MCQs on Antibodies, Immunoglobulin Classes, and Antigen Receptor Genetics – Medical Immunology

Antibody Structure

1	. Which j	nart (of tha	antihad	v mol	acula	datarr	ninac	ite a	ntigan-	hindi	na c	nacific	·itx/2
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- A) Heavy chain constant region
- B) Light chain constant region
- C) Variable regions of both heavy and light chains
- D) Fc region
- E) Hinge region

2. The Fc region of an antibody is responsible for:

- A) Antigen binding
- B) Complement activation and Fc receptor binding
- C) Somatic recombination
- D) Affinity maturation
- E) Antigen presentation

3. Which type of bond stabilizes the structure between antibody heavy and light chains?

- A) Hydrogen bonds
- B) Ionic bonds
- C) Peptide bonds
- D) Disulfide bonds
- E) Hydrophobic interactions

4. The hinge region of IgG antibodies provides:

- A) Complement fixation site
- B) Antigen specificity
- C) Flexibility between Fab and Fc regions

	D) Cross-linking with antigen
	E) Heavy-light chain association
	atient's serum electrophoresis shows a single monoclonal IgG peak. This finding ypical of:
	A) Normal immune response
	B) Multiple myeloma
	C) Common variable immunodeficiency
	D) Chronic infection
	E) Polyclonal activation
Antib	ody Classes and Effector Functions
6. Wh fetu	ich antibody class can cross the placenta to provide passive immunity to the is?
	A) IgA
	B) IgD
	C) IgE
	D) IgG
	E) IgM
7. Wh	ich antibody class predominates in mucosal secretions such as saliva and tears?
	A) IgA
	B) IgE
	C) IgG
	D) IgM
	E) IgD
8. IgM	is particularly effective in complement activation because:
	A) It has high affinity
	B) It binds Fc receptors strongly
	C) It is a pentamer with multiple C1q-binding sites
	D) It is the smallest antibody

E) It persists long-term	
9. Which antibody is most responsible for allergic reactions and defense again helminths?	ıst
A) IgA	
B) IgD	
C) IgE	
D) IgG	
E) IgM	
10. In a patient with selective IgA deficiency, which of the following is expected	d?
A) Increased mucosal infections	
B) Defective complement activation	
C) Impaired opsonization	
D) Increased cytotoxic T-cell activity	
E) Autoimmune hemolysis	
Primary and Secondary Immune Responses	
1. Which immunoglobulin class predominates in the primary immune respon	se?
A) IgA	
B) IgD	
C) IgE	
D) IgG	
E) IgM	
2. Which of the following best describes the secondary immune response?	
A) Slow and low affinity	
B) Mediated by naïve B cells	
C) Rapid and high-affinity IgG response	
D) Involves only T cells	
E) Independent of memory cells	

3. The lag phase before detectable antibody levels is shorter in the secondary response because:

- A) T cells are more active
- B) Memory B cells rapidly respond
- C) Complement is faster
- D) Antigen is larger
- E) IgM is pre-formed

4. A vaccine booster is given to:

- A) Enhance innate immunity
- B) Induce tolerance
- C) Stimulate memory B cells for stronger response
- D) Prevent autoimmunity
- E) Inhibit hypersensitivity

5. In early infection, IgM predominates because:

- A) It has higher affinity
- B) It is secreted first by plasma cells
- C) It crosses placenta
- D) It binds to Fc receptors
- E) It has longest half-life

Antigen Receptor Genetics and Somatic Mutations

1. V(D)J recombination occurs during:

- A) T-cell activation
- B) B-cell antigen encounter
- C) Lymphocyte development in primary lymphoid organs
- D) Somatic mutation
- E) Class switching

2. The enzyme responsible for DNA cleavage during V(D)J recombination is:

A) DNA polymerase
B) RAG1/2 recombinase
C) AID enzyme
D) DNA ligase IV
E) Terminal transferase
3. Somatic hypermutation occurs in:
A) Bone marrow
B) Thymus
C) Germinal centers
D) Liver
E) Red pulp of spleen
4. Class switching of immunoglobulin genes is mediated by:
A) RAG enzymes
B) DNA helicase
C) Activation-induced deaminase (AID)
D) RNA polymerase II
E) Exonuclease
5. A patient with AID deficiency would most likely show:
A) Hyper-IgM syndrome
B) Increased IgE levels
C) Absence of T-cell receptors
D) Loss of complement activation
E) Reduced IgM
6. Which enzyme seals the DNA breaks during V(D)J recombination?
A) DNA ligase IV
B) Polymerase δ

C) RNase H
D) RAG2
E) TdT
7. Junctional diversity in antibodies arises mainly due to:
A) Addition or deletion of nucleotides at gene joins
B) Class switching
C) Somatic hypermutation
D) RAG protein inactivation
E) IgM pentamer formation
8. Terminal deoxynucleotidyl transferase (TdT) increases diversity by:
A) Adding nucleotides to VDJ junctions
B) Splicing mRNA
C) Repairing thymine dimers
D) Degrading unused DNA
E) Methylating CpG islands
9. Affinity maturation of antibodies occurs due to:
A) Class switching
B) Somatic hypermutation and selection of high-affinity clones
C) RAG enzyme action
D) Complement activation
E) IL-2 signaling
10. Mutation in RAG1/2 genes causes:
A) SCID phenotype due to failure of antigen receptor gene rearrangement
B) Increased antibody affinity
C) Hyper-IgM syndrome
D) Complement overactivation

E) IgE-mediated allergy

Answers and Explanations

Antibody Structure

1. Answer: C

Explanation: The antigen-binding site (paratope) is formed by the variable regions of both heavy and light chains.

2. Answer: B

Explanation: The Fc region mediates effector functions such as complement activation and binding to Fc receptors on immune cells.

3. Answer: D

Explanation: Disulfide bonds stabilize the antibody's quaternary structure by linking heavy and light chains.

4. Answer: C

Explanation: The hinge region gives flexibility between Fab arms, allowing simultaneous binding to multiple epitopes.

5. Answer: B

Explanation: A monoclonal immunoglobulin peak (M-protein) indicates plasma cell proliferation, as in multiple myeloma.

Antibody Classes and Effector Functions

1. Answer: D

Explanation: IgG is the only antibody class that crosses the placenta through FcRn receptors.

2. Answer: A

Explanation: Secretory IgA provides mucosal defense by preventing microbial adherence.

3. Answer: C

Explanation: The pentameric structure of IgM allows strong activation of the classical complement pathway.

4. Answer: C

Explanation: IgE binds FceRI on mast cells and basophils, triggering histamine release during allergy and anti-helminth responses.

5. Answer: A

Explanation: IgA deficiency causes recurrent mucosal infections, especially of the respiratory and gastrointestinal tracts.

Primary and Secondary Immune Responses

1. Answer: E

Explanation: IgM is produced first during a primary immune response by naïve B cells.

2. Answer: C

Explanation: Memory B cells generate a rapid, high-affinity IgG response upon re-exposure to antigen.

3. Answer: B

Explanation: Memory B cells differentiate quickly into plasma cells upon re-exposure to antigen.

4. Answer: C

Explanation: Booster doses re-activate memory B cells to strengthen and prolong the immune response.

5. Answer: B

Explanation: IgM is secreted first by activated plasma cells before class switching occurs.

Antigen Receptor Genetics and Somatic Mutations

1. Answer: C

Explanation: V(D)J recombination assembles variable region genes in developing lymphocytes within bone marrow and thymus.

2. Answer: B

Explanation: RAG1/2 mediates cleavage at recombination signal sequences to enable gene segment joining.

3. Answer: C

Explanation: Somatic hypermutation takes place in germinal centers to enhance antibody affinity after antigen stimulation.

4. Answer: C

Explanation: AID enzyme catalyzes cytidine deamination in switch regions, initiating recombination and isotype switching.

5. Answer: A

Explanation: AID deficiency results in hyper-IgM syndrome due to inability to perform class switching and somatic hypermutation.

6. Answer: A

Explanation: DNA ligase IV completes the recombination process by sealing DNA ends after RAG cleavage.

7. Answer: A

Explanation: Random addition/deletion of nucleotides at junctions increases receptor diversity.

8. Answer: A

Explanation: TdT randomly adds nucleotides at the V(D)J junction, enhancing antigen receptor diversity.

9. Answer: B

Explanation: Somatic hypermutation introduces point mutations; B cells with higher affinity are selected in germinal centers.

10. Answer: A

Explanation: RAG mutations prevent B- and T-cell receptor formation, leading to severe combined immunodeficiency (SCID).