NEET PG Microbiology Sample Paper-9

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 high-risk patient presenting with subacute endocarditis grows a Gram-positive, catalase-negative coccus that is intrinsically resistant to vancomycin ($MIC > 256\,\mu g/mL$). Genetic analysis confirms the presence of the $vanC$ operon phenotype. Which of the following organisms is the most likely causative pathogen?

(A) Enterococcus faecium

(B) Enterococcus gallinarum

(C) Staphylococcus aureus

(D) Streptococcus pneumoniae

Q2. An intensive care unit outbreak of ventilator-associated pneumonia involves an organism that produces a Class D carbapenemase (OXA-23). The isolates are non-fermenting Gram-negative coccobacilli, strictly aerobic, and oxidase-negative. What is the definitive mechanism of collateral beta-lactam resistance observed in this strain?

(A) Hyper-production of AmpC beta-lactamase

(B) Loss of $OprD$ outer membrane porins

(C) Activation of the MexAB-OprM efflux pump

(D) Metallo-beta-lactamase bla_{NDM-1} expression

Q3. A 28-year-old chef with a previous history of terminal ileum resection presents with severe bloody diarrhea. Stool culture on Skirrow's medium at 42°C under microaerophilic conditions reveals darting motility. This organism possesses a cytolethal distending toxin that primarily halts host cells in which phase of the cell cycle?

(A) G_1/S transition phase

(B) S phase replication period

(C) G_2/M transition phase

(D) Late M phase anaphase split

Q4. A patient clinically diagnosed with suspected neurosyphilis undergoes CSF analysis. The VDRL test is reported negative, but the clinical suspicion remains exceptionally high. What immunological mechanism accounts for a false-negative non-treponemal serological result when antigen-antibody concentrations are extremely skewed?

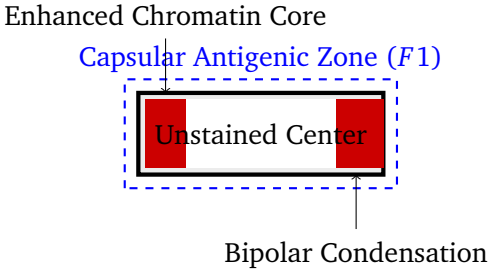
(A) Prozone phenomenon due to antibody excess

(B) Postzone phenomenon due to antigen excess

(C) Interstitial blocking factor activation

(D) Paradoxical antigen-masking effect

Q5. A clinical researcher evaluates a Gram-negative bacterium isolated from a patient with a chronic, non-healing tropical ulcer. The organism demonstrates distinct bipolar staining described as a "safety-pin" appearance. Identify the structural arrangement or virulence mechanism highlighted in the diagnostic morphology representation below:



(A) Pseudomonas aeruginosa pyocyanin core

(B) Yersinia pestis capsular safety-pin complex

(C) Klebsiella pneumoniae hypermucoviscosity locus

(D) Calymmatobacterium granulomatis Donovan body

Q6. A bone marrow biopsy from a patient with persistent pyrexia of unknown origin reveals granulomas. Culture yields small Gram-negative intracellular coccobacilli that require erythritol for optimal metabolic growth. The patient's serum agglutination test shows cross-reactivity with Francisella tularensis. What is the definitive species targeting this metabolic profile?

(A) Brucella melitensis

(B) Coxiella burnetii

(C) Bartonella henselae

(D) Orientia tsutsugamushi

Q7. A 35-year-old structural worker presents with suspected cutaneous anthrax. The virulence of Bacillus anthracis depends on a tripartite toxin plasmid ($pXO1$) and a capsule plasmid ($pXO2$). What amino acid comprises the unique homopolymeric capsule that inhibits host phagocytosis?

(A) D-Glutamic acid

(B) L-Lysine

(C) D-Alanine

(D) N-Acetylglucosamine

Q8. An infant presenting with severe bronchiolitis is diagnosed with Respiratory Syncytial Virus (RSV). The therapeutic use of Palivizumab is indicated for high-risk prophylaxis. Which specific viral structural surface component is directly targeted and neutralized by this monoclonal antibody?

(A) Hemagglutinin-Neuraminidase (HN) glycoprotein

(B) Fusion (F) glycoprotein

(C) Large (L) polymerase protein

(D) Matrix (M) structural protein

Q9. A 40-year-old patient with HIV develops a progressive neurological deficit presenting with altered mental status and ataxia. A brain MRI demonstrates asymmetric demyelination without mass effect. PCR of the CSF confirms JC virus activation. This virus executes viral entry into human oligodendrocytes by binding to which receptor complex?

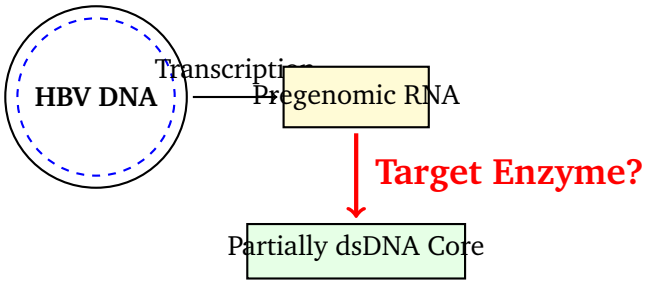
(A) CD4 molecule and CCR5 chemokine receptor

(B) α-2,6-linked sialic acid receptors

(C) 5-hydroxytryptamine (5-HT_{2A}) serotonin receptor

(D) Integrin $\alpha_v\beta_3$ surface receptor

Q10. An epidemiologist studies the replication cycle of a major blood-borne pathogen that carries a partially double-stranded circular DNA genome. The virus utilizes a unique reverse transcription step via pregenomic RNA inside the nucleocapsid core. Identify the enzymatic target denoted by the pathway structural block diagram shown below:



(A) RNA-dependent RNA polymerase

(B) DNA-dependent RNA polymerase II

(C) Reverse Transcriptase (RNA-dependent DNA polymerase)

(D) Integrase enzyme complex

Q11. A patient traveling to an endemic tropical area contracts Dengue virus. During a subsequent secondary infection with a different serotype, the patient

experiences a much higher risk of developing Dengue Hemorrhagic Fever. This phenomenon occurs due to which immunological mechanism?

(A) Antigenic drift induced immune evasion

(B) Antibody-Dependent Enhancement (ADE) via Fcγ receptors

(C) Molecular mimicry with endothelial cadherins

(D) Polyclonal T-cell superantigen activation

Q12. A child with recurrent, pyogenic staphylococcal infections is diagnosed with Chronic Granulomatous Disease (CGD). The nitroblue tetrazolium (NBT) dye reduction test shows no color change. Which precise enzymatic subunit of the NADPH oxidase complex is most frequently affected in the X-linked inherited form of this disease?

(A) $gp91^{phox}$ subunit

(B) $p22^{phox}$ subunit

(C) $p47^{phox}$ subunit

(D) $p67^{phox}$ subunit

Q13. An experimental assay tracks the activation of the classical complement pathway. A mutation prevents the auto-cleavage of the component that initiates enzymatic cleavage of both C4 and C2 substrates. Which specific subunit of the C1 macromolecular complex is defective in this setting?

(A) C1q recognition core

(B) C1r serine protease

(C) C1s acetyltransferase

(D) C1-inhibitor subunit

Q14. A newborn is evaluated for severe hypocalcemic tetany, structural cardiac outflow track anomalies, and an absent thymic shadow on a chest X-ray. Flow cytometry reveals a profound deficiency of CD3+ cells. This congenital defect is driven by a developmental arrest of which structures during embryogenesis?

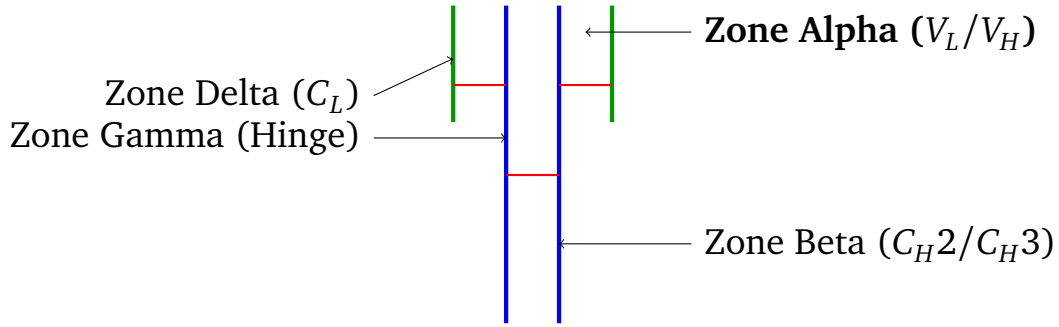
(A) First and second pharyngeal pouches

(B) Third and fourth pharyngeal pouches

(C) First and second branchial clefts

(D) Fourth and sixth branchial arches

Q15. A clinical immunology laboratory profiles a monoclonal antibody configuration to establish therapeutic target affinity. The diagrammatic scheme outlines the standard structural domains of an immunoglobulin molecule. Identify the exact location containing the hypervariable complementarity-determining regions (CDRs) that dictate antigen binding specificity:



(A) Zone Alpha (V_L/V_H)

(B) Zone Beta (C_H2/C_H3)

(C) Zone Gamma (Hinge)

(D) Zone Delta (C_L)

Q16. A patient experiences acute hyperacute rejection minutes after an allogeneic renal transplantation. This catastrophic immunological event is mediated by which pre-existing host mechanism?

(A) Host Donor-specific CD8+ Cytotoxic T lymphocytes

(B) Recipient Pre-formed Circulating Antibodies against ABO/HLA

(C) De novo synthesis of Interleukin-2 by Th1 cells

(D) Macrophage-induced delayed-type hypersensitivity

Q17. A bone marrow transplant recipient develops fever, dyspnea, and hemoptysis. A chest CT demonstrates a "halo sign." Sputum culture on Sabouraud dextrose

agar yields dichotomously branching hyphae at an angle of approximately 45°. What is the main fungal cell wall element targeted by the primary empirical echinocandin drug therapy required for this condition?

(A) Ergosterol synthesis pathway

(B) β-(1,3)-D-glucan synthase enzyme

(C) Chitin polymer microfibrils

(D) Squalene epoxidase component

Q18. A 34-year-old HIV-infected patient with a CD4+ T-lymphocyte count of 45/μL presents with progressive weight loss, diarrhea, and generalized lymphadenopathy. A bone marrow aspirate stained with Giemsa stain shows numerous small intracellular oval yeast cells within macrophages, each featuring a narrow base of budding. Which geographical niche or transmission source is most strongly correlated with this organism?

(A) Pigeon droppings enrichment

(B) Bat and bird excreta in river valleys

(C) Arid alkaline desert soil exposure

(D) Sphagnum moss handling injuries

Q19. A patient presenting with right upper quadrant pain, high fever, and leukocytosis is found to have a large solitary hepatic abscess. Microscopic analysis of an aspirate reveals trophozoites with ingested erythrocytes (erythrophagocytosis). What is the specific pathogenic factor responsible for the tissue destruction and anchoring ability of this parasite in the colonic epithelium before hepatic migration?

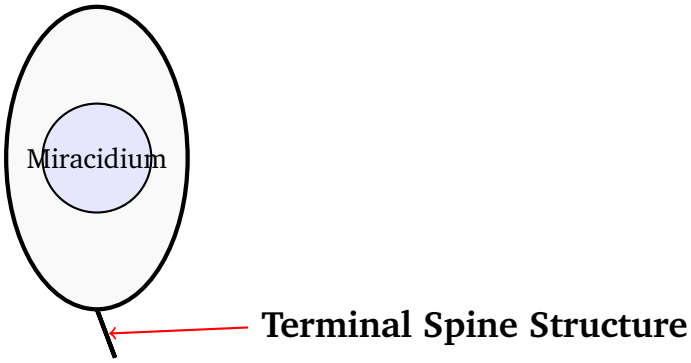
(A) Gal/GalNAc lectin adherence molecule

(B) Major Basic Protein release

(C) Variable Surface Glycoprotein (VSG) shift

(D) KALA antigen sequence

Q20. A clinical case of chronic hematuria in an immigrant patient from Egypt is evaluated. A biopsy of the urinary bladder mucosal wall reveals distinct parasitic ova featuring a characteristic terminal spine. Identify the helminthic life cycle stage or anatomical structure morphologically mapped out in the diagram below:



(A) Schistosoma mansoni egg

(B) Schistosoma japonicum egg

(C) Schistosoma hematobium egg

(D) Clonorchis sinensis egg

Detailed Solutions

Q1.

Solution

Concept: The genus *Enterococcus* comprises Gram-positive, catalase-negative cocci. While resistance to vancomycin in *Enterococcus faecium* and *Enterococcus faecalis* is typically acquired (via transferable plasmids carrying the *vanA* or *vanB* operons), certain enterococcal species possess an intrinsic, chromosomally mediated, low-level resistance characterized by the *vanC* operon.

Solution:

Let's analyze the microbiological characteristics specified in the vignette:

(a) The patient has subacute infective endocarditis caused by a Gram-positive, catalase-negative coccus that demonstrates high-level or intrinsic resistance to vancomycin.

(b) Genetic profile screening explicitly identifies the **$vanC$ operon**. The $vanC$ gene cluster is responsible for the intrinsic production of peptidoglycan precursors terminating in D-alanyl-D-serine (D-Ala-D-Ser) instead of the normal D-alanyl-D-alanine (D-Ala-D-Ala), reducing the binding affinity of vancomycin.

(c) This specific $vanC$ phenotype is a defining, species-specific **intrinsic characteristic of *Enterococcus gallinarum*** and *Enterococcus casseliflavus*.

(d) Conversely, *Enterococcus faecium* typically acquires high-level resistance via the $vanA$ or $vanB$ gene complexes (modifying precursors to D-Ala-D-Lac). *Staphylococcus aureus* and *Streptococcus pneumoniae* are structurally distinct and do not characteristically carry the intrinsic $vanC$ operon pathway.

Final Answer: Enterococcus gallinarum

Answer: (B)

Go Back to Question 1

Q2.

Solution

Concept: *Acinetobacter baumannii* is a non-fermenting, strictly aerobic, oxidase-negative, Gram-negative coccobacillus. Its remarkable multi-drug resistance (MDR) profile stems from the interplay of acquired carbapenemases (such as Class D OXA-23) alongside intrinsic resistance pathways.

Solution:

Let's break down the diagnostic markers and mechanisms of β-lactam resistance:

(a) The phenotypic description—non-fermenting, strictly aerobic, oxidase-negative Gram-negative coccobacilli causing a VAP outbreak—is pathognomonic for *Acinetobacter baumannii*. The production of the OXA-23 enzyme directly confers resistance to carbapenems.

(b) For collateral resistance against broad-spectrum cephalosporins, *A. baumannii* relies heavily on the **hyper-production of its intrinsic AmpC β-lactamase** (also known as Acinetobacter DNA-derived cephalosporinase, or ADC). Insertion sequence elements (such as IS*Aba1*) frequently insert upstream of the cephalosporinase gene, acting as a strong promoter that drives massive transcriptional upregulation.

(c) Let's evaluate why the alternative mechanisms belong to other pathogens:

- Loss of $OprD$ outer membrane porins and hyper-activation of the MexAB-OprM efflux system are classic resistance mechanisms seen in *Pseudomonas aeruginosa*, not *Acinetobacter*.
- While $bla_{\text{NDM}-1}$ is a Class B metallo-β-lactamase, the question establishes that the primary enzymatic driver here is a Class D OXA-23 enzyme.

Final Answer: Hyper-production of AmpC beta-lactamase

Answer: (A)

Go Back to Question 2

Q3.

Solution

Concept: *Campylobacter jejuni* is a primary bacterial cause of gastroenteritis. It grows optimally at 42°C on selective media (e.g., Skirrow's medium) under microaerophilic conditions, displays a characteristic "darting" motility, and produces several distinct virulence factors.

Solution:

Let's examine the cellular targeting mechanism of the toxin described:

(a) The clinical description focuses on a chef with severe bloody diarrhea whose stool culture confirms the isolation of *Campylobacter jejuni*.

(b) A key virulence factor of *C. jejuni* is the **cytolethal distending toxin (CDT)**, a tripartite (CdtA-CdtB-CdtC) protein complex. The active subunit, CdtB, acts as a functional analog to eukaryotic DNase I, executing double-stranded DNA breaks inside the host cell nucleus.

(c) The cellular response to this massive DNA damage activates the ataxia telangiectasia mutated (ATM) pathway, which triggers downstream inhibitory phosphorylation of Cdc25C. This blocks the activation of the Cyclin B-CDK1 complex, **halting host cells specifically at the G_2/M transition phase** of the cell cycle, preventing mitosis and leading to progressive cellular distention and apoptotic death of the intestinal epithelium.

Final Answer: G_2/M transition phase

Answer: (C)

Go Back to Question 3

Q4.

Solution

Concept: Flocculation and agglutination serological assays require an optimal ratio of antigen to antibody (the zone of equivalence) to form visible immune complexes.

Solution:

Let's analyze the immunological basis of the false-negative serology:

(a) Non-treponemal tests, such as the VDRL and RPR, detect anti-cardiolipin antibodies in patients with syphilis. In secondary or early neurosyphilis, the systemic antibody titer can be exceptionally high.

(b) When an extreme excess of patient antibody binds to the fixed amount of reagent antigen, every individual epitope is rapidly saturated by standalone antibodies. This prevents the cross-linking of antigens necessary to form a visible lattice network or precipitate.

(c) This false-negative result due to **antibody excess** is called the **prozone phenomenon**. The solution to confirm the true clinical status is to serially dilute the patient's serum sample, which lowers the antibody concentration into the zone of equivalence, allowing visible precipitation to occur.

(d) The *postzone phenomenon* refers to a lack of lattice formation due to *antigen excess*, which is not seen in standard VDRL screening where the antigen concentration is tightly controlled by the assay reagent kit.

Final Answer: Prozone phenomenon due to antibody excess

Answer: (A)

Go Back to Question 4

Q5.

Solution

Concept: *Yersinia pestis*, the causative agent of the plague, is a zoonotic Gram-negative coccobacillus that displays a characteristic bipolar ("safety-pin") appearance on Wayson or Giemsa stains.

Solution:

Let's evaluate the structural features and matching species:

(a) The schematic diagram demonstrates a classic safety-pin morphology with dense chromatin condensation at the poles and an unstained center, surrounded by a distinct outer capsule labeled as the **Fraction 1 ($F1$) capsular antigen**.

(b) This specific layout corresponds to **_Yersinia pestis_**. The $F1$ capsular antigen is encoded on the pFrg/pMT1 plasmid and is expressed optimally at 37°C inside the mammalian host, where it acts as a gel-like antiphagocytic shield against neutrophils.

(c) Let's evaluate why the alternative selections do not fit the layout:

- *Pseudomonas aeruginosa* is a simple rod that does not feature an F1 capsular matrix.
- *Klebsiella pneumoniae* exhibits a thick polysaccharide capsule but lacks safety-pin bipolar condensation.
- *Calymmatobacterium granulomatis* (reclassified as *Klebsiella granulomatis*) forms intracellular clusters within macrophages called Donovan bodies rather than this standalone extracellular capsular complex.

Final Answer: Yersinia pestis capsular safety-pin complex

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept: The genus *Brucella* contains fastidious, small, Gram-negative intracellular coccobacilli that cause brucellosis (undulant fever), demonstrating specific tissue and metabolic tropism.

Solution:

Let's trace the biochemical and serological features of the pathogen:

(a) The presentation describes a patient with a fever of unknown origin showing granulomas on bone marrow biopsy. The isolation of small intracellular Gram-negative rods points toward a zoonotic intracellular pathogen.

(b) *Brucella* species possess a unique metabolic affinity for **erythritol**, a sugar-alcohol concentrated in animal placenta, uterus, and fetal membranes. This metabolic tropism drives the localization of the pathogen in these tissues in livestock, leading to contagious abortion.

(c) Furthermore, due to similarities in the O-antigen chain of the smooth lipopolysaccharide (sLPS) layer, *Brucella melitensis* shares significant structural epitopes with *Francisella tularensis* and *Vibrio cholerae*, leading to **serological cross-reactivity** during standard serum agglutination testing (SAT).

Final Answer: Brucella melitensis

Answer: (A)

Go Back to Question 6

Q7.

Solution

Concept: *Bacillus anthracis* is a large, spore-forming, Gram-positive rod whose virulence is mediated by two large plasmids, $pXO1$ (encoding the protective antigen, lethal factor, and edema factor toxins) and $pXO2$ (encoding the capsule).

Solution:

Let's analyze the biochemical configuration of the bacterial capsule:

(a) Virtually all pathogenic bacteria that produce a protective capsule construct it from complex polysaccharide polymers (e.g., *Streptococcus pneumoniae*, *Neisseria meningitidis*).

(b) *Bacillus anthracis* is a classic exception to this rule. Its capsule is uniquely composed of a homopolymer of **D-glutamic acid** [poly-γ-D-glutamic acid].

(c) This polypeptide capsule prevents phagocytosis by host macrophages because its D-stereoisomer configuration resists degradation by normal proteolytic lysosomal enzymes, allowing the bacterium to disseminate unimpeded through the bloodstream and tissues.

Final Answer: D-Glutamic acid

Answer: (A)

Go Back to Question 7

Q8.

Solution

Concept: Respiratory Syncytial Virus (RSV), a member of the *Pneumoviridae* family, utilizes specific surface envelope proteins to mediate target host cell attachment and entry into the respiratory epithelium.

Solution:

Let's evaluate the target of the prophylactic monoclonal antibody:

(a) Palivizumab is a humanized monoclonal antibody indicated for the prevention of serious lower respiratory tract disease caused by RSV in high-risk pediatric patients (such as premature infants or those with chronic lung disease).

(b) The antibody **directly targets and neutralizes the Fusion (F) glycoprotein** on the surface of the RSV virion.

(c) By binding to an epitope on the F protein, Palivizumab prevents the conformational change required for the viral lipid envelope to fuse with the host cell membrane, blocking viral entry and inhibiting the cell-to-cell fusion that leads to syncytia formation.

Final Answer: Fusion (F) glycoprotein

Answer: (B)

Go Back to Question 8

Q9.

Solution

Concept: The John Cunningham (JC) virus is a member of the *Polyomaviridae* family that targets oligodendrocytes in the CNS of severely immunocompromised hosts, causing Progressive Multifocal Leukoencephalopathy (PML).

Solution:

Let's trace the molecular receptor specificity of the JC virus:

(a) The clinical picture—an HIV patient with altered mental status, ataxia, and an asymmetric demyelinating pattern on brain MRI—presents the classic clinical presentation of PML.

(b) To enter human oligodendrocytes and astrocytes, the JC virus utilizes its major capsid protein, VP1, to engage host cell surface receptors.

(c) The virus binds to α-2,6-linked sialic acid on host cells, but viral entry requires a specific cellular coreceptor: the **5-hydroxytryptamine (5-HT_{2A}) serotonin receptor**. Binding to this receptor triggers clathrin-dependent endocytosis, delivering the viral genome to the nucleus for replication.

Final Answer: 5-hydroxytryptamine (5-HT_{2A}) serotonin receptor

Answer: (C)

Go Back to Question 9

Q10.

Solution

Concept: Hepatitis B Virus (HBV) replicates its partially double-stranded circular DNA genome via an RNA intermediate called pregenomic RNA (pgRNA), a defining characteristic of the *Hepadnaviridae* family.

Solution:

Let's analyze the enzymatic step indicated in the block diagram:

(a) The diagram traces the replication pathway of the HBV genome, where full-length genomic DNA is transcribed by host machinery into a single-stranded **Pregenomic RNA** template.

(b) This pgRNA intermediate is then encapsulated into core nucleocapsid structures in the cytoplasm. To complete the lifecycle, this template must be converted back into the partially double-stranded DNA core of the progeny virion.

(c) The enzyme responsible for translating this RNA sequence back into a DNA strand is the viral **Reverse Transcriptase (RNA-dependent DNA polymerase)**. This multi-functional viral polymerase binds to pgRNA, uses its reverse transcriptase activity to synthesize the minus-strand DNA, degrades the RNA template via its RNase H activity, and then synthesizes the partial plus-strand DNA.

Final Answer: Reverse Transcriptase (RNA-dependent DNA polymerase)

Answer: (C)

Go Back to Question 10

Q11.

Solution

Concept: Dengue virus is a single-stranded positive-sense RNA flavivirus with four distinct antigenic serotypes (DENV-1 to DENV-4). Exposure to one serotype provides lifelong immunity against that specific serotype but increases the risk of severe disease upon secondary infection with a heterologous serotype.

Solution:

Let's evaluate the immunological mechanism behind this phenomenon:

(a) During a primary Dengue infection, the host generates neutralizing IgG antibodies against that specific viral serotype.

(b) Upon a secondary infection with a *different* serotype, these pre-existing antibodies bind to the new virus but cannot neutralize it because the surface epitopes differ slightly. Instead, they form non-neutralizing immune complexes.

(c) This leads to **Antibody-Dependent Enhancement (ADE)**. The constant (Fc) regions of the bound antibodies attach to **Fcγ receptors (FcγR)** on circulating monocytes and macrophages. Rather than destroying the virus, this facilitates viral entry into these cells, leading to a massive increase in viral replication, a cytokine storm, and downstream endothelial destruction that manifests as Dengue Hemorrhagic Fever.

Final Answer: Antibody-Dependent Enhancement (ADE) via Fcγ receptors

Answer: (B)

Go Back to Question 11

Q12.

Solution

Concept: Chronic Granulomatous Disease (CGD) is a primary immunodeficiency caused by mutations in the NADPH oxidase complex, which prevents phagocytes from generating the superoxide radicals required for the respiratory burst.

Solution:

Let's focus on the molecular genetics of the inherited forms of CGD:

(a) The clinical presentation involves a child with recurrent pyogenic staphylococcal infections who shows an abnormal (clear/negative) result on a nitroblue tetrazolium (NBT) slide test, confirming a diagnosis of CGD.

(b) The multi-protein NADPH oxidase complex is composed of several membrane-bound and cytosolic subunits, including $gp91^{phox}$, $p22^{phox}$, $p47^{phox}$, and $p67^{phox}$.

(c) The most common clinical presentation of CGD ($\approx 65-70\%$ of cases) is inherited in an **X-linked recessive pattern**. This form is driven by mutations in the *CYBB* gene, which encodes the membrane-bound **$gp91^{phox}$ subunit** (β-subunit of cytochrome b558). The other subunits ($p22^{phox}$, $p47^{phox}$, $p67^{phox}$) are typically associated with rarer autosomal recessive forms of the disease.

Final Answer: $gp91^{phox}$ subunit

Answer: (A)

Go Back to Question 12

Q13.

Solution

Concept: The classical complement cascade is initiated when the macromolecular C1 complex (composed of one C1q molecule, two C1r molecules, and two C1s molecules) binds to the Fc regions of IgM or IgG immune complexes.

Solution:

Let's track the sequence of enzymatic activation within the C1 complex:

(a) The C1q core acts as the initial sensor, physically binding to the surface of an antibody-antigen complex. This binding induces a structural shift that is transmitted to the attached proenzymes.

(b) This structural change causes the activation of the **C1r serine protease**, which undergoes an initial catalytic auto-cleavage to transform into an active serine protease enzyme.

(c) The active C1r enzyme then cleaves and activates the adjacent **C1s proenzyme subunit**. Once activated, C1s acts as the functional downstream protease that cleaves both C4 and C2 substrates to form the C3 convertase (C4b2a). Thus, the initial internal activation step depends on the auto-cleavage of C1r.

Final Answer: C1r serine protease

Answer: (B)

Go Back to Question 13

Q14.

Solution

Concept: DiGeorge syndrome (22q11.2 deletion syndrome) is a congenital disorder caused by abnormal migration and development of neural crest cells into the branchial/pharyngeal structures during early embryonic development.

Solution:

Let's analyze the embryological origin of the affected organs:

(a) The patient presented with hypocalcemic tetany (due to parathyroid hypoplasia), structural cardiac outflow tract anomalies (such as truncus arteriosus or Tetralogy of Fallot), and an absent thymic shadow with a profound loss of circulating CD3+ T cells. This is the classic presentation of DiGeorge syndrome.

(b) During embryogenesis, the thymus and the parathyroid glands develop from the endodermal lining of the pharyngeal pouches.

(c) Specifically, a developmental arrest of the **third and fourth pharyngeal pouches** prevents the normal differentiation of the inferior parathyroid glands and thymus (from the 3rd pouch) and the superior parathyroid glands (from the 4th pouch), leading to the clinical manifestations of the syndrome.

Final Answer: Third and fourth pharyngeal pouches

Answer: (B)

Go Back to Question 14

Q15.

Solution

Concept: Immunoglobulins consist of two identical heavy chains and two identical light chains linked by disulfide bonds, divided into variable (V) and constant (C) structural domains.

Solution:

Let's identify the antigen-binding zone on the immunoglobulin molecule:

(a) Antigen binding specificity is determined by the specific sequence of amino acids within the complementarity-determining regions (CDRs). These are hypervariable loops embedded within the variable domains of the antibody.

(b) In the provided schematic, **Zone Alpha (V_L/V_H)** represents the paired variable domains located at the amino-terminal tips of both the light (V_L) and heavy (V_H) chains, forming the Antigen-Binding Fragment (Fab).

(c) The other labeled zones handle different structural functions: Zone Beta represents the constant heavy domains (C_H2/C_H3) that form the constant fragment (Fc) region involved in complement fixation and macrophage FcR binding; Zone Gamma marks the flexible hinge region; and Zone Delta marks the constant light (C_L) domain.

Final Answer: Zone Alpha (V_L / V_H)

Answer: (A)

Go Back to Question 15

Q16.

Solution

Concept: Hyperacute graft rejection occurs almost immediately (within minutes to hours) after blood flow is established to the transplanted organ, presenting as a severe type II hypersensitivity reaction.

Solution:

Let's analyze the pathophysiological mechanism of hyperacute rejection:

(a) This rapid rejection is mediated by **pre-formed, circulating antibodies in the recipient's serum** directed against ABO blood group antigens or Major Histocompatibility Complex (MHC/HLA) Class I molecules present on the donor vascular endothelium.

(b) As soon as the donor organ is anastomosed and blood flows through it, these antibodies immediately bind to the vascular endothelial cells of the graft.

(c) This binding activates the classical complement pathway, leading to endothelial damage, denudation, and the recruitment of neutrophils. This triggers thrombosis, vascular occlusion, and hemorrhagic infarction of the graft, turning the organ cyanotic and non-functional on the operating table.

Final Answer: Recipient Pre-formed Circulating Antibodies against ABO/HLA

Answer: (B)

Go Back to Question 16

Q17.

Solution

Concept: Echinocandins (such as caspofungin, micafungin, and anidulafungin) are a class of antifungal agents that target the fungal cell wall rather than the cell membrane, providing narrow-spectrum activity against specific fungal pathogens.

Solution:

Let's analyze the mycological features and drug mechanism:

(a) The clinical description—a bone marrow transplant recipient with fever, hemoptysis, and a "halo sign" on chest CT, showing dichotomously branching hyphae at 45° on Sabouraud agar—is diagnostic for invasive pulmonary **aspergillosis**.

(b) Echinocandins are often utilized as primary or salvage therapy in these high-risk settings. They function by non-competitively inhibiting the **β-(1,3)-D-glucan synthase enzyme** complex.

(c) This enzyme is responsible for synthesizing β-(1,3)-D-glucan, a major structural polysaccharide polymer that provides tensile strength and rigidity to the fungal cell wall. Inhibiting this enzyme weakens the cell wall, leaving the fungus vulnerable to osmotic lysis.

(d) Alternative mechanisms, such as ergosterol synthesis or squalene epoxidase inhibition, are targeted by polyenes, azoles, and allylamines (like terbinafine), which act on the fungal cell membrane rather than the cell wall.

Final Answer: β-(1,3)-D-glucan synthase enzyme

Answer: (B)

Go Back to Question 17

Q18.

Solution

Concept: *Histoplasma capsulatum* is a dimorphic fungus that exists as a mold in the environment and transforms into a small intracellular yeast at core body temperature (37°C) within host macrophages.

Solution:

Let's link the pathognomonic morphology to its ecological niche:

(a) The presentation describes an advanced HIV patient with a low CD4 count presenting with systemic symptoms. A bone marrow biopsy stained with Giemsa reveals numerous **small intracellular oval yeast cells within macrophages**, a classic finding for disseminated histoplasmosis.

(b) *Histoplasma capsulatum* is endemic to the **Ohio and Mississippi River valleys**. The fungus grows as a mold in soil enriched with **bat or bird excreta** (which contains high nitrogen levels that support its growth).

(c) Exposure typically occurs through the inhalation of microconidia aerosolized during activities like exploring caves (spelunking), clearing chicken coops, or remodeling old buildings in endemic regions.

Final Answer: Bat and bird excreta in river valleys

Answer: (B)

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Q19.

Solution

Concept: *Entamoeba histolytica* is a pathogenic intestinal protozoan that causes amebic dysentery and can metastasize hematogenously via the portal circulation to form amebic liver abscesses.

Solution:

Let's identify the molecular factor responsible for the colonization and pathogenicity of the parasite:

(a) The clinical description—a patient with a solitary right lobe hepatic abscess containing trophozoites that demonstrate **erythrophagocytosis** (ingested red blood cells)—is diagnostic for an infection caused by *Entamoeba histolytica*.

(b) To colonize the host, the amoeba must first attach to the colonic mucosa. This initial adherence is mediated by a specialized surface protein complex called the **galactose/N-acetyl-D-galactosamine (Gal/GalNAc) lectin**.

(c) This lectin binds specifically to galactose and GalNAc residues on the surface mucins and carbohydrates of host intestinal epithelial cells. This adherence is a required step before the amoeba can deploy its downstream cytolytic factors (like amoebapores and cysteine proteases) to invade tissue, cause "flask-shaped" mucosal ulcers, and migrate to the liver.

Final Answer: Gal/GalNAc lectin adherence molecule

Answer: (A)

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Q20.

Solution

Concept: The genus *Schistosoma* contains blood flukes (trematodes) whose eggs possess unique morphological features that allow for species-specific identification.

Solution:

Let's analyze the morphological features shown in the diagram:

(a) The presentation describes an immigrant patient from Egypt experiencing chronic hematuria, with a bladder biopsy revealing parasitic eggs containing a fully formed miracidium and a prominent **terminal spine**.

(b) This specific morphology—a large, elongated egg with a sharp, terminal spine at one pole—is diagnostic for ***Schistosoma hematobium***.

(c) Adults of *S. hematobium* reside in the venous plexuses of the urinary bladder and pelvis. The terminal spine helps the egg penetrate through the bladder wall into the lumen, allowing it to be excreted in urine. This process causes chronic inflammation, hematuria, and increases the long-term risk of squamous cell carcinoma of the bladder.

(d) For comparison, *Schistosoma mansoni* produces eggs with a prominent *lateral spine*, while *Schistosoma japonicum* produces rounder eggs with a small, inconspicuous *lateral knob*.

Final Answer: Schistosoma hematobium egg

Answer: (C)

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Answer Key

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