosis.

### 11. C - 6.8

After strong acid addition, the new concentrations are  $[\text{HCO}_3^-] = 12 \text{ mM}$  and  $[\text{H}_2\text{CO}_3] = 2.4 \text{ mM}$ . Using Henderson-Hasselbalch:  $\text{pH} = \text{pK}_a + \log\left(\frac{[\text{HCO}_3^-]}{[\text{H}_2\text{CO}_3]}\right) = 6.1 + \log(12/2.4) = 6.1 + \log(5) = 6.1 + 0.70 = 6.80$ . The pH decreased from 7.4 to 6.8, a drop of 0.6 pH units. This demonstrates the buffer's effectiveness—the added acid was partially neutralized by the buffer, preventing a more dramatic pH drop. Without buffering, adding the same amount of strong acid would have decreased pH far more severely. The ratio changed from 20:1 to 5:1, and the pH change reflects the logarithm of this ratio change. This represents metabolic acidosis (accumulation of acid) that would trigger compensatory mechanisms including increased respiratory rate to blow off  $\text{CO}_2$ .

### 12. B - The buffer capacity is greatest when $\text{pH} = \text{pK}_a$

Buffer capacity—the ability to resist pH changes when acid or base is added—is maximum when pH equals the  $\text{pK}_a$ , where  $[\text{A}^-] = [\text{HA}]$  and the ratio is 1:1. At this point, the buffer has optimal amounts of both the acid form (to neutralize added base) and the base form (to neutralize added acid). Buffers work effectively within approximately  $\pm 1$  pH unit of the  $\text{pK}_a$ , where the  $[\text{A}^-]/[\text{HA}]$  ratio ranges from 10:1 to 1:10. At pH 8.2, one full pH unit above the  $\text{pK}_a$  of 7.2, the ratio  $[\text{HPO}_4^{2-}]/[\text{H}_2\text{PO}_4^-]$  would be 10:1, meaning

the buffer exists mostly in the base form with relatively little acid form available to neutralize added base. The buffer is much less effective outside its optimal range. Answer A is nonsensical (phosphate toxicity isn't related to pH-dependent buffer effectiveness). Answer C is wrong (phosphate can accept protons at any pH where the acid form exists; the issue is concentration, not ability). Answer D is wrong (the Henderson-Hasselbalch equation applies at all pH values, though buffer effectiveness varies).

### 13. A - Respiratory alkalosis with increased blood pH

Hyperventilation removes CO<sub>2</sub> from the blood more rapidly than it's produced by metabolism. This shifts the equilibrium  $\text{CO}_2 + \text{H}_2\text{O} \rightleftharpoons \text{H}_2\text{CO}_3 \rightleftharpoons \text{HCO}_3^- + \text{H}^+$  to the left, consuming H<sup>+</sup> ions and increasing pH (making blood more alkaline). With [H<sub>2</sub>CO<sub>3</sub>] decreasing from 1.2 mM to 0.6 mM while [HCO<sub>3</sub><sup>-</sup>] initially remains at 24 mM, the ratio increases from 20:1 to 40:1. Using Henderson-Hasselbalch:  $\text{pH} = 6.1 + \log(40) = 6.1 + 1.60 = 7.70$ , showing significant alkalosis. This is termed "respiratory" alkalosis because it's caused by changes in breathing (respiration), and "alkalosis" because pH increases above the normal 7.35-7.45 range. This can cause dizziness, tingling in extremities, and muscle spasms. Answer B is opposite (acidosis means low pH, and this is definitely not acidosis). Answer C is wrong (pH definitely changes when the buffer component ratio changes, even if bicarbonate concentration doesn't change initially). Answer D describes metabolic acidosis, which would result from acid accumulation, not hyperventilation.

### 14. B - Tris buffer (pKa = 8.1)

For optimal buffering, select a buffer with pKa close to the desired pH—ideally within ±0.5 pH units, and certainly within ±1 pH unit. For maintaining pH 7.8, Tris buffer with pKa = 8.1 is only 0.3 pH units away, placing it within the optimal buffering range. At pH 7.8 with pKa 8.1, the Henderson-Hasselbalch equation gives:  $7.8 = 8.1 + \log([A^-]/[HA])$ , so  $\log([A^-]/[HA]) = -0.3$ , meaning  $[A^-]/[HA] = 0.50$  or approximately 1:2. This is an excellent ratio for buffering capacity, with substantial amounts of both acid and base forms present. Answer A (acetic acid, pKa 4.76) is about 3 pH units below the target—far too low to buffer effectively at pH 7.8. Answer C (bicarbonate, pKa 6.1) is 1.7 units below the target—marginal effectiveness at best. Answer D (citric acid, pKa<sub>1</sub> 3.1) is nearly 5 units below the target—essentially no buffering capacity at pH 7.8. Tris is commonly used in biochemical applications requiring pH in the 7-9 range.

### 15. D - Competitive inhibitor

Inhibitor X increases the apparent K<sub>m</sub> from 2 mM to 8 mM while V<sub>max</sub> remains unchanged at 100 μmol/min. This is the diagnostic pattern for competitive inhibition. Competitive inhibitors structurally resemble the substrate and compete with it for binding to the enzyme's active site. When inhibitor is bound, substrate cannot bind, and vice versa. Increasing substrate concentration overcomes the inhibition because at sufficiently high [S], substrate outcompetes inhibitor for active site binding, allowing the enzyme to reach normal V<sub>max</sub>. The increased apparent K<sub>m</sub> reflects that more substrate is needed to achieve half-maximal velocity in the presence of competitive inhibitor. The passage confirms that "high substrate concentrations overcame inhibition." Answer A (noncompetitive inhibitor) would decrease V<sub>max</sub> while leaving K<sub>m</sub> unchanged. Answer B (uncompetitive inhibitor) would decrease both K<sub>m</sub> and V<sub>max</sub> proportionally. Answer C (allosteric activator) would increase activity, not maintain it while requiring more substrate.

### 16. B - Binds to an allosteric site

Inhibitor Y decreases  $V_{max}$  from 100 to 50  $\mu\text{mol}/\text{min}$  while  $K_m$  remains unchanged at 2 mM. This pattern indicates noncompetitive inhibition, where the inhibitor binds to a site distinct from the active site (an allosteric site). Since the inhibitor doesn't compete with substrate for the active site, substrate affinity (reflected in  $K_m$ ) is unchanged. However, when inhibitor is bound to its allosteric site, it reduces the enzyme's catalytic efficiency or the amount of functional enzyme available, decreasing  $V_{max}$ . High substrate concentrations cannot overcome this inhibition because substrate and inhibitor bind at different locations—there's no competition. The data shows "high substrate concentrations couldn't overcome inhibition," confirming noncompetitive behavior. Answer A (competes with substrate) describes competitive inhibition, which would change  $K_m$ . Answer C (binds to ES complex) describes uncompetitive inhibition, which would decrease both parameters. Answer D (permanent modification) suggests irreversible inhibition, but the kinetic pattern specifically indicates reversible noncompetitive inhibition.

### 17. B - Uncompetitive inhibitor

Inhibitor Z decreases both  $V_{max}$  (100 to 60  $\mu\text{mol}/\text{min}$ ) and apparent  $K_m$  (2.0 to 1.2 mM) proportionally. This is the signature of uncompetitive inhibition, where the inhibitor binds only to the enzyme-substrate (ES) complex, not to free enzyme. When inhibitor binds to ES, it removes that complex from the reaction pathway, preventing product formation. This decreases  $V_{max}$  (less enzyme available to form product). Because the inhibitor binding removes ES from solution, it shifts the equilibrium  $E + S \rightleftharpoons ES$  to the right, appearing to increase substrate affinity (decrease  $K_m$ ). The key observation that "inhibition increased with substrate concentration" confirms uncompetitive inhibition—more substrate means more ES complex forms, providing more targets for the inhibitor to bind. Answer A (competitive) would increase  $K_m$ , not decrease it. Answer C (mixed inhibitor) would affect  $K_m$  unpredictably and not maintain proportional changes. Answer D (irreversible) doesn't explain the specific and proportional kinetic pattern observed.

### 18. A - 50 $\mu\text{mol}/\text{min}$

Using the Michaelis-Menten equation:  $v = (V_{max}[S])/(K_m + [S])$ . When  $[S] = K_m = 2 \text{ mM}$ :  $v = (100 \times 2)/(2 + 2) = 200/4 = 50 \mu\text{mol}/\text{min}$ . This result demonstrates the definition of  $K_m$ : it is the substrate concentration at which the reaction velocity equals half the maximum velocity ( $V_{max}/2$ ). At this substrate concentration, exactly half of the enzyme's active sites are occupied by substrate on average. Below  $K_m$ , velocity increases almost linearly with  $[S]$ . Above  $K_m$ , the enzyme approaches saturation and velocity increases more slowly, eventually plateauing at  $V_{max}$ . Answer B (25  $\mu\text{mol}/\text{min}$ ) would be the velocity at  $[S] = K_m/3$  or when  $V_{max} = 100$  and we're at 25% of maximum. Answer C (100  $\mu\text{mol}/\text{min}$ ) is  $V_{max}$ , achieved only at saturating substrate concentrations ( $[S] \gg K_m$ ). Answer D (33  $\mu\text{mol}/\text{min}$ ) would be  $v$  at approximately  $[S] = K_m/2$ .

### 19. C - +33.4 kJ/mol (endothermic)

The temperature decrease from 25.0°C to 20.0°C ( $\Delta T = -5.0^\circ\text{C}$ ) indicates an endothermic dissolution process—heat was absorbed from the surroundings (water) by the dissolving  $\text{NH}_4\text{NO}_3$ . Calculating heat lost by water:  $q_{\text{water}} = mc\Delta T = (100 \text{ g})(4.18 \text{ J}/(\text{g}\cdot^\circ\text{C}))(-5.0^\circ\text{C}) = -2090 \text{ J}$ . The dissolution process absorbed this heat:  $q_{\text{dissolution}} = -q_{\text{water}} = +2090 \text{ J} = +2.09 \text{ kJ}$  (for 5.0 g of  $\text{NH}_4\text{NO}_3$ ). Converting to molar basis: moles of  $\text{NH}_4\text{NO}_3 = 5.0 \text{ g} \div 80 \text{ g/mol} = 0.0625 \text{ mol}$ . Molar enthalpy of dissolution:  $\Delta H = 2.09 \text{ kJ} \div 0.0625 \text{ mol} = 33.4 \text{ kJ/mol}$ . The positive value confirms the process is endothermic. Many ionic salts dissolve endothermically because the lattice energy (energy required to separate ions in the solid, endothermic) exceeds the hydration energy (energy released when ions are surrounded by water, exothermic). Despite being endothermic, the dissolution is spontaneous because of the large positive entropy change (increased disorder). Answer A uses incorrect values or calculation. Answer B has the wrong sign (process is endothermic, not exothermic). Answer D incorrectly adds or uses wrong mass.

## 20. C - 16.0 kJ

In a bomb calorimeter, the heat released by the combustion reaction is absorbed by the calorimeter and its contents. Heat released equals:  $q_{\text{rxn}} = C_{\text{cal}}\Delta T = (10.0 \text{ kJ}/^\circ\text{C})(26.6^\circ\text{C} - 25.0^\circ\text{C}) = (10.0 \text{ kJ}/^\circ\text{C})(1.6^\circ\text{C}) = 16.0 \text{ kJ}$ . This represents the actual heat released by burning 1.00 g of glucose under these conditions. The calorimeter's heat capacity (10.0 kJ/°C) accounts for all components that absorb heat—the water, the bomb itself, the thermometer, and other parts of the apparatus. Answer A incorrectly uses  $\Delta T = 1.0^\circ\text{C}$  instead of 1.6°C. Answer B miscalculates or uses wrong values. Answer D incorrectly adds initial and final temperatures or uses wrong formula. Bomb calorimeters measure heat at constant volume ( $\Delta E$ ), which for reactions without gas mole changes equals  $\Delta H$ .

## 21. A - Yes, it's very close to -2803 kJ/mol

Converting the experimental result to a molar basis:  $1.00 \text{ g glucose} \div 180 \text{ g/mol} = 0.00556 \text{ mol glucose}$  burned. Heat released per mole:  $16.0 \text{ kJ} \div 0.00556 \text{ mol} = 2878 \text{ kJ/mol}$ . Comparing to the theoretical value of 2803 kJ/mol: the experimental value is within about 3% of theory  $[(2878-2803)/2803 \times 100\% = 2.7\%]$ , which represents excellent agreement for calorimetry. The slight discrepancy could arise from incomplete combustion, heat transfer inefficiencies, measurement errors, or calorimeter calibration issues. For glucose combustion ( $\text{C}_6\text{H}_{12}\text{O}_6 + 6\text{O}_2 \rightarrow 6\text{CO}_2 + 6\text{H}_2\text{O}$ ),  $\Delta n(\text{gas}) = 6 - 6 = 0$ , meaning  $\Delta H = \Delta E$ , so bomb calorimeter measurements (constant volume, measuring  $\Delta E$ ) can be directly compared to standard enthalpy values (constant pressure,  $\Delta H$ ). Answer B is wrong (experimental is slightly higher, not much higher). Answer C is wrong (experimental is very close, not much lower). Answer D is incorrect (units can be converted for comparison, and  $\Delta n = 0$  makes  $\Delta H = \Delta E$  for this reaction).

## 22. B - 2.67 kJ

For the neutralization reaction, total solution volume = 50.0 mL + 50.0 mL = 100.0 mL. With density 1.0 g/mL, the total mass = 100.0 g. Temperature change:  $\Delta T = 31.4^\circ\text{C} - 25.0^\circ\text{C} = 6.4^\circ\text{C}$ . Heat released:  $q = mc\Delta T = (100.0 \text{ g})(4.18 \text{ J}/(\text{g}\cdot^\circ\text{C}))(6.4^\circ\text{C}) = 2675 \text{ J} \approx 2.67 \text{ kJ}$ . The positive temperature change indicates an exothermic reaction—acid-base neutralization releases heat. This heat warms the solution, which is why the temperature increased. Strong acid-strong base neutralization is always exothermic because bond formation ( $\text{H}_2\text{O}$ ) releases more energy than is required to break the existing bonds. Answer A uses the wrong temperature change value. Answer C incorrectly doubles the correct value. Answer D is far too high and represents a calculation error.

**23. A - -53.4 kJ/mol**

First, determine moles of water formed, which equals moles of the limiting reagent. Both HCl and NaOH are present in equal molar amounts: moles = (1.0 M)(0.0500 L) = 0.0500 mol each. The neutralization reaction is 1:1:  $\text{HCl} + \text{NaOH} \rightarrow \text{NaCl} + \text{H}_2\text{O}$ , so 0.0500 mol  $\text{H}_2\text{O}$  forms. Molar enthalpy of neutralization:  $\Delta H = -2.67 \text{ kJ} \div 0.0500 \text{ mol} = -53.4 \text{ kJ/mol}$ . The negative sign indicates the reaction is exothermic (releases heat). This value is typical for strong acid-strong base neutralization reactions, which all release approximately 55-57 kJ/mol because the net ionic equation is always the same:  $\text{H}^+(\text{aq}) + \text{OH}^-(\text{aq}) \rightarrow \text{H}_2\text{O}(\text{l})$ . The slight variation from the typical -56 kJ/mol could reflect experimental error or heat losses. Answer B is half the correct value. Answers C and D have the wrong sign—neutralization is exothermic, not endothermic, which is why the solution temperature increased.

**24. C - 50 cm (the patient's far point)**

For myopia (nearsightedness) correction, the corrective lens must create a virtual image at the patient's far point when the object is at infinity (distant objects). Patient A's far point is 50 cm—the farthest distance at which they can see clearly without correction. When looking at a distant object (object at infinity,  $d_o = \infty$ ), the diverging lens creates a virtual image at 50 cm in front of the lens. The patient's eye then focuses on this virtual image at 50 cm, which is within their range of clear vision (0 to 50 cm). This allows the patient to see distant objects clearly. Answer A is wrong (infinity is where the object is, not where the image forms). Answer B (25 cm) is the normal near point for reading, not relevant for distance vision. Answer D misunderstands the correction mechanism (the lens creates an image the eye can focus on; it doesn't directly create an image on the retina).

**25. B - -50 cm**

Using the relationship between lens power and focal length:  $P = 1/f$ , where P is in diopters and f is in meters. For Patient A's lens with  $P = -2.0 \text{ D}$ :  $f = 1/P = 1/(-2.0 \text{ D}) = -0.50 \text{ m} = -50 \text{ cm}$ . The negative focal length confirms this is a diverging (concave) lens, which is used to correct myopia. Diverging lenses spread light rays apart, creating virtual images closer to the lens than the object, allowing myopic patients to see distant objects by forming images at their far point. Answer A has the wrong sign (positive focal length would be a converging lens used for hyperopia). Answers C and D represent  $f = \pm 0.5 \text{ m} = \pm 50 \text{ cm}$  but are stated incorrectly, and C has the wrong sign. The power in diopters equals the reciprocal of focal length in meters.

**26. C - 75 cm**

For hyperopia (farsightedness) correction when reading, the converging lens must create a virtual image at the patient's actual near point when the object is at the desired reading distance. Patient B's near point is 75 cm (cannot focus closer than this). Using the thin lens equation  $1/f = 1/d_o + 1/d_i$ , where  $f = 1/P = 1/(+2.7 \text{ D}) = 0.370 \text{ m} = 37 \text{ cm}$ , and  $d_o = 25 \text{ cm}$  (normal reading distance):  $1/37 = 1/25 + 1/d_i$ . Solving:  $1/d_i = 1/37 - 1/25 = (25-37)/(37 \times 25) = -12/925$ , so  $d_i = -925/12 \approx -77 \text{ cm}$ . The negative sign indicates a virtual image (formed on the same side as the object), and the distance of approximately 77 cm is close to the patient's near point of 75 cm. The lens allows the patient to read at 25 cm by forming a virtual image at ~75 cm where their eye can actually focus. Answer A is the lens's focal length, not the image distance. Answer B would place the image too close. Answer D places the image too far.

**27. C - -400×**

For a compound microscope, total magnification  $M = m_{\text{objective}} \times m_{\text{eyepiece}}$ . The objective's magnification when the object is near its focal point is approximated as:  $m_o \approx -d_i/f_o = -160 \text{ mm} / 4 \text{ mm} = -40\times$ . The eyepiece acts as a magnifying glass, with magnification:  $m_e = 25 \text{ cm} / f_e = 250 \text{ mm} / 25 \text{ mm} = 10\times$  (using the standard near point distance of 25 cm). Total magnification:  $M = m_o \times m_e = (-40)(10) = -400\times$ . The negative sign indicates the final image is inverted relative to the object. Answer A would require a much longer objective focal length. Answer B would require different optical parameters. Answer D would require a shorter eyepiece focal length or different objective parameters. Standard laboratory microscopes typically provide magnifications in the range of  $40\times$  to  $1000\times$ .

**28. C - 40 cm in front of the lens (virtual image)**

Using the thin lens equation  $1/f = 1/d_o + 1/d_i$  with  $f = -50 \text{ cm}$  (from the  $-2.0 \text{ D}$  lens) and  $d_o = 200 \text{ cm}$  (object at 2.0 m):  $1/(-50) = 1/200 + 1/d_i$ . Solving:  $1/d_i = -1/50 - 1/200 = -4/200 - 1/200 = -5/200 = -1/40$ , giving  $d_i = -40 \text{ cm}$ .

**29. B - 12.3 L**

Using the ideal gas law  $PV = nRT$ , where  $n = 2.0 \text{ mol}$ ,  $R = 0.0821 \text{ L}\cdot\text{atm}/(\text{mol}\cdot\text{K})$ ,  $T = 27^\circ\text{C} = 300 \text{ K}$  (convert Celsius to Kelvin by adding 273), and  $P = 4.0 \text{ atm}$ . Solving for volume:  $V = nRT/P = (2.0 \text{ mol})(0.0821 \text{ L}\cdot\text{atm}/(\text{mol}\cdot\text{K}))(300 \text{ K}) / (4.0 \text{ atm}) = 49.26 \text{ L}\cdot\text{atm} / 4.0 \text{ atm} = 12.3 \text{ L}$ . This represents the volume occupied by the gas under these conditions. Remember that temperature must always be in Kelvin for gas law calculations—this is a common source of error. Answer A uses incorrect temperature or calculation. Answer C would be correct for half the pressure (2.0 atm). Answer D would be correct if pressure were 1.0 atm instead of 4.0 atm. Always check unit consistency and temperature conversion.

**30. D - 3.0 atm**

According to Dalton's law of partial pressures, each gas in a mixture contributes to the total pressure in proportion to its mole fraction. The passage states there are equal moles of He,  $\text{N}_2$ , and  $\text{O}_2$ , so each gas has a mole fraction of  $1/3$ . The partial pressure of each component:  $P_{\text{gas}} = \chi_{\text{gas}} \times P_{\text{total}} = (1/3)(9.0 \text{ atm}) = 3.0 \text{ atm}$ . This relationship holds regardless of the gases' molecular masses—partial pressure depends only on mole fraction, not on molecular weight. The fact that  $\text{N}_2$  (28 g/mol) is heavier than He (4 g/mol) or lighter than  $\text{O}_2$  (32 g/mol) is irrelevant to partial pressure calculation. Answer A would be correct if there were six components with equal moles. Answer B would be correct for four equal components. Answer C would be correct for two equal components. The key is recognizing that partial pressure is proportional to mole fraction, not mass fraction.

**31. C - 16 g/mol**

Graham's law of effusion states that the rate of effusion is inversely proportional to the square root of molar mass:  $\text{Rate}_1/\text{Rate}_2 = \sqrt{M_2/M_1}$ . The unknown gas effuses 2.0 times slower than He, so:  $\text{Rate}_{\text{unknown}}/\text{Rate}_{\text{He}} = 1/2.0$ . Substituting into Graham's law:  $1/2.0 = \sqrt{(M_{\text{He}}/M_{\text{unknown}})} = \sqrt{(4/M_{\text{unknown}})}$ . Squaring both sides:  $1/4 = 4/M_{\text{unknown}}$ . Cross-multiplying:  $M_{\text{unknown}} = 4 \times 4 = 16 \text{ g/mol}$ . This molar mass corresponds to methane ( $\text{CH}_4$ ) or an oxygen atom. The inverse square root

relationship explains why lighter molecules effuse faster—they have higher average velocities at the same temperature. Answer A (1 g/mol) would be hydrogen, which effuses twice as fast as helium, not slower. Answer B (8 g/mol) would effuse  $\sqrt{(8/4)} = \sqrt{2} \approx 1.4$  times slower than He. Answer D (32 g/mol, O<sub>2</sub>) would effuse  $\sqrt{(32/4)} = \sqrt{8} \approx 2.83$  times slower than He.

### 32. A - Intermolecular attractive forces reducing pressure

At high pressure, gas molecules are forced close together, and intermolecular attractive forces (van der Waals forces, dipole interactions) become significant. These attractive forces pull molecules toward each other, reducing the force of their collisions with the container walls, thereby decreasing the measured pressure below what the ideal gas law predicts. Real gases deviate from ideal behavior when: (1) pressure is high (molecules close together, making attractive forces significant), and (2) temperature is low (molecules moving slowly, allowing more time for attractions to affect motion). The van der Waals equation corrects for this:  $(P + a(n/V)^2)(V - nb) = nRT$ , where the "a" term accounts for intermolecular attractions. For CO<sub>2</sub>, which has a substantial dipole moment and polarizability, these attractive forces are particularly strong. Answer B is too vague and doesn't explain the mechanism. Answer C is extreme (liquefaction would be obvious and represents a phase change, not just deviation). Answer D is unsupported and unlikely given proper experimental technique.

### 33. D - 0.118 V

For a concentration cell, the Nernst equation simplifies to:  $E_{\text{cell}} = (0.0592/n)\log([\text{concentrated}]/[\text{dilute}])$ . For the silver concentration cell where  $n = 1$  ( $\text{Ag}^+ + e^- \rightarrow \text{Ag}$ ):  $E_{\text{cell}} = (0.0592/1)\log(1.0 \text{ M} / 0.010 \text{ M}) = 0.0592 \times \log(100) = 0.0592 \times 2.00 = 0.118 \text{ V}$ . The 100-fold concentration difference provides the driving force for electron flow from the dilute compartment (where Ag is oxidized) to the concentrated compartment (where Ag<sup>+</sup> is reduced), despite both electrodes being made of the same material. This demonstrates that concentration gradients alone can drive electrochemical reactions. Answer A (0.000 V) would only occur when concentrations are equal (at equilibrium). Answer B uses the wrong concentration ratio or  $n$  value. Answer C uses  $\log(10) = 1$  instead of  $\log(100) = 2$ , calculating for a 10-fold rather than 100-fold difference.

### 34. A - 0.059 V

For the copper concentration cell,  $\text{Cu}^{2+} + 2e^- \rightarrow \text{Cu}$ , so  $n = 2$ . Using the Nernst equation:  $E_{\text{cell}} = (0.0592/2)\log(0.100 \text{ M} / 0.001 \text{ M}) = 0.0296 \times \log(100) = 0.0296 \times 2.00 = 0.059 \text{ V}$ . The two-electron transfer process ( $n = 2$ ) reduces the cell potential by half compared to a one-electron system with the same concentration ratio. This is because the Nernst equation divides by  $n$ —more electrons transferred per reaction means smaller voltage per electron. The concentration ratio is also 100-fold (like the silver cell), but because copper involves two electrons, the potential is approximately half that of the silver cell. Answer B would be for a different concentration ratio. Answer C incorrectly combines values. Answer D would be correct only if  $n = 1$  instead of  $n = 2$ .

### 35. B - The electrode in 0.010 M Ag<sup>+</sup> solution

In any concentration cell, oxidation (loss of electrons) occurs at the electrode in the more dilute solution, making it the anode. At the dilute electrode (0.010 M Ag<sup>+</sup>), the reaction  $\text{Ag}(s) \rightarrow \text{Ag}^+(\text{aq}) + e^-$  occurs,

increasing the  $\text{Ag}^+$  concentration in that compartment. Simultaneously, at the concentrated electrode (1.0 M  $\text{Ag}^+$ ), reduction occurs:  $\text{Ag}^+(\text{aq}) + \text{e}^- \rightarrow \text{Ag}(\text{s})$ , decreasing the  $\text{Ag}^+$  concentration. The cell spontaneously operates to equalize the concentration difference. Electrons flow through the external circuit from anode (dilute, 0.010 M) to cathode (concentrated, 1.0 M), while cations flow through the salt bridge from anode to cathode to maintain electrical neutrality. Answer A misunderstands concentration cell operation—the concentration difference, not identical materials, is what matters. Answer C is backwards (concentrated solution is the cathode where reduction occurs). Answer D violates electrochemical principles (one must be anode, one must be cathode).

### 36. C - -94 mV

For a single ion crossing a membrane, the Nernst equation gives the equilibrium potential:  $E = (RT/zF)\ln([\text{ion}]_{\text{out}}/[\text{ion}]_{\text{in}})$ , where  $z = +1$  for  $\text{K}^+$ ,  $R = 8.314 \text{ J}/(\text{mol}\cdot\text{K})$ ,  $T = 310 \text{ K}$  ( $37^\circ\text{C}$ ),  $F = 96,485 \text{ C}/\text{mol}$ . Substituting:  $E = [(8.314)(310)/(1)(96,485)]\ln(4/140) = (0.0267)\ln(0.0286) = (0.0267)(-3.555) = -0.0949 \text{ V} = -94.9 \text{ mV} \approx -94 \text{ mV}$ . The negative value indicates that at equilibrium, the inside of the cell is negative relative to the outside. This  $\text{K}^+$  equilibrium potential (about -94 mV) is close to typical neuronal resting membrane potentials (-70 mV), reflecting  $\text{K}^+$ 's dominant role in setting resting potential. The actual resting potential is somewhat less negative than  $E_{\text{K}}$  because of small  $\text{Na}^+$  and  $\text{Cl}^-$  contributions. Answer A uses  $25^\circ\text{C}$  instead of  $37^\circ\text{C}$ . Answer B might result from calculation errors. Answer D is too negative, suggesting wrong concentration ratio or calculation error.

### 37. D - The cell potential decreases as concentrations equalize

As the concentration cell operates over time, the spontaneous reaction proceeds: at the anode (dilute solution), metal is oxidized, producing ions and increasing concentration; at the cathode (concentrated solution), ions are reduced to metal, decreasing concentration. The concentration gradient progressively diminishes. Since the Nernst equation shows  $E_{\text{cell}} = (RT/nF)\ln([\text{concentrated}]/[\text{dilute}])$ , as the ratio  $[\text{concentrated}]/[\text{dilute}]$  approaches 1, the natural logarithm approaches  $\ln(1) = 0$ , making  $E_{\text{cell}}$  approach zero. At equilibrium, when concentrations are equal, the cell potential reaches zero and no further net reaction occurs—the cell is "dead." This explains why batteries eventually die. Answer A is opposite to what occurs. Answer B is opposite (gradient decreases, not increases). Answer C incorrectly suggests potential increases (work output actually decreases as the system approaches equilibrium and available free energy diminishes).

### 38. D - Lower due to increased kinetic energy of faster-moving blood

Bernoulli's equation states:  $P + (1/2)\rho v^2 + \rho gh = \text{constant}$ . The kinetic energy term is  $(1/2)\rho v^2$ . When blood velocity increases from 26.5 cm/s in the normal aorta to 106 cm/s in the narrowed region (about  $4\times$  faster), the kinetic energy term increases by a factor of 16 (since  $\text{KE} \propto v^2$ ). To maintain the constant sum in Bernoulli's equation, pressure  $P$  must decrease significantly. In the narrowed atherosclerotic region, blood accelerates (continuity equation requires higher velocity in narrower vessels), converting pressure energy into kinetic energy. This pressure drop can be problematic—reduced pressure may impair perfusion of tissues downstream, and very high velocities can damage blood vessel walls. This is why severe stenosis (narrowing) can be dangerous even if total blood flow is maintained. Answer A is opposite. Answer B identifies an effect but not the correct mechanism. Answer C is wrong (pressure changes when velocity changes according to Bernoulli's equation).

**39. B - 0.033 cm/s**

Using the continuity equation for incompressible fluids:  $Q = Av$  (flow rate = area  $\times$  velocity). The flow rate must be constant throughout the circulatory system:  $Q_{\text{aorta}} = Q_{\text{capillaries}}$ . Given  $Q = 83 \text{ cm}^3/\text{s}$  and total capillary cross-sectional area =  $2500 \text{ cm}^2$ :  $v_{\text{cap}} = Q/A = 83 \text{ cm}^3/\text{s} \div 2500 \text{ cm}^2 = 0.033 \text{ cm/s}$ . This dramatic velocity decrease in capillaries (from  $\sim 26 \text{ cm/s}$  in aorta to  $0.033 \text{ cm/s}$  in capillaries—about  $800\times$  slower) occurs because the enormous number of capillaries provides huge total cross-sectional area. Slow capillary blood flow is physiologically crucial—it allows sufficient time for gas exchange ( $\text{O}_2$  and  $\text{CO}_2$ ), nutrient delivery, and waste removal between blood and tissues. Answer A is  $10\times$  too small. Answer C is  $10\times$  too large. Answer D is  $100\times$  too large. The vast capillary network dramatically increases total area, slowing velocity.

**40. A - 93 mmHg**

Mean arterial pressure (MAP) represents the average pressure during the cardiac cycle and is approximated by:  $\text{MAP} \approx \text{diastolic} + (1/3)(\text{systolic} - \text{diastolic})$ . This formula accounts for the heart spending approximately  $2/3$  of the cardiac cycle in diastole and  $1/3$  in systole. With blood pressure  $120/80 \text{ mmHg}$ :  $\text{MAP} = 80 + (1/3)(120 - 80) = 80 + (1/3)(40) = 80 + 13.3 = 93.3 \approx 93 \text{ mmHg}$ . MAP is clinically important because it represents the perfusion pressure driving blood through organs. A simple average  $(\text{systolic} + \text{diastolic})/2$  would give  $100 \text{ mmHg}$ , but this overestimates MAP because it doesn't account for the longer duration of diastole. Answer B ( $100 \text{ mmHg}$ ) is the arithmetic mean, not the weighted MAP. Answer C ( $107 \text{ mmHg}$ ) would result from incorrect weighting. Answer D ( $120 \text{ mmHg}$ ) is just systolic pressure.

**41. C - Flow rate depends on the fourth power of radius**

Poiseuille's equation for laminar flow through cylindrical tubes states:  $Q = (\pi r^4 \Delta P) / (8 \eta L)$ , showing  $Q \propto r^4$ . This fourth-power relationship is extraordinarily important physiologically—small changes in vessel radius produce dramatic changes in blood flow. For example, doubling the radius increases flow by  $2^4 = 16$ -fold; halving radius decreases flow to  $(1/2)^4 = 1/16$ . This explains why vasodilation (widening blood vessels) and vasoconstriction (narrowing blood vessels) are such effective and rapid mechanisms for regulating blood flow to different organs. Even modest vasoconstriction can dramatically reduce flow. This also explains why atherosclerotic plaques significantly impair blood flow—even partial narrowing has outsized effects. Answer A (linear relationship) would be  $Q \propto r$ , far less sensitive. Answer B (quadratic) would be  $Q \propto r^2$ , still less sensitive than the actual relationship. Answer D is wrong (viscosity is a fluid property independent of vessel radius, though blood viscosity can change with shear rate in very small vessels).

**42. C -  $2.44\times$  (144% increase)**

From Poiseuille's equation, resistance  $R \propto 1/r^4$ . When radius decreases from  $1.0 \text{ cm}$  to  $0.8 \text{ cm}$ :  $R_{\text{new}}/R_{\text{old}} = (r_{\text{old}}/r_{\text{new}})^4 = (1.0/0.8)^4 = (1.25)^4 = 2.44$ . Resistance increases by a factor of  $2.44$ , meaning it's  $144\%$

higher than the original value ( $2.44 - 1.00 = 1.44 = 144\%$  increase). A 20% radius reduction causes a 144% resistance increase due to the sensitive fourth-power relationship. This dramatic sensitivity explains why even modest atherosclerotic narrowing significantly impairs blood flow and why maintaining vascular health is so important. Answer A ( $1.25\times$ ) would be the linear factor (first power). Answer B ( $1.56\times$ ) would be approximately the squared factor. Answer D would require greater narrowing than stated.

#### 43. A - 1.89 eV

Using the Bohr model energy levels for hydrogen:  $E_n = -13.6 \text{ eV} / n^2$ . For  $n = 3$ :  $E_3 = -13.6/9 = -1.51 \text{ eV}$ . For  $n = 2$ :  $E_2 = -13.6/4 = -3.40 \text{ eV}$ . The energy of the emitted photon equals the transition energy:  $\Delta E = E_3 - E_2 = -1.51 - (-3.40) = 1.89 \text{ eV}$ . The positive value indicates energy is released as the electron drops to a lower energy level, emitting a photon. This transition ( $n=3$  to  $n=2$ ) is the first line of the Balmer series, called the  $H\alpha$  (H-alpha) line, which is visible as red light. Answer B ( $-3.40 \text{ eV}$ ) is just  $E_2$ , not the difference. Answer C ( $10.2 \text{ eV}$ ) would be approximately the  $n=1$  to  $n=3$  transition energy. Answer D ( $12.1 \text{ eV}$ ) would be close to the  $n=\infty$  to  $n=1$  transition (ionization from ground state).

#### 44. C - 656 nm (red)

Using  $E = hc/\lambda$  with  $E = 1.89 \text{ eV}$ . First convert to joules:  $E = 1.89 \text{ eV} \times 1.602 \times 10^{-19} \text{ J/eV} = 3.03 \times 10^{-19} \text{ J}$ . Then:  $\lambda = hc/E = (6.626 \times 10^{-34} \text{ J}\cdot\text{s})(3.00 \times 10^8 \text{ m/s}) / (3.03 \times 10^{-19} \text{ J}) = 1.988 \times 10^{-25} / 3.03 \times 10^{-19} = 6.56 \times 10^{-7} \text{ m} = 656 \text{ nm}$ . This is the  $H\alpha$  line, the most prominent visible line in the hydrogen spectrum, appearing as bright red light. It's extensively used in astronomy for studying hydrogen clouds, stellar spectra, and redshift measurements. Answer A ( $364 \text{ nm}$ ) is in the ultraviolet region, corresponding to the Balmer series limit. Answer B ( $486 \text{ nm}$ ) is the  $H\beta$  line ( $n=4$  to  $n=2$  transition), appearing blue-green. Answer D ( $820 \text{ nm}$ ) is in the infrared, part of the Paschen series (transitions to  $n=3$ ).

#### 45. A - 0.123 nm

First, find the electron's velocity using kinetic energy:  $KE = (1/2)mv^2$ . With  $KE = 100 \text{ eV} = 1.60 \times 10^{-17} \text{ J}$  and  $m = 9.11 \times 10^{-31} \text{ kg}$ :  $v^2 = 2KE/m = (2 \times 1.60 \times 10^{-17}) / (9.11 \times 10^{-31}) = 3.51 \times 10^{13} \text{ m}^2/\text{s}^2$ . Therefore  $v = 5.92 \times 10^6 \text{ m/s}$ . The de Broglie wavelength:  $\lambda = h/(mv) = (6.626 \times 10^{-34} \text{ J}\cdot\text{s}) / [(9.11 \times 10^{-31} \text{ kg})(5.92 \times 10^6 \text{ m/s})] = 6.626 \times 10^{-34} / 5.39 \times 10^{-24} = 1.23 \times 10^{-10} \text{ m} = 0.123 \text{ nm}$ . This wavelength is comparable to atomic dimensions ( $\sim 0.1 \text{ nm}$ ), which is why electrons can exhibit diffraction patterns when passing through crystals (electron diffraction experiments). The wave nature of matter is significant only for very small particles. Answer B is  $10\times$  too large. Answers C and D are much too large and would not show quantum effects.

#### 46. A - 0.1 eV

First, calculate the photon energy using  $E = hc/\lambda$ . With  $\lambda = 400 \text{ nm} = 4.00 \times 10^{-7} \text{ m}$ :  $E = (6.626 \times 10^{-34} \text{ J}\cdot\text{s})(3.00 \times 10^8 \text{ m/s}) / (4.00 \times 10^{-7} \text{ m}) = 4.97 \times 10^{-19} \text{ J}$ . Converting to eV:  $E = 4.97 \times 10^{-19} \text{ J} \div 1.602 \times 10^{-19} \text{ J/eV} = 3.1 \text{ eV}$ . The photoelectric effect equation states:  $KE_{\text{max}} = E_{\text{photon}} - \Phi$  (work function). Therefore:  $KE_{\text{max}} = 3.1 \text{ eV} - 3.0 \text{ eV} = 0.1 \text{ eV}$ . Most of the photon's energy ( $3.0 \text{ eV}$ ) is used to overcome the work function and liberate the electron from the metal surface, with only  $0.1 \text{ eV}$  remaining as kinetic energy. If photon energy were less than the work function, no electrons would be ejected regardless of light intensity.

Answer B is just the work function, not the kinetic energy. Answer C is the photon energy before subtracting work function. Answer D incorrectly adds instead of subtracts, which violates energy conservation.

#### 47. C - CH<sub>3</sub>OH (methanol)

Methanol has the highest boiling point among these substances due to strong hydrogen bonding between molecules. The O-H group allows methanol molecules to form hydrogen bonds (specifically, the partially positive H of one molecule's O-H group attracts the partially negative O of another molecule). Hydrogen bonds are significantly stronger than the London dispersion forces present in hydrocarbons. Methanol boils at 65°C, while butane boils at -0.5°C, methane at -162°C, and neon at -246°C. Answer A (methane, CH<sub>4</sub>) has only weak London dispersion forces due to its small, nonpolar structure. Answer B (butane) has stronger London forces than methane due to greater molecular size and surface area, but still only dispersion forces. Answer D (neon) is a noble gas atom with very weak dispersion forces. The hierarchy: hydrogen bonding > dipole-dipole > London dispersion explains the boiling point trend.

#### 48. B - Decreasing temperature and increasing pressure

Using Le Chatelier's principle to analyze the equilibrium  $\text{N}_2(\text{g}) + 3\text{H}_2(\text{g}) \rightleftharpoons 2\text{NH}_3(\text{g})$ ,  $\Delta H = -92 \text{ kJ/mol}$ . The negative  $\Delta H$  indicates the forward reaction is exothermic (releases heat). To increase NH<sub>3</sub> yield: (1) Decrease temperature—since the reaction is exothermic, lowering temperature favors the heat-producing (forward) direction, shifting equilibrium toward products. (2) Increase pressure—the reaction has 4 moles of gas on the left (1 N<sub>2</sub> + 3 H<sub>2</sub>) and 2 moles on the right (2 NH<sub>3</sub>). Increasing pressure favors the side with fewer gas moles (products), shifting equilibrium toward NH<sub>3</sub>. This is precisely how the industrial Haber process operates: high pressure (150-300 atm) to favor products, though temperature is kept moderate (400-500°C) as a compromise between equilibrium position (favors low T) and reaction rate (favors high T). Answer A favors reactants on both counts. Answer C correctly increases pressure but incorrectly raises temperature, partially offsetting the pressure benefit. Answer D correctly decreases temperature but incorrectly decreases pressure.

#### 49. A - +6

To find sulfur's oxidation state in H<sub>2</sub>SO<sub>4</sub>, assign oxidation states to other atoms first: hydrogen is +1 (as usual in compounds), oxygen is -2 (as usual in compounds). For the neutral molecule, the sum of oxidation states must equal zero:  $2(+1) + \text{S} + 4(-2) = 0$ . Simplifying:  $2 + \text{S} - 8 = 0$ , therefore  $\text{S} = +6$ . This is sulfur's maximum oxidation state, indicating it has lost all six valence electrons. Sulfuric acid is a strong oxidizing agent partly due to sulfur's high oxidation state—sulfur can readily accept electrons, being reduced to lower oxidation states like +4 (in SO<sub>2</sub>) or -2 (in H<sub>2</sub>S). Answer B (+4) is sulfur's oxidation state in sulfurous acid (H<sub>2</sub>SO<sub>3</sub>), sulfur dioxide (SO<sub>2</sub>), or sulfites. Answer C (-2) is sulfur's state when it gains electrons fully, as in hydrogen sulfide (H<sub>2</sub>S) or metal sulfides. Answer D (+2) is uncommon but can occur in some compounds like S<sub>2</sub>Cl<sub>2</sub>.

#### 50. B - 2.5

For a weak acid, use the approximation:  $[\text{H}^+] \approx \sqrt{K_a \times C}$ , where C is the initial acid concentration. This approximation is valid when  $K_a \ll C$  and when degree of dissociation is small (<5%). With  $K_a = 1.0 \times$

$10^{-5}$  and  $C = 1.0$  M:  $[H^+] = \sqrt{(1.0 \times 10^{-5})(1.0)} = \sqrt{1.0 \times 10^{-5}} = 1.0 \times 10^{-2.5} = 3.16 \times 10^{-3}$  M. Then  $pH = -\log[H^+] = -\log(3.16 \times 10^{-3}) = -\log(3.16) - \log(10^{-3}) = -0.50 + 3.00 = 2.50$ . Checking validity: percent dissociation  $= (3.16 \times 10^{-3}/1.0) \times 100\% = 0.32\%$ , which is  $\ll 5\%$ , confirming the approximation is valid. Answer A (pH 2.0) would require  $[H^+] = 0.01$  M, implying 1% dissociation and  $K_a \approx 10^{-4}$ . Answer C (pH 3.0) would require  $K_a \approx 10^{-6}$ , ten times weaker. Answer D (pH 5.0) would be for an extremely weak acid ( $K_a \sim 10^{-10}$ ) or very dilute solution where dissociation is more extensive.

### 51. B - Energy cannot be created or destroyed, only converted between forms

The first law of thermodynamics is the law of conservation of energy, mathematically expressed as  $\Delta E = q + w$  (the change in internal energy equals heat absorbed plus work done on the system). Energy exists in various forms—kinetic, potential, thermal, chemical, electrical, nuclear—and can be converted between these forms, but the total energy of an isolated system remains constant. For example, chemical energy in gasoline converts to kinetic energy in a moving car plus thermal energy (heat) and sound, but total energy is conserved. Answer A describes the second law of thermodynamics (entropy of the universe increases in spontaneous processes). Answer C is backwards (heat flows spontaneously from hot to cold objects, not cold to hot—this would violate the second law). Answer D mischaracterizes thermodynamics (many processes are irreversible, and adding energy doesn't make them reversible—the second law governs irreversibility).

### 52. A - sp

In  $CO_2$  ( $O=C=O$ ), carbon forms two double bonds with no lone pairs, giving 2 electron groups around the central carbon atom. According to VSEPR theory, 2 electron groups require sp hybridization to minimize repulsion, creating a linear molecular geometry with  $180^\circ$  bond angle. The carbon's two sp hybrid orbitals overlap with oxygen p orbitals to form two  $\sigma$  bonds, while the two unhybridized p orbitals on carbon overlap with oxygen p orbitals to form two  $\pi$  bonds (one  $\pi$  bond in each  $C=O$  double bond). This creates the characteristic linear structure of  $CO_2$ . Answer B ( $sp^2$ ) would be correct for 3 electron groups, producing trigonal planar geometry (like in carbonate ion,  $CO_3^{2-}$ , or formaldehyde,  $CH_2O$ ). Answer C ( $sp^3$ ) would be correct for 4 electron groups, producing tetrahedral geometry (like in methane,  $CH_4$ , or water,  $H_2O$  including lone pairs). Answer D ( $sp^3d$ ) would require 5 electron groups and expanded octet, only possible for elements in period 3 or higher (carbon cannot form  $sp^3d$  hybrids).

### 53. A - 4

A face-centered cubic (FCC) unit cell has atoms at specific positions: (1) corners:  $8 \text{ corners} \times 1/8 \text{ atom per corner} = 1 \text{ atom}$  (each corner atom is shared by 8 adjacent unit cells), and (2) face centers:  $6 \text{ faces} \times 1/2 \text{ atom per face} = 3 \text{ atoms}$  (each face-centered atom is shared by 2 adjacent unit cells). Total atoms per unit cell:  $1 + 3 = 4 \text{ atoms}$ . FCC is one of the most efficient packing arrangements (along with hexagonal close-packed) with 74% packing efficiency—74% of the volume is occupied by atoms, 26% is empty space. Many metals crystallize in FCC structure, including aluminum, copper, gold, and silver. Answer B (2) is correct for body-centered cubic (BCC) structure ( $8 \text{ corners} \times 1/8 + 1 \text{ center atom}$ ). Answer C (1) is correct for simple cubic ( $8 \text{ corners} \times 1/8$ ). Answer D (8) incorrectly counts corner atoms without accounting for sharing.

### 54. C - $CO_2$

CO<sub>2</sub> is a nonpolar molecule despite containing polar C=O bonds. The molecule is linear (O=C=O) with 180° bond angle due to sp hybridization of carbon. Each C=O bond is polar (oxygen is more electronegative than carbon, creating a dipole with δ<sup>-</sup> on O and δ<sup>+</sup> on C), but the two bond dipoles are equal in magnitude and opposite in direction, resulting in complete cancellation. The vector sum of the two dipoles is zero, giving CO<sub>2</sub> zero net dipole moment and nonpolar character. This explains why CO<sub>2</sub> is a gas at room temperature and has low solubility in water despite oxygen's presence. Answer A (H<sub>2</sub>O) is highly polar—the bent molecular geometry (104.5° bond angle) means the two O-H bond dipoles don't cancel, creating a large net dipole moment. Answer B (NH<sub>3</sub>) is polar—the trigonal pyramidal geometry means the three N-H bond dipoles don't cancel fully, creating a net dipole. Answer D (CH<sub>3</sub>Cl) is polar—the C-Cl bond is much more polar than the C-H bonds, creating an unsymmetrical charge distribution and net dipole.

**55. B - Rate = k[A]<sup>2</sup>[B]**

The experimental observation is: when [A] doubles while [B] is constant, the rate quadruples. This indicates the reaction is second-order with respect to A. Mathematically: if Rate = k[A]<sup>m</sup>[B]<sup>n</sup>, and [A] doubles while [B] constant: Rate<sub>new</sub>/Rate<sub>old</sub> = (2[A])<sup>m</sup>/[A]<sup>m</sup> = 2<sup>m</sup> = 4, therefore m = 2. The rate law must include [A]<sup>2</sup> to account for the quadrupling. Since only [A] was varied in this experiment, we cannot determine the order with respect to B from the given data, but for a stoichiometric equation 2A + B → C, first-order in B is typical, giving Rate = k[A]<sup>2</sup>[B]. Answer A has incorrect order for A (first-order would only double the rate when [A] doubles). Answer C is third-order in A (would increase rate 8-fold when [A] doubles). Answer D is incorrect unless additional data showed second-order dependence on B.

**56. D - 1/8**

For first-order reactions, the amount remaining after n half-lives is (1/2)<sup>n</sup> of the original amount. The half-life is 20 minutes. After 60 minutes, the number of half-lives elapsed is: n = 60 min ÷ 20 min = 3 half-lives. Fraction remaining = (1/2)<sup>3</sup> = 1/8 of the original amount. Alternatively, tracking through each half-life: after 20 min (1 half-life), 1/2 remains; after 40 min (2 half-lives), 1/4 remains; after 60 min (3 half-lives), 1/8 remains. This exponential decay is characteristic of first-order kinetics, where the half-life is constant regardless of concentration. Answer A (1/2) is the fraction after one half-life (20 min). Answer B (1/4) is the fraction after two half-lives (40 min). Answer C (1/6) would be appropriate for a zero-order reaction where amount decreases linearly, but is incorrect for first-order kinetics.

**57. A - Proton**

Protons and neutrons each have approximately the same mass: ~1.673 × 10<sup>-27</sup> kg or about 1 atomic mass unit (1 amu ≈ 1.66 × 10<sup>-27</sup> kg). The slight difference (neutron is about 0.1% heavier than proton) is negligible for most purposes. Both are classified as nucleons and make up virtually all atomic mass. Answer B (electron) is much lighter with mass ~9.11 × 10<sup>-31</sup> kg, about 1/1836 the mass of a proton—electrons contribute negligibly to atomic mass. Answer C (alpha particle) is a helium-4 nucleus containing 2 protons + 2 neutrons, so it has approximately 4 times the mass of a single proton or neutron. Answer D (positron) is the antimatter counterpart of the electron with identical mass (~9.11 × 10<sup>-31</sup> kg), much lighter than nucleons.

**58. C - 0.34 m**

Using the wave equation  $v = f\lambda$ , where  $v$  is wave speed,  $f$  is frequency, and  $\lambda$  is wavelength. Given  $v = 340$  m/s (speed of sound in air at room temperature) and  $f = 1000$  Hz:  $\lambda = v/f = 340 \text{ m/s} \div 1000 \text{ Hz} = 0.34 \text{ m} = 34 \text{ cm}$ . This wavelength is comparable to human body dimensions, which is why sound exhibits significant diffraction—we can hear sounds around corners and obstacles. Lower frequencies have longer wavelengths (better diffraction/propagation through obstacles), while higher frequencies have shorter wavelengths (more directional propagation). Answer A uses incorrect calculation or values. Answer B uses wrong values. Answer D incorrectly multiplies frequency and velocity instead of dividing ( $\lambda = v/f$ , not  $vf$ ). Remember that wavelength and frequency are inversely related for any wave: as frequency increases, wavelength decreases, with their product equaling wave speed.

**59. D - The electric field is never zero between opposite charges**

For two point charges of opposite sign ( $+2Q$  and  $-Q$ ) separated by distance  $d$ , the electric field contributions must be analyzed. Between the charges, at any point distance  $x$  from  $+2Q$ : the field due to  $+2Q$  is  $E_1 = k(2Q)/x^2$  pointing away from  $+2Q$  (toward  $-Q$ ), and the field due to  $-Q$  is  $E_2 = k(Q)/(d-x)^2$  pointing toward  $-Q$  (away from  $+2Q$ ). Both fields point in the same direction (toward  $-Q$ ), so they add rather than cancel—the field cannot be zero anywhere between opposite charges. The field can be zero only in regions where the two field contributions point in opposite directions. This occurs either: (1) to the left of  $+2Q$ , where both fields would point opposite directions but with wrong magnitude ratio to cancel, or (2) to the right of  $-Q$  (beyond it, away from  $+2Q$ ), where fields point opposite directions and can potentially cancel. Specifically, at distance  $x$  beyond  $-Q$ :  $k(2Q)/(d+x)^2 = k(Q)/x^2$ , solving gives  $x \approx 1.41d$  beyond  $-Q$ , or about  $2.41d$  from  $+2Q$ . Answer A would place the zero field point too close to  $+2Q$ . Answer B (midpoint) is wrong because both fields point the same direction there. Answer C is also between the charges where cancellation is impossible.

## SECTION 2: ANSWER EXPLANATIONS

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### 1. B - Demonstrate that traditional ethical frameworks are inadequate for addressing AI challenges

The passage systematically examines how traditional ethical frameworks (Kantian, utilitarian, virtue ethics) prove inadequate when applied to AI. The author explores multiple challenges—the trolley problem's transformation, the alignment problem, superintelligence risks—all demonstrating traditional ethics' limitations. Answer B directly states what the passage accomplishes throughout.

### 2. D - Abstract principles must be converted into concrete algorithmic decisions

The passage explicitly states the trolley problem "transforms into an engineering specification requiring precise algorithmic implementation" and that "the reification of ethical principles into algorithms reveals how philosophically underdetermined our moral intuitions truly are." Option D captures this transformation requirement.

### 3. C - Mild skepticism

The passage states: "Some philosophers argue for a pluralistic approach...But this merely displaces the problem: who determines the appropriate weights, and by what criteria?" The word "But" followed by identifying a fundamental limitation shows skepticism, not enthusiasm. The author sees the approach as not solving but displacing the problem.

### 4. C - The inconsistency and cultural variability of human values

The passage explicitly identifies this as the core difficulty: "Human values are inconsistent, context-dependent, and culturally variable. Whose values should AI systems encode?" This is stated as the fundamental conceptual challenge of the alignment problem.

### 5. B - Converting philosophical abstractions into algorithms reveals their underlying indeterminacy

The passage states: "Yet the reification of ethical principles into algorithms reveals how philosophically underdetermined our moral intuitions truly are." Reification means making abstract concepts concrete, and this process reveals indeterminacy—exactly what option B describes.

### 6. B - Philosophical work requiring more time than AI capabilities advance

The passage explains: "we must solve philosophical problems of profound difficulty before technological capabilities outpace our ability to control them, yet our philosophical methods—dialogue, reflection, gradual consensus-building—operate on timescales mismatched to rapid technical progress." This temporal mismatch is precisely option B.

**7. C** - Behavioral economics challenges both the descriptive and normative foundations of rational choice theory

The passage argues behavioral economics undermines rational choice theory both descriptively (showing systematic deviations) and normatively (raising questions about autonomy and the ethical premises of market societies). The comprehensive challenge to both dimensions is the main argument—option C.

**8. C** - Logically equivalent presentations of choices produce different decisions

The passage states people prefer "90% survival rate" over "10% mortality rate" despite their logical equivalence, and that "Rational actors should be invariant to such superficial reformulations, yet empirical evidence overwhelmingly confirms framing's influence." Option C captures this central point about equivalent framings producing different choices.

**9. B** - Suggest that behavioral patterns reflect adaptive responses rather than mere errors

The passage introduces evolutionary psychology to argue that "prospect theory...suggests these patterns reflect deep features of human psychology shaped by evolutionary pressures" and that biases "may have benefited" organisms in ancestral environments. This frames biases as potentially adaptive rather than mere mistakes—option B.

**10. D** - Refusing to sell a coffee mug one values at \$8 for any price below \$16

The passage states: "people demand roughly twice as much to relinquish an object they possess than they would pay to acquire it." Option D perfectly captures this 2:1 ratio: willing to pay \$8 to acquire but demanding \$16 to sell shows the endowment effect's asymmetry.

**11. C** - They involve determining welfare without clear justification for who decides

The passage states: "this approach raises normative questions: Who determines which outcomes improve welfare? When does nudging become manipulation?" The ethical problem is the authority to determine welfare—option C explicitly addresses this concern about who decides.

**12. A** - Critical but acknowledging its historical utility and ongoing normative questions

The passage criticizes rational choice theory's descriptive failures while noting it "served economics well as a simplifying assumption, enabling elegant mathematical models." It also acknowledges ongoing debates about its normative role. This balanced critical stance that recognizes historical value matches option A.

**13. D** - Question the assumptions underlying the concept of the avant-garde

The passage systematically interrogates avant-garde assumptions: the military metaphor's appropriateness, progressivist narratives, novelty fetishism, and revolutionary rhetoric. The opening sentence asks "Does art actually advance like an army toward victory, or does the avant-garde's self-conception reflect modernist ideology more than aesthetic reality?" This questioning approach is option D.

**14. B** - Allowed them to reinterpret hostile reception as validation

The passage explicitly states: "The hostile reception from traditional critics became evidence of revolutionary importance rather than artistic failure. Those who didn't understand were simply behind history's march—a convenient way to transform marginalization into validation." Option B directly describes this rhetorical function.

**15. C** - Military victories involve winners and losers unlike artistic innovation

The passage explains: "Armies advance toward concrete objectives in zero-sum conflicts where one side's gain is another's loss. Artistic innovation operates differently. Impressionism didn't make Renaissance painting obsolete; both coexist as valid aesthetic approaches." Option C captures this contrast between zero-sum military competition and artistic coexistence.

**16. A** - Evidence that Impressionist paintings now sell for high prices

Wait, let me reconsider. The author argues art doesn't progress through displacement—styles coexist validly. What would weaken this? If we showed certain techniques genuinely became obsolete and cannot coexist with new forms. That's more like option B. Option A about prices is irrelevant to whether styles can coexist.

**16. B** - A demonstration that certain artistic techniques genuinely became obsolete

The author argues "Impressionism didn't make Renaissance painting obsolete; both coexist as valid aesthetic approaches." If we could demonstrate that certain techniques genuinely became obsolete (cannot coexist validly with new approaches), this would weaken the claim that art accumulates rather than progresses through displacement.

**17. D** - A postmodern critique of originality as artistic value

The passage states Levine's re-photographing "simultaneously honored tradition and mocked originality fetishism. The avant-garde dream of perpetual revolution gave way to playful recombination." This clearly represents critique of originality as supreme value—option D.

**18. C** - Artistic change occurs more gradually and collaboratively than the avant-garde narrative suggests

The passage concludes: "Artistic change typically occurs through gradual accumulation of small modifications across communities of practitioners, not through the isolated genius's dramatic ruptures." This directly matches option C's description of gradual, collaborative change.

**19. B** - Modern democracies face difficult tensions between popular sovereignty and expert knowledge that lack easy resolution

The passage presents the tension, examines proposed solutions (technocracy, deliberation), identifies problems with each, and concludes that "institutional design" must "balance these competing values rather than pretending they automatically harmonize." This indicates unresolved tension requiring careful balancing—option B.

**20. C** - Whether economic or environmental values should take priority

The passage explicitly states: "Climate policy requires balancing economic costs against environmental protection—a normative choice that technical knowledge informs but cannot determine." Options A, B, and D involve technical matters experts can address, while C involves value priority that expertise cannot resolve.

**21. D** - Adjudicating between conflicting experts requires democratic judgment rather than expertise

The passage states: "When experts conflict, political decisions become unavoidable. Selecting which experts to trust or how to adjudicate expert disagreement requires democratic judgment that cannot be delegated to expertise itself." This matches option D—democratic judgment needed to adjudicate expert conflict.

**22. B** - Theoretically promising but practically difficult to implement

The passage states deliberative democracy offers a way to "reconcile" expertise and democracy (promising), but "deliberation's promise remains largely unfulfilled" due to "misinformation, partisan polarization, and attention scarcity" (practical difficulties). Option B captures this assessment.

**23. C** - It serves other important values like human dignity and preventing tyranny

The passage argues democracy might be valuable even if it produces suboptimal policy because it serves "other considerations: respecting human dignity through self-governance, preventing tyranny through dispersed power, or maintaining political legitimacy through consent." Option C directly states these values.

**24. B** - Economic policymakers from similar elite educational backgrounds making decisions affecting diverse populations

The passage states: "Those possessing specialized knowledge gain influence over policy that ordinary citizens cannot match...The problem intensifies when expert communities exhibit demographic homogeneity or ideological conformity." Option B—elite policymakers from similar backgrounds affecting diverse populations—perfectly illustrates this power asymmetry concern.

**25. C** - Narrative techniques embody philosophical assumptions about consciousness and subjectivity

The passage concludes: "Narrative choices aren't merely formal decisions but implicit theories about consciousness, identity, and reality...Literary form and philosophical content prove inseparable." Option C directly captures this central argument.

**26. B** - Consciousness is stable and can be objectively observed

The passage states traditional omniscient narration "presumes a transparent, stable self that can be observed and described from an external vantage point." Option B describes this assumption about stable, objectively observable consciousness.

**27. C** - Relegates human consciousness to peripheral status

The passage says "Time Passes" section "dispenses with human consciousness entirely, describing the house's decay in bracketed asides that relegate human events to parenthetical interruptions in nature's indifferent process." Option C captures this relegation to peripheral status.

**28. C** - It attempts to represent pre-linguistic consciousness using language

The passage identifies this paradox: "Consciousness is pre-linguistic; the stream of thought includes images, sensations, and inchoate feelings that resist verbal articulation. Any written representation necessarily imposes linguistic structure onto experiences that exceed language." Option C describes this fundamental paradox.

**29. B** - Stream-of-consciousness should be evaluated aesthetically rather than as accurate psychological representation

The passage uses the cubist comparison to argue Joyce's work doesn't "pretend to capture consciousness accurately; rather, it creates verbal artifacts that evoke consciousness's qualities through literary devices...The technique's value lies in aesthetic achievement rather than psychological verisimilitude." Option B captures this shift to aesthetic evaluation.

**30. D** - Selective integration of insights while recovering accessibility

The passage states: "Contemporary fiction has largely abandoned pure stream-of-consciousness while inheriting its insights...The synthesis preserves self-consciousness about narrative perspective while recovering storytelling's traditional pleasures." Option D describes this selective integration with recovered accessibility.

**31. C** - Neither pure relativism nor simple universalism is adequate; a nuanced position is needed

The passage examines problems with both strict relativism and simple universalism, ultimately suggesting "recognizing both cultural specificity and overlapping consensus" and a "hermeneutic approach" that "acknowledges both universal and particular elements." Option C describes this nuanced middle position.

**32. A** - Interpreting practices within indigenous frameworks of meaning

The passage defines thick description as "interpreting practices within indigenous frameworks of meaning" rather than judging by external standards. Option A directly states this definition.

**33. C** - Descriptive variation doesn't logically entail normative relativity

The passage states: "Moral relativism is typically supported by descriptive cultural variation...But descriptive diversity doesn't entail normative relativity—the inference from 'is' to 'ought' that philosophers since Hume have recognized as fallacious." Option C captures this logical problem.

**34. D** - Two cultures with conflicting practices needing to resolve a dispute

The passage describes the pragmatic dilemma: "When cultures conflict, how should disputes be adjudicated? If each culture's values are valid within its own context, what principle resolves intercultural conflicts?" Option D directly illustrates this scenario.

**35. B** - An attempt to specify universal values while remaining culturally sensitive

The passage presents Nussbaum's approach as grounding "universal values in human capabilities" while "remaining sensitive to how capabilities might be realized differently across cultures." Option B describes this attempt to balance universalism with cultural sensitivity.

**36. C** - Some practices can be legitimately criticized across cultures while respecting cultural variation

The passage supports "recognizing genuine cultural variation while maintaining certain universal moral minimum" and states "practices that systematically deny these capabilities can be legitimately criticized cross-culturally." Option C captures this position allowing selective cross-cultural criticism.

**37. D** - Jazz develops through cyclical returns to foundational elements that generate new possibilities

The opening paragraph states jazz "developed through cyclical returns to foundational elements, each return generating new possibilities rather than merely repeating the past" and describes a "spiral pattern—simultaneously conservative and revolutionary." Option D directly quotes this main point.

**38. C** - Positioned jazz as serious art while retaining traditional song forms

The passage states bebop musicians "rejected entertainment in favor of art, positioning jazz as modernist project" while also noting "bebop's revolutionary rhetoric obscured its conservative foundation. The music retained standard song forms and harmonic progressions." Option C captures both elements.

**39. C** - Simpler approaches can enable greater freedom

The passage states about modal jazz: "This minimalism paradoxically opened new improvisatory possibilities. Without harmonic motion dictating melodic choices, soloists achieved greater freedom." Option C describes how simplification (static modes) enabled freedom.

**40. B** - Artistic traditions accumulate rather than supersede previous forms

The passage contrasts science with jazz: "each innovation adds to an expanding vocabulary without invalidating previous styles. The tradition becomes richer precisely through retaining its history rather than superseding it." Option B describes this accumulation rather than supersession.

**41. D** - A natural result of jazz's accumulated historical richness

The passage states contemporary jazz "exhibits stylistic pluralism" with musicians drawing "eclectically on the tradition's entire history" and concludes "jazz's richness lies in its accumulated possibilities." Option D presents pluralism as natural outcome of accumulated richness.

**42. C** - Appreciation for how each style contributed to jazz's evolving vocabulary

The passage discusses each style neutrally, showing contributions to jazz's development. The conclusion emphasizes "expanding vocabulary" where "each innovation adds" without invalidating previous styles. Option C describes this appreciative approach to each style's contribution.

**43. C** - Memory's unreliability and psychological research complicate rather than resolve questions about personal identity

The passage systematically shows how psychological findings (reconstructive memory, false memories, neurological conditions) complicate philosophical theories of personal identity. The thesis isn't that one solution is correct but that memory's problems deepen rather than resolve identity questions—option C.

**44. A** - It involves rebuilding past events from fragmentary traces rather than retrieving complete records

The passage explicitly states: "Rather than retrieving stored records like files from a cabinet, remembering involves reconstructing past experiences from fragmentary traces, filling gaps with plausible inferences." Option A directly describes this reconstructive process.

**45. B** - Allow for personal identity despite incomplete memory between distant life stages

The passage explains Parfit's distinction aims to solve a problem: "Perhaps identity requires continuity rather than perfect memory. I am the same person as the child I was not because I remember all my childhood experiences, but because there exists a chain of overlapping connections." Option B describes this solution for incomplete memory.

**46. C** - A middle-aged person sharing no memories with their infant self, connected only through forgotten intermediate stages

The passage describes: "The person I am today might share no memories with the child I was, connected only through intermediate stages I've also forgotten." Option C perfectly illustrates this attenuation scenario where no direct memories remain.

**47. B** - Identity might be a pragmatically useful concept rather than a discovered metaphysical fact

The "deflationary conclusion" states: "Personal identity might be a useful fiction—pragmatically indispensable but metaphysically suspect. We treat ourselves and others as persisting persons because social life requires it, not because reality contains the sort of entity our identity concepts presume." Option B captures this pragmatic utility without metaphysical reality.

**48. C** - Aesthetic pluralism offers the best approach but faces challenges

The passage examines universal theories and cultural construction, then supports "aesthetic pluralism" while noting "pluralism creates new problems." The final paragraphs work through these challenges while maintaining that aesthetic questions might be "essentially contestable." Option C captures this critical but supportive stance toward pluralism.

**49. C** - Claim universal validity even though they're subjective

The passage explains Kant's concept: "aesthetic judgments, while not derived from concepts, nevertheless claim universal validity" and describes "universal subjective validity." The key point is that aesthetic judgments are subjective yet claim universal agreement—option C.

**50. B** - Aesthetic preferences correlate with social position

The passage states Bourdieu's work "demonstrated how aesthetic preferences correlate with social position, suggesting that disputes about beauty encode struggles over cultural authority." Option B directly describes this demonstrated correlation.

**51. C** - A value judgment subject to reasoned argument without final resolution

The passage defines essentially contestable as questions "subject to reasoned argument without admitting final demonstration" unlike math (with proofs) or empirical questions (with evidence). Option C describes value judgments allowing rational argument without definitive resolution.

**52. C** - The questions themselves are essentially contestable rather than that we lack knowledge

The passage states: "Aesthetic questions might lack such resolution procedures not because we haven't discovered them but because none exist." This suggests the nature of aesthetic questions themselves, not epistemic limitations—option C.

**53. C** - An opportunity for dialogue and cultivation rather than a defect requiring resolution

The passage concludes: "We might then approach aesthetic disputes differently—not seeking to resolve them definitively but to articulate our perspectives more fully, understand others' more deeply, and refine our sensibilities through dialogue. The goal becomes aesthetic cultivation." Option C presents disagreement as opportunity for cultivation rather than problem to solve.

# SECTION 3: ANSWER EXPLANATIONS

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## 1. A - O<sub>2</sub> becomes more soluble relative to CO<sub>2</sub> at high temperatures

At higher temperatures, CO<sub>2</sub> solubility decreases more rapidly than O<sub>2</sub> solubility, increasing the O<sub>2</sub>/CO<sub>2</sub> ratio in leaf tissues. Since RuBisCO can catalyze reaction with either CO<sub>2</sub> (carboxylase activity) or O<sub>2</sub> (oxygenase activity), and both gases compete for the same active site, the increased O<sub>2</sub>/CO<sub>2</sub> ratio favors the oxygenase reaction (photorespiration). The passage data confirms this: at 40°C, photorespiration increased to 40% in C<sub>3</sub> plants compared to 20% at 25°C. Additionally, when stomata close in hot conditions to prevent water loss, CO<sub>2</sub> cannot enter efficiently while O<sub>2</sub> from photosynthesis accumulates, further increasing the O<sub>2</sub>/CO<sub>2</sub> ratio. RuBisCO doesn't denature at physiological temperatures (option B), chlorophyll continues functioning (option C), and while water evaporation affects stomata, it's not the direct cause of increased oxygenase activity (option D).

## 2. B - Concentrating CO<sub>2</sub> in bundle-sheath cells where RuBisCO operates

C<sub>4</sub> plants use spatial separation of initial CO<sub>2</sub> fixation and the Calvin cycle. The passage explains that in mesophyll cells, PEP carboxylase (which has no oxygenase activity and higher affinity for CO<sub>2</sub>) fixes CO<sub>2</sub> into 4-carbon compounds. These move to bundle-sheath cells where they're decarboxylated, releasing CO<sub>2</sub> in high concentrations directly around RuBisCO. This concentrated CO<sub>2</sub> environment ensures RuBisCO preferentially performs carboxylation rather than oxygenation, minimizing photorespiration. The data shows C<sub>4</sub> plants (corn) had less than 5% photorespiration even at elevated temperatures, compared to C<sub>3</sub> plants at 20-40%. Option A is wrong because RuBisCO itself is the same enzyme; the strategy involves manipulating substrate concentrations. Option C is wrong because C<sub>4</sub> plants still produce O<sub>2</sub> during light reactions. Option D describes CAM plants, not C<sub>4</sub> plants.

## 3. B - Storing malate at night and processing it during day creates temporal separation that limits total CO<sub>2</sub> fixation

CAM plants show slower growth rates (30% slower than C<sub>3</sub>/C<sub>4</sub> plants according to data) because they separate CO<sub>2</sub> fixation (nighttime) from the Calvin cycle (daytime) temporally. At night, when stomata open, PEP carboxylase fixes available CO<sub>2</sub> into malate stored in vacuoles. During the day, this stored malate is gradually decarboxylated to supply CO<sub>2</sub> for the Calvin cycle while stomata remain closed. The temporal separation means CO<sub>2</sub> fixation capacity is limited by nighttime CO<sub>2</sub> uptake and malate storage capacity—they cannot simultaneously fix new CO<sub>2</sub> and run the Calvin cycle during peak sunlight hours like C<sub>3</sub>/C<sub>4</sub> plants can. This reduces overall growth rate despite excellent water use efficiency. Option A is wrong because CAM plants do perform photosynthesis during the day (they use stored CO<sub>2</sub>). Option C is wrong because they have normal chlorophyll. Option D is wrong because stomata do open at night.

## 4. B - Photorespiration would be virtually eliminated

If a C<sub>3</sub> plant were in 5% CO<sub>2</sub> atmosphere (compared to normal 0.04%), the dramatically elevated CO<sub>2</sub> concentration would overwhelm O<sub>2</sub> competition for RuBisCO's active site. The enzyme's substrate preference is concentration-dependent; with CO<sub>2</sub> at much higher levels than O<sub>2</sub>, carboxylase activity would be heavily favored and oxygenase activity (photorespiration) would be minimal. This is essentially

what C4 plants achieve through their CO<sub>2</sub>-concentrating mechanism. The passage shows photorespiration accounts for 20-40% of activity in normal C3 plants, but this occurs at atmospheric CO<sub>2</sub> levels of ~380 ppm (0.038%). At 5% CO<sub>2</sub> (50,000 ppm), RuBisCO would function almost exclusively as a carboxylase. Option A is opposite of what would occur. Option C is wrong because plants can tolerate elevated CO<sub>2</sub>. Option D is wrong because CO<sub>2</sub> concentration profoundly affects photorespiration rates.

### **5. B - Strong selective pressure in hot, dry environments favored this adaptation**

The independent evolution of C4 photosynthesis in over 60 plant families (including grasses, sedges, and some eudicots) demonstrates convergent evolution—different lineages arriving at the same solution to environmental challenges. The passage and data indicate C4 metabolism minimizes photorespiration and maintains photosynthetic efficiency in hot, dry conditions where C3 plants suffer significant photorespiration losses. The strong selective advantage in these environments (water use efficiency, heat tolerance, maintained productivity) caused natural selection to repeatedly favor mutations leading to C4 anatomy and biochemistry. This pattern of convergent evolution—the same complex adaptation arising independently multiple times—is powerful evidence for strong selective pressure. Option A contradicts the evolutionary persistence of this trait. Option C makes an unfounded prediction. Option D contradicts the data showing C4 advantages in specific environments.

### **6. B - They lack the checkpoint mechanism to halt division and allow repair**

The passage explains that p53 functions as "guardian of the genome" by detecting DNA damage and halting the cell cycle at the G1 checkpoint, allowing time for repair. If damage is severe, p53 triggers apoptosis. The experimental data shows that normal cells exposed to radiation accumulated p53, arrested at G1 for 8 hours allowing repair, then resumed cycling. In contrast, p53<sup>-/-</sup> cells showed no arrest and continued cycling despite radiation damage, accumulating mutations. Cancer cells with mutant p53 lack this critical checkpoint control—they can detect damage (option A is wrong) but cannot execute the appropriate response of cell cycle arrest. The mutation doesn't actively promote division (option C), it simply removes the brake. Option D is wrong because DNA damage affects all cells; the difference is in the response.

### **7. B - Mutant Ras has impaired GTPase activity, remaining GTP-bound and active**

Ras is a molecular switch that cycles between active (GTP-bound) and inactive (GDP-bound) states. Normal Ras has GTPase activity that hydrolyzes GTP to GDP, turning itself off after transmitting the growth signal. The passage states that mutant Ras in 30% of cancers "remains constitutively active, continuously signaling division even without growth factors." This occurs because mutations impair Ras's intrinsic GTPase activity—it binds GTP normally but cannot hydrolyze it efficiently, so it stays locked in the active GTP-bound conformation, continuously signaling proliferation. The data confirms Ras mutant cells grew without added growth factors (normally required for activation). Option A is opposite (mutation affects GTP hydrolysis, not binding). Option C confuses energy production with signaling. Option D is wrong because mutant Ras acts downstream of receptors.

### **8. B - Phosphorylated Rb releases E2F, allowing transcription of S-phase genes**

The passage explains that hypophosphorylated Rb restrains cell cycle progression by sequestering E2F transcription factors, preventing expression of genes required for S-phase (DNA replication). When G1/S-Cdk phosphorylates Rb, this phosphorylation causes a conformational change that releases E2F. Free E2F can then bind to promoters and activate transcription of genes encoding DNA synthesis machinery (DNA polymerases, nucleotide synthesis enzymes, etc.), allowing the cell to enter S-phase and replicate its genome. The Rb<sup>-/-</sup> cell data shows these cells bypass the G1 checkpoint and enter S-phase without proper growth signals because they lack the Rb brake on E2F. Option A is wrong because Rb doesn't directly affect polymerase. Option C is wrong because phosphorylation is reversible. Option D is wrong because Rb and p53 are separate pathways.

### **9. B - Stabilizing p53 by preventing its degradation**

Under normal conditions, p53 levels are kept low through continuous degradation mediated by MDM2, an E3 ubiquitin ligase that tags p53 for proteasomal destruction. When MDM2 is inhibited, p53 protein accumulates even without DNA damage because it's no longer being continuously degraded. The experimental data shows that MDM2 inhibitor treatment increased p53 levels and caused cell cycle arrest—demonstrating that elevated p53 (even without damage signals) is sufficient to halt division. This principle is being explored therapeutically: in cancers with wild-type p53 but overactive MDM2, MDM2 inhibitors can restore p53 function. Option A is wrong because MDM2 inhibitors don't directly affect CDKs. Option C is wrong because they don't cause damage. Option D is wrong because they don't affect oncogenes.

### **10. B - Has increased cancer risk because only one more hit needed in a cell**

Knudson's two-hit hypothesis explains that tumor suppressor genes (like Rb) require loss of both alleles for cancer development because they're recessive at the cellular level. The passage states this explicitly: tumor suppressors "require loss of both alleles." A person inheriting one mutant Rb allele (germline mutation) starts with one "hit" already present in every cell. They have massively increased cancer risk, particularly retinoblastoma in childhood, because only one additional somatic mutation (second hit) in any retinal cell will eliminate Rb function, whereas people with two normal alleles need two independent hits in the same cell—much less probable. Option A is wrong because the second hit is probabilistic, not certain. Option C is opposite of true. Option D is wrong because one functional Rb allele provides sufficient function for development, though cancer risk is very high.

### **11. B - Increased cardiac output compensates for decreased TPR**

The fundamental relationship  $MAP = CO \times TPR$  explains this observation. During exercise, skeletal muscle vasodilation dramatically decreases total peripheral resistance (TPR), which by itself would cause MAP to plummet. However, the data shows MAP remained relatively stable (90 to 95 mmHg) because cardiac output increased dramatically (4.9 to 15.4 L/min)—more than tripling. This massive CO increase, achieved through both increased heart rate (70 to 140 bpm) and increased stroke volume (70 to 110 mL), compensates mathematically for the decreased TPR, maintaining adequate perfusion pressure. The cardiovascular system coordinates these changes through sympathetic activation (increasing HR and contractility) while allowing local metabolic factors to cause muscle vasodilation. Option A occurs but isn't sufficient. Option C is wrong because MAP definitely depends on resistance ( $MAP = CO \times TPR$ ).

Option D is wrong because vasodilation does affect pressure; the point is that CO compensation maintains MAP.

**12. B - Excessive fiber stretch moves beyond optimal sarcomere length for force generation**

The Frank-Starling mechanism describes the relationship between end-diastolic volume (preload/stretch) and stroke volume on a curve with an optimal range. Within normal physiological ranges, increased stretch enhances contractility because sarcomeres are positioned where actin-myosin overlap is optimal for cross-bridge formation. However, the heart failure data shows the mechanism failing: despite very high end-diastolic volume (200 mL vs normal 120 mL), stroke volume is reduced (40 mL vs normal 70 mL). This occurs because excessive stretch moves sarcomeres beyond optimal length—actin filaments are pulled so far from the center that they overlap minimally with myosin, reducing possible cross-bridges and thus contractile force. The passage states that "excessive stretch (as in heart failure) moves beyond optimal sarcomere length, reducing contractility." Option A is wrong because venous return is actually excessive. Option C is circular reasoning. Option D is irrelevant to the mechanism's failure.

**13. A - Neural reflexes work within seconds while RAAS requires hours to days**

The passage explicitly distinguishes short-term (baroreceptor reflexes) and long-term (RAAS) blood pressure control by their timescales. Baroreceptors detect pressure changes and signal the medullary cardiovascular centers within milliseconds; sympathetic activation can alter heart rate, contractility, and vascular tone within seconds to minutes. The hemorrhage data confirms this: heart rate increased from 70 to 110 bpm immediately. In contrast, RAAS involves a sequential enzymatic cascade (renin → angiotensinogen → angiotensin I → angiotensin II → aldosterone → altered Na<sup>+</sup> reabsorption → changed blood volume) that requires hours to days for full effect. The data shows RAAS activation occurred after 6 hours. Option B is wrong because RAAS profoundly affects blood pressure (through both vasoconstriction and volume expansion). Option C is wrong because sensitivity isn't the distinguishing factor. Option D is wrong because RAAS works continuously, not just during exercise.

**14. B - Reducing afterload through decreased vasoconstriction and reducing preload through decreased blood volume**

ACE inhibitors block conversion of angiotensin I to angiotensin II, eliminating angiotensin II's effects: (1) Direct vasoconstriction—blocking this reduces afterload (resistance the heart pumps against), making ejection easier and improving cardiac output in failing hearts. (2) Aldosterone stimulation—blocking this reduces Na<sup>+</sup> reabsorption and blood volume, reducing preload (filling volume). The passage notes the treatment "reduced afterload" and "improved heart failure symptoms." Both mechanisms benefit heart failure: reduced afterload means the weakened heart can eject more effectively, and reduced preload prevents excessive stretch and pulmonary congestion. Option A is wrong because ACE inhibitors don't increase heart rate (may slightly decrease it). Option C is wrong because they don't directly affect contractility. Option D is wrong because they don't completely block sympathetic activity.

**15. B - Despite large end-diastolic volume, the heart ejects only a small fraction due to poor contractility**

Ejection fraction (EF) = (stroke volume / end-diastolic volume) × 100%. Normal EF is about 55-60%, meaning the heart ejects most of its filled volume with each beat. The heart failure data shows: end-diastolic volume = 200 mL, stroke volume = 40 mL, so EF = (40/200) × 100% = 20%. This low EF indicates that despite the heart filling with a large volume (actually overfilling—200 mL vs normal 120 mL), it's ejecting only a small fraction because the myocardium has poor contractility. The heart is dilated and overstretched but cannot generate sufficient force to pump effectively. This distinguishes systolic heart failure (pumping problem—low EF) from diastolic heart failure (filling problem—preserved EF but reduced filling). Option A is opposite (filling is excessive). Option C is wrong because 20% EF is severely reduced. Option D is wrong because contractility is reduced, not excessive.

### **16. B - Absence of pancreatic lipase prevents triglyceride hydrolysis**

Pancreatic lipase is essential for fat digestion—it hydrolyzes triglycerides into monoglycerides and fatty acids, which can then be absorbed. The passage explains that lipase is contained in pancreatic secretions, and the clinical data shows that pancreatic insufficiency causes steatorrhea (fatty stools) because undigested fats pass through the GI tract. Without lipase, dietary triglycerides remain intact and cannot be absorbed (the intestinal epithelium cannot absorb intact triglycerides). The observation that protein and carbohydrate absorption remains normal indicates the problem is specifically with fat digestion, not with intestinal absorption capacity. Pancreatic amylase and proteases can digest carbohydrates and proteins normally. Option A is wrong because the stomach doesn't significantly digest fats. Option C is wrong because pancreatic insufficiency doesn't stop bile production. Option D is wrong because the products of fat digestion (fatty acids, monoglycerides) can be absorbed—the problem is lack of digestion.

### **17. B - Without bile salt emulsification, lipid digestion and micelle formation are impaired**

Bile salts are amphipathic molecules that emulsify large lipid droplets into small micelles, dramatically increasing surface area for lipase action and also forming micelles that ferry lipophilic products to the brush border for absorption. The passage explains this process explicitly. Cholestatic disease (blocked bile ducts) prevents bile salts from reaching the intestine, so: (1) Large fat droplets aren't emulsified, limiting lipase effectiveness even if lipase is present, and (2) Even if some fat is digested, without micelles to solubilize fatty acids and fat-soluble vitamins, absorption is impaired. The clinical data confirms steatorrhea and fat-soluble vitamin deficiencies despite normal pancreatic function. Option A is wrong because bile doesn't contain vitamins (the liver produces them separately). Option C is wrong because pancreatic enzymes can enter the duodenum even if bile doesn't (separate ducts). Option D is wrong because the liver continues producing vitamins; the problem is their intestinal absorption.

### **18. B - Unabsorbed lactose creating osmotic gradient that retains water in lumen**

Lactose intolerance occurs when lactase deficiency leaves lactose undigested. The passage explains that undigested lactose remains in the intestinal lumen and "creates osmotic diarrhea as water follows the solute gradient." Water cannot be absorbed if osmotically active solutes remain in the lumen—water follows osmotic gradients, and the high concentration of lactose in the lumen prevents water reabsorption or actually draws water into the lumen from tissues. Additionally, the passage mentions bacterial fermentation of lactose in the colon produces gas, causing bloating and cramping. The clinical data confirms high stool osmolarity (due to unabsorbed lactose). Option A is wrong because lactose doesn't

directly stimulate motility. Option C is wrong because lactose isn't toxic. Option D is wrong because lactose intolerance isn't an immune/allergic reaction (that's milk protein allergy).

**19. B - The Na<sup>+</sup>-glucose symporter uses Na<sup>+</sup> gradient (established by Na<sup>+</sup>/K<sup>+</sup>-ATPase) to drive glucose uptake**

The passage explains that glucose and galactose use "Na<sup>+</sup>-glucose symporters (secondary active transport)" for absorption. This symporter binds both Na<sup>+</sup> and glucose; Na<sup>+</sup> moving down its concentration gradient (high outside, low inside enterocyte) provides the energy to move glucose against its gradient (into the cell even when cellular glucose is higher than luminal glucose). The Na<sup>+</sup> gradient is established by the Na<sup>+</sup>/K<sup>+</sup>-ATPase on the basolateral membrane pumping Na<sup>+</sup> out. This secondary active transport allows complete glucose absorption even when intestinal glucose concentration becomes very low. The mechanism is essential for efficient nutrient absorption. Option A is wrong because they don't chemically bind permanently. Option C is wrong because Na<sup>+</sup> doesn't chemically activate glucose. Option D is backwards (Na<sup>+</sup>/K<sup>+</sup>-ATPase creates the Na<sup>+</sup> gradient; glucose doesn't create it).

**20. A - They are too large to enter intestinal capillaries**

The passage explicitly states that after triglycerides are resynthesized in enterocytes and packaged into chylomicrons, these large lipoprotein particles "enter lymphatic vessels (not directly into blood) because they're too large for intestinal capillaries." Chylomicrons are 75-1200 nm in diameter, much larger than the fenestrations in capillary endothelium. Lymphatic capillaries (lacteals in intestinal villi) have large openings that accommodate chylomicrons. The lymph eventually drains into the bloodstream via the thoracic duct at the subclavian vein, so chylomicrons do reach the blood but indirectly. This explains why a fatty meal causes lymph to appear milky (chylous). Option B is wrong because blood transports lipids extensively (as lipoproteins). Option C is nonsensical. Option D is wrong because there's no active rejection—it's purely a size issue.

**21. B - Without insulin, cells metabolize fats for energy, producing ketone bodies**

The passage explains that Type 1 diabetes eliminates insulin production, so "cells cannot take up glucose despite hyperglycemia." Unable to use glucose for energy, cells break down fats (lipolysis) and proteins as alternative fuels. Beta-oxidation of fatty acids in liver produces acetyl-CoA faster than the citric acid cycle can oxidize it, leading to ketone body synthesis (acetoacetate, β-hydroxybutyrate, acetone). These ketone bodies accumulate in blood and lower pH, causing metabolic acidosis (diabetic ketoacidosis). The patient data confirms: hyperglycemia, undetectable insulin, ketones in blood and urine, and significant weight loss (from fat and protein catabolism). Ketoacidosis is a medical emergency requiring insulin administration to restore glucose utilization and stop ketone production. Option A is backwards (glucose cannot be converted to ketones). Option C is wrong because glucagon doesn't directly produce ketones. Option D is wrong because kidneys do excrete ketones (causing ketonuria), but this doesn't prevent acidosis.

**22. A - Insulin resistance requires more insulin to achieve the same glucose-lowering effect**

Type 2 diabetes involves target cells becoming less responsive to insulin—insulin receptors may be downregulated or post-receptor signaling pathways may be impaired, often related to obesity and

inflammation. The passage states "Pancreas initially compensates by secreting more insulin (hyperinsulinemia)." To achieve the same effect of glucose uptake that normal insulin levels would produce in healthy individuals, the pancreas must produce much more insulin to overcome the resistance. The patient data confirms: elevated fasting glucose (160 mg/dL) despite insulin levels 3× normal—demonstrating that despite high insulin, glucose remains elevated because cells aren't responding adequately. Eventually, β-cells cannot maintain this excessive secretion rate and fail, progressing to insulin-dependent diabetes. Option B uses vague language without explaining mechanism. Option C is wrong because glucagon doesn't inhibit insulin degradation. Option D is incomplete (dietary factors contribute to resistance but don't fully explain hyperinsulinemia).

### **23. B - Thyroid hormones increase metabolic rate, burning more calories**

The passage states thyroid hormones "regulate metabolic rate" and "increase cellular respiration." The hyperthyroid patient data shows BMR increased 40% and weight loss of 15 lbs in 6 weeks despite increased appetite. Elevated T4 (4× normal) stimulates cellular metabolism throughout the body: increased oxygen consumption, increased ATP production and utilization, enhanced protein synthesis and degradation, increased cardiac output and heart rate. This elevated metabolism burns more calories than dietary intake provides, despite increased appetite, resulting in weight loss. The passage notes hyperthyroidism causes "weight loss despite increased appetite" as a classic presentation. Option A is wrong because digestion is normal. Option C is wrong because hyperthyroid patients don't typically develop diabetes (though they have reduced glucose tolerance). Option D is dismissive and wrong—the appetite increase is real but insufficient to match the increased energy expenditure.

### **24. B - Activating glycogen breakdown and gluconeogenesis in liver**

Glucagon is the primary counterregulatory hormone to insulin, released when blood glucose falls. The passage explains it "activates G-protein coupled receptors, increasing cAMP, which activates protein kinase A (PKA)," which then phosphorylates enzymes to promote: (1) Glycogenolysis—breaking down stored glycogen to glucose, and (2) Gluconeogenesis—synthesizing new glucose from amino acids, lactate, and glycerol. Both processes occur primarily in liver and release glucose into blood. The glucagon response test data confirms: blood glucose increased from 90 to 120 mg/dL within 30 minutes, glycogen phosphorylase activity increased 5-fold, and liver glycogen content decreased. Option A is wrong because glucagon doesn't affect intestinal absorption (it acts on stored/synthesized glucose). Option C is wrong because glucagon doesn't block insulin receptors directly (they're opposing hormones released in different conditions). Option D is wrong because proteins must be broken down to amino acids, transported to liver, and converted through gluconeogenesis.

### **25. B - Blood glucose exceeds renal reabsorption capacity**

Normally, glucose is freely filtered at the glomerulus but completely reabsorbed in the proximal tubule via Na<sup>+</sup>-glucose cotransporters (SGLT2 and SGLT1). These transporters have a maximum transport capacity (T<sub>m</sub>). At normal blood glucose (~90 mg/dL), filtered glucose doesn't exceed this capacity. However, in uncontrolled diabetes with blood glucose of 350 mg/dL (as in the patient data), the amount of filtered glucose exceeds the transporters' maximum reabsorption capacity. The excess glucose that cannot be reabsorbed remains in the tubular fluid and appears in urine (glucosuria). The passage states urinary glucose was "present (exceeded renal threshold)" in the Type 1 diabetic patient. This glucose in

urine creates osmotic diuresis, contributing to polyuria (excessive urination) and polydipsia (excessive thirst) in diabetics. Option A is wrong in untreated cases (though chronic diabetes does damage kidneys). Option C is nonsensical. Option D is wrong because insulin doesn't direct anything to urine.

### **26. A - Only males are affected and there's no male-to-male transmission**

The passage defines X-linked recessive traits as "affect[ing] males predominantly because males have one X chromosome" and states "Male-to-male transmission never occurs for X-linked traits because fathers give Y chromosomes to sons." The Family A pedigree data shows: affected males in generations I and III, no male-to-male transmission (affected males don't have affected sons), and carrier females in generation II who transmit to affected sons. These are diagnostic features of X-linked recessive inheritance. Affected males ( $X^rY$ ) have the recessive allele on their single X chromosome. Carrier females ( $X^RX^r$ ) are phenotypically normal but can pass the affected allele to sons (50% chance). Affected male  $\times$  normal female produces carrier daughters and unaffected sons. Option B is wrong because not everyone is affected. Option C is wrong because females can carry the trait. Option D is wrong because X-linked recessive traits typically skip generations.

### **27. B - IAi and IBi (both heterozygous)**

The ABO blood system has three alleles: IA, IB, and i. IA and IB are codominant ( $IAIB = \text{Type AB}$ ), both dominant over i. Type O requires ii genotype. If a child is Type O (ii), they must have inherited one i allele from each parent. Therefore: Mother (Type A) must be IAi, not IAIA (if she were IAIA, she couldn't pass i to offspring). Father (Type B) must be IBi, not IBIB (if he were IBIB, he couldn't pass i). Both parents are heterozygous carriers of the recessive i allele. Their children could be: 25% Type A (IAi), 25% Type B (IBi), 25% Type AB (IAIB), or 25% Type O (ii). Option A is wrong because two homozygous parents couldn't produce Type O offspring. Option C is impossible (can't have an IAIB parent and Type O child unless non-paternity). Option D is wrong because it can be determined from a Type O child.

### **28. B - The genes assort independently**

The 9:3:3:1 ratio is the classic dihybrid F2 ratio predicted by Mendel's law of independent assortment. In the cross described: F1 ( $RrYy$ )  $\times$  F1 ( $RrYy$ ), if the genes for seed shape (R/r) and color (Y/y) assort independently, we get: 9/16 round yellow ( $R\_Y\_$ ), 3/16 round green ( $R\_yy$ ), 3/16 wrinkled yellow ( $rrY\_$ ), 1/16 wrinkled green ( $rryy$ ). This ratio only occurs when genes are on different chromosomes or far apart on the same chromosome (unlinked), so alleles segregate independently during meiosis. The passage states independent assortment "applies to genes on different chromosomes or far apart on the same chromosome." Option A is wrong because linked genes would show parental combinations more frequently, distorting the ratio. Option C is wrong because incomplete dominance would show intermediate phenotypes, not these discrete categories. Option D is wrong because sex-linked traits show different ratios in male vs. female offspring.

### **29. B - 1/4 (each child is independent event)**

Probability for each birth is independent of previous births. The genotype probabilities from  $Aa \times Aa$  are: 1/4 AA, 1/2 Aa, 1/4 aa. If the trait is recessive, affected children are aa (1/4), and unaffected are AA or Aa (3/4). The fact that the first three children were unaffected doesn't change the probability for the fourth

child—each child independently has 1/4 chance of being affected and 3/4 chance of being unaffected. This is analogous to coin flips: flipping three heads doesn't change the probability that the fourth flip will be tails (still 50%). This independence is fundamental to Mendelian genetics—alleles segregate randomly during meiosis for each gamete formation event. Option A commits the gambler's fallacy. Option C gives the probability of being unaffected. Option D would be for a different cross (like  $Aa \times aa$ ).

### **30. B - Heterozygous (Aa)**

A test cross involves crossing an individual with dominant phenotype (unknown genotype: could be AA or Aa) with a homozygous recessive individual (aa). The passage explains this is done "to determine if an individual with dominant phenotype is homozygous or heterozygous." If the unknown is AA:  $AA \times aa$  produces all Aa offspring (100% dominant phenotype). If the unknown is Aa:  $Aa \times aa$  produces 50% Aa (dominant phenotype) and 50% aa (recessive phenotype). The data shows 50% dominant and 50% recessive offspring, which can only occur if the unknown parent is heterozygous (Aa). Each Aa parent gamete has 50% chance of carrying A or a, so crossed with aa (which can only contribute a), offspring are 50% Aa and 50% aa. Option A is wrong because homozygous dominant would produce 100% dominant offspring. Option C is wrong because that's recessive phenotype. Option D is wrong because the test cross definitively determines genotype.

### **31. B - Lysogenic cycle allows viral genome persistence when conditions favor host survival**

The passage explains that lambda phage can follow either lytic or lysogenic pathways. Under optimal growth conditions (nutrient-rich environment), the lysogenic pathway is favored because the host bacterium will continue dividing, replicating the integrated prophage with each cell division. This allows the viral genome to persist and propagate without killing the host. The lysogenic strategy is advantageous when environmental conditions are good and hosts are healthy—the virus "bets" on host survival and reproduction carrying the prophage into many descendant cells. Environmental stress (UV exposure in the data) triggers the prophage to excise and enter lytic cycle, producing new virions before the host dies from stress. This is evolutionarily adaptive: lysogeny during good times, lytic cycle during bad times. Option A is nonsensical. Option C is wrong because lambda can perform lytic cycle (UV induces it). Option D is wrong because it has lytic genes.

### **32. A - Only retroviruses use reverse transcriptase to synthesize DNA from RNA**

Retroviruses like HIV have RNA genomes but replicate through a DNA intermediate. The passage explains they "use reverse transcriptase to synthesize DNA from RNA genomes, integrating into host chromosomes as proviruses." This is unique to retroviruses—no other viruses or cellular organisms normally synthesize DNA from an RNA template (the central dogma is  $DNA \rightarrow RNA \rightarrow protein$ ). Host cells use DNA polymerases ( $DNA \rightarrow DNA$ ) and RNA polymerases ( $DNA \rightarrow RNA$ ) but not reverse transcriptase. Because this enzyme is unique to retroviruses, drugs targeting it (like AZT, a nucleoside analog) specifically inhibit retroviruses with minimal effect on host polymerases. The data shows AZT reduced HIV viral load by 90%, confirming this targeting. Option B is wrong because only retroviruses have reverse transcriptase. Option C is wrong because host cells definitely use DNA polymerase. Option D is wrong because retroviruses do contain RNA (they're not "lacking" DNA, they have RNA instead).

### **33. A - Both are needed for complete viral entry**

The passage states HIV's gp120 "binds CD4 and coreceptors (CCR5 or CXCR4)" and the experimental data shows "HIV can infect cells expressing CD4 but not CCR5/CXCR4: No infection." This demonstrates that both receptors are required—binding to CD4 alone is insufficient for entry. The entry process involves sequential binding: gp120 first binds CD4, which triggers conformational changes exposing the coreceptor binding site; gp120 then binds the coreceptor (CCR5 or CXCR4), triggering membrane fusion. Both binding events are necessary for the fusion process that allows viral entry. Individuals with CCR5 mutations are resistant to HIV infection despite having normal CD4, demonstrating the coreceptor's essential role. This dual requirement explains HIV's specificity for CD4+ T cells, macrophages, and dendritic cells that express both receptors. Option B is wrong because CD4 alone is insufficient (data proves this). Option C is wrong because the coreceptor is required, not optional. Option D contradicts the passage.

#### **34. B - Preventing viral release; virions remain attached to infected cell surface**

Neuraminidase is a viral surface enzyme that cleaves sialic acid residues. After new influenza virions assemble and bud from infected cells, they initially remain attached to the cell surface via hemagglutinin binding to sialic acid residues. Neuraminidase cleaves these bonds, releasing virions to infect new cells. The passage states "Neuraminidase inhibitors prevent influenza virion release." The experimental data confirms: "Virions produced but unable to release from infected cells. Accumulated at cell surface." Without neuraminidase, virions are trapped on the infected cell surface, preventing spread to other cells and limiting infection. This is the mechanism of drugs like oseltamivir (Tamiflu) and zanamivir. Option A is wrong because that's hemagglutinin's function, and neuraminidase acts after budding. Option C is wrong because it doesn't destroy RNA. Option D is wrong because it doesn't block immune response.

#### **35. A - Influenza viruses undergo antigenic drift (mutations in surface proteins)**

The passage explains that influenza requires annual vaccines while measles provides lifelong immunity, and the data confirms "Influenza: Annual vaccines needed (hemagglutinin and neuraminidase drift)" vs. "Measles: Single vaccination provides lifelong immunity (stable proteins)." Influenza virus has high mutation rates in genes encoding surface proteins (hemagglutinin and neuraminidase). This antigenic drift—accumulation of point mutations—allows the virus to evade immune recognition. Each year's circulating strains have slightly different surface proteins that antibodies from previous infections/vaccinations don't recognize well, requiring updated vaccines. In contrast, measles virus has stable surface proteins with low mutation rates; antibodies generated from vaccination recognize the virus throughout life. This difference reflects RNA polymerase fidelity: influenza (segmented negative-sense RNA) has error-prone replication, while measles (non-segmented negative-sense RNA) has more stable proteins. Option B is wrong because frequency doesn't explain the immunological difference. Option C is wrong because influenza does stimulate immunity (just against that specific strain). Option D is nonsensical.

#### **36. B - Carriers (Aa) represent 2pq while affected (aa) represent q<sup>2</sup>, and q<sup>2</sup> << 2pq when q is small**

The passage explains Hardy-Weinberg genotype frequencies: p<sup>2</sup> (AA), 2pq (Aa), q<sup>2</sup> (aa). For rare recessive diseases, q is very small. The PKU example calculates: disease frequency (aa) = q<sup>2</sup> = 0.0001, so q = 0.01.

Carrier frequency =  $2pq = 2(0.99)(0.01) = 0.0198 \approx 2\%$ . Carriers are about 200 times more common than affected individuals (0.02 vs. 0.0001). Mathematically, when  $q$  is small,  $2pq \gg q^2$  because:  $2pq \approx 2q$  (when  $p \approx 1$ ) while  $q^2$  is  $q$  multiplied by itself. For example, if  $q = 0.01$ , then  $2q \approx 0.02$  but  $q^2 = 0.0001$ —a 200-fold difference. This has major implications: most recessive disease alleles exist hidden in heterozygous carriers, making it impossible to eliminate these alleles from populations through selection (which only acts on expressed phenotypes). Option A is nonsensical. Option C is wrong because affected individuals are born alive (though survival depends on the specific disease). Option D is wrong because carriers are generally asymptomatic by definition.

### **37. B - Heterozygote advantage maintaining balanced polymorphism**

The passage explicitly describes sickle cell as an example of heterozygote advantage: "HbA/HbS heterozygotes resist malaria better than HbA/HbA homozygotes while avoiding sickle cell disease (HbS/HbS). Both alleles persist at intermediate frequencies." The data shows HbS frequency is 10-15% in malaria regions but <1% in non-malaria regions, demonstrating that the allele persists specifically where its heterozygote advantage exists. In malaria-endemic regions, natural selection favors the heterozygote over both homozygotes: HbA/HbA individuals are susceptible to malaria, HbS/HbS individuals have sickle cell disease, but HbA/HbS individuals are malaria-resistant and healthy. This balancing selection maintains both alleles in the population. Option A (drift) is wrong because the pattern is clearly adaptive, not random. Option C (mutation pressure) is wrong because mutation rates don't differ between regions. Option D (founder effect) is wrong because the correlation with malaria prevalence indicates selection, not random founding.

### **38. A - The small founding group didn't represent the mainland genetic diversity**

Founder effects occur when a small group establishes a new population, carrying only a subset of the original population's genetic variation. By chance, allele frequencies in the founders may differ substantially from the source population. The passage states "Founder effects occur when small groups establish new populations, carrying only subset of original genetic variation." The data shows the island (founded by 20 individuals) has allele B frequency of 0.30 compared to mainland frequency of 0.05—a 6-fold difference. This occurred because, by chance, the 20 founders included individuals carrying multiple B alleles. If selection or local conditions favored B on the island, we'd expect gradual increase over generations, not immediate high frequency. Option B is wrong because there's no evidence of selection (and the question attributes it to founding). Option C is wrong because mutation rates don't differ by location. Option D is wrong because population size doesn't systematically affect allele frequencies in that direction.

### **39. C - 0.48**

Using Hardy-Weinberg formula with  $p = 0.6$  and  $q = 0.4$  (and noting  $p + q = 1.0$ ): Heterozygote frequency =  $2pq = 2(0.6)(0.4) = 2(0.24) = 0.48$ . This means 48% of the population is heterozygous. To verify:  $p^2 = (0.6)^2 = 0.36$  (36% homozygous dominant),  $q^2 = (0.4)^2 = 0.16$  (16% homozygous recessive), and  $2pq = 0.48$  (48% heterozygous). Total:  $0.36 + 0.48 + 0.16 = 1.00 \checkmark$ . Option A (0.16) is the  $q^2$  term (homozygous recessive frequency). Option B (0.36) is the  $p^2$  term (homozygous dominant frequency). Option D (0.64) might result from calculation error.

#### **40. B - Most copies are hidden in heterozygotes who have normal phenotypes**

For a recessive lethal allele, selection can only act against homozygous recessive individuals (aa) who die or have reduced fitness. The passage states "Slow decline because most alleles hidden in heterozygotes." Heterozygotes (Aa) have normal phenotype (by definition of recessive) and normal fitness, so natural selection cannot "see" and eliminate the recessive allele they carry. When  $q$  is small (recessive alleles are rare), the heterozygote frequency ( $2pq$ ) greatly exceeds the homozygote frequency ( $q^2$ ), meaning most recessive alleles exist in heterozygotes. The data confirms: starting from  $q = 0.10$ , after one generation of complete selection against aa,  $q$  only decreased to 0.09—a very slow decline. Mathematically,  $\Delta q = -q^2/(1+q)$  per generation for recessive lethal, and when  $q$  is small, this approaches zero. Option A is true but isn't the reason for slow decline. Option C is wrong (lethal alleles don't provide advantages). Option D is wrong (selection definitely affects recessive alleles, just very slowly).

#### **41. B - Ribosomes recognize and bind the cap to initiate translation**

The passage states "Initiation begins when the small ribosomal subunit binds mRNA's 5' cap, scans for the start codon (AUG), and initiates at the first AUG." The experimental data confirms: "Without 5' cap: Translation efficiency decreased 90%." The 5' cap (7-methylguanosine) is recognized by eukaryotic initiation factors that recruit the small ribosomal subunit to the mRNA. This cap-dependent initiation ensures ribosomes bind at the correct end of mRNA and begin scanning for the start codon from the 5' end. Without the cap, ribosomes cannot efficiently locate and bind the mRNA, dramatically reducing translation. Some alternative initiation mechanisms exist (IRES-mediated) but are much less efficient. Option A is wrong because the cap doesn't contain the start codon (AUG is downstream). Option C is wrong because the cap doesn't encode anything (it's modified guanosine). Option D is too extreme—uncapped mRNA is degraded faster but the data shows 10% translation efficiency remains, indicating some stability.

#### **42. A - Blocking elongation, preventing ribosome translocation**

The passage states "Cycloheximide treatment (blocks eEF2 elongation factor)" and data shows "Translation stopped mid-chain. Completed proteins released normally. Nascent chains remained attached to ribosomes." eEF2 (eukaryotic elongation factor 2) catalyzes ribosome translocation—the movement of the ribosome three nucleotides along mRNA after peptide bond formation. By blocking eEF2, cycloheximide prevents this translocation step, stalling ribosomes with partially completed polypeptides still attached. Proteins that were already complete were released normally (termination proceeded), but new elongation cycles couldn't complete. This explains why cycloheximide is used experimentally to freeze translation at a particular moment—allowing study of which proteins are actively being synthesized. Option B is wrong because it doesn't remove the cap. Option C is wrong because it doesn't degrade mRNA directly. Option D is wrong because it acts during elongation, not termination (the data shows completed proteins release normally).

#### **43. A - Inosine can base pair flexibly with different nucleotides**

Wobble base pairing occurs at the third codon position (5' position of the anticodon) where non-Watson-Crick base pairing is tolerated. Inosine, a modified nucleoside found in some tRNAs, can base pair with U, C, or A (though not with G), allowing a single tRNA to recognize multiple codons differing in the third

position. The passage states "The third codon position shows wobble; non-standard base pairing allows single tRNAs to recognize multiple codons." The data shows tRNA with anticodon 3'-IAU-5' recognizes codons AUA, AUC, and AUU—all encoding isoleucine but differing in the third position. This wobble reduces the number of tRNAs required (organism doesn't need 61 different tRNAs for 61 sense codons). Option B is wrong because inosine is distinct from adenine (it's hypoxanthine ribonucleoside). Option C is wrong because the ribosome doesn't change mRNA sequence. Option D is wrong because wobble position absolutely affects which codons are recognized (it enables the flexible pairing).

#### **44. B - Degradation of regulatory proteins like cyclins is required for cycle progression**

The passage states that ubiquitin "tags proteins for degradation via proteasomes." The experimental data shows "Proteasome inhibition: Ubiquitinated proteins accumulated. Cell cycle arrest (cyclins not degraded)." Cyclins are regulatory proteins that must be degraded at specific cell cycle points to allow progression to the next phase. For example, M-cyclins must be degraded to exit mitosis and enter G1. When proteasomes are inhibited, cyclins accumulate and cannot be destroyed at the appropriate time, preventing cell cycle progression. This demonstrates that protein degradation is not just waste disposal but an active regulatory mechanism—controlled proteolysis is as important as synthesis for regulating cellular processes. ER stress increased because misfolded proteins that should be degraded accumulated. Option A is backwards (proteasomes degrade, not synthesize). Option C is wrong because proteasomes don't produce ATP. Option D is wrong because DNA replication doesn't directly require proteasomes (though cell cycle progression to S-phase does).

#### **45. B - Causes premature termination, producing truncated protein**

Nonsense mutations change a codon specifying an amino acid into a stop codon (UAA, UAG, or UGA). The passage states these are "termination codons" and the experimental data shows: "Normal: AUG-UAC-CAG → Met-Tyr-Gln. Mutant: AUG-UAG-CAG → Met-STOP (truncated protein). Mutant protein: Nonfunctional." When the ribosome encounters the premature stop codon at position 2, it terminates translation immediately, releasing a truncated polypeptide containing only the amino acids encoded before the stop codon. In this example, only methionine is incorporated. Truncated proteins are usually nonfunctional because they lack most of their sequence, losing critical domains, proper folding, or stability. Nonsense mutations often cause severe genetic diseases (like some forms of cystic fibrosis or muscular dystrophy) by eliminating functional protein production. Option A is opposite. Option C is wrong because the effect is severe. Option D describes a missense mutation, not nonsense.

#### **46. B - Light-activated cascade closes cGMP-gated Na<sup>+</sup> channels, stopping depolarizing current**

The passage explains the phototransduction cascade: light activates rhodopsin → activates transducin → activates PDE → hydrolyzes cGMP → cGMP-gated Na<sup>+</sup> channels close. In darkness, high cGMP keeps Na<sup>+</sup> channels open, allowing Na<sup>+</sup> influx that depolarizes the cell to about -40 mV. When light hits, cGMP is hydrolyzed, channels close, Na<sup>+</sup> influx stops, and the cell hyperpolarizes to about -70 mV (closer to K<sup>+</sup> equilibrium potential). The data confirms: "Dark-adapted rods: High cGMP, Na<sup>+</sup> channels open, depolarized (-40 mV). Light exposure: cGMP decreased, channels closed, hyperpolarized (-70 mV)." This hyperpolarization reduces glutamate release onto bipolar cells. The unusual aspect is that the stimulus (light) causes hyperpolarization rather than depolarization—most sensory cells depolarize with stimulation. Option A is wrong because light doesn't destroy channels. Option C is wrong because

rhodopsin doesn't pump ions. Option D is wrong because light doesn't directly affect membrane potential in all cells.

#### **47. A - Detection of single photons**

The passage emphasizes amplification: "one activated rhodopsin activates hundreds of transducin molecules; each PDE hydrolyzes thousands of cGMP molecules. This allows detection of single photons." Without amplification, a single photon would only cause one rhodopsin molecule to isomerize, producing a signal too small to affect the cell's membrane potential detectably. The cascade amplifies the initial signal by factors of 100-1000 at each step, so one photon ultimately closes enough  $\text{Na}^+$  channels to produce a measurable electrical response. This extraordinary sensitivity explains why we can see in very dim conditions—rods can respond to single photons, though conscious perception requires multiple photons. Option B is wrong because color vision depends on cone opsins, not amplification. Option C is wrong because focusing involves the lens, not signal transduction. Option D is wrong because pupil constriction involves iris muscles, not phototransduction amplification.

#### **48. B - Endolymph has unusually high $[\text{K}^+]$ , making $\text{K}^+$ equilibrium potential positive**

The passage explains this unusual situation: " $\text{K}^+$  influx depolarizes hair cells (unusually,  $\text{K}^+$  influx depolarizes because endolymph has high  $\text{K}^+$  concentration)." Normally,  $\text{K}^+$  influx would hyperpolarize cells because  $[\text{K}^+]$  is higher inside than outside, so  $\text{K}^+$  flows out. However, cochlear endolymph contains about 150 mM  $\text{K}^+$  (similar to intracellular concentration), making the  $\text{K}^+$  equilibrium potential around 0 mV rather than the typical -90 mV. When mechanically-gated channels open and  $\text{K}^+$  flows from endolymph into hair cells, it depolarizes the cell from its resting potential of about -50 mV toward 0 mV. This depolarization opens voltage-gated  $\text{Ca}^{2+}$  channels, triggering neurotransmitter release. This unique ionic environment allows hair cells to use  $\text{K}^+$  channels for excitation rather than inhibition. Option A is wrong because  $\text{K}^+$  direction depends on concentration gradients. Option C is wrong because they are  $\text{K}^+$ -permeable channels. Option D is wrong because polarity isn't reversed, just the  $\text{K}^+$  gradient is unusual.

#### **49. B - Each odorant activates a unique combination of receptors**

The passage states: "Humans have ~400 different olfactory receptor genes; each olfactory neuron expresses only one receptor type, achieving specificity. Odor discrimination arises from combinatorial coding—each odorant activates multiple receptor types in unique patterns." The experimental data illustrates this: octanol activates receptors A and B, hexanol activates A and B, geraniol activates B and C, citronellal activates only C. Each odorant creates a specific activation pattern across receptors, and the brain recognizes odors by the combination of receptors activated. This combinatorial code allows discrimination of thousands of odors with just hundreds of receptors—similar to how English uses 26 letters to encode many words through different combinations. Option A is wrong because receptors are not perfectly specific (they're broadly tuned). Option C is wrong because receptors definitely discriminate. Option D is wrong because ~400 receptor types exist.

#### **50. A - The base responds preferentially to high frequencies**

The passage explains cochlear tonotopy: "High-frequency sounds: Basilar membrane vibration at cochlear base. Low-frequency sounds: Vibration at apex." The basilar membrane has mechanical properties that

vary along its length: at the base (near the oval window) it's narrow and stiff, responding best to high frequencies; at the apex it's wide and flexible, responding best to low frequencies. When hair cells at the base are damaged (by noise exposure, ototoxic drugs, or aging), sensitivity to high frequencies is lost while low-frequency hearing remains intact. The data confirms: "Hair cell damage at base: High-frequency hearing loss." This frequency mapping allows the auditory system to perform spectral analysis—different frequencies activate different locations, enabling frequency discrimination. Option B is opposite (high frequencies cause maximal vibration at base). Option C is wrong because the base can detect sounds (just high-frequency ones). Option D is wrong because frequency definitely relates to location (place coding).

### **51. A - DNA polymerase can only synthesize 5' to 3'**

DNA polymerase adds nucleotides only to the 3'-OH group of a growing strand, meaning synthesis proceeds 5' to 3'. At the replication fork, the two template strands are antiparallel: one runs 3' to 5' (allowing continuous leading strand synthesis in the 5' to 3' direction toward the fork), while the other runs 5' to 3' (requiring discontinuous synthesis away from the fork in short Okazaki fragments). The lagging strand must be synthesized discontinuously because polymerase cannot synthesize 3' to 5'. Each Okazaki fragment requires a new RNA primer from primase, then DNA polymerase extends from that primer. Later, primers are removed and gaps filled by DNA polymerase I and sealed by DNA ligase. Option B describes the leading strand template. Option C is wrong because primase's discontinuous activity is a consequence, not cause. Option D is wrong because leading strand synthesis doesn't physically prevent lagging strand synthesis.

### **52. B - Releases free energy ( $\Delta G < 0$ ) and can occur spontaneously**

Exergonic reactions have negative  $\Delta G$  (Gibbs free energy change), meaning they release free energy and are thermodynamically favorable (spontaneous). The term "exergonic" literally means "energy outward"—the system loses free energy to the surroundings. Examples include: cellular respiration, ATP hydrolysis, and most catabolic pathways. Spontaneity ( $\Delta G < 0$ ) doesn't necessarily mean fast—kinetically slow reactions can still be thermodynamically favorable. Many spontaneous reactions require activation energy input to overcome barriers, but once initiated, they proceed with net energy release. Option A describes endergonic reactions ( $\Delta G > 0$ ), which require energy input. Option C describes equilibrium ( $\Delta G = 0$ ). Option D is wrong because many exergonic reactions don't involve ATP (some produce it).

### **53. B - Partial pressure of O<sub>2</sub> is higher in alveolar air than in deoxygenated blood**

Gas exchange in lungs occurs by passive diffusion down concentration gradients (more precisely, partial pressure gradients). Alveolar air has PO<sub>2</sub>  $\approx$  100 mmHg, while deoxygenated blood entering pulmonary capillaries has PO<sub>2</sub>  $\approx$  40 mmHg. This 60 mmHg gradient drives O<sub>2</sub> diffusion from alveoli into blood across the respiratory membrane (alveolar epithelium, basement membrane, capillary endothelium). Similarly, PCO<sub>2</sub> is higher in blood ( $\sim$ 46 mmHg) than alveolar air ( $\sim$ 40 mmHg), driving CO<sub>2</sub> diffusion from blood into alveoli for exhalation. The large surface area of alveoli ( $\sim$ 70 m<sup>2</sup>) and thin respiratory membrane ( $\sim$ 0.5  $\mu$ m) facilitate rapid diffusion. Option A is wrong because O<sub>2</sub> moves by passive diffusion, not active transport. Option C is wrong because hemoglobin doesn't "pull" O<sub>2</sub> (it binds O<sub>2</sub> that has diffused into blood). Option D is wrong because blood pressure doesn't directly force gases across membranes.

#### 54. C - Missense mutation

A missense mutation is a point mutation (single nucleotide change) that alters a codon so it encodes a different amino acid. For example: GAA (Glu) → GUA (Val) causes sickle cell anemia. Effects vary: conservative substitutions (similar amino acids) may have minimal impact, while nonconservative substitutions can severely affect protein function if they occur in critical regions. Option A (silent mutation) changes a codon but still encodes the same amino acid due to genetic code degeneracy (e.g., GAA → GAG both encode Glu)—no phenotypic effect. Option B (nonsense mutation) introduces a stop codon, causing premature termination. Option D (frameshift mutation) involves insertion or deletion of nucleotides not in multiples of three, shifting the reading frame and usually causing completely different amino acid sequence downstream.

#### 55. B - The amino acid sequence

Primary structure is the linear sequence of amino acids connected by peptide bonds—the fundamental level of protein organization specified directly by mRNA sequence. It's written N-terminus to C-terminus. Secondary structure (option C) refers to local folding patterns like  $\alpha$ -helices and  $\beta$ -sheets stabilized by hydrogen bonding between backbone atoms. Tertiary structure (option A) is the overall 3D shape from interactions between side chains (disulfide bonds, hydrophobic interactions, electrostatic interactions). Quaternary structure (option D) involves multiple polypeptide subunits assembling into functional protein complexes (e.g., hemoglobin has four subunits). The Anfinsen principle states that primary structure determines all higher-order structures under appropriate conditions—the amino acid sequence contains information specifying how the protein folds.

#### 56. B - Target bacterial cell walls, which human cells lack

Penicillin and related  $\beta$ -lactam antibiotics inhibit bacterial cell wall synthesis by blocking transpeptidase enzymes that cross-link peptidoglycan strands. Bacterial cell walls (peptidoglycan) are essential for withstanding osmotic pressure and maintaining cell shape—without intact walls, bacteria lyse. Human and other eukaryotic cells completely lack cell walls (we have cell membranes only), so penicillin has no target in human cells and causes minimal toxicity (except allergic reactions in sensitive individuals). This selective toxicity principle—targeting structures or processes present in pathogens but absent in hosts—is fundamental to antimicrobial chemotherapy. Option A is wrong because penicillin does cross membranes. Option C is wrong because temperature doesn't affect specificity. Option D is wrong because penicillin is quite stable in human body.

#### 57. C - Fats (lipids)

Fats provide approximately 9 kcal/gram, while carbohydrates and proteins both provide about 4 kcal/gram. This more than 2× energy density makes fats the most efficient energy storage molecules. The high energy content results from fats being highly reduced (many C-H bonds) and anhydrous (no water of hydration required for storage like glycogen needs). Adipose tissue stores energy as triglycerides, allowing animals to maintain large energy reserves without excessive weight. During fasting or endurance exercise, fatty acid  $\beta$ -oxidation provides sustained energy. Option A (carbohydrates) stores less energy per gram and requires water for glycogen storage. Option B (proteins) are not primarily energy storage (used for structure/function). Option D (nucleic acids) are informational molecules, not energy stores.

### **58. C - Human chorionic gonadotropin (hCG)**

After ovulation, the corpus luteum forms from the ruptured follicle and produces progesterone (and estrogen) that maintains the uterine lining. Without pregnancy, the corpus luteum degenerates after ~14 days, progesterone drops, triggering menstruation. During pregnancy, the developing embryo (specifically the trophoblast/placenta) secretes hCG, which is structurally similar to LH and binds LH receptors on the corpus luteum, maintaining its function. This hCG "rescue" of the corpus luteum ensures continued progesterone production essential for maintaining pregnancy until the placenta can produce sufficient progesterone (around week 10). Home pregnancy tests detect hCG in urine. Option A (FSH) stimulates follicle development before ovulation. Option B (LH) triggers ovulation and initially forms corpus luteum but doesn't maintain it in pregnancy. Option D (progesterone) is produced by corpus luteum but doesn't maintain itself.

### **59. C - 10%**

The 10% rule (also called 10% energy transfer efficiency) states that when energy is transferred from one trophic level to the next in a food chain/web, approximately 10% is incorporated into biomass while 90% is lost as heat through cellular respiration, inefficient digestion, and other metabolic processes. For example, if primary producers capture 10,000 kcal of solar energy, primary consumers (herbivores) obtain ~1,000 kcal, secondary consumers ~100 kcal, and tertiary consumers ~10 kcal. This explains why food chains rarely exceed 4-5 trophic levels—insufficient energy remains to support higher levels. It also explains biomass pyramids in ecosystems. The low efficiency reflects the second law of thermodynamics—energy transformations are never 100% efficient. Option A (90%) is the amount lost, not transferred. Options B and D don't match ecological data.

# SECTION 4: ANSWER EXPLANATIONS

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## 1. B - Sleep actively consolidates memories beyond mere passage of time

Group A (sleep condition) recalled 85% compared to Group C (same time awake) at 75%, demonstrating that sleep provides benefits beyond simply allowing time to pass. If memory consolidation were merely a time-dependent passive process, Groups A and C should perform similarly since both had equivalent time intervals between learning and testing. The superior performance after sleep indicates that sleep actively engages consolidation processes—the hippocampal-neocortical dialogue described in the passage where neural patterns replay and strengthen during sleep. Group C's 75% recall (better than sleep-deprived Group B's 55%) shows some consolidation occurs with time, but Group A's 85% demonstrates sleep's active enhancement. Option A is incorrect because passive decay would predict worse memory after sleep (more time for forgetting). Option C is extreme and contradicted by Group C's reasonable performance. Option D is wrong because the comparison between Groups A and C controls for circadian factors—both tested at same time of day.

## 2. B - Slow-wave sleep is specifically important for declarative memory consolidation

The passage states the dual-process hypothesis proposes that "declarative memories consolidate primarily during slow-wave sleep, while procedural memories consolidate during REM sleep." The experimental data directly tests this: Group Y (deprived of slow-wave sleep but with preserved REM) showed impaired declarative memory, while Group X (deprived of REM but with preserved slow-wave sleep) showed normal declarative memory consolidation. This selective impairment pattern demonstrates that slow-wave sleep specifically supports declarative memory—removing it hurts declarative memory even when total sleep time and REM sleep are preserved. If all sleep stages contributed equally (option C), removing any stage should impair memory equivalently. Option A is wrong because the results show REM deprivation didn't impair declarative memory in this experiment (though REM is important for procedural memory). Option D is opposite of the findings.

## 3. A - REM sleep facilitates creative problem-solving and novel associations

The passage states "REM sleep may facilitate emotional memory processing and creative problem-solving through novel connections formed during cortical activation." The insight problem data supports this: morning naps (REM-rich due to circadian timing) led to 45% solving the insight problem compared to only 20% after afternoon naps (slow-wave-rich). Sleep overall improved problem-solving (60% after full night vs. 25% for wake controls), but the nap timing effect specifically implicates REM sleep since morning naps contain more REM while afternoon naps contain more slow-wave sleep. Insight problems require seeing problems in new ways and making non-obvious connections—processes that REM's cortical activation and novel neural associations may facilitate. Option B is wrong because slow-wave sleep has important functions (declarative memory), just not specifically for creative insight. Option C is contradicted by data showing napping improved outcomes. Option D is wrong because time of day clearly affects sleep stage composition and thus cognitive outcomes.

## 4. B - Transfer hippocampal-dependent memories to neocortex for long-term storage

The passage explains that during slow-wave sleep, "the hippocampus replays neural patterns from recent experiences, strengthening cortical representations and gradually making memories independent of hippocampal retrieval." This describes systems consolidation—the process by which memories initially dependent on the hippocampus become gradually represented in neocortical networks for permanent storage. The hippocampus serves as a temporary storage site for new declarative memories, and sleep allows the transfer of these memories to distributed cortical networks where they become stable and less vulnerable to hippocampal damage. Sleep spindles facilitate this information transfer. This explains why older memories (already consolidated to cortex) survive hippocampal damage while recent memories (still hippocampal-dependent) are lost. Option A is wrong because consolidation strengthens existing memories, not creates imaginary ones. Option C misunderstands the process—memories transfer but aren't deleted from hippocampus. Option D is wrong because REM sleep (not slow-wave) features dreaming prominently.

### **5. B - Cumulative consolidation deficits impair both encoding and retrieval of learned material**

The passage states "Chronic sleep restriction accumulates deficits, affecting academic performance, workplace productivity, and safety" and that "sleep-deprived individuals show reduced hippocampal activation during encoding, predicting poor subsequent memory." Chronic sleep restriction creates multiple problems: (1) impaired consolidation of previously learned material (each night's poor sleep fails to consolidate that day's learning), (2) impaired encoding of new material (sleep deprivation reduces hippocampal function needed for forming new memories), and (3) cumulative effects that compound over time. Students who chronically restrict sleep experience accumulated deficits—their learning from weeks ago wasn't consolidated properly, yesterday's learning wasn't consolidated, and today they can't encode effectively. Option A is nonsensical and opposite of evidence. Option C is directly contradicted by extensive research showing sleep-learning relationships. Option D is wrong because while caffeine may reduce subjective sleepiness, it doesn't restore consolidation processes that occur specifically during sleep.

### **6. C - Can emerge from mere categorization into groups**

The minimal group paradigm's key finding is that even arbitrary, meaningless categorization (like preference for Klee vs. Kandinsky, or dot estimation accuracy) produces ingroup favoritism without any history, interaction, competition, or realistic conflict between groups. The passage states "minimal group paradigm experiments demonstrate that even arbitrary group assignment causes ingroup favoritism" and that "This suggests that mere categorization, without competition or prior hostility, is sufficient for intergroup bias." The data confirms: 72% allocated more resources to their ingroup despite the groups being completely meaningless and newly created. This is theoretically important because it shows that intergroup bias doesn't require the conditions predicted by other theories—it emerges from the basic cognitive process of categorization combined with the motivation to maintain positive social identity. Option A is wrong because minimal groups have no history. Option B is wrong because realistic conflict theory is contradicted by minimal group findings. Option D is wrong because the phenomenon is well-replicated in labs.

### **7. B - Motivation to enhance relative ingroup status even at absolute cost**

When participants chose ingroup advantage over maximum joint profit (sacrificing total resources to maintain relative advantage), this demonstrates that social identity motivation isn't about absolute gain but

relative status. The passage notes "58% chose ingroup advantage even at cost to total reward." For example, choosing to give ingroup member \$3 and outgroup \$1 (difference of +\$2) over giving ingroup \$4 and outgroup \$5 (difference of -\$1), despite the latter providing more total money. This irrational economic choice serves the psychological function of maintaining positive social identity through favorable social comparison—the ingroup "wins" relative to the outgroup even though everyone gets less money. This supports social identity theory's prediction that people are motivated to achieve positive distinctiveness for their group to enhance self-esteem. Option A is wrong because economically rational behavior would maximize absolute gain. Option C is insulting and wrong. Option D is wrong because participants specifically chose unequal outcomes favoring ingroup.

### **8. B - Ingroup favoritism serves to maintain or restore positive social identity and self-esteem**

Social identity theory proposes that group membership contributes to self-esteem, and that people engage in ingroup favoritism partly to maintain positive social identity. The experimental data directly tests this: participants who received negative feedback (threat to self-esteem) were given opportunity to show ingroup favoritism (Group A) or not (Group B). Group A's self-esteem recovered to baseline after the intergroup task allowing favoritism, while Group B's remained depressed. This shows that discriminating in favor of one's ingroup served the psychological function of restoring self-esteem damaged by the negative feedback—by making one's group look good relative to the outgroup, participants could feel better about themselves as group members. This causal evidence (manipulation → outcome) is stronger than mere correlation. Option A is contradicted by the evidence. Option C is wrong because the entire experiment manipulates group membership to affect self-esteem. Option D is wrong because Group A's self-esteem did recover.

### **9. A - Contact alone is insufficient; cooperation toward shared goals is necessary**

The passage distinguishes between realistic conflict theory (predicting that cooperation toward superordinate goals reduces conflict) and mere contact. The experimental data shows that Condition 1 (contact without shared goals—just eating lunch together) produced no prejudice reduction or even slight increase, while Condition 2 (cooperative tasks toward shared goals) reduced prejudice by 40% and increased cross-group friendships. This supports Allport's contact hypothesis, which specifies conditions under which contact reduces prejudice: equal status, common goals, intergroup cooperation, and institutional support. Mere proximity or contact without these conditions may actually increase prejudice by providing opportunities for negative interactions. The Robbers Cave study mentioned in the passage showed the same pattern: hostility reduced when groups worked on superordinate goals (fixing water supply, getting stuck truck moving) requiring cooperation. Option B is wrong because some contact conditions do reduce prejudice. Option C is wrong because Condition 2 showed reduction. Option D is backward—cooperation, not competition, improved relations.

### **10. C - The context determines which identity is salient and influences behavior accordingly**

Self-categorization theory proposes that people have multiple social identities that become salient depending on context. The passage states "identity shifts along a continuum from personal to social depending on context" and "When social identity is salient, group norms strongly influence behavior." The experimental manipulation demonstrates this: when gender identity was made salient (Condition A), gender biases emerged in competence ratings; when university identity was salient (Condition B), gender

bias disappeared but university affiliation bias emerged. The same person holds both identities (woman/man AND university student), but which identity influences judgment depends on which is activated by the situation. This has important implications: discrimination isn't necessarily about fixed prejudices but about which categorization is cognitively active. Interventions might reduce bias by making different identities salient—for example, emphasizing common superordinate identity. Options A and B are wrong because people have multiple identities. Option D is contradicted by the data showing clear effects.

### **11. B - The HPA axis involves a hormonal cascade requiring time for synthesis and secretion**

The passage explains that "When the brain perceives a stressor, the paraventricular nucleus of the hypothalamus releases CRH, which stimulates the anterior pituitary to secrete ACTH. ACTH travels through the bloodstream to the adrenal cortex, stimulating cortisol release. This cascade occurs over minutes." The data shows cortisol peaks at +20 minutes, not immediately at stressor onset, because the sequential hormonal signaling takes time: hypothalamus must synthesize and release CRH, CRH must travel to pituitary, pituitary must synthesize and secrete ACTH, ACTH must travel through blood to adrenal glands, and adrenal cortex must synthesize and secrete cortisol. This contrasts with the sympathetic nervous system (fight-or-flight) which acts within seconds via neural signaling. The time course is characteristic of endocrine versus neural responses. Option A is nonsensical. Option C is backward. Option D is unwarranted skepticism—the time course is well-established.

### **12. B - Prolonged cortisol exposure damages hippocampus, impairing HPA regulation**

The passage states "Prolonged cortisol exposure can damage hippocampal neurons, impairing negative feedback and creating a vicious cycle of overactivation. Hippocampal atrophy observed in chronic stress and depression may result from this cortisol neurotoxicity combined with reduced neurogenesis." The caregivers showed both hippocampal volume reduction (8% smaller) and HPA dysregulation (higher baseline cortisol, flatter diurnal rhythm). Normal cortisol regulation requires the hippocampus to detect elevated cortisol and inhibit further HPA activation (negative feedback). When chronic stress damages the hippocampus, this feedback mechanism fails, leading to chronic overactivation—the system cannot turn itself off properly. The flatter diurnal rhythm (which normally shows high morning cortisol declining through the day) indicates dysregulation. This creates a pathological cycle: stress → cortisol → hippocampal damage → impaired feedback → more cortisol → more damage. Option A is contradicted by the data. Option C is wrong because hippocampal size definitely relates to stress. Option D is wrong because baseline cortisol was actually higher, not absent.

### **13. A - Early adversity programs HPA axis sensitivity, creating lasting vulnerability**

The passage describes the "fetal programming hypothesis" that "prenatal stress exposure alters HPA axis set points" and states "childhood adversity predicts exaggerated cortisol responses and increased vulnerability to stress-related disorders." The data shows adults with childhood abuse history had 50% higher cortisol peaks and prolonged recovery (90 min vs. 60 min) to laboratory stressors—decades after the adversity ended. This demonstrates that early experiences during development permanently alter stress physiology, creating heightened reactivity that persists into adulthood. The mechanisms likely involve epigenetic changes during sensitive developmental periods when stress systems are being established. This early programming creates vulnerability: the same objective stressor produces larger physiological

responses in those with adversity history, contributing to their 3× higher lifetime depression risk. Option B is contradicted by extensive evidence. Option C is wrong because the groups differed systematically. Option D is too deterministic—genetics contribute but early experience has documented effects.

#### **14. A - Cognitive appraisal influences physiological stress responses**

The passage states "Stress appraisal—how individuals interpret situations—profoundly affects HPA activation" and describes how challenge vs. threat appraisals produce different responses. The experimental data demonstrates this causally: identical stressor (public speech) but different framing—threat frame: "this will be stressful" versus challenge frame: "opportunity to showcase skills." Challenge frame produced lower cortisol (18 vs. 25 ng/mL), better performance (7.8 vs. 6.2), and different subjective experience (excitement vs. anxiety). This shows that cognitive interpretation of situations (top-down processing) influences bottom-up physiological responses—not just conscious experience but actual hormone secretion. This has therapeutic implications: cognitive reappraisal techniques can reduce physiological stress responses, as mentioned in the passage. The mind-body interaction is bidirectional and powerful. Option B is contradicted by the data. Option C is wrong because the relationship between cortisol and performance is complex and inverted-U shaped. Option D represents outdated dualism—psychological and physiological processes interact constantly.

#### **15. B - Terminate the stress response after the threat has passed**

The passage explains that cortisol "affects the brain, influencing memory consolidation, emotional processing, and the stress response itself through negative feedback loops—cortisol binds receptors in the hippocampus and hypothalamus, inhibiting further HPA activation once the stressor passes." Negative feedback is a homeostatic regulatory mechanism: when cortisol reaches sufficient levels, it binds to glucocorticoid receptors in the hippocampus and hypothalamus, which detect the elevated cortisol and send inhibitory signals to shut down further CRH and ACTH release. This prevents indefinite stress response activation after the threat is resolved. The acute stress data shows this working normally: cortisol peaks at +20 min then declines toward baseline by +60 min as negative feedback takes effect. In chronic stress, this feedback mechanism becomes impaired (as discussed in question 12), leading to persistent elevation. Option A is opposite of negative feedback function. Option C is wrong because the system needs to respond to future stressors. Option D is opposite—negative feedback decreases production.

#### **16. B - Why gambling persists despite losses—unpredictable rewards create persistent behavior**

The passage states "Variable-ratio schedules (reinforcement after unpredictable number of responses) produce the highest response rates with greatest extinction resistance—explaining gambling persistence." The data confirms variable-ratio produced 200 pecks during extinction compared to 15 (continuous) and 80 (fixed-ratio). Slot machines and other gambling operate on variable-ratio schedules: players cannot predict which pull will win, but wins occur often enough to maintain behavior. This schedule is so effective because: (1) the unpredictability prevents the learner from detecting when reinforcement has stopped (extinction), and (2) each response could potentially be the one that's reinforced, motivating continued responding. This explains why gambling problems are difficult to treat—the learning history on variable-ratio creates extremely persistent behavior that continues long after it stops being profitable. Option A is wrong because continuous reinforcement actually produces the fastest extinction. Option C is

wrong because variable-ratio learning is slowest (40 trials vs. 20 for continuous). Option D is irrelevant to the question.

### **17. B - Behaviors generalized and maintained through secondary reinforcement mechanisms**

The passage notes that after token economy removal, "Behaviors decreased but remained 30% above original baseline," indicating partial maintenance. This suggests several processes: (1) Some behaviors became intrinsically reinforcing or maintained by natural consequences (social approval, feeling better), (2) Secondary reinforcers (social praise from staff) that were paired with tokens during the program retained some reinforcing value, and (3) Stimulus control—behaviors became associated with the setting and continued partially even without explicit tokens. The passage discusses how "secondary reinforcers acquire value through association with primary reinforcers via classical conditioning," and the token economy likely established multiple conditioned reinforcers. Complete return to baseline would suggest no learning occurred; maintenance above baseline indicates that the intervention produced lasting changes beyond simple contingency control. Option A is wrong because there were lasting effects. Option C is wrong because 30% maintenance demonstrates some permanence. Option D is wrong because tokens are secondary, not primary reinforcers.

### **18. B - Systematic application of reinforcement and shaping effectively teaches complex skills**

The passage describes ABA as using "breaking skills into small steps, using prompting and fading, and providing immediate reinforcement" and states "intensive early ABA significantly improves outcomes." The data shows dramatic effects: ABA group averaged 25-point IQ increase versus 5 points for control, and 47% achieved normal educational placement versus 10%. This demonstrates that operant principles—breaking complex behaviors into component parts, shaping through successive approximations, immediate reinforcement for correct responses, prompting and fading to promote independence—can effectively teach skills that don't develop spontaneously in autism. The intensity matters (40 hrs/week vs. 10 hrs/week) because learning requires sufficient practice opportunities. Critics debate whether ABA addresses underlying deficits versus teaching specific behaviors, but the data clearly shows it produces measurable improvements in functioning. Option A is contradicted by results. Option C is wrong because individual differences exist but group effects are clear. Option D is contradicted by the intensity comparison showing dose-response relationship.

### **19. B - External rewards can undermine intrinsic motivation when applied to inherently enjoyable activities**

The overjustification effect, demonstrated when Group A's drawing time decreased 50% after rewards were removed while Group B remained stable, shows that providing external rewards for intrinsically motivated activities can reduce inherent interest. The passage states this occurs when "external rewards decrease intrinsic interest in inherently enjoyable activities." The mechanism involves attribution: children initially drew because it was fun (intrinsic motivation), but when rewards were introduced, they began attributing their behavior to the external reward rather than inherent enjoyment (external motivation). When rewards were removed, they had lost touch with their intrinsic motivation. This has important educational implications: excessive use of rewards for inherently interesting activities (reading, learning) may backfire. However, rewards remain effective for initially uninteresting activities or when used appropriately (informational feedback rather than controlling). Option A is wrong because rewards

sometimes undermine motivation. Option C is unsupported. Option D is contradicted by extensive data on reinforcement effects.

**20. B - It involves removing an aversive stimulus rather than adding a pleasant one, but both increase behavior**

The passage carefully defines: "Positive reinforcement increases behavior by adding a pleasant stimulus; negative reinforcement increases behavior by removing an aversive stimulus. Both increase behavior frequency but through different mechanisms." This is a common source of confusion—the terms "positive" and "negative" refer to whether something is added or removed (mathematical sense), not whether the experience is good or bad. Examples: Positive reinforcement = giving a child praise for good grades (add something pleasant). Negative reinforcement = taking aspirin to remove a headache (remove something aversive)—you're more likely to take aspirin in the future because it removes pain. Both processes strengthen behavior, just through different operations. Option A is the key misconception—negative reinforcement increases behavior just like positive reinforcement. Option C makes an empirical claim not supported by research—both are effective depending on circumstances. Option D confuses negative reinforcement with punishment.

**21. B - Some semantic processing of unattended information occurs, especially for personally relevant stimuli**

The passage discusses the cocktail party effect: "hearing one's name in unattended conversation—challenges pure early selection, suggesting some semantic processing occurs for unattended stimuli." The data shows that while 95% couldn't report any left ear content (supporting selective filtering), 33% noticed their own name when inserted in the unattended ear. Names are semantically meaningful and personally relevant, so noticing them requires semantic-level processing, not just physical feature analysis. This contradicts pure early selection models like Broadbent's filter theory, which proposed that unattended information is blocked before semantic processing. Late selection models better account for this: all information receives some semantic analysis, with attention determining what enters awareness. The brain appears to monitor unattended channels for important information (like your name, or "fire!") even while focused elsewhere. Option A is contradicted by name detection. Option C is dismissive. Option D is wrong because name recognition requires semantic processing (knowing what your name means), not just acoustic feature detection.

**22. B - Parallel, pre-attentive processing for simple feature differences**

The passage describes how in pop-out search (Condition A), search time was "~500ms regardless of number of distractors (parallel processing)" contrasted with conjunction search (Condition B) where "search time increased ~40ms per distractor (serial processing)." When the target differs from distractors by a single simple feature (color—red among green), the visual system processes all items simultaneously (in parallel) across the visual field, and the target "pops out" automatically without needing attention focused on each item sequentially. This pre-attentive processing is fast and capacity-unlimited for simple features. In contrast, conjunction search (finding a target defined by a combination of features, like red and vertical) requires attention to bind features together, so items must be examined serially. This distinction supports feature integration theory. The flat function (no effect of number of distractors) is the key signature of parallel processing. Option A describes serial processing (conjunction search). Option C

is nonsensical. Option D is wrong because attention is necessary for conjunction search, just not for simple feature detection.

**23. B - Without attention directed to an event, even obvious stimuli may not reach awareness**

Inattention blindness, dramatically demonstrated by only 50% noticing the gorilla during the counting task (while 95% noticed when just watching), shows that attention is not merely a highlighter that enhances already-conscious stimuli but a gateway to consciousness itself. The gorilla was fully visible and objectively obvious (95% noticed when not counting), but when attention was engaged by the counting task, it literally didn't reach conscious awareness for half the participants. The passage states this illustrates that "without attention directed to an event, even obvious stimuli may not reach awareness." This has practical implications: drivers on cell phones may miss pedestrians or traffic signals not because of impaired vision but because attention is elsewhere (as shown in the driving data in question 24). It challenges the intuition that we consciously see everything in our visual field—we actually see what we attend to. Option A is contradicted by all the research showing attention is limited. Option C is absurd. Option D is wrong because the counting specifically directed attention away from the gorilla.

**24. B - Conversation requires verbal-cognitive resources that compete with driving's attentional demands**

The passage explains "dual-task interference occurs when tasks compete for the same limited resource" and describes multiple resource theory: "separate pools for verbal versus spatial processing." The data shows cell phone conversation impaired driving significantly (reaction time +150ms, 18% miss rate) compared to podcast listening (+20ms, 4% miss rate) or no task. Conversation requires verbal-cognitive resources: understanding speech, formulating responses, holding conversational context in working memory—the same resources needed for driving-related cognition like route planning, interpreting traffic patterns, and decision-making. Podcast listening is more passive and requires fewer cognitive resources, allowing more to remain available for driving. Notably, this suggests hands-free cell phone use isn't much safer than handheld—the problem isn't manual manipulation but cognitive demand. Option A is contradicted by the data showing different effects. Option C is absurd. Option D is wrong because conversation and passive listening differ in cognitive demands.

**25. A - The right parietal cortex is critical for spatial attention, especially to contralateral space**

The passage states "right parietal damage causes left-sided neglect (inability to attend to left space)" and describes attention networks including "the frontal eye fields and intraparietal sulcus (spatial orienting)." Hemispatial neglect is a striking syndrome where patients ignore the contralesional (opposite side) space: they may eat only the right side of a plate, draw only the right half of objects, or fail to attend to people on their left. This isn't blindness (visual fields may be intact) but an attention deficit—they don't orient attention to that space. The right parietal cortex appears dominant for spatial attention to both sides, while left parietal mainly attends to right space, explaining why right parietal damage causes severe neglect while left damage causes milder neglect. This neurological evidence demonstrates that attention has clear neural substrates in parietal and frontal regions. Option B is contradicted by this and other evidence. Option C is incomplete—vision involves occipital cortex but attention to visual space involves parietal. Option D is wrong because neglect is specifically an attention deficit, not motor impairment (though motor neglect can co-occur).

## **26. B - Resources and stressors associated with SES influence health across the entire spectrum**

The passage emphasizes that "The SES-health gradient is not merely a poverty threshold effect—health improves continuously across the entire SES spectrum, from poorest to wealthiest." The data demonstrates this strikingly: mortality rates decline at each successively higher income quintile (12.3 → 9.8 → 7.6 → 6.1 → 4.2 per 1000), with the gradient existing even between upper-middle and upper class. If only poverty mattered (option A), we'd see a threshold—health would be poor for those in poverty and equivalent for everyone above poverty. Instead, the linear gradient suggests that resources (healthcare access, nutrition, housing quality, stress levels, sense of control) and risks vary continuously with SES, affecting health at every level. Even comparing people with \$80k vs. \$120k annual income shows health differences. This is important theoretically because it suggests SES operates through multiple mechanisms that scale with resources, not just presence/absence of deprivation. Option C is backward. Option D is contradicted by extensive evidence.

## **27. B - Factors beyond SES, including discrimination and residential segregation, contribute to racial health disparities**

The passage states "African Americans experience higher rates of hypertension, diabetes, infant mortality, and lower life expectancy compared to White Americans, even after controlling for SES," and explains this partly through "chronic stress from discrimination" and "residential segregation concentrates poverty and limits access to quality healthcare, healthy foods, and safe environments." The mortality data shows a 3.9-year Black-White life expectancy gap overall, but this varies dramatically by neighborhood (5.2-year gap between high and low SES neighborhoods for Black Americans). If disparities were purely about individual SES, they should disappear after controlling for SES. The persistence indicates additional factors: weathering from chronic discrimination stress, residential segregation creating different environmental exposures, healthcare system bias (shown in the pain management data), and potential gene-environment interactions. This doesn't mean genetics are irrelevant but that social factors account for substantial portions of observed differences. Option A is too deterministic and reductionist. Option C is irrelevant. Option D denies documented reality.

## **28. B - Chronic stress from discrimination contributes to poor health through psychophysiological pathways**

The passage describes the "weathering hypothesis" that "accumulated stress from racism accelerates biological aging (allostatic load)" and notes that discrimination effects remained "after controlling for SES, health behaviors." The data shows people reporting high discrimination had 2.1× higher hypertension and 1.8× higher depression. The mechanisms likely involve chronic HPA axis activation (discussed in Passage 3), elevated cortisol and inflammation, and physiological wear-and-tear from repeated stress responses. Discrimination is a unique stressor—chronic, unpredictable, and uncontrollable, affecting fundamental identity. The health effects aren't merely about conscious distress but involve unconscious physiological processes: blood pressure reactivity, immune function, metabolic dysregulation. The finding that effects persist after controlling for SES and behaviors suggests direct psychophysiological pathways, not merely that discrimination leads to poverty or unhealthy behaviors. Option A is contradicted by evidence. Option C is irrelevant to the finding. Option D is too deterministic—genetic predisposition interacts with environmental stress.

### **29. B - Implicit bias affects clinical treatment decisions, creating disparities in care quality**

The passage states "implicit bias affects provider-patient interactions and treatment decisions" and notes "racial minorities receive less aggressive pain management, fewer cardiac procedures, and lower quality care even within the same healthcare systems, suggesting bias affects clinical decision-making beyond access issues." The emergency department data shows significant disparities: 74% of White patients received opioid analgesia versus 57% Black and 55% Hispanic patients, despite similar pain severity ratings. Since patients presented to the same emergency departments with the same condition and similar pain levels, access isn't the explanation—something about clinical decision-making differed by patient race. Implicit bias (unconscious stereotypes) may lead providers to underestimate pain in minority patients, distrust their reports, or have different prescribing thresholds. This is concerning because it indicates disparities persist even when access barriers are removed. Option A makes unsupported claims about biological differences in pain. Option C is contradicted by the data showing unequal distribution. Option D represents an idealized view contradicted by extensive research on implicit bias.

### **30. B - Addressing social determinants through community-level interventions can reduce health disparities**

The passage argues "Understanding these social determinants is essential for addressing health inequalities and improving population health." The intervention program tested this by addressing multiple social determinants: community health workers (healthcare access, health literacy), improved transit (reducing transportation barriers), and food assistance (addressing food insecurity). Results showed substantial improvements: 28% better diabetes control and 35% fewer ER visits in intervention neighborhoods versus minimal changes in controls. This demonstrates that health problems with apparent medical solutions (diabetes, ER overuse) actually respond to social interventions addressing root causes. Rather than just treating diabetes with medication (medical model), addressing food access, transportation, and healthcare navigation (social determinants model) produces better outcomes. This supports policies addressing housing, education, economic opportunity, and neighborhood resources as health interventions. Option A represents overly individualistic thinking contradicted by the intervention's success. Option C is extreme. Option D is contradicted by extensive research linking neighborhood characteristics to health.

### **31. B - The amygdala is specifically critical for fear processing and fear learning**

The passage states "The amygdala, particularly its basolateral complex, plays a central role in emotional learning and fear processing" and notes it "enables rapid 'low road' emotional responses" to threats. Patient S.M.'s profile demonstrates selective impairment: complete inability to recognize fear in faces, failure to show fear conditioning (no conditioned response to tone paired with loud noise), and inappropriate approach to dangerous situations—yet normal recognition of other emotions (happiness, sadness, disgust). This double dissociation (fear impaired, other emotions intact) shows the amygdala's specificity for fear rather than emotions generally. If the amygdala processed all emotions equally (option A), S.M. should show deficits across all emotions. The selective fear deficit, combined with fear conditioning deficits, demonstrates the amygdala's specialized role in threat detection and fear learning. This makes evolutionary sense—rapid fear responses to threats have obvious survival value. Option C is absurd. Option D is wrong because the data shows clear specialization.

### **32. B - The amygdala modulates memory consolidation for emotional events, enhancing their retention**

The passage states "The amygdala also modulates memory consolidation for emotional events—arousal-enhanced memory. Emotionally arousing events activate amygdala, which modulates hippocampal consolidation, explaining why emotional memories are vivid and persistent." The data shows 78% recall of emotional pictures versus 42% for neutral, with strong amygdala activation during encoding of emotional pictures predicting later memory. The amygdala doesn't store memories itself but modulates the hippocampal memory system—when emotional arousal activates the amygdala, it signals the hippocampus that "this is important, consolidate strongly." This explains flashbulb memories (vivid memories of emotionally significant events like 9/11) and why we remember emotionally charged events better than neutral ones. This has adaptive value: remembering threats and rewards better than neutral information improves survival. The correlation between encoding-phase amygdala activation and later memory provides strong evidence for this modulatory role. Option A is opposite. Option C is contradicted by the integration between systems. Option D is opposite.

### **33. B - Top-down prefrontal control can regulate amygdala-based emotional responses**

The passage explains "The prefrontal cortex exerts top-down control over the amygdala through inhibitory projections—the neural basis of emotion regulation." The reappraisal data demonstrates this mechanism: when participants used cognitive reappraisal ("Imagine less negative interpretation"), prefrontal activation increased while amygdala activation decreased, accompanied by 45% reduction in subjective negative emotion and reduced physiological arousal. This shows the prefrontal cortex can downregulate amygdala reactivity through cognitive control—interpreting stimuli differently reduces automatic emotional responses. This is the neural basis of emotion regulation strategies used in cognitive-behavioral therapy and other interventions. The inverse relationship (prefrontal up, amygdala down) suggests inhibitory connections. Damage to prefrontal regions impairs emotion regulation, leading to emotional lability. Option A is contradicted by evidence. Option C is wrong because effective regulation was demonstrated. Option D is wrong because the data shows clear interaction via inhibitory pathways.

### **34. B - Social pain recruits neural systems also involved in physical pain processing**

The passage states "The anterior cingulate cortex...activates during social exclusion (Cyberball paradigm), physical pain, and cognitive conflict, suggesting common neural substrates for social and physical pain." The Cyberball data shows that social exclusion (being excluded from virtual ball-toss game) activated anterior cingulate and insula—the same regions activated during physical pain—with activation correlating with self-reported distress. This overlap suggests social pain isn't merely metaphorical but involves actual pain processing systems. This makes evolutionary sense: social connection was critical for survival in ancestral environments, so exclusion from groups represented genuine threat. The brain may have co-opted physical pain systems to signal social threats. This explains why social rejection "hurts" and why social support helps with physical pain. Importantly, some studies show analgesics (acetaminophen) reduce social pain, further supporting overlapping mechanisms. Option A is contradicted by the shared activation. Option C trivializes documented experience. Option D is backward—physical pain has clear physiological and psychological components.

### **35. B - Rapid emotional responses to potential threats before detailed cortical processing**

The passage explains that "The amygdala enables rapid 'low road' emotional responses: thalamic input reaches the amygdala before cortical processing, allowing quick responses to potential threats before conscious recognition." This subcortical pathway (thalamus → amygdala) bypasses cortical processing, enabling reactions within ~12 milliseconds—much faster than the cortical "high road" (thalamus → visual cortex → amygdala) which takes ~300ms. This speed advantage is crucial for survival: beginning to jump away from a snake-like object before consciously identifying it as a snake versus a stick could save your life. The system can afford false alarms (reacting to sticks as if they're snakes) because the cost of false negatives (failing to react to actual snakes) is much higher. Consciousness and detailed analysis happen afterward via cortical processing. This two-pathway system balances speed (subcortical) with accuracy (cortical). Option A is wrong because the low road specifically operates pre-consciously. Option C is opposite—the point is responding before complete analysis. Option D is wrong because positive emotions use different pathways more.

**36. B - Dispositional attributions are strongly preferred even when situational explanations are obvious**

The fundamental attribution error (correspondence bias) describes the tendency to overestimate dispositional factors when explaining others' behavior. The passage notes this occurs despite situational information. The essay data demonstrates this strikingly: even though participants were explicitly told "Writer was randomly assigned position and required to argue it"—a clear situational explanation—78% still rated the writer's true attitudes as consistent with the essay position. This should be zero—if the position was assigned, it tells us nothing about actual attitudes. Yet participants made dispositional inferences despite obvious situational constraints. The small difference from control condition (85% vs. 78%) shows that situational information has minimal impact. This bias occurs because: (1) behavior is perceptually salient while situations are less visible, (2) cognitive efficiency favors dispositional explanations, and (3) cultural factors in individualistic societies emphasize personality over context. Option A is wrong because consideration was minimal. Option C is irrelevant. Option D is patronizing.

**37. B - Perspective influences attribution—people have different information and attention for own versus others' behavior**

The actor-observer bias shows asymmetry: 65% situational attributions for self ("I chose biology because of interesting courses") versus 62% dispositional for roommate ("She chose biology because she's science-oriented"). The passage explains: "This occurs because actors have more information about situational constraints on their own behavior and different attentional focus (actors attend to situation, observers attend to actor)." Actors experience the situations influencing their choices and remember past variations in their behavior across contexts, while observers see only the behavior without access to situational pressures or behavioral variability. Attention differs too: actors looking outward see situational features, while observers looking at actors see the actor as figure against situational background. The finding that people explain their past behavior more dispositionally (mentioned in passage) supports the attention/information explanation—with temporal distance, you become more like an observer of your past self. Option A is contradicted by the bias. Option C overstates. Option D is nonsensical.

**38. B - Protect self-esteem and maintain sense of control**

The self-serving bias involves taking credit for success (82% internal attributions) while blaming failure on external factors (74% external attributions). The passage states this "protects self-esteem and maintains sense of control." By attributing success to internal factors (ability, effort), people can feel good about themselves and maintain confidence in future success. By attributing failure to external factors (bad luck, test unreliability), people avoid self-blame and maintain self-esteem despite poor outcomes. The motivation isn't conscious deception but automatic self-protective cognition. This bias has adaptive aspects (maintaining motivation and self-confidence) but also costs (reducing accurate self-assessment and preventing learning from mistakes). The finding that depressed individuals show reversed patterns (question 39) supports the self-esteem protection function—when self-protective biases fail, depression may result or worsen. Option A is secondary effect. Option C overstates. Option D is too negative—the bias may impair learning but serves psychological functions.

### **39. B - Maladaptive attribution patterns contribute to maintaining depressive symptoms**

The passage states "depressed individuals show reversed pattern—attributing success externally and failure internally—contributing to depressive cognitive style." The data confirms: depressed participants attributed success externally (63%) but failure internally (78%)—opposite of non-depressed participants. This attribution pattern maintains depression: when success is attributed externally ("I succeeded because of luck"), it doesn't improve self-esteem or hopelessness; when failure is attributed internally to stable traits ("I failed because I'm incompetent"), it damages self-esteem and creates hopelessness about change. Beck's cognitive theory of depression identifies these attributions as key maintaining factors. The passage mentions "Attributional retraining programs teach adaptive attribution patterns to improve motivation and performance"—these CBT-based interventions help depressed individuals adopt healthier attribution patterns. Importantly, this pattern may be both cause and consequence of depression (bidirectional). Option A is contradicted by the data. Option C makes unsupported claim about accuracy. Option D is contradicted by clear relationships between attribution and mood.

### **40. B - People distort perceptions of victims to preserve belief that the world is fair**

The just-world hypothesis proposes that "people get what they deserve," providing sense of predictability and control but leading to victim-blaming. The passage states "When bad things happen to people, observers may attribute it to victims' actions or character to maintain belief in a just world." The victim derogation study provides experimental evidence: the innocent victim (Condition A—shocked for research purposes, not by choice) was rated less attractive and received less sympathy than the same victim in Condition B (volunteered). This defensive attribution occurs because acknowledging that bad things happen to innocent people is threatening—if victims did nothing to deserve their suffering, then the observer could also suffer randomly, creating anxiety about controllability. By subtly blaming victims ("she must be less worthy somehow"), observers maintain the comforting belief that they can avoid similar fates through their own choices. This has serious consequences: reduced help for people in poverty, crime victims, and illness sufferers. Option A is wrong because ratings were distorted. Option C represents the bias itself. Option D misses the mechanism.

### **41. B - Nativist theory—innate mechanisms guide acquisition rather than explicit instruction**

The passage discusses Chomsky's nativist theory proposing "an innate Language Acquisition Device (LAD) containing universal grammar" and presents as evidence that children acquire complex grammar

without explicit instruction. The "poverty of stimulus" data shows that explicit grammar corrections occur for only 3% of children's grammatical errors (with most corrections focusing on truth rather than form), yet children achieve grammatical competence by age 5. If language learning depended primarily on explicit instruction and correction (behaviorist theory, option A), children couldn't acquire grammar given this sparse corrective input. The nativist explanation is that innate grammatical principles guide children to extract rules from input—they're predisposed to notice patterns relevant to grammar and construct grammatical systems. The rapid acquisition across diverse language environments despite limited explicit teaching supports innate contributions to language learning. Option C is wrong because errors do occur during development. Option D is contradicted by the data showing minimal explicit teaching.

#### **42. B - Critical period hypothesis—language acquisition is constrained by developmental timing**

The passage states "The critical period hypothesis proposes that language acquisition must occur before puberty for native-like proficiency" with "proficiency decreases with age of acquisition after puberty." The Korean/Chinese adoptee data provides strong evidence: near-perfect grammaticality (98%) when adopted before age 3, declining linearly to 78% when adopted ages 13-16. This declining function demonstrates that language learning ability changes with age—the brain's plasticity for language acquisition decreases after early childhood, with particularly sharp declines after puberty. Similar patterns appear in other domains (phonological accent, syntax) though lexical learning remains more open-ended. The critical period likely reflects neural development: during early development, language areas are maximally plastic and can be shaped by input, but plasticity gradually closes, making late learning more difficult and less successful. Evidence from feral children (like Genie) and late second-language learners converges on this conclusion. Option A is contradicted by data. Option C is backward. Option D is irrelevant—the pattern holds within each language.

#### **43. B - Social-cognitive factors like shared attention facilitate word learning**

The passage's interactionist perspective "emphasizes caregiver input and social context" including "Joint attention—caregiver and child focusing on the same object—scaffolds word learning." The experimental data demonstrates this: 18-month-olds learned novel words after just 3 exposures when the adult looked at the target object while naming it (Condition A), but failed to learn even after 12 exposures when the adult looked away (Condition B). This shows word learning isn't simple association of sounds with objects in view (behaviorist prediction) but involves social understanding—children assume adults are naming what they're attending to, not arbitrary objects in the environment. This requires Theory of Mind precursors: understanding others' attention and intentions. Joint attention emerges around 9-12 months and scaffolds language learning throughout early development. This social-pragmatic account complements nativist grammar acquisition—social cognition for word learning, innate mechanisms for syntax. Option A is contradicted by the necessity of joint attention. Option C is absurd. Option D is wrong because the looking direction was the experimental manipulation.

#### **44. B - Language categories can influence perceptual discrimination through linguistic mediation**

The passage discusses linguistic relativity (Sapir-Whorf hypothesis), distinguishing strong version (linguistic determinism, rejected) from weak version (language influences some domains, supported). The Russian color discrimination data supports the weak version: Russian speakers showed 124ms faster reaction times when distinguishing blues crossing the goluboy/siniy boundary compared to within-

category blues, while English speakers (who call both "blue") showed no boundary effect. Critically, verbal interference eliminated this advantage, indicating linguistic mediation: Russians were unconsciously accessing linguistic categories ("those are different colors by name") to facilitate discrimination. However, the fact that interference eliminated the advantage shows the effect involves language use rather than permanent perceptual restructuring. Option A is contradicted by the boundary effect. Option C makes unfounded claim. Option D is too strong—the data shows language influences perception in specific ways, not that perception is entirely innate or entirely determined by language.

#### **45. B - Children actively impose grammatical structure, supporting innate grammatical capacity**

The passage presents creole languages as evidence for innate grammar: "Creole languages—fully grammatical languages created by children from pidgin input—demonstrate children's innate grammatical capacity." Pidgins are communication systems without full grammar, used when adults speaking different languages need to interact. Remarkably, when children grow up exposed to pidgins, they transform them into creoles—languages with complete grammatical systems including complex syntax, consistent word order, morphology, etc. This grammaticization happens in one generation and doesn't come from adult input (which was the ungrammatical pidgin). The only explanation is that children bring grammatical principles to language learning, actively imposing structure rather than passively absorbing input patterns. This is powerful evidence against pure empiricist accounts and supports Chomsky's proposal of innate universal grammar. Option A is contradicted by the creative restructuring. Option C is wrong because pidgins lack full grammar by definition. Option D is contradicted by successful creole creation.

#### **46. B - Substantial intergenerational persistence—poverty tends to reproduce across generations**

The passage discusses intergenerational mobility and presents striking data: 43% of children born to parents in the lowest income quintile remain in that quintile as adults, while only 4% rise to the highest quintile. If mobility were perfect (the American Dream ideal), each quintile should have 20% chance (random distribution). The strong diagonal (43% stay lowest, 40% stay highest) demonstrates substantial immobility—economic position substantially predicts children's adult position. The passage attributes this to multiple mechanisms: limited opportunities, inferior schools in low-income neighborhoods, lack of social capital, and compound disadvantages (mentioned as "advantages compound while disadvantages accumulate"). The fact that only 4% of those born in poverty reach the top quintile suggests that structural barriers limit mobility despite individual effort. This persistence of inequality across generations raises questions about meritocracy and equal opportunity. Option A is contradicted by data. Option C is wrong because the pattern is systematic. Option D is contradicted by the strong intergenerational correlation.

#### **47. B - Lower mobility in the U.S.—parental income more strongly predicts child's income**

The passage presents "correlation between father's and son's earnings (higher = less mobility)" comparing countries. Higher correlation means children's incomes more strongly reflect parents' incomes—less movement between quintiles, less mobility. The U.S. correlation (0.47) exceeds Denmark (0.15), Canada (0.19), Germany (0.32), and France (0.41), indicating lower mobility than these comparison countries. This contradicts the American Dream narrative that emphasizes opportunity and mobility. The higher U.S. correlation may reflect: greater inequality (larger gaps to cross), less social safety net, educational funding tied to property taxes (perpetuating neighborhood advantages), and weaker labor market institutions. Ironically, countries with more active government redistribution show higher mobility. Option A is

opposite. Option C is contradicted by the numerical differences. Option D is wrong because the correlation coefficient precisely measures mobility.

**48. B - Non-economic social assets transmitted through families create advantages independent of merit**

Bourdieu's cultural capital concept explains how upper-class advantages transmit across generations beyond money. The passage explains cultural capital as "non-financial social assets like education, speech patterns, cultural knowledge, and social skills that facilitate upward mobility." The data shows students' cultural activities (museum visits, classical music, reading) predicted by parental education/occupation, and these cultural activities predicted college attendance even controlling for academic achievement (2.1× higher college attendance for high cultural capital). Additionally, high cultural capital students received better grades for equivalent work quality—suggesting teacher bias favoring students who display "refined" cultural tastes and communication styles. This shows that class reproduction operates through subtle mechanisms: children from educated families inherit cultural knowledge, tastes, and dispositions that educational systems reward, providing advantages that appear meritocratic but actually reflect family background. Option A is contradicted by the "controlling for achievement" finding. Option C is wrong because cultural capital is clearly valuable in educational contexts. Option D is contradicted by the substantial group differences.

**49. B - Cross-cultural consensus exists about occupational value, though stratification systems differ**

The passage notes "Occupational prestige ranking" with physicians (86), lawyers (75), teachers (64), police (60), and retail workers (28), stating the "Pattern consistent across most societies despite different economic systems." This remarkable consistency suggests some universal basis for occupational evaluation—perhaps based on required education/training, power/autonomy, societal contribution, or income. However, the consistency doesn't mean the rankings are objectively correct or functionally necessary (as Davis-Moore thesis claims). Conflict theorists would argue the rankings reflect power relations and ideological justification. The consistency does demonstrate that prestige is a dimension of stratification distinct from income (teachers have moderate prestige but often low income relative to education). The cross-cultural pattern suggests cognitive or social psychological universals in status perception. Option A is wrong because consistency across cultures contradicts arbitrariness. Option C is obviously wrong. Option D is contradicted by the integration of prestige into stratification systems.

**50. B - Extreme concentration of resources at the top of the stratification system**

The passage presents wealth inequality data showing top 1% owns 32%, top 10% owns 70%, while bottom 50% owns just 2% of total wealth. This extreme concentration demonstrates high inequality—wealth is distributed very unevenly. For comparison, perfect equality would mean top 10% owns 10%, bottom 50% owns 50%. The passage notes "Wealth inequality increased substantially over 40 years," indicating growing concentration. Wealth (total assets minus debt) is more unequally distributed than income because it accumulates over time and generates returns—the rich can invest, earning passive income and compounding wealth, while the poor may have negative net worth. This concentration has implications: political power (wealth enables lobbying and campaign contributions), intergenerational advantage (inheritance and gifts transfer wealth), and social mobility (starting wealth affects opportunities). Conflict

theorists view this concentration as reflecting power relations more than productivity or merit. Option A is obviously wrong. Option C is absurd. Option D understates the societal implications.

### **51. B - Conservation**

Piaget's conservation tasks assess understanding that quantitative properties (number, volume, mass, length) remain constant despite perceptual changes. Classic demonstrations include: pouring liquid from a short, wide glass into a tall, narrow glass (same volume despite different appearance), spreading out a row of coins (same number despite different spacing), or rolling a ball of clay into a snake (same amount despite different shape). Preoperational children (ages 2-7) fail conservation, focusing on perceptual appearances (centration) and reasoning "the tall glass has more because it's taller." Concrete operational children (ages 7-11) achieve conservation, understanding that transformations don't change quantity—they can mentally reverse operations or recognize compensation (taller but narrower). Conservation is a watershed achievement indicating logical thinking. Option A (object permanence) is the understanding that objects continue existing when out of sight, achieved in infancy. Option C (animism) is attributing life to inanimate objects. Option D (centration) is focusing on one perceptual dimension while ignoring others—what prevents conservation.

### **52. A - Diffusion of responsibility**

The bystander effect, famously studied after Kitty Genovese's murder where 38 witnesses reportedly failed to help, describes decreased likelihood of helping when more bystanders are present. The explanation involves diffusion of responsibility: when multiple people witness an emergency, each feels less personal responsibility because responsibility is psychologically distributed across all present ("someone else will help"). In ambiguous situations, pluralistic ignorance also contributes: people look to others to define the situation, and when everyone appears calm (because everyone is looking at everyone else), the situation seems less serious. Additionally, evaluation apprehension (fear of appearing foolish if misinterpreting the situation) and audience inhibition (self-consciousness about helping publicly) contribute. Experimental studies (Darley & Latané) show that increasing bystander number dramatically reduces helping rates and increases response latency. Option B is backward—more people reduces helping. Option C might limit helping in some situations but doesn't explain the psychological mechanism. Option D (groupthink) is a different phenomenon about group decision-making.

### **53. C - Dopamine**

Dopamine is the primary neurotransmitter in reward and motivation pathways, particularly the mesolimbic pathway from ventral tegmental area to nucleus accumbens. This pathway signals reward prediction and reward receipt, motivating approach behavior. All major drugs of abuse increase dopamine: cocaine and amphetamines increase dopamine directly, alcohol and opioids do so indirectly. Repeated drug use causes neuroadaptations: dopamine receptors downregulate, natural rewards become less satisfying (anhedonia), and the drug becomes necessary for normal dopamine function—the neurobiological basis of addiction. Dopamine isn't about pleasure per se but about "wanting" (incentive salience) versus "liking." Parkinson's disease (dopamine cell loss) causes movement problems and reduced motivation. Antipsychotic medications block dopamine (particularly D2 receptors) to reduce positive symptoms of schizophrenia. Option A (serotonin) is involved in mood, sleep, and appetite. Option B (GABA) is the main inhibitory neurotransmitter. Option D (acetylcholine) is involved in muscle control and memory.

#### **54. A - Improved performance on simple/well-learned tasks in the presence of others**

Social facilitation (Zajonc) describes the effect of others' presence on performance: presence of others enhances performance on simple or well-practiced tasks but impairs performance on difficult or novel tasks. The mechanism involves arousal: others' presence increases arousal (evaluation apprehension, distraction, or mere presence), and arousal enhances dominant responses—the most likely behaviors. For well-learned tasks, dominant responses are correct (facilitating performance), but for difficult tasks, dominant responses are often errors (impairing performance). Examples: experienced pool players perform better with audience, while novices perform worse; simple math improves with observers, while complex problem-solving suffers. This explains why practicing alone but performing publicly can be optimal. Option B is half right (impairment on difficult tasks) but wrong about simple tasks. Option C describes helping behavior. Option D (social loafing) is decreased individual effort in groups—related but distinct phenomenon.

#### **55. B - Obsessive-compulsive disorder (OCD)**

OCD features recurrent, intrusive thoughts (obsessions) that cause anxiety, and repetitive behaviors or mental acts (compulsions) performed to reduce the anxiety. Common obsessions include contamination fears, doubting (did I lock the door?), need for symmetry, and taboo thoughts. Compulsions include washing, checking, counting, and arranging. The person typically recognizes the thoughts and behaviors as excessive or unreasonable but feels unable to control them. DSM-5 criteria require time-consuming obsessions/compulsions (>1 hour/day) causing significant distress or impairment. The disorder involves orbitofrontal cortex, anterior cingulate, and basal ganglia dysfunction. Treatment includes exposure and response prevention (ERP) therapy and SSRIs. Option A (generalized anxiety disorder) involves excessive worry but not obsessions/compulsions. Option C (schizophrenia) involves psychosis (hallucinations, delusions). Option D (major depression) involves persistent low mood and anhedonia.

#### **56. B - Physiological, safety, love/belonging, and esteem needs**

Maslow's hierarchy of needs proposes a pyramid with physiological needs (food, water, sleep) at the base, followed by safety (security, stability), love/belonging (relationships, connection), esteem (respect, achievement), and self-actualization (realizing potential, creativity) at the peak. The theory proposes that lower needs must be substantially satisfied before higher needs become motivating—a starving person focuses on food, not self-actualization. While hierarchical progression is generally true, exceptions exist: artists may pursue creative self-actualization despite poverty (violating the hierarchy), and some cultures prioritize belonging over individual achievement. Research support is mixed—the hierarchy captures important motivational principles but may be more fluid than the strict pyramid suggests. Self-actualization (peak experiences, growth, authenticity) represents full human potential but requires solid foundation in more basic needs. Option A is too limited. Option C contradicts the hierarchical premise. Option D is insufficient.

#### **57. A - Groups one belongs to (ingroup) versus groups one doesn't belong to (outgroup)**

Ingroups are social groups with which individuals identify and feel membership ("we," "us"), while outgroups are groups individuals don't belong to ("they," "them"). These categorizations are psychological and context-dependent—the same person is an ingroup member in one context (gender, nationality,

occupation) and outgroup member in another. Ingroup/outgroup distinctions drive multiple phenomena: ingroup favoritism (preferring and helping ingroup members), outgroup homogeneity effect (seeing outgroup members as more similar than ingroup members—"they all look alike"), and stereotyping. Social identity theory (Passage 2) explains how ingroup/outgroup dynamics serve self-esteem through favorable social comparison. The minimal group paradigm shows these dynamics emerge from mere categorization. Option B is literal misinterpretation. Option C is sometimes but not necessarily true (numerical majority/minority is distinct from ingroup/outgroup). Option D is too specific—groups can be neither friends nor enemies.

### **58. C - Executive functions including planning, impulse control, and decision-making**

The prefrontal cortex, particularly dorsolateral and ventromedial regions, implements executive functions: working memory, cognitive flexibility, planning, inhibitory control, decision-making, and self-regulation. This region continues developing into the mid-20s through synaptic pruning and myelination, explaining why adolescents show incomplete executive control: increased risk-taking, impulsivity, susceptibility to peer influence, and difficulty with long-term planning. The protracted development has legal and policy implications: should juveniles be held to adult standards when their brains aren't fully mature? Damage to prefrontal cortex causes dysexecutive syndrome: impaired planning, perseveration, disinhibition, and poor decision-making despite intact intellect. The famous case of Phineas Gage (railroad spike through prefrontal cortex) demonstrated this dissociation. Option A (basic reflexes) involves brainstem/spinal cord. Option B (vision/hearing) involves occipital/temporal lobes. Option D (heart rate) involves brainstem autonomic centers.

### **59. B - Defining and treating conditions as medical problems that were not previously considered medical**

Medicalization is the social process by which non-medical conditions become defined and treated as medical problems requiring medical intervention. Examples include: childbirth (shifted from home to hospital), aging (anti-aging medicine), shyness (social anxiety disorder), unhappiness (depression), and normal grief (prolonged grief disorder proposed for DSM). Medicalization can be beneficial (legitimizing suffering, providing treatment, reducing stigma) but also problematic (pathologizing normal variation, increasing medical control, pharmaceutical industry influence, ignoring social causes). The process involves physicians, pharmaceutical companies, insurance, and cultural factors. Demedicalization also occurs (homosexuality removed from DSM). Medicalization doesn't necessarily mean conditions are "not real"—suffering is real—but questions whether medical framework is always appropriate. Option A describes healthcare access. Option C describes infrastructure. Option D describes workforce training.