QUESTION

What is the function of IL-8?

A **Chemotaxis** CORRECT

B Fever

C Lymphocyte proliferation

D TH1 activation

RIGHT ANSWER

OK **A. Chemotaxis**

WHY THIS IS RIGHT

IL-8 is a potent neutrophil chemoattractant, guiding them toward sites of acute inflammation. It also activates neutrophils, enhancing their phagocytic function. Other options do not match its primary role-IL-1/IL-6 cause fever, IL-2 drives lymphocyte proliferation, and IL-12 promotes TH1 activation.

QUESTION

The Nobel Prize for Physiology and Medicine in 2025 was given for:

A Receptor for touch and temperature

B Discovery of genomes of extinct hominins

C Discovery of tissue mRNA and its role in post-transcriptional modification

D **Peripheral immune tolerance** CORRECT

RIGHT ANSWER

OK

D. Peripheral immune tolerance

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Which of the following antibodies is the most reliable marker for diagnosing an acute infection?

A IgA

B IgG

C IgE

D **IgM** CORRECT

OK

RIGHT ANSWER

D. IgM

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

A 22-year-old woman presents to the emergency department with high-grade fever, diffuse erythematous rash, hypotension, and shock. She started menstruating 3 days ago and reports using superabsorbent tampons. Which of the following is the most likely reason responsible for her condition?

A Lipoteichoic acid

B **Superantigen like TSST-1** CORRECT

C Hypovolemia

D Exotoxin

OK RIGHT ANSWER

B. Superantigen like TSST-1

WHY THIS IS RIGHT

The presentation is classic for toxic shock syndrome associated with tampon use.
It is caused by TSST-1 produced by *Staphylococcus aureus*, which acts as a superantigen.
Superantigens cause non-specific activation of a large number of T cells with massive cytokine release.
This cytokine storm leads to fever, rash, hypotension, and shock.
TSST-1 (superantigen)
↓
Bridges MHC II and TCR (non-specific)
↓
Massive T-cell activation
↓
Cytokine storm:
• IL-1 -> fever
• IL-2 -> T-cell proliferation
• TNF-α -> hypotension, shock
↓
Toxic Shock Syndrome

Feature	Normal Antigen	Superantigen
Processing	Required	Not required
T-cell activation	Specific (\~0.01%)	Massive (20-30%)
Binding	Specific peptide-MHC	Direct MHC-TCR binding
Cytokines	Controlled	Explosive (storm)

QUESTION

A patient has adult respiratory distress syndrome. Which of the following is the action of IL-8 in the pathogenesis in this patient?

A Endothelial injury

B **Neutrophil activation** CORRECT

C Fibrin deposition

D Macrophage activation

OK RIGHT ANSWER

B. Neutrophil activation

WHY THIS IS RIGHT

Injury (sepsis, trauma, infection)
↓
Cytokine release (TNF, IL-1, IL-8)
↓
IL-8 recruits neutrophils
↓
Neutrophil activation
↓
Release of ROS + proteases
↓
Alveolar damage
↓
ARDS

IL-8 is a potent chemokine responsible for recruitment and activation of neutrophils.
In ARDS, IL-8-mediated neutrophil activation leads to release of proteases and reactive oxygen species. This contributes to alveolar-capillary membrane damage and increased vascular permeability.
Endothelial injury and fibrin deposition are downstream effects, not the primary action of IL-8.

Cytokine	Function
IL-8	Neutrophil recruitment & activation
TNF-α	Inflammation
IL-1	Fever, inflammation
IFN-γ	Macrophage activation

QUESTION

All of the following interactions between T cells and APC (Antigen-Presenting Cell) are required for T cell activation/differentiation except:

A T cell receptor and MHC II

B CD28 and CD80

C **PD-PDL1** CORRECT

D Cytokine secretion

OK RIGHT ANSWER

C. PD-PDL1

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

Which of the following is not an example of passive immunity?

A **Toxoid** CORRECT

B Antitoxin

C IgA

D Monoclonal antibodies

OK RIGHT ANSWER

A. Toxoid

WHY THIS IS RIGHT

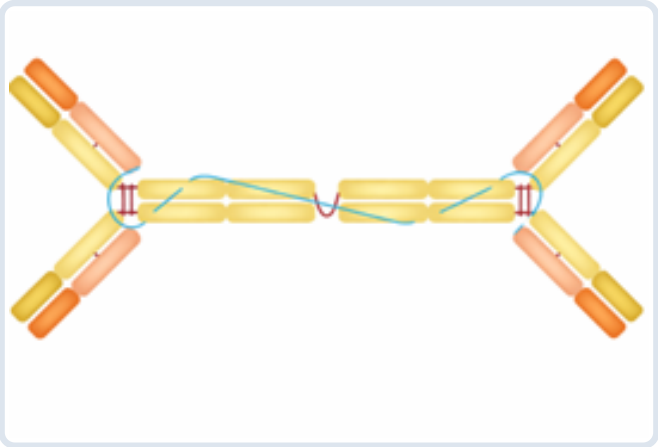
Passive immunity involves transfer of preformed antibodies without activation of the recipient's immune system.

Feature	Active Immunity	Passive Immunity
Source	Self-produced	External antibodies
Onset	Slow	Immediate
Memory	Present	Absent
Duration	Long-lasting	Short-lived
Examples	Vaccines (toxoid)	IgA, antitoxin, mAbs

Antitoxins, monoclonal antibodies, and IgA in breast milk provide immediate protection and are classic examples of passive immunity.
Toxoids are inactivated toxins used as vaccines that stimulate endogenous antibody production.
Hence, toxoid induces active immunity and is not an example of passive immunity.

QUESTION

Based on the given structural features, identify the type of antibody depicted.



A IgG

B **IgA** CORRECT

C IgM

D IgE

OK RIGHT ANSWER

B. IgA

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

Innate immunity forms the 1st line of defence. Why is it faster than adaptive immunity?

A It is acquired genetically and is present since birth

B **It recognises pre-determined patterns against common pathogens** CORRECT

C It cannot differentiate between the self and the foreign antigens

D It forms a memory response following a previous infection

OK RIGHT ANSWER

B. It recognises pre-determined patterns against common pathogens

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1d” here and does not include a written explanation for this item.

QUESTION

Allelic exclusion during B-cell maturation is ensured by:

A V(D)J rearrangements — CORRECT

B Alternate splicing

C Alternate polyadenylation

D Somatic hypermutation

RIGHT ANSWER

OK

A. V(D)J rearrangements

WHY THIS IS RIGHT

Allelic exclusion ensures that each B cell expresses a single antigen receptor specificity by allowing productive rearrangement of only one immunoglobulin heavy-chain allele and one light-chain allele.
• During B-cell development in the bone marrow, V(D)J recombination rearranges variable (V), diversity (D), and joining (J) gene segments.
• Once a functional Ig heavy chain is produced, further rearrangement of the other allele is inhibited, ensuring single antibody specificity.

Why not other options:
B) Alternative splicing: Produces different forms of Ig (IgM vs IgD) from the same transcript. Does not ensure single allele expression.
C) Alternative polyadenylation: Determines membrane-bound vs secreted antibody forms. Not related to allelic exclusion.
D) Somatic hypermutation: Occurs after antigen stimulation in germinal centers to increase antibody affinity. Not involved in initial allele selection.

QUESTION

Which of the following about γδ T-cells is incorrect?

A Kills extracellular bacteria by granulysin and perforin

B **Need antigen processing and presentation for its recognition** CORRECT

C Direct killing of infected macrophages

D Provide protection against mycobacteria

OK RIGHT ANSWER

B. Need antigen processing and presentation for its recognition

WHY THIS IS RIGHT

γδ T cells are a subset of T lymphocytes that act at the interface of innate and adaptive immunity.
• Their T-cell receptor (γδ TCR) recognizes antigens without the need for antigen processing or presentation by MHC molecules.
• They can directly recognize microbial phospholipids and stress-induced molecules.
• They kill infected cells or macrophages using perforin and granulysin.
• Play an important role in defense against intracellular pathogens, including mycobacteria.

Therefore, the incorrect statement is that γδ T cells require antigen processing and presentation, because they recognize antigens directly without MHC restriction.

QUESTION

Antibody-dependent cellular cytotoxicity (ADCC) is seen with:

1. Natural killer (NK) cells
2. Macrophages
3. Neutrophils
4. Eosinophils
5. CD4 lymphocytes

A 1, 2, 3, 4, 5

B 2, 5

C **1, 3, 4** CORRECT

D 1, 2, 5

OK RIGHT ANSWER

C. 1, 3, 4

WHY THIS IS RIGHT

Antibody-dependent cellular cytotoxicity (ADCC) is an immune mechanism in which effector cells destroy antibody-coated target cells.
• Target cells are coated with IgG antibodies.
• Immune cells bind the Fc portion of IgG via Fc receptors.
• This triggers release of cytotoxic substances, leading to destruction of the target cell.

Cells involved in ADCC:
• Natural killer (NK) cells
• Neutrophils
• Eosinophils (especially against parasites)

Not involved:
• CD4 T lymphocytes, which function mainly in helper and regulatory roles, not cytotoxic killing.

QUESTION

What is the correct sequence of cellular events of inflammation?
1. Selectin
2. ICAM-1
3. CD31
4. IL-8

A **1, 2, 3, 4** CORRECT

B 2, 1, 4, 3

C 4, 1, 2, 3

D 3, 4, 1, 2

OK RIGHT ANSWER

A. 1, 2, 3, 4

WHY THIS IS RIGHT

The cellular events of leukocyte recruitment in inflammation occur in a specific sequence:

1. Selectins -> Mediate rolling of leukocytes along the endothelium.
2. ICAM-1 (Intercellular Adhesion Molecule-1) -> Interacts with integrins to cause firm adhesion of leukocytes to the endothelium.
3. CD31 (PECAM-1) -> Facilitates diapedesis/transmigration of leukocytes through the endothelial junctions.
4. IL-8 (chemokine) -> Directs chemotaxis, guiding leukocytes to the site of tissue injury.

Correct order: Selectin -> ICAM-1 -> CD31 -> IL-8

QUESTION

Among the following, which cells have both MHC class I and MHC class II?
1. NK cells
2. B-lymphocytes
3. Dendritic cells
4. Platelets
5. RBC

A 4, 5

B 1, 3, 4

C **2, 3** CORRECT

D 1, 2, 3

OK RIGHT ANSWER

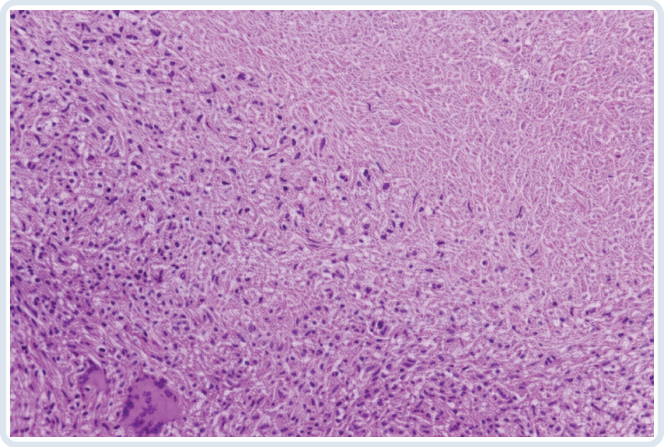
C. 2, 3

WHY THIS IS RIGHT

MHC Class I molecules are expressed on all nucleated cells, whereas MHC Class II molecules are expressed mainly on professional antigen-presenting cells (APCs).
Cells expressing both MHC I and MHC II:
· B lymphocytes
· Dendritic cells
· (Also macrophages, though not listed here)
Other cells:
· NK cells: Express MHC I only, not MHC II.
· Platelets: Limited or no typical MHC II expression.
· RBCs: No MHC molecules because they are anucleate.

QUESTION

A 32-year-old man presents with a 3-month history of weight loss, fever, and a persistent cough. Imaging of the chest reveals bilateral apical opacities with cavitation. A lung biopsy is performed, which is shown below. Which of the following cell surface markers is most specific for the cells predominantly found surrounding the necrotic tissue?



A CD4

B CD34

C **CD14** CORRECT

D CD8

RIGHT ANSWER

OK

C. CD14

WHY THIS IS RIGHT

The biopsy shows caseating granulomatous inflammation, typical of pulmonary tuberculosis. Granulomas consist of epithelioid macrophages and Langhans giant cells surrounding central caseous necrosis.

Key cells around the necrotic center:
• Activated macrophages (epithelioid cells) derived from monocytes.

CD14 is a marker for monocytes/macrophages, which are the predominant cells forming granulomas.

Other markers:
• CD4: Helper T lymphocytes.
• CD8: Cytotoxic T cells.
• CD34: Hematopoietic stem cells/endothelial progenitors.

QUESTION

Arrange the following sentences in the correct order for activation of B cells.

1. T-helper cells interact with the presented antigen peptides.
2. Improved quality of the immune response with affinity maturation.
3. Somatic hypermutation and immunoglobulin isotype class switching occur.
4. Antigen binding to B cell receptor.
5. Binding of CD40 ligand on T cells to CD 40 on B cells.

A 1, 2, 3, 4, 5

B 4, 1, 3, 2, 5

C **4, 1, 5, 3, 2** CORRECT

D 1, 2, 4, 3, 5

OK

RIGHT ANSWER

C. 4, 1, 5, 3, 2

WHY THIS IS RIGHT

Activation of B cells in T-dependent humoral immunity occurs through a sequence of antigen recognition and T-cell help.
Correct sequence:
1. Antigen binds to B-cell receptor (BCR) -> B cell internalizes and processes antigen.
2. B cell presents antigen peptide on MHC II to T-helper cells.
3. CD40L on T cells binds CD40 on B cells, providing the essential co-stimulatory signal.
4. This triggers somatic hypermutation and immunoglobulin class switching in germinal centers.
5. Result is affinity maturation, producing antibodies with higher antigen affinity.

QUESTION

Casoni reaction is seen in patients with hydatid cyst. Which of the following is incorrect about the immunoglobulin causing this reaction?

A **It is the only heat stable antibody** CORRECT

B Levels are raised in helminthic infections

C It does not pass the placental barrier

D It has a half-life of 1-5 days

OK RIGHT ANSWER

A. It is the only heat stable antibody

WHY THIS IS RIGHT

The Casoni test for hydatid cyst involves a Type I hypersensitivity reaction mediated by IgE antibodies against Echinococcus antigens.

Key features of IgE:
• Levels increase in helminthic infections and allergic conditions.
• Does not cross the placenta (unlike IgG).
• Has a short serum half-life (\~1-5 days).
• Binds strongly to mast cells and basophils, triggering histamine release on antigen exposure.

The statement “It is the only heat-stable antibody” is incorrect because IgE is heat-labile, not heat-stable.

QUESTION

Which of the following is the most potent stimulator of naive T-cells?

A **Mature dendritic cells** CORRECT

B Follicular dendritic cells

C B lymphocytes

D NK cell

OK RIGHT ANSWER

A. Mature dendritic cells

WHY THIS IS RIGHT

Mature dendritic cells are the most potent antigen-presenting cells (APCs) for activation of naïve T cells.
• Express high levels of MHC class I and II molecules.
• Provide strong co-stimulatory signals (B7/CD80-CD86) required for T-cell activation.
• Secrete cytokines that help differentiate T cells.
• Efficiently capture antigens in peripheral tissues and migrate to lymph nodes to present them to naïve T cells.

Other cells:
• B lymphocytes: Present antigen mainly to already activated/helper T cells.
• Follicular dendritic cells: Present antigen to B cells in germinal centers.
• NK cells: Part of innate immunity, not antigen-presenting cells.

QUESTION

Which of the following mediators of inflammation is an example of a C-X-C chemokine?

A Lipoxin LXA-4

B **IL-8** CORRECT

C Lymphotactin

D Fractalkine

OK RIGHT ANSWER

B. IL-8

WHY THIS IS RIGHT

Chemokines are cytokines that regulate leukocyte migration (chemotaxis) during inflammation and are classified based on the arrangement of cysteine (C) residues.

CXC chemokines (α-chemokines):
• Have one amino acid separating two cysteine residues (C-X-C).
• Primarily attract neutrophils.
• Example: IL-8 (CXCL8), an important mediator in acute inflammation.

Other options:
• Lipoxin LXA-4: Anti-inflammatory lipid mediator, not a chemokine.
• Lymphotactin: Member of the C chemokine family.
• Fractalkine: A CX3C chemokine.

QUESTION

Which of the following statements are TRUE about Leukotriene C4 (LTC□)?
1. It is not inhibited by aspirin
2. It causes chemotaxis
3. It produces bronchoconstriction
4. It causes vasoconstriction
5. It increases vascular permeability

A 1, 2, 3

B 2, 4

C 1, 3, 5

D **1, 3, 4, 5** CORRECT

OK RIGHT ANSWER

D. 1, 3, 4, 5

WHY THIS IS RIGHT

Leukotriene C□ (LTC□) is a cysteinyl leukotriene produced from arachidonic acid via the 5-lipoxygenase pathway in mast cells, eosinophils, and basophils.
Key actions of LTC□ (along with LTD□ and LTE□):
• Not inhibited by aspirin because aspirin blocks the cyclooxygenase (COX) pathway, not the lipoxygenase pathway.
• Causes bronchoconstriction (important in asthma).
• Causes vasoconstriction.
• Increases vascular permeability, leading to edema.

Chemotaxis is mainly mediated by Leukotriene B□ (LTB□), not LTC□.

QUESTION

Which of the following provides the primary co-stimulatory signal required for naïve T-cell activation?

A CD40-CD40L

B ICOS-ICOSL

C **CD28-B7** CORRECT

D PD-1-PD-L1

OK RIGHT ANSWER

C. CD28-B7

WHY THIS IS RIGHT

Activation of naïve T cells requires two signals from antigen-presenting cells (APCs):
1. Signal 1: Recognition of antigen
• T-cell receptor (TCR) binds peptide-MHC complex on the APC.

2. Signal 2 (Co-stimulatory signal):
• CD28 on T cells binds B7 (CD80/CD86) on APCs.

This CD28-B7 interaction is essential for full T-cell activation, proliferation, and IL-2 production.
If the co-stimulatory signal is absent, the T cell becomes anergic (functionally inactive).

QUESTION

Lipopolysaccharide of Leptospira is recognized by which Toll-like receptor?

A TLR1

B **TLR2** CORRECT

C TLR4

D TLR5

RIGHT ANSWER

OK

B. TLR2

WHY THIS IS RIGHT

Toll-like receptors (TLRs) are pattern recognition receptors of innate immunity that detect microbial components.

The lipopolysaccharide (LPS) of Leptospira has a unique structure and is primarily recognized by TLR2, unlike the typical Gram-negative bacterial LPS, which is recognized by TLR4.

Other options:
• TLR2: Recognizes lipoproteins, peptidoglycan, and leptospiral LPS.
• TLR4: Recognizes classical Gram-negative LPS.
• TLR5: Recognizes bacterial flagellin.
• TLR1: Forms heterodimers with TLR2 to recognize certain microbial lipoproteins.

QUESTION

Activation of RIG-I-like receptors leads to production of which key antiviral cytokines?

A IL-1β and IL-6

B TNF-α and IL-8

C **Type I interferons (IFN-α, IFN-β)** CORRECT

D IL-4 and IL-10

OK RIGHT ANSWER

C. Type I interferons (IFN-α, IFN-β)

WHY THIS IS RIGHT

RIG-I-like receptors (RLRs) are cytosolic pattern recognition receptors that detect viral RNA inside infected cells.

When activated by viral RNA, RLRs trigger signaling through MAVS (mitochondrial antiviral signaling protein), which activates transcription factors such as IRF3 and IRF7 --> This leads to production of type I interferons (IFN-α and IFN-β).

Functions of Type I interferons:
• Induce an antiviral state in neighboring cells
• Inhibit viral replication
• Increase MHC class I expression
• Activate natural killer (NK) cells

QUESTION

All of the following interaction occurs between antigen antibody reaction except:

A Ionic bond

B **Covalent bond** CORRECT

C Hydrogen bond

D Vander Waals forces

RIGHT ANSWER

OK **B. Covalent bond**

WHY THIS IS RIGHT

Antigen-antibody interactions are non-covalent and reversible, allowing antibodies to bind and release antigens efficiently. The binding between epitope (antigen) and paratope (antibody) occurs through weak forces such as:

- Ionic bonds
- Hydrogen bonds
- Van der Waals forces
- Hydrophobic interactions

These weak interactions collectively provide high specificity and affinity.
Covalent bonds are not involved, because they are strong, irreversible bonds and would prevent normal immune complex dynamics.

QUESTION

A 20-year-old medical student went for a screening health test and was injected with PPD intradermally. Identify the immune cell in the skin that picks up the PPD to present it to T cells.

A Langhans cells

B Follicular dendritic cells

C **Langerhans cells** CORRECT

D Natural killer cells

RIGHT ANSWER

OK

C. Langerhans cells

WHY THIS IS RIGHT

Langerhans cells are specialized dendritic antigen-presenting cells located in the epidermis.
• They capture antigens entering through the skin and migrate to regional lymph nodes to present antigen via MHC II to T lymphocytes, initiating a cell-mediated immune response.
• In the PPD (Mantoux) test, tuberculin protein is injected intradermally.
• Langerhans cells capture the antigen and present it to sensitized T cells, resulting in a type IV (delayed-type) hypersensitivity reaction mediated by Th1 cells.

> Why not the other options:
> A. Langhans cells: Multinucleated giant cells seen in granulomas (e.g., tuberculosis); not antigen-presenting cells in the epidermis.
>
> B. Follicular dendritic cells: Located in germinal centers of lymph nodes and spleen; present antigen to B cells, not involved in skin antigen capture.
>
> D. Natural killer cells: Part of innate immunity, responsible for killing virus-infected or tumor cells, not antigen presentation.

QUESTION

Which type of macrophages predominantly contributes to tissue repair and resolution of inflammation in patients with chronic obstructive pulmonary disease complicated by pneumonia?

A M1 macrophages

B **M2 macrophages** CORRECT

C Both M1 and M2 macrophages

D Resting macrophages

OK RIGHT ANSWER

B. M2 macrophages

WHY THIS IS RIGHT

Macrophages can polarize into two main functional types during inflammation:

M1 macrophages (classically activated):
• Induced by IFN-γ and microbial products
• Produce pro-inflammatory cytokines (IL-1, TNF, IL-6)
• Involved in microbial killing and tissue damage

M2 macrophages (alternatively activated):
• Induced by IL-4 and IL-13
• Promote tissue repair, fibrosis, and resolution of inflammation
• Secrete anti-inflammatory cytokines (IL-10, TGF-β) and growth factors

> In conditions like pneumonia complicating COPD, M2 macrophages play the dominant role in repairing damaged lung tissue and resolving inflammation.

QUESTION

A 6-year-old child is vaccinated with a polysaccharide antigen without protein conjugation. After vaccination, antibody titers rise initially but fall rapidly over the next few months. On re-exposure to the same antigen one year later, there is no significant anamnestic response. Which of the following immunological features is most consistent with the mechanism responsible for this response?

A Predominant IgG1 production with germinal center formation

B Requirement of CD40-CD40L interaction for B-cell activation

C **Absence of class switching and lack of memory B cells** CORRECT

D Somatic hypermutation occurring in secondary lymphoid organs

OK RIGHT ANSWER

C. Absence of class switching and lack of memory B cells

WHY THIS IS RIGHT

Pure polysaccharide vaccines (without protein conjugation) stimulate T-cell-independent type 2 (TI-2) immune responses.

Key characteristics of T-independent responses:
• Direct B-cell activation without T-helper cell involvement
• Predominantly IgM production
• Minimal or no class switching
• No germinal center formation
• Little or no memory B-cell generation

Because memory B cells are not formed, antibody levels decline quickly, and re-exposure does not produce a strong anamnestic (secondary) response.

QUESTION

Patients with which of the following conditions are more susceptible to infections caused by catalase-positive organisms:

A **Chronic granulomatous disease** CORRECT

B Hyper IgM syndrome

C Job syndrome

D Chediak-Higashi syndrome

OK RIGHT ANSWER

A. Chronic granulomatous disease

WHY THIS IS RIGHT

Susceptibility to catalase-positive organisms is a hallmark of Chronic Granulomatous Disease (CGD).
• Defect in NADPH oxidase -> impaired respiratory burst
• Phagocytes cannot generate reactive oxygen species to kill ingested microbes

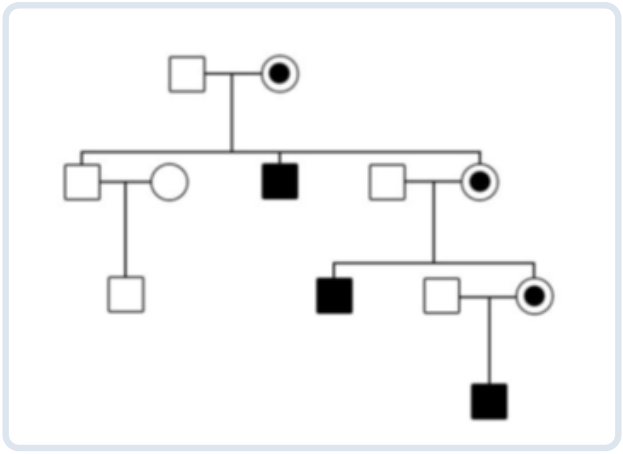
Catalase-positive organisms degrade their own hydrogen peroxide using catalase. Phagocytes cannot utilize microbial H□O□ -> failure of intracellular killing
Common organisms:
• Staphylococcus aureus
• Serratia marcescens
• Aspergillus species
• Nocardia

Differentiation:
• Hyper IgM -> defective class switching
• Job syndrome -> impaired neutrophil chemotaxis, recurrent staph infections but not specifically catalase-positive pattern

Chediak-Higashi -> lysosomal trafficking defect

QUESTION

Which of the following is the likely disease looking at the pattern of inheritance as provided in the image?



A **Wiskott Aldrich syndrome** — CORRECT

B Wilson's disease

C Achondroplasia

D Leber disease

RIGHT ANSWER

OK **A. Wiskott Aldrich syndrome**

WHY THIS IS RIGHT

Classic triad of cute baby wearing a TIE:
• Thrombocytopenia
• Recurrent infections
• Eczema

The pedigree shows an X-linked recessive inheritance pattern with affected males and carrier females.
There is no male-to-male transmission, and affected sons are born to carrier mothers.
Wiskott-Aldrich syndrome is an X-linked recessive immunodeficiency fitting this pattern.

Pattern	Key Feature	Example
XLR	Males affected	Wiskott-Aldrich
AR	Both sexes	Wilson
AD	Vertical transmission	Achondroplasia
Mitochondrial	Maternal only	Leber

QUESTION

An infant of 3 months presents with recurrent severe infections, including pneumonia, chronic diarrhea, and oral candidiasis. He has an absence of thymic shadow on chest X-ray. Laboratory tests reveal very low levels of adenosine deaminase (ADA). Which of the following is the most likely diagnosis?

A. X-linked agammaglobulinemia

B. Hyper-IgE syndrome

C. **Severe combined immunodeficiency (SCID)** — CORRECT

D. Common variable immunodeficiency (CVID)

RIGHT ANSWER

OK

C. Severe combined immunodeficiency (SCID)

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

A child presents with recurrent upper respiratory tract infections (URTIs) and laboratory findings reveal absence of B cells, T cells, and NK cells, along with ADA deficiency. What is the most likely underlying immunodeficiency disorder responsible for these findings?

A Bruton's disease

B Myeloperoxidase deficiency

C **Severe combined immunodeficiency disorder** CORRECT

D DiGeorge syndrome

RIGHT ANSWER

OK

C. Severe combined immunodeficiency disorder

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

Which of the following immunodeficiency disorders is least likely to cause rejection of a mismatched transplant due to absent or severely impaired immune function?

A Chediak-Higashi syndrome

B Wiskott-Aldrich syndrome

C **Bare lymphocyte syndrome** CORRECT

D Job syndrome

OK RIGHT ANSWER

C. Bare lymphocyte syndrome

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

A child presents with recurrent infections. Laboratory investigations reveal low levels of both B and T lymphocytes. Genetic testing demonstrates a mutation in the adenosine deaminase (ADA) gene. What is the most likely diagnosis?

A X-linked agammaglobulinemia

B Common variable immunodeficiency (CVID)

C **Severe Combined Immunodeficiency (SCID)** CORRECT

D DiGeorge syndrome

RIGHT ANSWER

OK

C. Severe Combined Immunodeficiency (SCID)

WHY THIS IS RIGHT

A mutation in adenosine deaminase (ADA) leading to low B and T lymphocytes is characteristic of Severe Combined Immunodeficiency (SCID).
• ADA deficiency -> accumulation of deoxyadenosine and dATP
• These metabolites are toxic to lymphocytes
• Inhibit DNA synthesis -> failure of lymphocyte proliferation

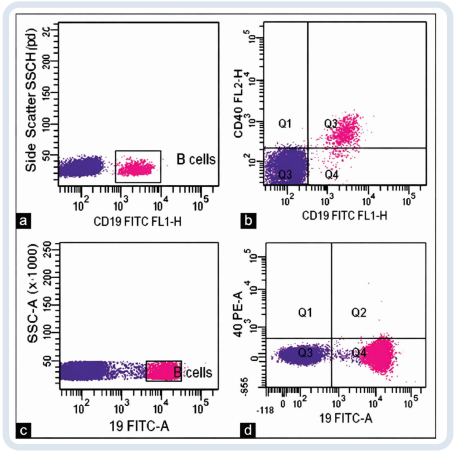
Immunological consequence:
• ↓ T cells + ↓ B cells -> combined immunodeficiency
• Leads to severe, recurrent infections early in life

Clinical features:
• Recurrent bacterial, viral, and fungal infections
• Failure to thrive
• Absent lymphoid tissue (e.g., tonsils)

Why other options are incorrect:
• X-linked agammaglobulinemia: Only B-cell deficiency
• CVID: Primarily B-cell dysfunction, later onset
• DiGeorge syndrome: T-cell deficiency due to thymic aplasia (not ADA defect)

QUESTION

A 3-year-old boy presents with recurrent bacterial infections and Pneumocystis jiroveci pneumonia. The flowcytometry analysis of the boy (Boxes c & d) is shown below (Boxes a & b are normal). What is the most likely diagnosis?



A Bare lymphocytes

B Chronic granulocyte deficiency

C **Hyper IgM syndrome** CORRECT

D DiGeorge syndrome

RIGHT ANSWER

OK

C. Hyper IgM syndrome

WHY THIS IS RIGHT

Hyper-IgM syndrome is an immunodeficiency caused by defective class switching of B cells, most commonly due to CD40 ligand (CD40L) deficiency on T-helper cells.
• B cells require CD40-CD40L interaction with T-helper cells to undergo class switching from IgM to IgG, IgA, and IgE.
• When this interaction is defective, B cells remain normal in number but produce mainly IgM, with low IgG, IgA, and IgE.

Clinical features:
• Recurrent bacterial infections
• Opportunistic infections (e.g., Pneumocystis jirovecii)
• Normal or increased B-cell count on flow cytometry
• Defective humoral immunity due to lack of class switching

QUESTION

Nude mice are not resistant to xenograft due to the absence of?

A B cell

B **T cell** CORRECT

C Both B and T cell

D INK cell

RIGHT ANSWER

OK

B. T cell

WHY THIS IS RIGHT

Nude mice have a congenital absence of the thymus due to a mutation in the Foxn1 gene, leading to failure of T-cell maturation.

Key features:
• Deficient T lymphocytes
• Impaired cell-mediated immunity
• B cells present but poorly functional due to lack of T-cell help
• Used in research because they accept xenografts (human tumor grafts) due to lack of T-cell-mediated rejection.

QUESTION

A 4-month-old baby was brought with history of recurrent infections in the past 4 months. On examination white patches were present in oral mucosa which could be scraped easily. Which of the following statements is false about this condition?

A. It has autosomal as well as X linked inheritance

B. Adenosine deaminase enzyme is deficient

C. **Tonsils are present** — CORRECT

D. B-cell count may be normal

RIGHT ANSWER

OK

C. Tonsils are present

WHY THIS IS RIGHT

This vignette describes Severe Combined Immunodeficiency (SCID), which presents in early infancy with recurrent infections and oral candidiasis (thrush).
Severe defect in both T-cell and B-cell immunity.

Common causes include:
• X-linked SCID due to IL2RG mutation
• Autosomal recessive SCID due to adenosine deaminase (ADA) deficiency.

Clinical features:
• Recurrent bacterial, viral, and fungal infections
• Oral thrush
• Failure to thrive
• Absent or very small lymphoid tissues (tonsils and lymph nodes)
• B-cell numbers may be normal in some forms (e.g., X-linked SCID) but functionally impaired due to lack of T-cell help.

QUESTION

3-year-old boy is brought to the emergency department for 2 days of fever, cough, and worsening shortness of breath. The patient's parents report that he recently recovered from prolonged diarrhea due to Giardia infection. Medical history is significant for recurrent ear infections treated with antibiotics since age 6 months and lobar pneumonia requiring hospitalization. Temperature is 38.7 C (101.7 F), pulse is 140/min, and respirations are 60/min. Physical examination reveals small tonsils and crackles in the lower lobe of the right lung. Which of the following is the most likely cause of this patient's recurrent infections?

A **Abnormal B-lymphocyte maturation** CORRECT

B Adenosine deaminase deficiency

C Complement deficiency

D Impaired oxidative burst

OK RIGHT ANSWER

A. Abnormal B-lymphocyte maturation

WHY THIS IS RIGHT

This presentation is characteristic of X-linked agammaglobulinemia (Bruton disease) due to defective B-cell maturation caused by mutation in the BTK (Bruton tyrosine kinase) gene.
• Failure of pre-B cells to mature into B lymphocytes
• Leads to markedly decreased immunoglobulins (IgG, IgA, IgM)
• Results in recurrent bacterial infections, especially after 6 months of age when maternal IgG declines.

Clinical clues:
• Recurrent otitis media and pneumonia
• Prolonged Giardia infection (due to low IgA)
• Small or absent tonsils and lymphoid tissue

QUESTION

18-month-old boy is brought to the emergency department due to blood in his stool, which the parents noticed when changing his diaper. The infant has had no previous bleeding and has been eating and drinking normally. He has a history of recurrent otitis media, frequent herpes labialis, and 2 episodes of pneumonia. On examination, the patient is well developed, well nourished, and has a fair complexion. He has eczema on his cheeks, trunk, and extremities. Scattered petechiae are also visible on his lower extremities. Laboratory studies show a platelet count of 24,000/mm³ and a leukocyte count of 9,000/mm³. Peripheral smear confirms the low platelet count and that the platelets are small. Genetic testing confirms the diagnosis. Which of the following processes is most likely affected by this patient's gene mutation?

A Antibody class switching

B **Cytoskeleton regulation** CORRECT

C DNA repair

D Maturation of T cells

RIGHT ANSWER

OK

B. Cytoskeleton regulation

WHY THIS IS RIGHT

This child has features of Wiskott-Aldrich syndrome, an X-linked immunodeficiency characterized by the triad:
• Eczema
• Recurrent infections
• Thrombocytopenia with small platelets

The disease is caused by mutation in the WAS gene, which encodes the Wiskott-Aldrich syndrome protein (WASP).
• WASP regulates actin cytoskeleton remodelling in hematopoietic cells.
• Defective cytoskeleton dynamics impair immune cell signalling, migration, and platelet formation, leading to immunodeficiency and microthrombocytopenia.

QUESTION

A 2-month-old male infant presents with recurrent severe bacterial, viral, and fungal infections, chronic diarrhea, and failure to thrive. Chest X-ray shows absence of thymic shadow. Flow cytometry reveals markedly reduced T cells and NK cells with normal or increased B cells. Which of the following genes is most commonly mutated?

A **IL2RG (common γ-chain of interleukin receptors)** CORRECT

B ADA (adenosine deaminase)

C BTK (Bruton tyrosine kinase)

D CD40 ligand

OK RIGHT ANSWER

A. IL2RG (common γ-chain of interleukin receptors)

WHY THIS IS RIGHT

Mutation of IL2RG (interleukin-2 receptor γ-chain) causes X-linked severe combined immunodeficiency (SCID).
• T cells ↓
• NK cells ↓
• B cells normal or increased but functionally impaired

Clinical features:
• Early infancy onset
• Recurrent severe bacterial, viral, and fungal infections
• Chronic diarrhea
• Failure to thrive
• Absent thymic shadow on chest X-ray

Why not the other options:
• ADA deficiency: Causes autosomal recessive SCID with T↓ B↓ NK↓ due to toxic purine metabolite accumulation.
• BTK mutation: Causes X-linked agammaglobulinemia with absent B cells and low immunoglobulins; T cells are normal.
• CD40 ligand deficiency: Causes Hyper-IgM syndrome with defective class switching (↑IgM, ↓IgG/IgA/IgE) but normal T and B cell numbers.

QUESTION

A 6-week-old infant presents with recurrent skin and mucosal bacterial infections. The umbilical cord fell off after 4 weeks. Examination shows absence of pus formation at infection sites. Laboratory tests reveal marked neutrophilia. Which of the following is the most likely underlying defect?

A Defective NADPH oxidase

B **Deficiency of CD18 on neutrophils** CORRECT

C Impaired myeloperoxidase activity

D Defective IL-12 receptor

OK RIGHT ANSWER

B. Deficiency of CD18 on neutrophils

WHY THIS IS RIGHT

This presentation is characteristic of Leukocyte Adhesion Deficiency type 1 (LAD-1), caused by deficiency of CD18 (β2-integrin) on neutrophils.
• CD18 is required for firm adhesion of neutrophils to endothelial cells during leukocyte extravasation.
• Without CD18, neutrophils cannot migrate from blood to infection sites, leading to impaired pus formation.

Key clinical features:
• Delayed separation of umbilical cord (>3 weeks)
• Recurrent bacterial infections of skin and mucosa
• Absence of pus at infection sites
• Marked neutrophilia in blood due to failure of tissue migration

> Why not other options:
>
> * NADPH oxidase defect: CGD -> defective killing, granulomas, normal pus.
> * Myeloperoxidase deficiency: Usually mild, mainly Candida infections.
> * IL-12 receptor defect: Mycobacterial infections (Th1 defect).

QUESTION

A 9-month-old male infant presents with persistent watery diarrhea since early infancy, failure to thrive, and recurrent skin rashes. He has been recently diagnosed with autoimmune hypothyroidism. Examination reveals eczema-like dermatitis over the trunk. Laboratory tests show elevated IgE levels. Sequencing of which of the following genes would confirm the diagnosis?

A PTPN22

B **FOXP3** CORRECT

C MTP

D CETP

OK RIGHT ANSWER

B. FOXP3

WHY THIS IS RIGHT

Mutation in the FOXP3 gene causes IPEX syndrome (Immune dysregulation, Polyendocrinopathy, Enteropathy, X-linked).
• FOXP3 is essential for the development and function of regulatory T cells (Tregs), which maintain immune tolerance and prevent autoimmunity.
• Loss of FOXP3 leads to failure of immune regulation, resulting in widespread autoimmune disease.
• Severe early-onset diarrhea due to autoimmune enteropathy
• Failure to thrive
• Eczema/dermatitis with elevated IgE
• Autoimmune endocrinopathies (e.g., type 1 diabetes, hypothyroidism)

Why not other options:
• PTPN22: Associated with autoimmune susceptibility (e.g., RA, T1DM), not severe early infant immune dysregulation.
• MTP: Causes abetalipoproteinemia (fat malabsorption, neurologic signs).
• CETP: Involved in lipid metabolism, affects HDL levels.

QUESTION

A 5-year-old boy presents with severe, chronic diarrhoea, failure to thrive, and a rash. His medical history includes a diagnosis of type 1 diabetes mellitus and thyroiditis. Laboratory tests show elevated levels of anti-enterocyte antibodies. Based on these findings, which mechanism is most likely impaired in this patient's condition?

A Central tolerance

B **Peripheral tolerance** CORRECT

C Anergy

D Hidden antigen

OK RIGHT ANSWER

B. Peripheral tolerance

WHY THIS IS RIGHT

Key clues in the question
• Severe chronic diarrhoea -> autoimmune enteropathy
• Failure to thrive
• Rash (eczema-like dermatitis)
• Type 1 diabetes + thyroiditis (polyendocrinopathy)
• Anti-enterocyte antibodies

This classic triad points to IPEX syndrome. (IPEX syndrome stands for: I - Immune dysregulation, P - Polyendocrinopathy, E - Enteropathy, X - X-linked)
IPEX occurs due to mutation of the FOXP3 gene, which is required for development of regulatory T cells (Tregs).
• Tregs normally suppress autoreactive T cells in peripheral tissues
• Loss of FOXP3 -> defective Treg function
• Result -> loss of peripheral immune tolerance

QUESTION

A patient with aplastic anemia undergoes bone marrow transplantation. Within 2 weeks, he presents with complaints of bloody diarrhea. Upon further examination, he was found to have jaundice and blistering rashes on the skin. What is the possible mechanism behind the patient's current clinical picture?

A **Transplanted cells attacking host tissues** — CORRECT

B Immune cells of the host attacking transplanted tissue

C Immune complex-mediated hypersensitivity reaction

D Preformed antibodies of the host attacking transplanted tissue

OK RIGHT ANSWER

A. Transplanted cells attacking host tissues

WHY THIS IS RIGHT

The source quiz contains the placeholder "$19" here and does not include a written explanation for this item.

QUESTION

Two weeks after undergoing allogeneic stem cell transplant for multiple myeloma, a 55- year-old man develops a severely pruritic rash, abdominal cramps, and profuse diarrhea. He appears lethargic. Physical examination shows yellow sclerae. There is a generalized maculopapular rash on his face, trunk, and lower extremities, and desquamation of both soles. His serum alanine aminotransferase is 115 U/L, serum aspartate aminotransferase is 97 U/L, and serum total bilirubin is 2.7 mg/dL. Which of the following is the most likely underlying cause of this patient's condition?

A **Donor T cells in the graft** CORRECT

B Newly formed anti-HLA antibodies

C Proliferating transplanted B cells

D Activated recipient T cells

OK RIGHT ANSWER

A. Donor T cells in the graft

WHY THIS IS RIGHT

This patient has acute graft-versus-host disease (GVHD) following allogeneic hematopoietic stem cell transplantation.
• Immunocompetent donor T cells present in the graft recognize recipient (host) tissues as foreign.
• These donor T cells become activated and attack host organs.

Common target organs:
• Skin: maculopapular rash, desquamation
• Gastrointestinal tract: abdominal pain, diarrhea
• Liver: elevated liver enzymes, jaundice

Typically occurs within weeks after transplantation.

QUESTION

A patient was diagnosed with bare lymphocytic disease. In this patient, graft vs host reaction does not occur due to:

A Complement deficiency

B Deficiency of CD4 T lymphocytes

C **MHC class II deficiency on lymphocytes** CORRECT

D MHC class I deficiency on lymphocytes

OK RIGHT ANSWER

C. MHC class II deficiency on lymphocytes

WHY THIS IS RIGHT

Bare lymphocyte syndrome type II is caused by deficiency of MHC class II expression on antigen-presenting cells due to mutations affecting transcription of MHC II genes.
• CD4 T-cell development is severely impaired because positive selection of CD4 T cells in the thymus requires MHC class II molecules.
• This results in marked reduction of CD4 T lymphocytes and severe immunodeficiency.

Why GVHD does not occur:

Donor T cells require recognition of host MHC molecules to initiate graft-versus-host reactions. In this condition, absence of MHC class II prevents antigen presentation, so donor T cells cannot become activated, preventing GVHD.

QUESTION

A 52-year-old man undergoes a renal transplant. On post-operative day 6, he develops sudden rise in serum creatinine and oliguria. Doppler shows good graft perfusion. Renal biopsy demonstrates capillary endothelial swelling, neutrophilic infiltration, fibrinoid necrosis of vessels, and linear C4d deposition along peritubular capillaries. Which of the following is the most likely mechanism?

A Direct cytotoxic T-cell-mediated injury

B Deposition of circulating immune complexes

C **Formation of donor-specific anti-HLA antibodies with complement activation** CORRECT

D Progressive intimal fibrosis due to smooth muscle proliferation

OK RIGHT ANSWER

C. Formation of donor-specific anti-HLA antibodies with complement activation

WHY THIS IS RIGHT

This presentation is characteristic of acute antibody-mediated (humoral) rejection after renal transplantation.
• Recipient forms donor-specific antibodies (DSA) against donor HLA antigens on graft endothelial cells.
• These antibodies activate the complement cascade, leading to endothelial injury, neutrophilic infiltration, and vascular damage.
• C4d deposition in peritubular capillaries is a key diagnostic marker of complement activation in antibody-mediated rejection.

Typical biopsy findings:
• Endothelial swelling
• Neutrophils in capillaries
• Fibrinoid necrosis of vessels
• Linear C4d staining in peritubular capillaries

*

QUESTION

A patient with AML is planned for an allogeneic hematopoietic stem cell transplant. As part of the pre transplant conditioning regimen, he receives myeloablative therapy using high dose chemotherapy and total body irradiation. Which of the following statements about myeloablation is false?

A Myeloablation increases the risk of graft failure if inadequate suppression of host immunity occurs.

B Myeloablation regimen can increase the incidence of post transplant lymphoproliferative disorders.

C Myeloablation conditioning destroys the recipient's bone marrow to allow engraftment of donor stem cells.

D **Myeloablation prevents graft versus host disease by suppressing donor T cells.** CORRECT

RIGHT ANSWER

OK

D. Myeloablation prevents graft versus host disease by suppressing donor T cells.

WHY THIS IS RIGHT

Myeloablative conditioning before allogeneic hematopoietic stem cell transplantation (HSCT) uses high-dose chemotherapy ± total body irradiation to:

1. Destroy the recipient's bone marrow, creating space for donor stem cell engraftment.
2. Suppress host immune cells to reduce graft rejection.
3. Eliminate residual malignant cells.

> Important points:
>
> * If host immunity is not adequately suppressed, graft failure or rejection can occur.
> * Intense immunosuppression can predispose to post-transplant lymphoproliferative disorders (PTLD), often related to EBV reactivation.

Myeloablation does NOT prevent graft-versus-host disease (GVHD) because GVHD is caused by donor T cells attacking recipient tissues. Prevention requires T-cell depletion or post-transplant immunosuppressive therapy, not myeloablation itself.

QUESTION

What is the function of IL-8?

A **Chemotaxis** CORRECT

B Fever

C Lymphocyte proliferation

D TH1 activation

RIGHT ANSWER

OK

A. Chemotaxis

WHY THIS IS RIGHT

IL-8 is a potent neutrophil chemoattractant, guiding them toward sites of acute inflammation. It also activates neutrophils, enhancing their phagocytic function. Other options do not match its primary role-IL-1/IL-6 cause fever, IL-2 drives lymphocyte proliferation, and IL-12 promotes TH1 activation.

QUESTION

The Nobel Prize for Physiology and Medicine in 2025 was given for:

A Receptor for touch and temperature

B Discovery of genomes of extinct hominins

C Discovery of tissue mRNA and its role in post-transcriptional modification

D **Peripheral immune tolerance** CORRECT

RIGHT ANSWER

OK

D. Peripheral immune tolerance

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Which of the following antibodies is the most reliable marker for diagnosing an acute infection?

A IgA

B IgG

C IgE

D **IgM** CORRECT

OK RIGHT ANSWER

D. IgM

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

A 22-year-old woman presents to the emergency department with high-grade fever, diffuse erythematous rash, hypotension, and shock. She started menstruating 3 days ago and reports using superabsorbent tampons. Which of the following is the most likely reason responsible for her condition?

A Lipoteichoic acid

B **Superantigen like TSST-1** CORRECT

C Hypovolemia

D Exotoxin

OK RIGHT ANSWER

B. Superantigen like TSST-1

WHY THIS IS RIGHT

The presentation is classic for toxic shock syndrome associated with tampon use.
It is caused by TSST-1 produced by *Staphylococcus aureus*, which acts as a superantigen.
Superantigens cause non-specific activation of a large number of T cells with massive cytokine release.
This cytokine storm leads to fever, rash, hypotension, and shock.
TSST-1 (superantigen)
↓
Bridges MHC II and TCR (non-specific)
↓
Massive T-cell activation
↓
Cytokine storm:
• IL-1 -> fever
• IL-2 -> T-cell proliferation
• TNF-α -> hypotension, shock
↓
Toxic Shock Syndrome

Feature	Normal Antigen	Superantigen
Processing	Required	Not required
T-cell activation	Specific (\~0.01%)	Massive (20-30%)
Binding	Specific peptide-MHC	Direct MHC-TCR binding
Cytokines	Controlled	Explosive (storm)

QUESTION

A patient has adult respiratory distress syndrome. Which of the following is the action of IL-8 in the pathogenesis in this patient?

A Endothelial injury

B **Neutrophil activation** CORRECT

C Fibrin deposition

D Macrophage activation

OK RIGHT ANSWER

B. Neutrophil activation

WHY THIS IS RIGHT

Injury (sepsis, trauma, infection)
↓
Cytokine release (TNF, IL-1, IL-8)
↓
IL-8 recruits neutrophils
↓
Neutrophil activation
↓
Release of ROS + proteases
↓
Alveolar damage
↓
ARDS

IL-8 is a potent chemokine responsible for recruitment and activation of neutrophils.
In ARDS, IL-8-mediated neutrophil activation leads to release of proteases and reactive oxygen species. This contributes to alveolar-capillary membrane damage and increased vascular permeability.
Endothelial injury and fibrin deposition are downstream effects, not the primary action of IL-8.

Cytokine	Function
IL-8	Neutrophil recruitment & activation
TNF-α	Inflammation
IL-1	Fever, inflammation
IFN-γ	Macrophage activation

QUESTION

All of the following interactions between T cells and APC (Antigen-Presenting Cell) are required for T cell activation/differentiation except:

A T cell receptor and MHC II

B CD28 and CD80

C **PD-PDL1** CORRECT

D Cytokine secretion

OK RIGHT ANSWER

C. PD-PDL1

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

Which of the following is not an example of passive immunity?

A **Toxoid** CORRECT

B Antitoxin

C IgA

D Monoclonal antibodies

RIGHT ANSWER

OK

A. Toxoid

WHY THIS IS RIGHT

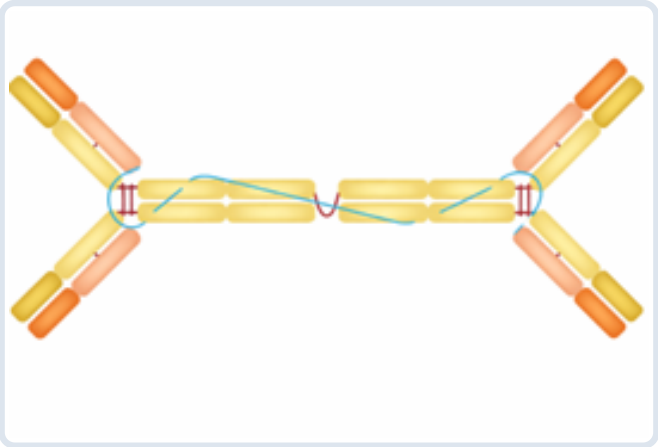
Passive immunity involves transfer of preformed antibodies without activation of the recipient's immune system.

Feature	Active Immunity	Passive Immunity
Source	Self-produced	External antibodies
Onset	Slow	Immediate
Memory	Present	Absent
Duration	Long-lasting	Short-lived
Examples	Vaccines (toxoid)	IgA, antitoxin, mAbs

Antitoxins, monoclonal antibodies, and IgA in breast milk provide immediate protection and are classic examples of passive immunity.
Toxoids are inactivated toxins used as vaccines that stimulate endogenous antibody production.
Hence, toxoid induces active immunity and is not an example of passive immunity.

QUESTION

Based on the given structural features, identify the type of antibody depicted.



A IgG

B **IgA** CORRECT

C IgM

D IgE

RIGHT ANSWER

OK **B. IgA**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

Innate immunity forms the 1st line of defence. Why is it faster than adaptive immunity?

A. It is acquired genetically and is present since birth

B. **It recognises pre-determined patterns against common pathogens** — CORRECT

C. It cannot differentiate between the self and the foreign antigens

D. It forms a memory response following a previous infection

OK

RIGHT ANSWER

B. It recognises pre-determined patterns against common pathogens

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1d” here and does not include a written explanation for this item.

QUESTION

Allelic exclusion during B-cell maturation is ensured by:

A **V(D)J rearrangements** CORRECT

B Alternate splicing

C Alternate polyadenylation

D Somatic hypermutation

RIGHT ANSWER

OK **A. V(D)J rearrangements**

WHY THIS IS RIGHT

Allelic exclusion ensures that each B cell expresses a single antigen receptor specificity by allowing productive rearrangement of only one immunoglobulin heavy-chain allele and one light-chain allele.
• During B-cell development in the bone marrow, V(D)J recombination rearranges variable (V), diversity (D), and joining (J) gene segments.
• Once a functional Ig heavy chain is produced, further rearrangement of the other allele is inhibited, ensuring single antibody specificity.

Why not other options:
B) Alternative splicing: Produces different forms of Ig (IgM vs IgD) from the same transcript. Does not ensure single allele expression.
C) Alternative polyadenylation: Determines membrane-bound vs secreted antibody forms. Not related to allelic exclusion.
D) Somatic hypermutation: Occurs after antigen stimulation in germinal centers to increase antibody affinity. Not involved in initial allele selection.

QUESTION

Patients with which of the following conditions are more susceptible to infections caused by catalase-positive organisms:

A **Chronic granulomatous disease** CORRECT

B Hyper IgM syndrome

C Job syndrome

D Chediak-Higashi syndrome

RIGHT ANSWER

OK **A. Chronic granulomatous disease**

WHY THIS IS RIGHT

Susceptibility to catalase-positive organisms is a hallmark of Chronic Granulomatous Disease (CGD).
• Defect in NADPH oxidase -> impaired respiratory burst
• Phagocytes cannot generate reactive oxygen species to kill ingested microbes

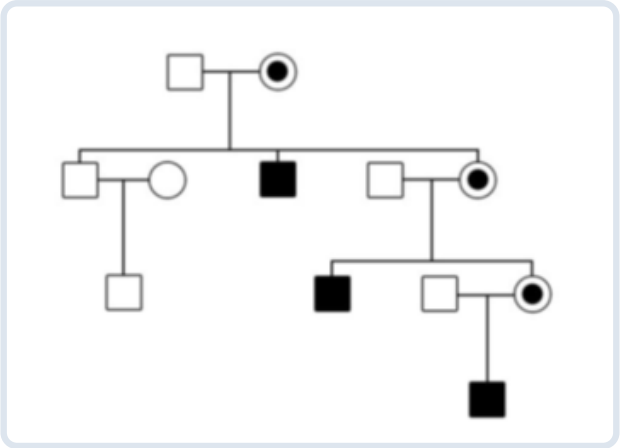
Catalase-positive organisms degrade their own hydrogen peroxide using catalase. Phagocytes cannot utilize microbial H□O□ -> failure of intracellular killing
Common organisms:
• Staphylococcus aureus
• Serratia marcescens
• Aspergillus species
• Nocardia

Differentiation:
• Hyper IgM -> defective class switching
• Job syndrome -> impaired neutrophil chemotaxis, recurrent staph infections but not specifically catalase-positive pattern

Chediak-Higashi -> lysosomal trafficking defect

QUESTION

Which of the following is the likely disease looking at the pattern of inheritance as provided in the image?



A **Wiskott Aldrich syndrome** CORRECT

B Wilson's disease

C Achondroplasia

D Leber disease

RIGHT ANSWER

OK **A. Wiskott Aldrich syndrome**

WHY THIS IS RIGHT

Classic triad of cute baby wearing a TIE:
• Thrombocytopenia
• Recurrent infections
• Eczema

The pedigree shows an X-linked recessive inheritance pattern with affected males and carrier females.
There is no male-to-male transmission, and affected sons are born to carrier mothers.
Wiskott-Aldrich syndrome is an X-linked recessive immunodeficiency fitting this pattern.

Pattern	Key Feature	Example
XLR	Males affected	Wiskott-Aldrich
AR	Both sexes	Wilson
AD	Vertical transmission	Achondroplasia
Mitochondrial	Maternal only	Leber

QUESTION

An infant of 3 months presents with recurrent severe infections, including pneumonia, chronic diarrhea, and oral candidiasis. He has an absence of thymic shadow on chest X-ray. Laboratory tests reveal very low levels of adenosine deaminase (ADA). Which of the following is the most likely diagnosis?

A X-linked agammaglobulinemia

B Hyper-IgE syndrome

C **Severe combined immunodeficiency (SCID)** CORRECT

D Common variable immunodeficiency (CVID)

OK RIGHT ANSWER

C. Severe combined immunodeficiency (SCID)

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1e” here and does not include a written explanation for this item.

QUESTION

A child presents with recurrent upper respiratory tract infections (URTIs) and laboratory findings reveal absence of B cells, T cells, and NK cells, along with ADA deficiency. What is the most likely underlying immunodeficiency disorder responsible for these findings?

A Bruton's disease

B Myeloperoxidase deficiency

C **Severe combined immunodeficiency disorder** CORRECT

D DiGeorge syndrome

OK RIGHT ANSWER

C. Severe combined immunodeficiency disorder

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1f” here and does not include a written explanation for this item.

QUESTION

Which of the following immunodeficiency disorders is least likely to cause rejection of a mismatched transplant due to absent or severely impaired immune function?

A Chediak-Higashi syndrome

B Wiskott-Aldrich syndrome

C **Bare lymphocyte syndrome** CORRECT

D Job syndrome

RIGHT ANSWER

OK

C. Bare lymphocyte syndrome

WHY THIS IS RIGHT

The source quiz contains the placeholder “$20” here and does not include a written explanation for this item.

QUESTION

A child presents with recurrent infections. Laboratory investigations reveal low levels of both B and T lymphocytes. Genetic testing demonstrates a mutation in the adenosine deaminase (ADA) gene. What is the most likely diagnosis?

A X-linked agammaglobulinemia

B Common variable immunodeficiency (CVID)

C **Severe Combined Immunodeficiency (SCID)** CORRECT

D DiGeorge syndrome

RIGHT ANSWER

OK **C. Severe Combined Immunodeficiency (SCID)**

WHY THIS IS RIGHT

A mutation in adenosine deaminase (ADA) leading to low B and T lymphocytes is characteristic of Severe Combined Immunodeficiency (SCID).
• ADA deficiency -> accumulation of deoxyadenosine and dATP
• These metabolites are toxic to lymphocytes
• Inhibit DNA synthesis -> failure of lymphocyte proliferation

Immunological consequence:
• ↓ T cells + ↓ B cells -> combined immunodeficiency
• Leads to severe, recurrent infections early in life

Clinical features:
• Recurrent bacterial, viral, and fungal infections
• Failure to thrive
• Absent lymphoid tissue (e.g., tonsils)

Why other options are incorrect:
• X-linked agammaglobulinemia: Only B-cell deficiency
• CVID: Primarily B-cell dysfunction, later onset
• DiGeorge syndrome: T-cell deficiency due to thymic aplasia (not ADA defect)

QUESTION

A patient with aplastic anemia undergoes bone marrow transplantation. Within 2 weeks, he presents with complaints of bloody diarrhea. Upon further examination, he was found to have jaundice and blistering rashes on the skin. What is the possible mechanism behind the patient's current clinical picture?

A **Transplanted cells attacking host tissues** CORRECT

B Immune cells of the host attacking transplanted tissue

C Immune complex-mediated hypersensitivity reaction

D Preformed antibodies of the host attacking transplanted tissue

OK RIGHT ANSWER

A. Transplanted cells attacking host tissues

WHY THIS IS RIGHT

The source quiz contains the placeholder "$21" here and does not include a written explanation for this item.