NEET PG Microbiology Sample Paper-3

Duration: 15 Minutes **Maximum Marks: 80**

Instructions

- This paper contains **20** Multiple Choice Questions.
- Each correct answer carries **+4** mark. Incorrect answer: **−1** marks. Only **one** correct option.
- Unattempted questions carry **0** marks.
- Use of mobile phones, smartwatches, or any electronic gadgets is strictly prohibited.

Q1. A 45-year-old patient presented with a painless, indurated penile ulcer. Dark-ground microscopy reveals actively motile spirochetes. The clinician wants to differentiate pathogenic *Treponema pallidum* subsp. *pallidum* from non-pathogenic commensal genital spirochetes based on structural motility characteristics. Which structural element provides the torque required for the distinctive corkscrew motility of this organism?

(A) Extracellular peritrichous flagella

(B) Endoflagella located within the periplasmic space

(C) Plasma membrane-anchored type IV pili

(D) Outer membrane lipopolysaccharide-driven gliding complexes

Q2. An outbreak of severe bloody diarrhea occurs in a daycare center. The isolated pathogen is a Gram-negative rod that is non-motile, does not ferment lactose, and does not produce gas from glucose. The toxin produced by this organism shares an identical molecular mechanism of action with which of the following diarrheagenic *Escherichia coli* pathotypes?

(A) Enterotoxigenic E. coli (ETEC)

(B) Enteropathogenic E. coli (EPEC)

(C) Enterohemorrhagic E. coli (EHEC)

(D) Enteroinvasive E. coli (EIEC)

Q3. A molecular biologist is studying antibiotic resistance mechanisms in a multi-drug resistant clinical isolate of *Enterococcus faecium*. The isolate shows high-level resistance to vancomycin ($MIC \geq 256 \mu g/mL$) and teicoplanin. This phenotype is mediated by the alteration of the terminal D-Ala-D-Ala of cell wall peptidoglycan precursors. Identify the structural modification configuration found in this specific *vanA* phenotype genotype:

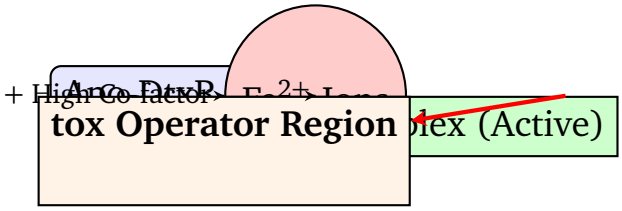
(A) Replacement of D-Ala-D-Ala with D-Ala-D-Lactate

(B) Replacement of D-Ala-D-Ala with D-Ala-D-Serine

(C) Methylation of the 23S rRNA binding pocket

(D) Acetylation of the aminoglycoside-binding site

Q4. A clinical trial evaluates the growth kinetics and toxin production profiles of *Corynebacterium diphtheriae*. The regulatory check of virulence gene expression is governed by an iron-dependent repressor (DtxR). Analyze the metabolic pathway cascade schematic shown below. Identify the exact physiological state under which the active functional repressor complex binds to the tox-operator sequence to completely inhibit transcription:



(A) Low inorganic intracellular iron concentration

(B) High inorganic intracellular iron concentration

(C) Complete absence of magnesium ions

(D) Acidic extracellular pH under anaerobic stagnation

Q5. A 34-year-old IV drug user presents with high-grade fever, chills, and a new systolic murmur. Echocardiography confirms vegetation on the tricuspid valve. Blood cultures grow Gram-positive cocci in clusters that are

catalase-positive and coagulase-positive. Which surface virulence factor of this organism specifically prevents opsonization by binding to the Fc portion of IgG antibodies?

(A) Teichoic acid

(B) Protein A

(C) Clumping factor A

(D) Fibronectin-binding protein

Q6. A sputum sample from a 52-year-old HIV-positive patient with chronic productive cough demonstrates acid-fast bacilli. To verify the diagnosis of *Mycobacterium tuberculosis* via phenotypic automated liquid cultures (MGIT), a biochemical confirmation test targeting the accumulation of nicotinic acid is ordered. Which metabolic assay match is specific for this biochemical feature?

(A) Negative Nitrate reduction test

(B) Positive Niacin accumulation test

(C) Heat-stable catalase production at 68°C

(D) Arylsulfatase production test within three days

Q7. A patient presents with a painless, expanding indurated lesion on the mandible with multiple draining sinuses discharging yellowish granules ("sulfur granules"). Histopathological evaluation reveals filamentous, branching Gram-positive bacilli that are obligate anaerobes and non-acid fast. What is the definitive primary therapeutic drug of choice for this condition?

(A) High-dose Penicillin G

(B) Trimethoprim-Sulfamethoxazole

(C) Amphotericin B

(D) Linezolid

Q8. An intensive care unit investigator evaluates the mutation dynamics of Influenza A virus strains causing seasonal epidemic shifts. The high rate of

antigenic drift in the hemagglutinin (HA) and neuraminidase (NA) genes is fundamentally driven by which molecular enzymatic characteristic?

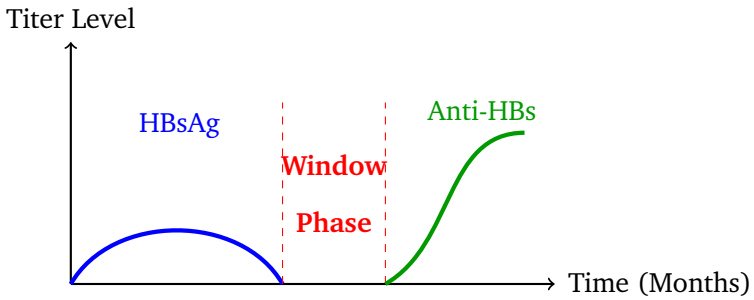
(A) High-fidelity proofreading mechanism of the viral DNA polymerase

(B) Lack of $3' \rightarrow 5'$ exonuclease proofreading activity in viral RNA-dependent RNA polymerase

(C) Homologous genetic recombination between non-segmented genomic elements

(D) Host-mediated alternative splicing of structural matrix mRNAs

Q9. A diagnostic virology lab performs serological screening for a patient suspected of chronic Hepatitis B virus infection. The assay maps specific antigen-antibody kinetics over a multi-month period. Based on the seromarker trajectory layout modeled in the diagram below, determine the exact clinical diagnosis of the patient:



(A) Chronic Active Hepatitis B carrier state

(B) Acute Hepatitis B Infection during the Window period

(C) Hyper-acute Fulminant Hepatic Failure

(D) Successful past immunization via Recombinant Vaccine

Q10. A neonate is born with microcephaly, chorioretinitis, sensorineural hearing loss, and periventricular calcifications. Shell vial culture of urine specimens demonstrates cytomegalic cytopathology ("owl's eye" intranuclear inclusions). Which antiviral agent targeting viral DNA polymerase processing is considered the primary first-line choice to manage this systemic congenital infection?

(A) Acyclovir

(B) Ganciclovir

(C) Ribavirin

(D) Oseltamivir

Q11. A 28-year-old traveler returns from Central Africa with severe hemorrhagic fever, myalgia, and maculopapular rash. Electron microscopy of blood samples reveals characteristic elongated, filamentous, enveloped RNA viruses that form "U"-shaped structures. What is the structural classification family of this pathogen?

(A) Filoviridae

(B) Flaviviridae

(C) Bunyaviridae

(D) Arenaviridae

Q12. A renal transplant recipient develops a sudden onset of fever, pleuritic chest pain, and hemoptysis. A chest CT scan reveals a nodular lesion surrounded by a crescent of attenuation ("halo sign"). A tissue biopsy shows acutely branching (45°), septate hyphae invading the pulmonary parenchyma. What is the identity of this fungal pathogen?

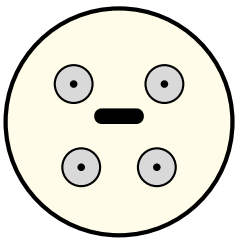
(A) *Mucor indoneesicus*

(B) *Aspergillus fumigatus*

(C) *Rhizopus oryzae*

(D) *Cryptococcus neoformans*

Q13. An analytical parasitology laboratory processes stool specimens from an asymptomatic food handler. Microscopic examination under iodine mount identifies a specific cyst morphology. Based on the structural internal arrangement and nuclear count demonstrated in the vector graphic below, identify the correct organism match:



(A) Mature cyst of *Entamoeba coli*

(B) Mature cyst of *Entamoeba histolytica*

(C) Trophozoite of *Giardia lamblia*

(D) Oocyst of *Cryptosporidium parvum*

Q14. A 39-year-old HIV patient with a CD4 count of $45/\mu L$ presents with progressive dyspnea and dry cough. Silver stains of bronchoalveolar lavage fluid demonstrate collapsed, cup-shaped/crushed ping-pong ball-like cystic forms. The drug of choice for prophylaxis and treatment acts by inhibiting folic acid synthesis. This drug is:

(A) Pentamidine

(B) Trimethoprim-Sulfamethoxazole

(C) Atovaquone

(D) Amphotericin B lipid complex

Q15. A muscle biopsy from a patient presenting with periorbital edema, severe myalgia, and eosinophilia reveals coiled, spiral encysted larvae within striated muscle fibers. The patient admits to eating undercooked wild boar meat during a recent hunting trip. Which nematode is responsible for this presentation?

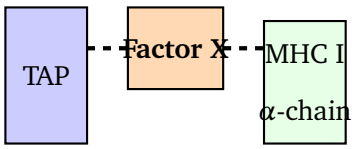
(A) *Wuchereria bancrofti*

(B) *Ancylostoma duodenale*

(C) *Trichinella spiralis*

(D) *Strongyloides stercoralis*

Q16. An immunologist charts the molecular assembly sequence of Major Histocompatibility Complex (MHC) Class I molecules within the endoplasmic reticulum. During the quality-control peptide-loading phase, a specific protein acts as a physical physical bridge between the MHC heterodimer and the Transporter associated with Antigen Processing (TAP) channel. Identify this bridging chaperone component labeled as "Factor X" in the schema below:



(A) Calnexin

(B) Calreticulin

(C) Tapasin

(D) β_2-microglobulin

Q17. A 2-year-old boy presents with recurrent severe pyogenic sinopulmonary infections caused by encapsulated bacteria. Laboratory evaluation shows profoundly low serum IgG, IgA, and IgM levels. Flow cytometry reveals an absolute absence of circulating mature CD19+ and CD20+ B lymphocytes, while CD3+ T cell numbers are normal. What is the fundamental molecular defect underlying this primary immunodeficiency?

(A) Defective CD40 ligand expression

(B) Mutation in the Bruton Tyrosine Kinase (BTK) gene

(C) Deletion of chromosome 22q11.2

(D) Missense mutation in the Adenosine Deaminase gene

Q18. A patient undergoes acute allograft rejection 10 days after a matched living-donor kidney transplantation. A biopsy of the graft shows extensive interstitial infiltration by CD8+ T lymphocytes along with inflammation of the renal tubular epithelium (tubulitis). Which hypersensitivity pathway profile best describes this specific cell-mediated immune damage?

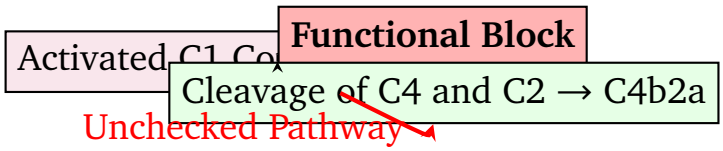
(A) Type I Immediate Hypersensitivity

(B) Type II Antibody-Dependent Cytotoxicity

(C) Type III Immune-Complex Mediated Inflammation

(D) Type IV Delayed-Type Hypersensitivity

Q19. A patient presents with recurrent episodes of angioedema without urticaria or pruritus. Laboratory tests reveal depleted classical complement pathway activity. The diagnostic workflow isolates a regulatory failure in the early classical complement initiation cascade modeled in the flow diagram below. Which factor corresponds to the missing regulatory inhibitor whose functional absence triggers uncontrolled C2 and C4 cleavage?



(A) Factor H

(B) C1 Inhibitor (C1-INH)

(C) Decay Accelerating Factor (DAF/CD55)

(D) Factor I

Q20. A neonate develops severe hypocalcemic tetany within 48 hours of birth. Physical examination reveals low-set ears, micrognathia, and a truncus arteriosus cardiac defect. Chest X-ray confirms the total absence of a thymic shadow. Which immunological deficiency parameter matches this patient's underlying developmental defect?

(A) Intact cell-mediated immunity with complete lack of humoral response

(B) Profound depletion of T cells with intact or variable B cell humoral reserves

(C) Isolated deficiency of mucosal secretory IgA antibodies

(D) Complete inability of neutrophils to generate oxidative burst respiratory radicals

Detailed Solutions

Q1.

Solution

Concept: *Treponema pallidum*, the causative spirochete of syphilis, possesses a highly specialized cellular anatomy that enables its characteristic corkscrew-like boring motility through viscous host extracellular matrices.

Solution:

Let's analyze the structural components driving spirochete motility:

(a) Unlike typical flagellated bacteria that possess external flagellar filaments exposed directly to the surrounding medium, spirochetes contain internal flagella.

(b) These structures are termed **endoflagella (or axial filaments)**. They are structurally anchored at the cellular poles and reside entirely within the **periplasmic space**, localized between the inner peptidoglycan-cytoplasmic membrane complex and the outer membrane.

(c) The coordinated rotation of these periplasmic endoflagella creates torque against the flexible outer sheath, causing the entire cell body to twist in a rigid, helical **corkscrew fashion**. This allows the pathogen to migrate through endothelial linings and connective tissues efficiently. Non-pathogenic surface commensals often vary significantly in their endoflagellar configurations and density.

Final Answer: Endoflagella located within the periplasmic space

Answer: (B)

Go Back to Question 1

Q2.

Solution

Concept: Bacillary dysentery is caused by *Shiga* species, producing a potent cytotoxin whose molecular mechanism is shared exactly by specific pathotypes of diarrheagenic *Escherichia coli*.

Solution:

Let's match the phenotypic markers and toxins described:

(a) The description of a Gram-negative, non-motile rod that does not ferment lactose or produce gas from glucose is characteristic of *Shiga dysenteriae*. It produces **Shiga toxin (Stx)**, an AB_5-subunit exotoxin.

(b) Shiga toxin catalyzes the specific cleavage of an adenine residue from the 28S rRNA component of the eukaryotic **60S ribosomal subunit**, completely arresting aminoacyl-tRNA binding and halting host protein synthesis, leading to mucosal cell death and bloody diarrhea.

(c) **Enterohemorrhagic *Escherichia coli* (EHEC)**, specifically strains like O157:H7, synthesizes **Shiga-like toxins (Stx1 and Stx2)** via lysogenic bacteriophages. These toxins share an identical structure and molecular mechanism of action, making EHEC infections highly prone to causing hemorrhagic colitis and Hemolytic Uremic Syndrome (HUS).

Final Answer: Enterohemorrhagic E. coli (EHEC)

Answer: (C)

Go Back to Question 2

Q3.

Solution

Concept: Glycopeptide resistance in *Enterococcus faecium* is mediated by plasmid-borne transposons that structurally reprogram cell wall precursors to eliminate antibiotic-binding affinity.

Solution:

Let's evaluate the structural alterations in the *vanA* operon:

(a) Vancomycin normally binds with high affinity to the terminal **D-Ala-D-Ala** residues of lipid-bound cell wall peptidoglycan precursors, sterically blocking transpeptidation and transglycosylation linkages.

(b) In the presence of the ***vanA* genotype**, the organism synthesizes alternative regulatory enzymes. The VanA ligase synthesizes an altered terminal precursor: **D-Alanyl-D-Lactate** (D-Ala-D-Lac).

(c) Replacing the terminal amide link with an ester link causes the loss of a critical hydrogen bond donor. This simple structural modification decreases the binding affinity of vancomycin and teicoplanin by over 1,000-fold, conferring high-level clinical resistance while maintaining structurally sound cell wall linkages.

Final Answer: Replacement of D-Ala-D-Ala with D-Ala-D-Lactate

Answer: (A)

Go Back to Question 3

Q4.

Solution

Concept: The expression of the structural *tox* gene encoding diphtheria toxin in *Corynebacterium diphtheriae* is tightly regulated by host-derived iron concentrations acting as a molecular co-repressor.

Solution:

Let's analyze the flowchart logic for the DtxR repressor:

(a) The DtxR protein is a functional transcription factor that exists naturally as an inactive monomer (Apo-DtxR) when isolated from co-factors.

(b) When environmental or **intracellular inorganic iron (Fe^{2+}) concentrations are high**, iron ions bind directly to the apo-enzyme as an activating co-factor. This induces dimerization and activation, forming the functional **Holo-DtxR complex**.

(c) The active Holo-DtxR complex then physically binds to the *tox* operator sequence with high affinity, blocking RNA polymerase from initiating transcription. Therefore, toxin production is completely shut off when iron is plentiful (high intracellular iron), and is heavily up-regulated in iron-starved settings, such as human tissues during active systemic infection.

Final Answer: High inorganic intracellular iron concentration

Answer: (B)

Go Back to Question 4

Q5.

Solution

Concept: *Staphylococcus aureus* uses specific cell-surface proteins to subvert host humoral immune responses and avoid phagocytosis.

Solution:

Let's evaluate the structural mechanisms of the listed staphylococcal surface elements:

(a) The presentation describes acute infective endocarditis of the tricuspid valve, typical of intravenous drug users, caused by *Staphylococcus aureus* (Gram-positive cocci in clusters, catalase/coagulase positive).

(b) A premier surface virulence factor of this pathogen is **Protein A**.

(c) Protein A contains specialized domains that specifically bind to the **Fc portion of IgG antibodies**, turning the antibody upside down. By coating itself in inverted antibodies, the bacterium conceals its antigen sites and leaves the Fab arms sticking outward. Because phagocytes recognize the Fc region to initiate opsonophagocytosis, this inversion prevents immune clearance.

Final Answer: Protein A

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept: *Mycobacterium tuberculosis* possesses distinct metabolic pathways that accumulate specific chemical products, which can be measured to differentiate it from non-tuberculous mycobacteria (NTM).

Solution:

Let's break down the metabolic pathways of mycobacteria:

(a) All mycobacteria synthesize nicotinic acid (niacin) as a baseline precursor for NAD synthesis.

(b) While most species possess functional enzymes to convert excess nicotinic acid into downstream metabolites, *Mycobacterium tuberculosis* lacks the specific enzyme configuration required to process it further.

(c) As a result, when growing in liquid media (like MGIT tubes), *M. tuberculosis* excretes and **accumulates large quantities of free niacin** into the surrounding environment. Measuring this accumulation via a **Positive Niacin accumulation test** serves as a reliable phenotypic confirmation marker for *M. tuberculosis*.

Final Answer: Positive Niacin accumulation test

Answer: (B)

Go Back to Question 6

Q7.

Solution

Concept: Cervicofacial actinomycosis is a chronic, granulomatous infection caused by anaerobic, branching Gram-positive bacilli that requires long-term targeted antibiotic therapy.

Solution:

Let's isolate the features of the pathogen and find its drug of choice:

(a) The presence of a mandate lesion with sinus tracts expressing yellowish "sulfur granules" made of tangled bacterial filaments is diagnostic for **Actinomyces israelii**.

(b) *Actinomyces* species are true prokaryotic bacteria (non-acid fast, Gram-positive, anaerobic) rather than fungi, meaning they are completely resistant to antifungal agents like amphotericin B.

(c) The definitive primary antimicrobial treatment of choice for actinomycosis is **high-dose Penicillin G**, typically administered intravenously for several weeks followed by oral penicillin therapy for an extended period to prevent disease recurrence in dense scarred tissues.

Final Answer: High-dose Penicillin G

Answer: (A)

Go Back to Question 7

Q8.

Solution

Concept: Antigenic drift in RNA viruses involves accumulation of point mutations within surface glycoprotein genes due to the inherent biochemical limitations of viral replication enzymes.

Solution:

Let's examine the replication properties of the influenza virus:

(a) Influenza A is an orthomyxovirus containing a segmented single-stranded RNA genome. Large structural swaps (antigenic shift) occur via segment reassortment.

(b) In contrast, gradual seasonal variations (**antigenic drift**) result from the steady accumulation of single nucleotide mutations within the hemagglutinin (HA) and neuraminidase (NA) genes.

(c) This continuous mutation rate is driven by the viral **RNA-dependent RNA Polymerase**, which **lacks a $3' \rightarrow 5'$ exonuclease proofreading activity**. Without this correction mechanism, base misincorporations during replication are left unrepaired, leading to rapid drift that allows the virus to evade host antibodies year over year.

Final Answer: Absence of $3' \rightarrow 5'$ exonuclease proofreading activity in the viral RNA-dependent RNA polymerase, resulting in a high mutation rate during genome replication.

Answer: (B)

Go Back to Question 8

Q9.

Solution

Concept: The serological "window period" of acute Hepatitis B virus (HBV) infection represents a specific timeframe where surface antigen shedding has ceased but detectable protective surface antibodies have not yet fully accumulated.

Solution:

Let's track the seromarker curves across the timeline:

(a) In an acute HBV infection that resolves successfully, Hepatitis B Surface Antigen (**HBsAg**) rises rapidly during early viremia and then steadily declines as the host immune response clears the virus, eventually dropping to zero (around month 3.5 to 4).

(b) Protective Hepatitis B Surface Antibodies (**Anti-HBs**) take several weeks to build up to a titer level that can be measured by standard assays, appearing later (around month 5.2).

(c) The gap between the disappearance of HBsAg and the appearance of Anti-HBs is known as the **Window Phase**. During this window, both markers test negative. A clinician can confirm an acute infection during this phase by testing for **Anti-HBc IgM**, which remains highly positive throughout this diagnostic gap.

Final Answer: Acute Hepatitis B Infection during the Window period

Answer: (B)

Go Back to Question 9

Q10.

Solution

Concept: Congenital Cytomegalovirus (CMV) infection presents with systemic abnormalities and requires specific nucleoside analogue therapy to limit long-term neurological sequelae.

Solution:

Let's select the correct antiviral agent for cytomegalic disease:

(a) The constellation of microcephaly, chorioretinitis, sensorineural deafness, and periventricular intracranial calcifications, paired with "owl's eye" inclusions in culture, is pathognomonic for congenital CMV.

(b) Acyclovir is highly effective against HSV and VZV because it is activated by their specific thymidine kinase. However, CMV lacks thymidine kinase, making acyclovir ineffective.

(c) The primary first-line antiviral drug of choice for managing symptomatic congenital CMV is **Ganciclovir** (or its oral prodrug valganciclovir). Ganciclovir is efficiently monophosphorylated by CMV's native UL97 protein kinase, allowing it to successfully inhibit viral DNA polymerase processing and protect hearing and developmental outcomes.

Final Answer: Ganciclovir

Answer: (B)

Go Back to Question 10

Q11.

Solution

Concept: Viral hemorrhagic fevers like Ebola and Marburg are caused by pathogens with unique filamentous structures that can be identified via electron microscopy.

Solution:

Let's review the structural classifications of hemorrhagic RNA viruses:

(a) The presentation describes a severe, acute hemorrhagic fever contracted in Central Africa, showing a characteristic maculopapular rash and profound constitutional symptoms.

(b) Under electron microscopy, the description notes highly distinct, pleomorphic, **enormously elongated filamentous threads** that wrap into "U"-shapes, "6"-shapes, or circular loops.

(c) This unique filamentous architecture is the defining structural trait of the **Filoviridae** family, which includes the Ebola virus and Marburg virus.

Final Answer: Filoviridae

Answer: (A)

Go Back to Question 11

Q12.

Solution

Concept: Invasive Aspergillosis is a dangerous angioinvasive fungal disease that typically develops in immunocompromised individuals, such as organ transplant recipients.

Solution:

Let's look at the radiographic and histopathological keys:

(a) The clinical picture describes an immunocompromised patient with chest pain, hemoptysis, and a chest CT scan showing a "halo sign" (a nodule surrounded by ground-glass hemorrhage). This strongly points to an angioinvasive mold infection.

(b) Biopsy findings show **septate hyphae that branch at uniform acute angles (approximately 45°)**.

(c) This specific structural layout—narrow, regular, septate hyphae branching at 45°—is characteristic of **_Aspergillus fumigatus_**. This helps rule out Mucor or Rhizopus species, which instead feature broad, ribbon-like, non-septate hyphae that branch at wider right angles (90°).

Final Answer: Aspergillus fumigatus

Answer: (B)

Go Back to Question 12

Q13.

Solution

Concept: The microscopic identification of intestinal protozoa relies on distinguishing specific internal structural features, such as nuclear count and chromatoid body geometry within the cyst stage.

Solution:

Let's analyze the internal structures shown in the diagram:

(a) The vector diagram illustrates a spherical cyst containing exactly **four distinct nuclei**, each featuring a small, centrally placed dot known as a central karyosome.

(b) Additionally, the cytoplasm contains an elongated, dark-staining structure with smooth, rounded margins: a **chromatoid bar** (cigar-shaped).

(c) This combination—exactly 1 to 4 nuclei with central karyosomes and rounded chromatoid bars—is diagnostic for a mature cyst of ***Entamoeba histolytica***. In contrast, non-pathogenic *Entamoeba coli* cysts typically contain up to eight nuclei with eccentric karyosomes and splintered, jagged chromatoid bars.

Final Answer: Mature cyst of Entamoeba histolytica

Answer: (B)

Go Back to Question 13

Q14.

Solution

Concept: *Pneumocystis jirovecii* pneumonia (PCP) is a classic opportunistic infection seen in patients with low CD4 counts, managed primarily with antifolates.

Solution:

Let's identify the pathogen and its corresponding treatment:

(a) The clinical picture describes a patient with advanced HIV and progressive respiratory symptoms whose BAL fluid shows cup-shaped or crushed-crescent structures on silver stain. This confirms a diagnosis of *Pneumocystis jirovecii*.

(b) The first-line drug for both the prophylaxis and active treatment of PCP is **Trimethoprim-Sulfamethoxazole (TMP-SMX)**.

(c) TMP-SMX operates by sequentially blocking two steps in the microbial **folic acid synthesis pathway**. Sulfamethoxazole inhibits dihydropteroate synthase, while trimethoprim inhibits dihydrofolate reductase, halting nucleotide synthesis.

Final Answer: Trimethoprim-Sulfamethoxazole

Answer: (B)

Go Back to Question 14

Q15.

Solution

Concept: Trichinellosis is a tissue zoonosis acquired by consuming raw or undercooked meats containing encysted larvae of the genus *Trichinella*.

Solution:

Let's break down the clinical signs and dietary history:

(a) The patient present with periorbital edema, muscle pain, and marked blood eosinophilia after consuming undercooked wild game (boar meat).

(b) A definitive diagnosis is established via muscle biopsy, which shows **coiled, spiral larvae** safely encysted within the sarcoplasm of striated muscle cells.

(c) The nematode responsible for this lifecycle pattern is ***Trichinella spiralis***. Adult worms live briefly in the host's intestinal mucosa to produce newborn larvae, which then migrate via the bloodstream to encyst inside skeletal muscle fibers.

Final Answer: Trichinella spiralis

Answer: (C)

Go Back to Question 15

Q16.

Solution

Concept: Peptide loading onto MHC Class I molecules within the endoplasmic reticulum is coordinated by a specialized multi-protein structure called the Peptide Loading Complex (PLC).

Solution:

Let's trace the interaction steps shown in the schema:

(a) Newly synthesized MHC Class I heavy chains fold in the ER with the help of chaperones like calnexin, and then associate with β_2-microglobulin.

(b) To load endogenous peptides, this heterodimer must be brought into close physical proximity with the **TAP transporter** channel, which pumps degraded cytosolic peptides directly into the ER lumen.

(c) The specialized transmembrane glycoprotein that acts as a structural **bridge**, physically linking the MHC Class I heterodimer directly to the TAP complex, is **Tapasin** (labeled as Factor X). Without tapasin, peptide loading is inefficient, resulting in unstable MHC structures that fail to reach the cell surface.

Final Answer: Tapasin

Answer: (C)

Go Back to Question 16

Q17.

Solution

Concept: X-linked Agammaglobulinemia (XLA) is a primary immunodeficiency caused by an inherited block in B cell development, leading to an absence of mature B cells and antibodies.

Solution:

Let's evaluate the cellular and immunological markers provided:

(a) The patient is a young boy presenting with recurrent infections from encapsulated bacteria, showing a severe reduction across all immunoglobulin classes (IgG, IgA, IgM).

(b) Flow cytometry isolates the problem: there is a **complete absence of circulating mature B cells ($CD19^+/CD20^+$)**, while T cell counts ($CD3^+$) are normal.

(c) This presentation is diagnostic for XLA, which is caused by a mutation in the **Bruton Tyrosine Kinase (*BTK*) gene**. BTK signaling is required for pro-B cells to develop into mature B cells. Without a functional BTK gene, B cell maturation halts in the bone marrow, leaving the patient without mature B lymphocytes or functional antibody production.

Final Answer: Mutation in the Bruton Tyrosine Kinase (BTK) gene

Answer: (B)

Go Back to Question 17

Q18.

Solution

Concept: Acute cellular allograft rejection occurs within days to weeks of a transplant and is primarily driven by cell-mediated adaptive immune destruction.

Solution:

Let's analyze the histopathology and immune mechanisms of transplant rejection:

(a) The renal graft biopsy demonstrates an extensive infiltration of **$CD8^+$ cytotoxic T lymphocytes** combined with direct inflammation of the renal tubular epithelial cells (tubulitis).

(b) This pattern describes acute cellular rejection, where host T cells recognize foreign HLA molecules on the donor tissue and execute targeted cellular destruction.

(c) This mechanism aligns directly with a **Type IV (Delayed-Type) Hypersensitivity** pathway profile. It is mediated entirely by T-lymphocytes rather than pre-formed antibodies or immune complex deposition.

Final Answer: Type IV Delayed-Type Hypersensitivity

Answer: (D)

Go Back to Question 18

Q19.

Solution

Concept: Hereditary angioedema (HAE) is an autosomal dominant condition caused by a deficiency in a key fluid-phase complement regulatory enzyme, leading to unchecked bradykinin release.

Solution:

Let's trace the complement cascade inhibition scheme:

(a) The diagram depicts an activated C1 complex ($C1qr_2s_2$) that cleaves C4 and C2 downstream to form the classical convertase (C4b2a).

(b) Under normal physiological conditions, this step is regulated by **C1 Inhibitor (C1-INH)**. C1-INH binds to and dissociates C1r and C1s from the active complex, halting further enzyme activity.

(c) When functional C1-INH is absent or deficient, the active C1 complex remains unchecked, causing continuous cleavage of C2 and C4. This pathway imbalance leads to the production of vasoactive peptides—principally **bradykinin**—inducing recurrent, life-threatening episodes of non-pitting angioedema without hives.

Final Answer: C1 Inhibitor (C1-INH)

Answer: (B)

Go Back to Question 19

Q20.

Solution

Concept: DiGeorge syndrome is a congenital disorder caused by abnormal development of the third and fourth pharyngeal pouches, resulting in defective thymic organogenesis.

Solution:

Let's match the clinical signs with the immune defect:

(a) The combination of hypocalcemic tetany (absent parathyroids), congenital heart defects (truncus arteriosus), facial dysmorphic features, and an **absent thymic shadow** on chest X-ray is classic for DiGeorge syndrome (22q11.2 deletion).

(b) The thymus is the primary lymphoid organ required for the maturation and selection of T lymphocytes.

(c) Because the thymic microenvironment fails to develop, the patient experiences a **profound depletion of mature, functional T cells**, severely crippling cell-mediated immunity. In contrast, B-cell production in the bone marrow remains structurally intact, meaning humoral antibody reserves are initially present but variable due to a lack of T-cell help.

Final Answer: Profound depletion of T cells with intact or variable B cell humoral reserves

Answer: (B)

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Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	B	2	C	3	A	4	B	5	B
6	B	7	A	8	B	9	B	10	B
11	A	12	B	13	B	14	B	15	C
16	C	17	B	18	D	19	B	20	B