NEET PG Microbiology Sample Paper-8

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 intensive care unit patient on mechanical ventilation for 12 days develops new-onset fever, purulent sputum, and worsening oxygenation. Sputum culture yields Gram-negative bacilli that are obligate aerobes, oxidase-negative, and non-motile. Molecular profiling reveals the presence of the $bla_{\text{OXA}-23}$ gene. Which of the following resistance mechanisms or characteristics is most uniquely tied to the definitive therapeutic management of this specific pathogen?

(A) Outer membrane porin loss via CarO downregulation causing high-level meropenem resistance

(B) Mutations in the $gyrA$ and $parC$ genes completely rendering cefiderocol ineffective

(C) Modification of the lipid A moiety via phosphoethanolamine transferase coded by $mcr-1$

(D) Expression of a specialized metallo-β-lactamase capable of hydrolyzing aztreonam exclusively

Q2. A 34-year-old individual presenting with chronic, non-healing ulcerated cutaneous lesions along the draining lymphatics undergoes a skin biopsy. Acid-fast staining reveals intracellular bacilli. The laboratory attempts cultivation on Lowenstein-Jensen media at various temperatures. Optimal

growth is achieved strictly at 30°C after 6 weeks, with no growth observed at 37°C. Which molecular or pathogenic mechanism accounts for this unique temperature restriction profile?

(A) Thermolabile nature of the mycolyltransferase enzyme complex (*Ag*85) at core body temperature

(B) Inability to synthesize phenolic glycolipids (PGL-1) when incubated above 33°C

(C) Heat sensitivity of the alternative sigma factor σ^{E} required for heat-shock regulation

(D) Intrinsic structural instability of its cell wall arabinogalactan core polymer at higher thermal thresholds

Q3. A specimen of cerebrospinal fluid from a neonate with suspected septic meningitis grows short Gram-positive rods that exhibit characteristic end-over-end tumbling motility at room temperature (22°C) but are non-motile at 37°C. The virulence of this pathogen depends heavily on its ability to escape the phagolysosome into the host cytoplasm. Which bacterial factor directly coordinates this escape by forming pore complexes activated specifically by vacuolar acidification?

(A) Internalin A (InlA)

(B) Listeriolysin O (LLO)

(C) Actin assembly inducing protein (ActA)

(D) Phosphatidylinositol-specific phospholipase C (PI-PLC)

Q4. An emergency department patient presents with acute, severe watery diarrhea described as "rice-water stools" accompanied by profound dehydration. Pathogenesis involves an enterotoxin ADP-ribosylating a regulatory G-protein subunit. Which specific biochemical event immediately follows this intracellular modification, directly driving the hypersecretion of electrolytes into the intestinal lumen?

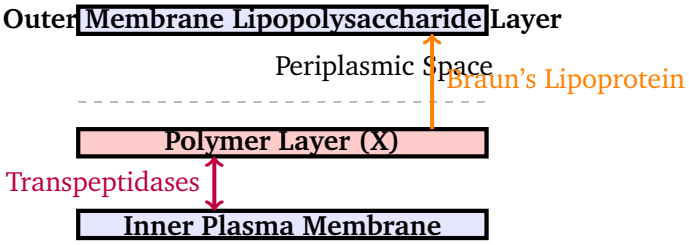
(A) Irreversible activation of adenylate cyclase leading to a massive intracellular spike in cAMP

(B) Persistent inhibition of guanylate cyclase preventing the formation of intracellular cGMP

(C) Downregulation of protein kinase A (PKA) activity across the brush border membrane

(D) Direct blocks on basolateral Na^{+}-K^{+}-$2Cl^{-}$ cotransporters via calcium channel blockades

Q5. A reference laboratory isolates a multi-drug resistant enteric pathogen and maps its cell envelope macromolecular layout to evaluate outer membrane integrity during beta-lactam stress. Examine the schematic topological cross-section of the cell wall layer provided below:



Identify the definitive structural component labeled as **Polymer Layer (X)** and its specific composition characteristically cleaved by the action of lysozyme molecules:

(A) Teichoic acid backbone repeating units linked via D-alanine esters

(B) Lipopolysaccharide core oligosaccharide bonded via lipid A anchors

(C) Peptidoglycan mesh network containing alternating β-(1,4) linked NAG and NAM residues

(D) Arabinogalactan-mycolic acid complex associated with porin channels

Q6. A 29-year-old human immunodeficiency virus (HIV) positive patient with a CD4 count of 45/μL presents with progressive neurological decline, cognitive deficits, and visual field defects. Cranial magnetic resonance imaging shows asymmetric, non-enhancing white matter demyelinating lesions without mass effect. PCR of the cerebrospinal fluid confirms JC virus DNA. Which specific central nervous system cell line is preferentially targeted for lytic replication by this polyomavirus?

(A) Microglial cells

(B) Perivascular macrophages

(C) Oligodendrocytes

(D) Proline-rich cortical neurons

Q7. An infant presenting with severe bouts of barking cough, inspiratory stridor, and respiratory distress is diagnosed with croup. The etiological viral agent belongs to the Paramyxoviridae family. During the viral entry and assembly pathway, which pair of surface glycoproteins handles host cell membrane receptor attachment and subsequent syncytia formation, respectively?

(A) Hemagglutinin-Neuraminidase (HN) and Fusion (F) protein

(B) Glycoprotein gp120 and gp41 complex

(C) Hemagglutinin (H) and Neuraminidase (N) independent subunits

(D) Glycoprotein E1 and E2 heterodimers

Q8. A clinical trial analyzes the replication kinetics of Hepatitis B Virus (HBV) in hepatocytes to establish the mechanism of a novel nucleoside analogue. Which of the following chronological sequences accurately outlines the intracellular lifecycle stages of the HBV genome immediately following uncoating and entry into the host nucleus?

(A) rcDNA conversion → cccDNA formation → Transcription of pgRNA → Reverse transcription inside capsid

(B) pgRNA packaging → Direct integration into host chromosome → Excision → dsDNA synthesis

(C) cccDNA formation → Translation of structural proteins → Splicing → RNA-templated DNA transposition

(D) rcDNA cleavage → Positive-strand synthesis → Assembly → Nuclear retrotransposition

Q9. A 58-year-old bone marrow transplant recipient develops severe, bilateral interstitial pneumonitis. Shell vial culture and subsequent immunohistochemistry demonstrate enlarged cells with eccentric nuclei surrounded by

a clear halo ("owl's eye" inclusion bodies). Which viral gene product represents the primary target of ganciclovir phosphorylation required to active its monophosphate form inside these infected cells?

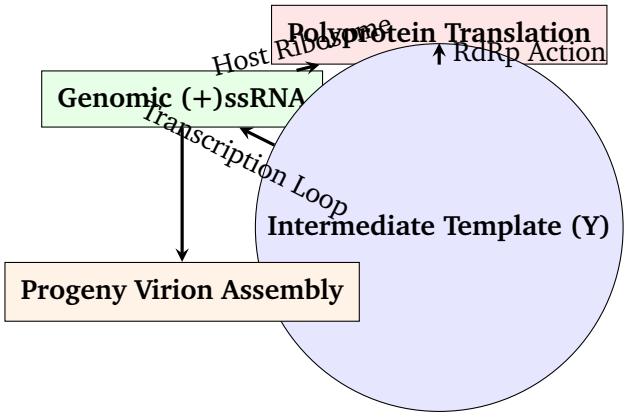
(A) Viral Thymidine Kinase coded by open reading frame $UL23$

(B) Protein Kinase phosphotransferase encoded by the $UL97$ gene

(C) Ribonucleotide reductase large subunit complex

(D) DNA polymerase catalytic subunit encoded by $UL54$

Q10. A virology diagnostics team creates a schematic kinetic vector mapping model for an asymmetric positive-sense single-stranded RNA virus (ssRNA) replicating inside host cytoplasm. Refer to the structural replication loop flow diagram below:



In this classic positive-strand viral genomic replication cycle, determine the exact molecular identity of the intermediate structure labeled as **Intermediate Template (Y)**:

(A) Double-stranded circular episomal DNA

(B) Complementary Negative-sense single-stranded RNA [(-)ssRNA]

(C) Integrated retroviral proviral DNA element

(D) Subgenomic messenger RNA loop segment

Q11. A 42-year-old farmer from a rural region presents with a chronic, granulomatous verrucous lesion on his right foot following a minor trauma caused by thorny vegetation several months ago. Histopathological examination

of a tissue biopsy reveals characteristic thick-walled, dark brown, multiseptate fungal structures resembling copper pennies (Medlar bodies). Which mycological condition is most consistently associated with these diagnostic features?

(A) Mycetoma

(B) Chromoblastomycosis

(C) Sporotrichosis

(D) Lobomycosis

Q12. An immunocompromised patient presenting with low-grade fever, non-productive cough, and dyspnea exhibits bilateral ground-glass opacities on a chest high-resolution CT scan. Silver staining of bronchoalveolar lavage fluid shows collapsed, cup-shaped cystic forms. Which unique biological element characterizes the cell membrane or wall of this specific fungal pathogen, distinguishing it from most conventional fungi?

(A) Total absence of ergosterol in the membrane, substituted instead by cholesterol

(B) High concentrations of alpha-(1,3)-glucan lacking any chitin networks

(C) Thick outer capsule composed purely of glucuronoxylomannan polymers

(D) Inability to synthesize beta-(1,3)-D-glucan chains under any conditions

Q13. A 38-year-old traveler returning from an extended safari in East Africa presents with an irregular remittent fever, severe headaches, and progressive somnolence. A peripheral blood film shows long, slender, flagellated trypomastigotes. This pathogen is renowned for its capacity to cause recurrent waves of parasitemia through decades of infection. What molecular mechanism enables this evasion of the host's adaptive immune system?

(A) Continuous antigenic variation driven by the sequential expression of thousands of distinct Variant Surface Glycoprotein (VSG) genes

(B) Intracellular replication within specialized parasitophorous vacuoles in splenic red pulp macrophages

(C) Direct proteolytic cleavage of host immunoglobulin heavy chains via secreted cysteine endopeptidases

(D) Shedding of decoy lipophosphoglycan complexes that fixed complement proteins away from the plasma membrane

Q14. A biopsy specimen of the small intestine from a patient suffering from severe, persistent malabsorptive diarrhea and steatorrhea is evaluated under microscopy. The specimen reveals flagellated, pear-shaped trophozoites with two nuclei and a distinct ventral sucking disk. Which immunological pathway is primarily responsible for clearing this non-invasive protozoan parasite from the mucosal surface of the upper gastrointestinal tract?

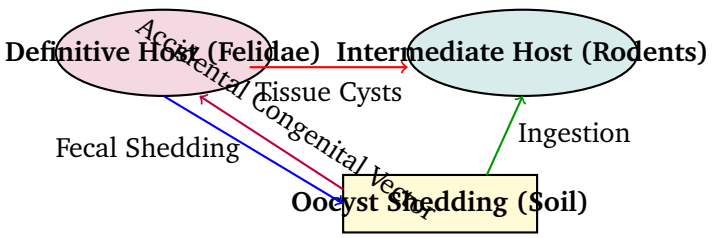
(A) Perforin-mediated lysis driven by CD8+ cytotoxic T lymphocytes

(B) Secretory IgA antibodies that block parasite attachment to the enterocyte brush border

(C) Complement-mediated lysis initiated via the alternative pathway

(D) Eosinophil degranulation releasing major basic protein directly onto target cuticles

Q15. An epidemiologist traces transmission dynamics for a tissue-dwelling protozoan parasite responsible for focal necrotizing retinochoroiditis and intracranial calcifications. Consider the life-cycle flow schematic layout mapping its host interaction checkpoints detailed below:



Identify this parasite, which undergoes sexual reproduction exclusively within the intestinal epithelial cells of its definitive host as diagrammed:

(A) *Leishmania donovani*

(B) *Toxoplasma gondii*

(C) *Cryptosporidium parvum*

(D) *Trypanosoma cruzi*

Q16. A newborn child exhibits recurrent pyogenic bacterial infections, delayed separation of the umbilical cord, and severe leukocytosis without pus formation at infection sites. Flow cytometry confirms a profound deficiency in leukocyte adhesion molecules. Which specific cell surface molecule or integrin subunit is characteristically absent or defective in this type 1 leukocyte adhesion deficiency (LAD-1)?

(A) CD11a/CD18 integrin complex (β_2 integrin subunit)

(B) Sialyl-Lewis X ligand on circulating neutrophils

(C) Intercellular Adhesion Molecule 1 (ICAM $-$ 1) on endothelial borders

(D) GlyCAM-1 addressins within high endothelial venules

Q17. A 5-year-old boy presents with recurrent, severe sinopulmonary infections caused by *Catalase-positive* organisms such as *Staphylococcus aureus* and *Aspergillus fumigatus*. A dihydrorhodamine (DHR) 123 flow cytometry assay demonstrates an absence of green fluorescence production upon neutrophil activation. Which specific multi-protein enzymatic complex is structurally disrupted or non-functional in this patient's phagocytes?

(A) Myeloperoxidase (MPO) enzyme matrix

(B) Nicotinamide adenine dinucleotide phosphate (NADPH) oxidase complex

(C) Lysosomal trafficking regulator protein (LYST)

(D) Acid sphingomyelinase path network

Q18. During a fundamental research seminar on the structural basis of adaptive immunity, the mechanism of somatic hypermutation is evaluated. This process alters the antigen-binding affinity of immunoglobulins within the germinal centers of peripheral lymphoid organs. Which enzyme is explicitly required to initiate this process by actively deaminating cytosine bases into uracil within single-stranded genomic DNA targets?

(A) Recombination Activating Gene 1 (RAG − 1)

(B) Terminal Deoxynucleotidyl Transferase (TdT)

(C) Activation-Induced Cytidine Deaminase (AID)

(D) Uracil DNA Glycosylase (UNG)

Q19. A patient presenting with recurrent episodes of angioedema without associated urticaria or pruritus is found to have low C4 complement levels. Further testing confirms a quantitative deficiency in C1 esterase inhibitor. Which of the following pathophysiological consequences directly ensues from this specific inhibitor deficiency, triggering the localized vascular permeability seen in these clinical presentations?

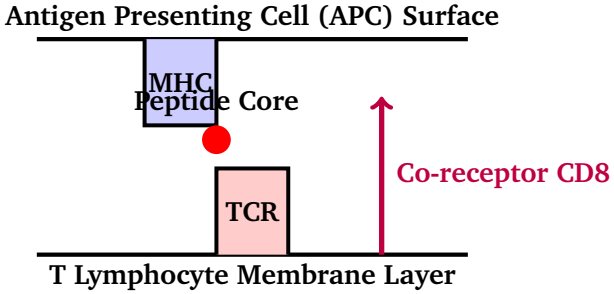
(A) Unchecked consumption of C2 and C4 leading to excessive generation of bradykinin

(B) Hyperactivation of the alternative complement loop driven by factor B stabilization

(C) Inability of decay-accelerating factor (DAF) to protect vascular endothelial tissues

(D) Accelerated assembly of the Membrane Attack Complex (C5b-9) on host surfaces

Q20. An immunochemistry group analyzes antigen processing pathways involved in cell-mediated immune responses. Examine the topological map depicting the trimolecular interaction complex between an Antigen-Presenting Cell (APC) and a T lymphocyte line:



Based on the inclusion of the **Co-receptor CD8** interaction arm depicted in this structural configuration, specify the restriction class of the matching

Major Histocompatibility Complex (MHC) molecule and its cellular processing source:

(A) MHC Class II molecule presenting exogenously derived peptides processed within endolysosomes

(B) MHC Class I molecule presenting endogenously synthesized viral or tumor proteins processed by proteasomes

(C) CD1d non-classical molecules displaying glycolipid fractions derived from mycobacterial walls

(D) MHC Class III molecular configurations handling soluble complement factor segments

Detailed Solutions

Q1.

Solution

Concept: *Acinetobacter baumannii* is an opportunistic, non-motile, oxidase-negative, obligately aerobic Gram-negative bacillus frequently causing ventilator-associated pneumonia (VAP) in intensive care settings, characterized by extensive multidrug-resistance profiles.

Solution:

Let's analyze the clinical scenario and the specific diagnostic markers described:

(a) The isolation of a non-motile, oxidase-negative, aerobic Gram-negative rod from a patient on prolonged mechanical ventilation points directly to *Acinetobacter baumannii*. The presence of the $**bla_{\text{OXA}-23}**$ gene indicates the production of an OXA-type Class D carbapenemase, which confers resistance to carbapenems.

(b) Resistance management in *Acinetobacter baumannii* is complex and often multi-factorial. High-level resistance to meropenem and imipenem in these strains is frequently compounded by **outer membrane porin loss via CarO downregulation**, which limits antibiotic entry into the periplasmic space.

(c) Let's evaluate why the alternative choices are incorrect:

- Mutations in $gyrA$ and $parC$ alter DNA gyrase and topoisomerase IV, causing resistance to fluoroquinolones, not the siderophore cephalosporin cefiderocol.
- Colistin resistance in *Acinetobacter* is driven by chromosomal mutations in the *pmrAB* two-component regulatory system rather than the plasmid-borne *mcr-1* gene typically found in *Enterobacteriaceae*.
- Metallo-β-lactamases (MBLs) are notable for their ability to hydrolyze almost all β-lactams *except* aztreonam.

Final Answer: Outer membrane porin loss via CarO downregulation causing high-level meropenem resistance

Answer: (A)

Go Back to Question 1

Q2.

Solution

Concept: Certain non-tuberculous mycobacteria (NTM), such as *Mycobacterium marinum* and *Mycobacterium ulcerans*, exhibit strict temperature-dependent growth phenotypes that restrict their pathology to the cooler peripheral extremities of the host body.

Solution:

Let's trace the pathophysiological basis of this thermal restriction:

(a) The clinical description—chronic skin ulcers demonstrating a "sporotrichoid" pattern along lymphatic channels, with acid-fast bacilli that grow optimally at 30°C but fail to grow at core body temperature (37°C)—is diagnostic for *Mycobacterium marinum*.

(b) This strict temperature restriction is due to the **thermolabile nature of its essential mycolyltransferase enzyme complex (Antigen 85 complex)**.

(c) At higher core body temperatures (37°C), these crucial cell wall synthesis enzymes undergo structural destabilization and inactivation, rendering the organism unable to assemble its mycolic acid-rich outer envelope and halting replication completely.

Final Answer: Thermolabile nature of the mycolyltransferase enzyme complex (Ag85) at core body temperature

Answer: (A)

Go Back to Question 2

Q3.

Solution

Concept: *Listeria monocytogenes* is a foodborne intracellular pathogen capable of crossing the blood-brain and placental barriers, using specialized virulence factors to escape host immune destruction.

Solution:

Let's analyze the intracellular survival mechanisms of this pathogen:

(a) The description of short Gram-positive rods causing neonatal meningitis with characteristic end-over-end **tumbling motility at 22°C** but non-motile behavior at 37°C is diagnostic for *Listeria monocytogenes*.

(b) Once phagocytosed by macrophages or non-professional phagocytes, the bacterium is enclosed within a primary phagosome. To survive, it must rapidly escape into the host cell cytoplasm before phagolysosomal fusion occurs.

(c) This escape is driven primarily by **Listeriolysin O (LLO)**, a cholesterol-dependent cytolysin. LLO is uniquely adapted to be **activated specifically by vacuolar acidification** (pH ≈ 5.5). It forms trans-membrane pore complexes in the phagosomal membrane, destabilizing the vacuole and allowing the bacterium to exit into the nutrient-rich cytoplasm.

Final Answer: Listeriolysin O (LLO)

Answer: (B)

Go Back to Question 3

Q4.

Solution

Concept: Cholera toxin, produced by *Vibrio cholerae*, is a classic AB_5-subunit enterotoxin that subverts host cell signaling pathways to cause massive, life-threatening secretory diarrhea.

Solution:

Let's map the biochemical cascade initiated by the toxin:

(a) The active A-subunit of cholera toxin is translocated into the cytosol of intestinal epithelial cells, where it catalyzes the **ADP-ribosylation of the $G_{\alpha s}$ regulatory protein subunit**.

(b) This modification permanently locks $G_{\alpha s}$ in its active, GTP-bound state by completely abolishing its intrinsic GTPase activity.

(c) The persistently active $G_{\alpha s}$ protein causes **irreversible activation of adenylate cyclase**, triggering an immediate and massive intracellular spike in **cyclic adenosine monophosphate (cAMP)**. High cAMP levels overactivate Protein Kinase A (PKA), which phosphorylates and opens the CFTR chloride channels, driving the hypersecretion of Cl^-, Na^+, and water into the intestinal lumen, producing characteristic "rice-water stools."

Final Answer: Irreversible activation of adenylate cyclase leading to a massive intracellular spike in cAMP

Answer: (A)

Go Back to Question 4

Q5.

Solution

Concept: The bacterial cell wall contains a robust, cross-linked glycan polymer meshwork that maintains osmotic stability and structural integrity, serving as the primary target for several cell wall-active agents and host defense enzymes.

Solution:

Let's evaluate the structural arrangement from the diagram:

(a) The cross-sectional diagram models a classic Gram-negative cell envelope, showing an inner plasma membrane, a thin intermediate polymer layer within the periplasmic space, and an outer lipopolysaccharide membrane linked by Braun's lipoprotein. This identifies **Polymer Layer (X)** as the **peptidoglycan (murein) layer**.

(b) Peptidoglycan is a complex heteropolymer consisting of alternating glycan strands of **N-acetylglucosamine (NAG) and N-acetylmuramic acid (NAM)** linked together by **β-(1,4) glycosidic bonds**, which are further reinforced by short cross-linking peptide side chains attached to the NAM residues.

(c) Host lysozyme molecules (found in tears, saliva, and mucus) specifically target and **cleave these β-(1,4) glycosidic bonds** between NAG and NAM. Breaking these bonds destabilizes the structural mesh network, leading to bacterial cell lysis under osmotic pressure.

Final Answer: Peptidoglycan meshwork composed of alternating *N*-acetylglucosamine (NAG) and *N*-acetylmuramic acid (NAM) residues linked by β-(1,4) glycosidic bonds.

Answer: (C)

Go Back to Question 5

Q6.

Solution

Concept: Progressive Multifocal Leukoencephalopathy (PML) is a fatal demyelinating disease of the central nervous system that occurs in severely immunosuppressed individuals due to the opportunistic reactivation of the John Cunningham (JC) polyomavirus.

Solution:

Let's analyze the cellular tropism of the pathogenic JC virus in the brain:

(a) The clinical presentation—an advanced HIV patient with a low CD4 count experiencing cognitive decline, paired with an MRI showing asymmetric, non-enhancing white matter lesions without mass effect—is the classic presentation of PML.

(b) Demyelination in PML occurs because the reactivated, neurotropic JC virus selectively targets and infects **oligodendrocytes**, the myelin-producing cells of the central nervous system.

(c) The virus undergoes highly productive, lytic replication within oligodendrocytes, leading to cell death. The loss of these cells prevents myelin maintenance, resulting in patchy, widespread white matter demyelination and progressive neurological failure.

Final Answer: Oligodendrocytes

Answer: (C)

Go Back to Question 6

Q7.

Solution

Concept: Human Parainfluenza Viruses, members of the *Paramyxoviridae* family, cause laryngotracheobronchitis (croup) in infants, utilizing specialized surface envelope glycoproteins to enter host cells and mediate cell-to-cell fusion.

Solution:

Let's examine the roles of the surface glycoproteins in paramyxoviruses:

(a) Paramyxoviruses are enveloped, negative-sense, single-stranded RNA viruses. Their lipid envelope contains two main surface projections that act sequentially during pathogenesis.

(b) The first is the **Hemagglutinin-Neuraminidase (HN)** glycoprotein (or H/G proteins in other genera), which recognizes and binds to sialic acid receptors on the surface of host respiratory epithelial cells to mediate attachment.

(c) The second is the **Fusion (F) protein**, which undergoes a structural change after attachment to fuse the viral envelope directly with the host plasma membrane. Crucially, the F protein is also expressed on the surface of infected cells, where it can fuse with adjacent uninfected cells at physiological pH, leading to the formation of multinucleated giant cells (**syncytia**).

Final Answer: Hemagglutinin-Neuraminidase (HN) and Fusion (F) protein

Answer: (A)

Go Back to Question 7

Q8.

Solution

Concept: Hepatitis B Virus (HBV) is a partially double-stranded DNA virus (Hepatnaviridae) that replicates its genome through an intracellular RNA intermediate using an error-prone reverse transcription step.

Solution:

Let's follow the chronological steps of the HBV genome lifecycle inside an infected hepatocyte nucleus:

(a) Upon entry and uncoating, the viral **relaxed circular DNA (rcDNA)** genome is delivered into the host nucleus.

(b) Host cellular repair enzymes patch the gaps in the rcDNA, converting it into a stable, supercoiled **covalently closed circular DNA (cccDNA)** episome.

(c) This cccDNA acts as the stable transcriptional template for host RNA polymerase II, which transcribes viral mRNAs, including the greater-than-genome-length **pregenomic RNA (pgRNA)**.

(d) The pgRNA is exported to the cytoplasm, where it binds to the viral polymerase and is packaged into emerging core viral capsids. Inside these capsids, the viral polymerase **reverse-transcribes the pgRNA** back into new progeny rcDNA.

Final Answer: Relaxed circular DNA (rcDNA) conversion → covalently closed circular DNA (cccDNA) formation → transcription of pregenomic RNA (pgRNA) → reverse transcription within the nucleocapsid (capsid).

Answer: (A)

Go Back to Question 8

Q9.

Solution

Concept: Cytomegalovirus (CMV) is a double-stranded DNA herpesvirus (β-herpesvirinae) that causes severe opportunistic diseases, such as pneumonitis and retinitis, in immunocompromised hosts.

Solution:

Let's examine the mechanism of action and activation of ganciclovir:

(a) The clinical description—interstitial pneumonitis in a bone marrow transplant recipient showing enlarged cells with "owl's eye" intranuclear inclusion bodies—is diagnostic for CMV.

(b) Ganciclovir is a nucleoside analogue that inhibits viral DNA synthesis. However, it is administered as a prodrug that must undergo three sequential phosphorylation steps to become active.

(c) Unlike Herpes Simplex Virus (HSV) and Varicella-Zoster Virus (VZV), which encode a viral thymidine kinase (*UL23*), CMV does not possess a thymidine kinase gene. Instead, the initial, rate-limiting monophosphorylation step is catalyzed by a specific **protein kinase phosphotransferase encoded by the viral *UL97* gene**. Once monophosphorylated by UL97, host cell enzymes convert it into the active ganciclovir triphosphate form, which competitively inhibits the viral DNA polymerase (*UL54*).

Final Answer: Protein Kinase phosphotransferase encoded by the UL97 gene

Answer: (B)

Go Back to Question 9

Q10.

Solution

Concept: Positive-sense, single-stranded RNA viruses [(+)ssRNA] can use their genomic RNA directly as mRNA for immediate translation upon entering the host cell cytoplasm, but they require a complementary negative-sense template for genomic replication.

Solution:

Let's analyze the replication loop modeled in the flow diagram:

(a) Upon entering the host cytosol, the incoming **Genomic (+)ssRNA** is recognized by host ribosomes and translated into a single large polyprotein, which is then cleaved into functional structural and non-structural proteins.

(b) Among these non-structural elements is the **RNA-dependent RNA Polymerase (RdRp)**. The RdRp binds to the genomic (+)ssRNA and transcribes a full-length, complementary **Negative-sense single-stranded RNA [(-)ssRNA]** strand, which serves as **Intermediate Template (Y)**.

(c) This negative-sense intermediate template then functions as the master strand for the synthesis of multiple new positive-sense ssRNA strands via the transcription loop. These progeny (+)ssRNA strands are either translated further or packaged into new virions.

Final Answer: Complementary Negative-sense single-stranded RNA [(-)ssRNA]

Answer: (B)

Go Back to Question 10

Q11.

Solution

Concept: Chromoblastomycosis is a chronic, subcutaneous fungal infection seen in tropical and subtropical regions, caused by the traumatic inoculation of pigmented (dematiaceous) environmental molds.

Solution:

Let's analyze the clinical and histopathological findings:

(a) The patient is an agricultural worker who developed a slow-growing, cauliflower-like, granulomatous verrucous skin lesion on the lower extremity following minor trauma from vegetation.

(b) A definitive diagnosis is established by the presence of **Medlar bodies (also known as sclerotic bodies or copper pennies)** in tissue section. These are thick-walled, dark brown, multiseptate fungal cells that divide by internal cleavage rather than budding.

(c) The production of these pigmented sclerotic bodies within subcutaneous granulomas is pathognomonic for **chromoblastomycosis**, typically caused by dematiaceous molds such as *Fonsecaea pedrosoi* or *Cladophialophora carrionii*.

Final Answer: Chromoblastomycosis

Answer: (B)

Go Back to Question 11

Q12.

Solution

Concept: *Pneumocystis jirovecii* is an atypical, opportunistic fungus that causes severe pneumonia in immunocompromised individuals, demonstrating unique structural differences in its cell membrane compared to conventional fungi.

Solution:

Let's review the structural biology of this unique fungal pathogen:

(a) The combination of bilateral ground-glass lung opacities on CT and the presence of collapsed, cup-shaped cystic forms on silver stain is diagnostic for *Pneumocystis jirovecii* pneumonia (PCP).

(b) While *P. jirovecii* is classified phylogenetically as a fungus due to its nucleic acid sequences and cell wall glucan components, its cell membrane contains a unique structural difference: it demonstrates a **total absence of ergosterol**, which is replaced instead by **cholesterol**.

(c) Because it lacks an ergosterol target, *P. jirovecii* is completely resistant to conventional empirical antifungal agents like amphotericin B and azoles, requiring management with anti-folate regimens (TMP-SMX) instead.

Final Answer: Total absence of ergosterol in the membrane, substituted instead by cholesterol

Answer: (A)

Go Back to Question 12

Q13.

Solution

Concept: *Trypanosoma brucei*, the extracellular protozoan agent of African Sleeping Sickness, evades host humoral immunity by continually changing its surface coat antigens.

Solution:

Let's analyze the mechanism behind the characteristic waves of parasitemia:

(a) The presentation describes a patient with East African trypanosomiasis (sleeping sickness), showing irregular fevers, headaches, somnolence, and slender flagellated trypomastigotes in a blood film.

(b) The host immune system mounts a robust IgM antibody response against the parasite's predominant surface coat antigen. However, just as the antibody titer clears the main population, a new wave of parasites emerges with a different surface antigen, leading to recurrent parasitemia.

(c) This evasion is driven by **continuous antigenic variation via the sequential expression of thousands of distinct Variant Surface Glycoprotein (VSG) genes**. The parasite genome contains a large repertoire of telomeric *VSG* genes, but expresses only one at a time. Periodic gene conversion and transcriptional switching swap the active *VSG* gene, allowing the parasite to continuously evade the adaptive immune response.

Final Answer: Continuous antigenic variation driven by the sequential expression of thousands of distinct Variant Surface Glycoprotein (VSG) genes

Answer: (A)

Go Back to Question 13

Q14.

Solution

Concept: *Giardia lamblia* is a non-invasive flagellated protozoan that colonizes the lumen of the duodenum and jejunum, requiring mucosal immune responses for control and clearance.

Solution:

Let's evaluate the primary immune mechanism used to clear this parasite:

(a) The presence of flagellated, pear-shaped trophozoites with two nuclei and a ventral sucking disk in a patient with malabsorptive diarrhea and steatorrhea is diagnostic for *Giardia lamblia*.

(b) Because *Giardia* is a non-invasive luminal parasite that attaches to enterocytes without invading tissue, internal cellular immune defenses like cytotoxic CD8+ T cells or alternative complement lysis are largely ineffective.

(c) The primary immunological pathway responsible for clearing *Giardia* is the production of **secretory IgA (sIgA) antibodies**. B cells in the lamina propria synthesize dimeric IgA, which is transported across the enterocytes into the gut lumen. These sIgA antibodies specifically bind to surface antigens on the trophozoite, physically blocking its ventral disk from attaching to the microvilli and allowing the parasite to be cleared by normal peristalsis.

Final Answer: Secretory IgA antibodies that block parasite attachment to the enterocyte brush border

Answer: (B)

Go Back to Question 14

Q15.

Solution

Concept: *Toxoplasma gondii* is an obligate intracellular apicomplexan protozoan with a complex lifecycle that relies on specific definitive and intermediate hosts for sexual and asexual reproduction.

Solution:

Let's analyze the host pathways shown in the lifecycle diagram:

(a) The question describes a tissue parasite that causes necrotizing retinochoroiditis and congenital intracranial calcifications.

(b) The diagram maps out a lifecycle where members of the **Felidae (cat) family serve as the definitive host**, while rodents or other mammals serve as intermediate hosts.

(c) ***Toxoplasma gondii*** undergoes sexual reproduction exclusively within the intestinal epithelial cells of felines, leading to the shedding of unembryonated **oocysts** into the environment via feces. Ingestion of these oocysts or tissue cysts by intermediate hosts leads to asexual replication (tachyzoites and bradyzoites). Humans can become infected accidentally through contact with oocysts in soil/feces or via congenital transmission.

Final Answer: Toxoplasma gondii

Answer: (B)

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Q16.

Solution

Concept: Leukocyte Adhesion Deficiency Type 1 (LAD-1) is an autosomal recessive primary immunodeficiency characterized by a defect in leukocyte integrins that prevents firm adhesion and transendothelial migration.

Solution:

Let's identify the molecular defect underlying this immunodeficiency:

(a) The clinical triad of recurrent pyogenic infections, **delayed detachment of the umbilical cord**, and severe leukocytosis/neutrophilia without pus formation is the classic presentation of LAD-1.

(b) Leukocytes from these patients roll normally along the vascular endothelium via selectins but are unable to bind tightly or arrest because they lack functional integrins.

(c) LAD-1 is caused by mutations in the *ITGB2* gene, which encodes **CD18 (the β_2 integrin subunit)**. CD18 normally pairs with various alpha subunits to form critical adhesion molecules, including LFA-1 (**CD11a/CD18**), Mac-1 (CD11b/CD18), and p150,95 (CD11c/CD18). Without the CD18 subunit, these complexes cannot assemble on the leukocyte cell surface, preventing tight binding to endothelial ICAM-1.

Final Answer: CD11a/CD18 integrin complex (β_2 integrin subunit)

Answer: (A)

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Q17.

Solution

Concept: Chronic Granulomatous Disease (CGD) is an immunodeficiency characterized by the inability of phagocytes to produce reactive oxygen species (ROS), leaving patients susceptible to recurrent infections with catalase-positive organisms.

Solution:

Let's break down the laboratory assay and the underlying defect:

(a) A patient presenting with recurrent sinopulmonary infections from catalase-positive organisms (*Staphylococcus aureus*, *Aspergillus*) who shows an **absence of green fluorescence in a dihydrorhodamine (DHR) 123 assay** has a diagnosis of CGD. The DHR flow cytometry assay measures the oxidation of rhodamine by respiratory burst products.

(b) The primary molecular defect in CGD is a mutation in one of the subunits of the **nicotinamide adenine dinucleotide phosphate (NADPH) oxidase complex** (most commonly the X-linked *CYBB* gene encoding gp91phox).

(c) The NADPH oxidase complex is responsible for converting molecular oxygen into superoxide anions ($\cdot O_2^-$), the initial step in the respiratory burst pathway. Without this functional complex, phagocytes cannot generate the microbicidal oxidative radicals required to kill ingested catalase-positive pathogens.

Final Answer: Nicotinamide adenine dinucleotide phosphate (NADPH) oxidase complex

Answer: (B)

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Q18.

Solution

Concept: Somatic hypermutation occurs within the germinal centers of peripheral lymphoid organs, introducing point mutations into the V regions of immunoglobulin heavy and light chain genes to drive affinity maturation.

Solution:

Let's identify the enzyme responsible for initiating this mutational process:

(a) Somatic hypermutation and class-switch recombination require a specialized enzyme to alter DNA sequence configuration within activated B cells.

(b) This master initiating enzyme is **Activation-Induced Cytidine Deaminase (AID)**.

(c) AID functions by directly **deaminating cytosine bases into uracil** (C → U) within single-stranded regions of targeting genomic DNA during active transcription. The resulting uracil-guanine mismatches are then processed by error-prone DNA repair pathways, generating the targeted point mutations that alter antibody-binding affinity.

Final Answer: Activation-Induced Cytidine Deaminase (AID)

Answer: (C)

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Q19.

Solution

Concept: Hereditary Angioedema (HAE) is an autosomal dominant disorder caused by a quantitative or qualitative deficiency in C1 esterase inhibitor (C1-INH), a key regulatory protein that controls both complement and contact activation pathways.

Solution:

Let's trace the link between C1-INH deficiency and vascular permeability:

(a) A patient presenting with recurrent episodes of non-pitting angioedema without hives, accompanied by chronically low C4 levels and a documented deficiency in C1-INH, has HAE.

(b) C1-INH normally downregulates the classical complement pathway by dissociating the active C1 complex. Consequently, its absence leads to the **unchecked consumption of C2 and C4**.

(c) Crucially, C1-INH is also the primary inhibitor of **plasma kallikrein** and coagulation factor XIIa within the contact activation system. Without C1-INH to suppress kallikrein activity, there is overproduction of the vasoactive peptide **bradykinin**. Bradykinin binds to endothelial B_2 receptors, promoting profound intracellular gap formation and localized vascular permeability that triggers angioedema.

Final Answer: Unchecked consumption of C2 and C4 leading to excessive generation of bradykinin

Answer: (A)

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Q20.

Solution

Concept: T-lymphocyte activation relies on specific coreceptor interactions that match the restriction class and intracellular processing source of the presented peptide antigen.

Solution:

Let's analyze the trimolecular synapse model illustrated in the diagram:

(a) The diagram depicts an Antigen-Presenting Cell (APC) surface presenting a core peptide molecule within an MHC pocket to a T-cell receptor (TCR). This interaction is stabilized by the **CD8 co-receptor** arm.

(b) The CD8 glycoprotein coreceptor binds specifically to the invariant α_3 domain of **MHC Class I molecules**. It cannot interact with MHC Class II molecules, which are restricted instead to CD4+ T helper cells.

(c) MHC Class I molecules present **endogenous antigens** (such as viral proteins or mutant tumor proteins synthesized directly within the cytoplasm). These cytosolic proteins are degraded by the **proteasome**, transported into the rough endoplasmic reticulum via the TAP complex, loaded onto MHC Class I heterodimers, and trafficked to the cell surface for presentation to CD8+ cytotoxic T lymphocytes.

Final Answer: MHC Class I molecule presenting endogenously synthesized viral or tumor proteins processed by proteasomes

Answer: (B)

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Answer Key

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