NEET PG Microbiology Sample Paper-2

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 44-year-old high-altitude trekker returns from an expedition with a deep, necrotic puncture wound on his right calf that rapidly develops crepitus, severe localized pain, and a foul-smelling serosanguinous discharge. Gas gangrene is suspected. Anaerobic cultivation on egg yolk agar demonstrates a distinct zone of opalescence surrounding the colonies, which is completely inhibited on the side of the plate pre-treated with specific anti-alpha-toxin. Which specific enzymatic mechanism explains the biochemical zone of opalescence observed in this diagnostic test?

(A) Cleavage of sphingomyelin into ceramide and phosphorylcholine

(B) Hydrolysis of phosphatidylcholine into diacylglycerol and phosphorylcholine

(C) Digestion of mucopolysaccharides via hyaluronidase activation

(D) Zinc-dependent metalloproteinase cleavage of ganglioside GM1 receptors

Q2. During a molecular surveillance investigation of nosocomial outbreaks in an intensive care unit, an isolate of *Klebsiella pneumoniae* is recovered that demonstrates high-level resistance to ceftazidime, aztreonam, and imipenem. Phenotypic confirmation confirms the production of a metallo-β-lactamase (MBL). Which of the following biochemical parameters or structural con-

figurations specifically characterizes this organism's resistance mechanism?

(A) Active-site serine-mediated nucleophilic attack inhibited by clavulanic acid

(B) D-Alanyl-D-Lactate peptidoglycan cell wall remodeling mediated by the vanA operon

(C) Utilization of divalent zinc ions to facilitate water molecule activation for hydroxyl attack on the β-lactam ring

(D) Covalent modifications via aminoglycoside-modifying enzymes co-translocated on the pKPC plasmid

Q3. A 31-year-old researcher accidentally punctures her finger while handling culture plates of an unknown Gram-negative coccobacillus isolated from a wild rodent carcass. Within 48 hours, she develops acute high fever, excruciatingly painful left axillary lymphadenopathy (bubo), and petechiae. Safety protocols require immediate rule-out of *Yersinia pestis*. Which specific virulence factor configuration allows this pathogen to directly resist phagocytosis by host macrophages during the early systemic phase?

(A) Thermolabile capsular F1 antigen expressed at 37°C combined with Yop H/E injection via Type III secretion system

(B) IgA1 protease cleavage coupled with lipid A modification by lipid IVA desaturase

(C) High-affinity iron acquisition via aerobactin siderophore up-regulation at low temperatures

(D) Cord factor-mediated disruption of mitochondrial oxidative phosphorylation inside neutrophilic phagosomes

Q4. A reference reference reference microbiology unit processes an isolate from a patient suspected of hosting multi-drug resistant *Corynebacterium diphtheriae*. Technicians perform an Elek immunodiffusion test to verify toxigenicity. Analyze the schematic diagram below showing the precipitin line geometries.

Identify the exact configuration component corresponding to a true control positive toxigenic reaction:

Intersection Precipitin Arc

(A) Continuous symmetrical line formation between Line 1 and Line 2 demonstrating complete identity

(B) Crossing or spur formation over Line 3 indicative of a non-specific cross-reaction

(C) Complete absence of precipitin bands along Line 2 due to prozone effects

(D) Vertical parallel lines running parallel to the filter paper strip boundaries

Q5. An infant presenting with severe recurrent pyogenic infections, persistent leukocytosis ($> 40,000/\mu$L), and delayed separation of the umbilical cord (at 6 weeks of age) is evaluated. Flow cytometry analysis reveals a complete absence of CD18 expression on the surface of polymorphonuclear leukocytes. Which step of the leukocyte extravasation and homing cascade is primarily and directly disrupted by this genetic molecular defect?

(A) Selectin-mediated rolling along the vascular endothelium

(B) CD31-mediated PECAM-1 transendothelial migration (diapedesis)

(C) Firm integrin-mediated adhesion to endothelial ICAM-1 molecules

(D) Sialyl-Lewis X configuration binding to E-selectin receptors

Q6. A 24-year-old female with a history of severe systemic lupus erythematosus (SLE) undergoes evaluation for deteriorating renal function. Laboratory tests indicate critically low serum levels of C3 and C4. Molecular analysis reveals a genetic deficiency in the early components of the classical complement

pathway. Which specific physiological function of the complement cascade is impaired, directly predisposing this patient to accelerated immune complex-mediated glomerulonephritis?

(A) Impaired assembly of the alternative pathway C3 convertase ($C3bBb$)

(B) Failure of C3b/C4b-mediated opsonization needed for CR1-bearing erythrocyte clearance of immune complexes in the spleen/liver

(C) Inability to synthesize properdin required for stabilizing fluid-phase amplification loops

(D) Defective protectin (CD59) expression preventing membrane attack complex (MAC) inhibition

Q7. An experimental mouse model is engineered with a selective knockout mutation in the gene encoding the Invariant Chain (I_i / CD74) within antigen-presenting cells. When these mice are exposed to exogenous viral protein antigens, which of the following immunologic processing alterations is most likely to be observed?

(A) Failure of MHC Class I molecules to bind endogenous cytoplasmic peptides inside the endoplasmic reticulum

(B) Defective loading of exogenous antigenic peptides onto MHC Class II molecules due to premature binding of self-peptides in the rough endoplasmic reticulum

(C) Complete block in the retro-translocation of ubiquitinated proteins through the TAP1/TAP2 heterodimer pore

(D) Inability of the Proteasome complex to perform immunoproteasome catalytic subunit switching

Q8. A clinical immunologist performs a functional assessment on a patient suspected of having an atypical form of Severe Combined Immunodeficiency (SCID). The laboratory discovers a loss-of-function mutation in the gene encoding Janus Kinase 3 (JAK3). Intracellular signaling downstream of which specific subset of interleukin receptors is fundamentally abolished by this molecular pathology?

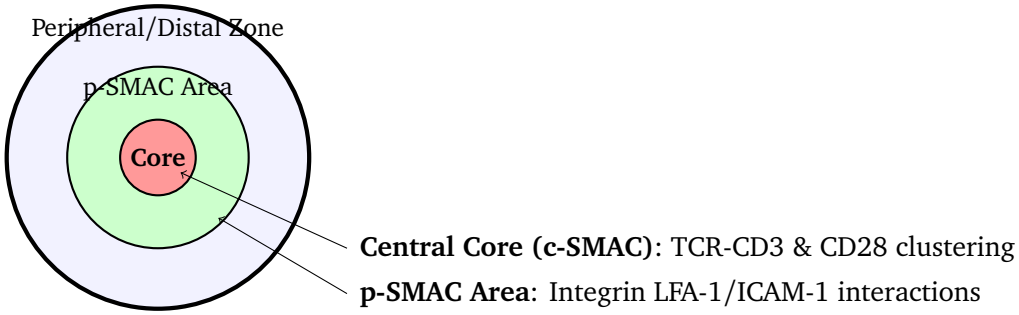
(A) Receptors utilizing the common cytokine receptor γ-chain (γ_c, CD132) including IL-2, IL-4, IL-7, IL-9, IL-15, and IL-21

(B) Heterodimeric receptors for IL-12 and IL-23 regulating Th1 and Th17 differentiation paths

(C) Homodimeric superfamily receptors binding Erythropoietin and Granulocyte Colony-Stimulating Factor

(D) Decoy receptors like IL-1R2 that lack functional intracellular signaling death domains

Q9. An immunological research assay explores T-cell activation dynamics by analyzing structural spatial rearrangements within the immunological synapse formed between a cytotoxic T lymphocyte (CTL) and a target tumor cell. The spatial topology maps into distinct concentric ring zones known as Supramolecular Activation Clusters (SMACs). Observe the architectural diagram below:



Which molecule pairs are specifically targeted and sequestered into the outermost boundary ring (**Distal-SMAC / d-SMAC**) during a stable cytotoxic interaction?

(A) TCR-CD3 complex interacting with peptide-MHC units

(B) LFA-1 binding to Intercellular Adhesion Molecule-1 (ICAM-1)

(C) Large, bulky filamentous glycoprotein structures like CD45 and CD43

(D) Perforin monomers polymerizing with granzyme B proteases

Q10. A 28-year-old HIV-positive patient with a CD4 count of 45/μL presents with progressive vision loss, retroorbital pain, and funduscopic evidence of "pizza-pie" necrotizing retinopathy. Treatment with intravenous Ganciclovir is

initiated. However, after 6 weeks, clinical progression continues, and genetic sequencing reveals a specific point mutation in the viral UL97 gene. What is the precise enzymatic step bypassed or disrupted by this drug resistance mechanism?

(A) Pyrophosphate analog competition with viral DNA polymerase

(B) Initial monophosphorylation step of Ganciclovir by the viral protein kinase

(C) Synthesis of structural glycoprotein gp120 during post-translational modification

(D) Cleavage of polyprotein precursors by HIV encoded aspartyl protease

Q11. A 4-year-old child presents with high fever, cough, conjunctivitis, coryza, and a diffuse maculopapular rash that began on the face and spread cranio-caudally. Microscopic examination of giant cells from the nasopharyngeal secretions reveals multinucleated syncytia with both intranuclear and intra-cytoplasmic inclusion bodies. Which viral surface structural component is directly responsible for mediating the cellular membrane fusion that forms these pathognomonic multinucleated syncytia?

(A) Hemagglutinin-Neuraminidase (HN) binding to sialic acid residues

(B) Hydrophobic Fusion (F) protein acting after attachment via the Hemagglutinin (H) glycoprotein

(C) Matrix (M) protein structural integrity alignment under the lipid bilayer membrane

(D) Non-structural NS1 protein-mediated inhibition of host interferon regulatory factor 3

Q12. A 58-year-old male with a history of chronic Hepatitis C infection underwent a liver biopsy showing bridging fibrosis and cirrhosis. Molecular profiling indicates he is infected with Hepatitis C Virus (HCV) Genotype 1b. Direct-acting antiviral (DAA) therapy containing Sofosbuvir is proposed. Sofosbuvir behaves as a potent inhibitor of the HCV NS5B protein. What is the funda-

mental catalytic identity of the target NS5B protein inhibited by this agent?

(A) NS3/4A Serine Protease responsible for polyprotein processing

(B) RNA-dependent RNA Polymerase responsible for viral genome replication

(C) NS5A Zinc-binding phosphoprotein managing viral assembly dynamics

(D) Host-derived cyclophilin A chaperone involved in replication complex stability

Q13. A basic virology laboratory monitors the replication kinetics of an unknown positive-sense single-stranded RNA (+ssRNA) arbovirus. The investigators note that during active replication within host cells, the viral genome acts directly as mRNA but cannot initiate negative-strand template synthesis without generating a distinct membrane-bound replication complex on the surface of modified endoplasmic reticulum vesicles. Which of the following structural or replication features applies uniquely to +ssRNA viruses over negative-sense single-stranded RNA (-ssRNA) viruses?

(A) Obligate requirement to carry a pre-formed, functional RNA-dependent RNA polymerase inside the mature virion core particle

(B) Purified naked genomic RNA extracted from the virion is inherently infectious when transfected directly into permissive host cells

(C) Transcription must transition through a double-stranded DNA integration intermediate via integrase enzymes

(D) Genome segmentation permits high-frequency antigenic shift via genetic reassortment mechanisms

Q14. A 35-year-old renal transplant recipient on intensive immunosuppression presents with severe dry cough, dyspnea, and low-grade fever. A high-resolution CT scan of the chest demonstrates bilateral ground-glass opacities. A bronchoalveolar lavage (BAL) is performed, and Silver stain (GMS) confirms the presence of collapsed crescentic or crushed-ping-pong-ball-like cystic structures. Molecular testing confirms *Pneumocystis jirovecii*. Which

structural cell wall component, unique compared to most clinical fungi, characterizes this organism and shapes its pharmacological response profiles?

(A) Ergoskeletal reliance on high levels of ergosterol within its plasma membrane

(B) Total absence of ergosterol in the cell membrane, substituting cholesterol instead, combined with a cell wall rich in β-1,3-glucan

(C) Outer wall capsule composed entirely of glucuronoxylomannan polymers

(D) Thick cell wall rich in chitin layers resistant to all polyene and azole antifungals

Q15. A 29-year-old missionary returning from a 6-month stay in rural Bolivia presents with a chronic, non-healing, painless cutaneous ulcer on his right forearm with raised indurated borders. A biopsy of the lesion border is obtained, and Giemsa staining reveals numerous small, round-to-oval intracellular organisms ($2-4\,\mu$m in diameter) inside macrophages, each containing a distinctive nucleus and a bar-like kinetoplast structure. Which morphological stage of this parasite was inoculated into the patient by the vector?

(A) Amastigote stage via the bite of a Blackfly (*Simulium*)

(B) Promastigote stage via the bite of a Sandfly (*Phlebotomus* or *Lutzomyia*)

(C) Epimastigote stage via the faeces of a Reduviid bug (*Triatoma*)

(D) Trypomastigote stage via the bite of a Tsetse fly (*Glossina*)

Q16. A 42-year-old HIV patient with poor compliance on antiretroviral therapy is admitted with a subacute onset of headache, confusion, and low-grade fever. Lumbar puncture reveals elevated opening pressure, low glucose, and elevated protein. An India ink preparation of the cerebrospinal fluid reveals encapsulated yeast cells with narrow-based budding. The clinician suspects Cryptococcal meningitis. Which major fungal virulence factor actively inhibits leukocyte migration and phagocytosis via its dense negative charge and steric hindrance profiles?

(A) Phenoloxidase-mediated melanin deposition within the inner layer cell wall

(B) Glucuronoxylomannan (GXM) capsular polysaccharide matrix

(C) Secreted aspartyl proteinases (SAPs) targeting baseline mucosal IgA antibodies

(D) Galactomannan cell wall side chains triggering aberrant TLR-4 signal transduction

Q17. A 23-year-old international student from Malawi presents to the emergency department with intermittent painless hematuria, terminal dysuria, and lower abdominal discomfort for three months. Cystoscopy demonstrates mucosal granulomas, ulcerations, and "sandy patches" inside the urinary bladder wall. Microscopic examination of the concentrated urine sediment reveals large, elongated, non-operculated helminth eggs measuring approximately $150\,\mu\text{m} \times 60\,\mu\text{m}$ with a sharp, terminal spine. What is the direct route of transmission for the infectious stage of this parasite?

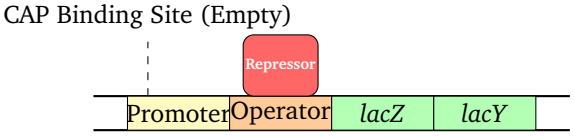
(A) Ingestion of undercooked freshwater fish containing metacercariae cyst clusters

(B) Active transdermal penetration of human skin by free-swimming fork-tailed cercariae in contaminated water

(C) Ingestion of aquatic vegetation harboring encysted metacercariae structures

(D) Accidental ingestion of embryonated eggs directly excreted in canine feces

Q18. A molecular research group studies the transcription kinetics of the lactose (*lac*) operon in mutant strains of *Escherichia coli* grown in varying concentrations of glucose and lactose. The diagram below maps the promoter/operator regulatory domain assembly state under a specific environmental configuration:



Analyze the bound and unbound status of the regulatory elements shown in the schematic. Which metabolic substrate environment corresponds directly to this exact molecular distribution state?

(A) High Glucose concentration + High Lactose concentration

(B) High Glucose concentration + Zero/No Lactose concentration

(C) Zero/No Glucose concentration + High Lactose concentration

(D) Zero/No Glucose concentration + Zero/No Lactose concentration

Q19. A 62-year-old male with a history of severe aortic stenosis undergoes prosthetic valve replacement surgery. Three months later, he presents with low-grade fever, weight loss, and new-onset heart murmurs. Blood cultures grow a Gram-positive, catalase-positive, coagulase-negative coccus that is found to be highly resistant to methicillin. The clinical team initiates continuous intravenous Vancomycin therapy. Which specific gene element or alteration facilitates phenotypic methicillin resistance in this pathogen?

(A) Chromosomal modification of DNA gyrase via mutation of the gyrA locus

(B) Acquisition of the mobile genetic cassette SCCmec carrying the mecA gene encoding PBP2a

(C) Plasmid-mediated synthesis of aminoglycoside acetyltransferase AAC(6')

(D) Over-expression of efflux pumps driven by multi-drug resistance operons (norA)

Q20. An epidemiological investigation identifies a cluster of cases of atypical pneumonia among elderly individuals who attended a convention in a modern hotel. Sputum analysis yields minimal results on routine Gram staining, but culture on Buffered Charcoal Yeast Extract (BCYE) agar supplemented with α-ketoglutarate and L-cysteine grows grey-white colonies with a cut-glass appearance. What is the fundamental mechanism used by this intracellular pathogen to avoid destruction within human alveolar macrophages?

(A) Escaping the phagosome into the host cell cytoplasm via a pore-forming hemolysin

(B) Direct inhibition of phagosome-lysosome fusion mediated by a Type IV secretion system (Dot/Icm)

(C) Enzymatic neutralization of reactive oxygen intermediates via surface-bound catalase-peroxidase

(D) Rapid degradation of host cell lysosomal membrane proteins using a highly active zinc metalloprotease

Detailed Solutions

Q1.

Solution

Concept: The Nagler reaction is a classical diagnostic culture method used to identify the alpha-toxin (α-toxin) produced by *Clostridium perfringens* on egg yolk agar.

Solution:

Let's trace the biochemical basis of the Nagler test:

(a) The alpha-toxin of *Clostridium perfringens* is a multi-functional zinc-dependent metalloenzyme acting as a principal virulence factor in gas gangrene (myonecrosis).

(b) Biochemically, it functions as a **Phospholipase C (lecithinase)**. Egg yolk agar naturally contains high levels of lecithin (phosphatidylcholine), which is clear and soluble when suspended in the medium.

(c) The enzyme breaks down **phosphatidylcholine into insoluble diacylglycerol (diglyceride) and water-soluble phosphorylcholine**. The precipitation of insoluble diacylglycerol in the medium produces the macroscopic "zone of opalescence" around the bacterial growth. Pre-treatment with specific antitoxin neutralizes the enzyme, preventing this hydrolysis and confirming the identity of the toxin.

Final Answer: Hydrolysis of phosphatidylcholine into diacylglycerol and phosphorylcholine

Answer: (B)

Go Back to Question 1

Q2.

Solution

Concept: Metallo-β-lactamases (MBLs) belong to Ambler Class B β-lactamases and confer broad-spectrum resistance to nearly all β-lactams, including carbapenems, while remaining characteristically structurally distinct from serine-based β-lactamases.

Solution:

Let's analyze the enzymatic configuration of Metallo-β-lactamases:

(a) Unlike Class A, C, and D enzymes that rely on an active-site serine residues to form a covalent acyl-enzyme intermediate, Class B MBLs are non-serine-dependent enzymes.

(b) They structurally incorporate **one or two divalent zinc (Zn^{2+}) ions** coordinated within their catalytic active site.

(c) These zinc ions polarize and anchor a **water molecule**, lowering its pK_a to generate a highly reactive nucleophilic hydroxyl group. This activated water directly attacks the carbonyl carbon of the β-lactam ring, hydrolyzing the bond without forming a covalent enzyme-drug complex. This structural property renders them completely insensitive to classic serine β-lactamase inhibitors like clavulanic acid.

Final Answer: Utilization of divalent zinc ions (Zn^{2+}) to activate a water molecule, enabling nucleophilic hydroxyl attack on the β-lactam ring and subsequent antibiotic hydrolysis.

Answer: (C)

Go Back to Question 2

Q3.

Solution

Concept: *Yersinia pestis*, the causative agent of the plague, is a zoonotic pathogen that uses thermal-regulated and secretion-driven virulence mechanisms to survive inside the mammalian host.

Solution:

Let's break down the virulence mechanisms that prevent early phagocytosis:

(a) When *Y. pestis* transfers from the flea vector (ambient temperature) to a mammalian host (37°C), it undergoes rapid transcription shifts to express host-evasion proteins.

(b) The **F1 (Fraction 1) capsular antigen** is a gel-like capsule synthesized preferentially at 37°C that sterically hinders phagocytic recognition and uptake.

(c) Simultaneously, the bacterium utilizes a **Type III Secretion System (T3SS)** to directly inject outer proteins (**Yops**, such as YopH and YopE) into nearby immune cells. YopH acts as a tyrosine phosphatase and YopE acts as a GTPase-activating protein; together they disrupt host actin cytoskeleton dynamics, crippling the macrophage's physical capability to execute phagocytosis.

Final Answer: Expression of the thermolabile capsular F1 antigen at 37°C together with delivery of YopH and YopE effector proteins into host cells through a Type III secretion system.

Answer: (A)

Go Back to Question 3

Q4.

Solution

Concept: The Elek immunodiffusion test is an *in vitro* precipitin assay used to differentiate toxigenic strains of *Corynebacterium diphtheriae* from non-toxigenic strains.

Solution:

Let's examine the mechanics of the immunodiffusion patterns shown in the schematic:

(a) In an Elek test, a filter paper strip impregnated with diphtheria antitoxin is placed across a culture plate. Organisms are streaked at right angles to this strip.

(b) Toxigenic strains secrete diphtheria toxin, which diffuses radially into the agar. Simultaneously, the antitoxin diffuses from the filter paper strip.

(c) Where the moving fronts of toxin and antitoxin meet at an optimal zone of equivalence, a visible precipitin line forms at roughly a 45° angle.

(d) Because Line 2 is a known reference positive toxigenic control, it establishes a specific precipitin line geometry. If the multi-drug resistant (MDR) strain under examination (Line 1) is also genuinely toxigenic, its precipitin line will merge continuously and symmetrically with the line from Line 2, forming a continuous **Intersection Precipitin Arc** (a reaction of complete identity).

Final Answer: Continuous symmetrical line formation between Line 1 and Line 2 demonstrating complete identity

Answer: (A)

Go Back to Question 4

Q5.

Solution

Concept: Leukocyte Adhesion Deficiency Type 1 (LAD-1) is an autosomal recessive primary immunodeficiency resulting from mutations in the *ITGB2* gene encoding the integrin β_2 subunit (CD18).

Solution:

Let's track the molecular steps of leukocyte extravasation to find the exact point of failure:

(a) **Step 1: Rolling:** Mediated by low-affinity interactions between selectins (E-selectin, P-selectin) and carbohydrate ligands (Sialyl-Lewis X). This step is intact in LAD-1.

(b) **Step 2: Firm Adhesion:** Triggered by chemokine activation, leukocytes must bind tightly to the activated endothelium. This requires **β_2-integrins** like LFA-1 (CD11a/CD18) and Mac-1 (CD11b/CD18) to attach to **ICAM-1** on endothelial cells.

(c) Because CD18 is completely absent, these high-affinity integrin complexes cannot form. Consequently, neutrophils are unable to execute **firm adhesion**, causing them to remain trapped in circulation. This results in severe peripheral leukocytosis alongside a total absence of pus formation at infection sites.

Final Answer: Firm integrin-mediated adhesion to endothelial ICAM-1 molecules

Answer: (C)

Go Back to Question 5

Q6.

Solution

Concept: Early classical complement components (C1, C4, C2) play a fundamental, non-redundant physiological role in processing and clearing soluble immune complexes from systemic circulation.

Solution:

Let's analyze why early complement deficiency accelerates lupus nephritis:

(a) Under normal conditions, the activation of the classical complement pathway by circulating IgG/IgM immune complexes leads to the covalent deposition of fragments **C3b and C4b** directly onto the surface of these networks.

(b) These bound fragments serve as ligands that engage **Complement Receptor 1 (CR1, CD35)** expressed on the membranes of circulating erythrocytes.

(c) The red blood cells bind the opsonized complexes and transport them safely through the bloodstream to the liver and spleen, where specialized resident macrophages safely strip away and destroy the immune complexes without triggering local inflammation. When early components are missing, immune complexes fail to be properly opsonized, remain free in circulation, and precipitate within narrow vascular beds like the renal glomerular basement membrane, inducing severe lupus nephritis.

Final Answer: Failure of C3b/C4b-mediated opsonization needed for CR1-bearing erythrocyte clearance of immune complexes in the spleen/liver

Answer: (B)

Go Back to Question 6

Q7.

Solution

Concept: The Invariant Chain (I_i / CD74) is a specialized chaperone protein that stabilizes newly synthesized MHC Class II $\alpha\beta$ heterodimers within the rough endoplasmic reticulum (RER).

Solution:

Let's isolate the molecular sorting events in the absence of the invariant chain:

(a) Inside the RER, MHC Class I molecules naturally bind endogenous cytosolic peptides delivered via the TAP transporter. In contrast, MHC Class II molecules must keep their antigen-binding cleft vacant while passing through the RER to save it for exogenous peptides encountered later in the endolysosomal network.

(b) The invariant chain sits directly within the peptide-binding groove of the MHC Class II molecule inside the RER, physically blocking any self-peptides or fragments from binding prematurely.

(c) Without the invariant chain, the empty binding cleft of the MHC Class II molecule is left exposed within the RER. **Self-peptides present inside the RER will bind prematurely to MHC Class II**, filling the groove and rendering the molecule stable but structurally locked. When it eventually reaches the endolysosome, it cannot load the target exogenous viral peptides.

Final Answer: Defective loading of exogenous antigenic peptides onto MHC Class II molecules due to premature binding of self-peptides in the rough endoplasmic reticulum

Answer: (B)

Go Back to Question 7

Q8.

Solution

Concept: Severe Combined Immunodeficiency (SCID) can arise from molecular defects in downstream cytokine signaling pathways, particularly the Janus Kinase 3 (JAK3) pathway.

Solution:

Let's map out the connection between JAK3 and interleukin receptors:

(a) Janus Kinase 3 (JAK3) is unique among the JAK family because its expression is highly restricted to hematopoietic cells, and it associates almost exclusively with a single receptor component.

(b) This component is the **common cytokine receptor γ-chain (γ_c, CD132)**. The γ_c subunit is a shared structural element across receptors for **IL-2, IL-4, IL-7, IL-9, IL-15, and IL-21**.

(c) When an interleukin binds any of these receptors, JAK3 must physically bind the γ_c tail to initiate STAT phosphorylation and gene transcription. A loss-of-function mutation in JAK3 completely shuts down signaling for all six of these interleukins. This phenotypically mirrors X-linked SCID (which is caused by a direct mutation in the γ_c gene itself), leading to a profound loss of T cells and NK cells due to absent IL-7 and IL-15 signaling.

Final Answer: Receptors that utilize the common cytokine receptor γ-chain (γ_c, CD132), including those for IL-2, IL-4, IL-7, IL-9, IL-15, and IL-21.

Answer: (A)

Go Back to Question 8

Q9.

Solution

Concept: The immunological synapse is organized into concentric spatial ring domains known as Supramolecular Activation Clusters (SMACs) that sort proteins based on their physical dimensions and functional roles.

Solution:

Let's examine the architectural zones of a stable immunological synapse:

(a) **Central-SMAC (c-SMAC):** The innermost core zone where signaling receptors cluster, including the TCR-CD3 complex, co-stimulatory molecules like CD28, and signaling kinases.

(b) **Peripheral-SMAC (p-SMAC):** The intermediate ring that provides structural stability. It contains adhesion molecules such as the integrin LFA-1 binding to its endothelial ligand ICAM-1.

(c) **Distal-SMAC (d-SMAC):** The outermost boundary ring. To allow the membranes of the T cell and target cell to pull tight against each other in the center (forming a narrow 15 nm gap for signaling), **large, bulky cell-surface glycoproteins with elongated ectodomains—such as CD45 and CD43**—must be sterically squeezed out of the core and moved to the outer periphery. This clean sorting prevents these large phosphatases from non-specifically dampening the signaling inside the core.

Final Answer: Large, bulky filamentous glycoprotein structures like CD45 and CD43

Answer: (C)

Go Back to Question 9

Q10.

Solution

Concept: Ganciclovir is a nucleoside analogue that acts against Cytomegalovirus (CMV) by blocking viral DNA synthesis, but it requires initial intracellular biotransformation to become active.

Solution:

Let's break down the activation pathway of ganciclovir and how mutations break it:

(a) Like many nucleoside analogues, ganciclovir is administered as an inactive prodrug. It cannot be utilized by host or viral DNA polymerases in its raw form; it must undergo three consecutive steps of phosphorylation.

(b) Human cells do not possess a native kinase that can efficiently perform the **initial monophosphorylation step** on ganciclovir. Instead, CMV carries its own specialized gene, **UL97**, which encodes a viral protein kinase dedicated to this step.

(c) Once the UL97 kinase adds the first phosphate group, host cellular kinases quickly add the next two to yield active ganciclovir triphosphate. A point mutation in the *UL97* gene alters this kinase, preventing it from performing the initial monophosphorylation. As a result, the drug remains inactive, allowing CMV retinitis to progress.

Final Answer: Initial monophosphorylation step of Ganciclovir by the viral protein kinase

Answer: (B)

Go Back to Question 10

Q11.

Solution

Concept: Measles virus (a member of the Paramyxoviridae family) produces multinucleated giant cells (Warthin-Finkeldey cells) that serve as a hallmark histological indicator of infection.

Solution:

Let's analyze the membrane fusion mechanisms of the measles virus:

(a) The measles virus uses two distinct glycoproteins embedded in its lipid envelope to enter host cells: the Hemagglutinin (H) protein and the Fusion (F) protein.

(b) The H protein handles initial attachment by binding to host cell surface receptors such as CD150 (SLAM) or CD46.

(c) Following this attachment, a conformational shift exposes the hydrophobic **Fusion (F) protein**. The F protein inserts its hydrophobic peptide directly into the host cell plasma membrane, pulling the viral envelope and host membrane together until they fuse. Crucially, this F protein remains active on the surface of infected host cells, allowing them to bind and fuse with neighboring uninfected host cells at a neutral pH, creating massive **multinucleated syncytia** with characteristic inclusion bodies.

Final Answer: Hydrophobic Fusion (F) protein acting after attachment via the Hemagglutinin (H) glycoprotein

Answer: (B)

Go Back to Question 11

Q12.

Solution

Concept: Direct-acting antiviral (DAA) regimens treat Hepatitis C Virus (HCV) by targeting specific non-structural (NS) viral proteins required for replication.

Solution:

Let's verify the functional identities of the HCV non-structural proteins:

(a) The HCV positive-sense RNA genome encodes a single large polyprotein that must be cleaved into individual functional units by both host and viral proteases (such as the NS3/4A serine protease).

(b) To replicate its positive-sense single-stranded RNA genome, the virus must assemble a replication complex that can synthesize an RNA strand directly from an RNA template.

(c) The non-structural protein **NS5B** serves as the catalytic engine of this complex, functioning as the viral **RNA-dependent RNA Polymerase (RdRp)**. Sofosbuvir is a uridine nucleotide analogue that acts as a chain terminator; once phosphorylated intracellularly, it is incorporated by NS5B into the growing RNA strand, blocking viral genome replication.

Final Answer: RNA-dependent RNA Polymerase responsible for viral genome replication

Answer: (B)

Go Back to Question 12

Q13.

Solution

Concept: The polarity of single-stranded RNA viral genomes dictates their initial replication strategy, protein expression sequence, and the infectious properties of their naked nucleic acids.

Solution:

Let's compare the functional properties of positive-sense (+ssRNA) and negative-sense (-ssRNA) single-stranded RNA viruses:

(a) A negative-sense single-stranded RNA genome is antisense to mRNA; it cannot be read or translated by host ribosomes. Therefore, -ssRNA viruses must package a pre-formed, structural RNA-dependent RNA polymerase (RdRp) inside their virion core to transcribe the negative strand into positive mRNA upon entry.

(b) In contrast, a **positive-sense single-stranded RNA (+ssRNA)** genome has the exact same configuration as host mature mRNA. It features a structure that host ribosomes can recognize immediately.

(c) Consequently, if **purified naked genomic RNA** from a +ssRNA virus is transfected directly into a permissive host cell without any accompanying viral proteins or capsids, the host ribosomes will immediately translate it to produce functional viral proteins, including the virus's own RdRp. This makes the naked genomic RNA inherently infectious on its own.

Final Answer: Purified naked genomic RNA extracted from the virion is inherently infectious when transfected directly into permissive host cells

Answer: (B)

Go Back to Question 13

Q14.

Solution

Concept: *Pneumocystis jirovecii* is an atypical opportunistic fungus that causes severe interstitial pneumonia in immunocompromised patients, featuring atypical cell membrane and wall compositions.

Solution:

Let's evaluate the unique structural biochemistry of *Pneumocystis jirovecii*:

(a) Most classic pathogenic fungi incorporate ergosterol as the primary sterol within their plasma membranes, making them sensitive to polyenes (like amphotericin B) and azoles.

(b) *Pneumocystis jirovecii* is atypical because its plasma membrane **completely lacks ergosterol**, incorporating **cholesterol** instead. Because it lacks the target sterol, standard anti-fungal drugs like azoles and polyenes are clinically ineffective against it.

(c) Despite having an atypical membrane, its cell wall still contains **β-1,3-glucan** polymers during its cystic life stage. Because of this distinct layout, first-line treatment relies on target metabolic inhibitors like Trimethoprim-Sulfamethoxazole (TMP-SMX) rather than standard antifungals.

Final Answer: Complete absence of ergosterol in the cell membrane, with cholesterol serving as the principal membrane sterol instead, together with a cell wall enriched in β-1,3-glucan.

Answer: (B)

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Q15.

Solution

Concept: Cutaneous leishmaniasis is caused by intracellular protozoan parasites of the genus *Leishmania*, which alternate between distinct morphological stages during their life cycle.

Solution:

Let's trace the lifecycle stages and vectors for *Leishmania*:

(a) The biopsy finding—showing small, round intracellular organisms ($2-4\,\mu$m) containing a nucleus and a bar-like kinetoplast within host macrophages—describes the **amastigote stage**. This is the form that replicates inside the mammalian host.

(b) The parasite is transmitted through the bite of an infected female **sandfly** (*Phlebotomus* species in the Old World, or *Lutzomyia* species in the New World).

(c) Inside the sandfly vector, the parasite develops into a flagellated, elongated, motile form known as the **promastigote stage**. When the sandfly takes a blood meal from a human, it regurgitates these infectious promastigotes into the skin. The promastigotes are then phagocytosed by macrophages, where they lose their flagella and transition into amastigotes.

Final Answer: Promastigote stage via the bite of a Sandfly (*Phlebotomus* or *Lutzomyia*)

Answer: (B)

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Q16.

Solution

Concept: *Cryptococcus neoformans* is an encapsulated, opportunistic yeast that causes life-threatening meningitis in patients with advanced HIV/AIDS.

Solution:

Let's analyze the virulence properties of the cryptococcal capsule:

(a) The diagnostic finding of an encapsulated yeast showing narrow-based budding on an India ink mount of cerebrospinal fluid confirms a diagnosis of cryptococcal meningitis.

(b) The primary virulence factor of this pathogen is its thick extracellular polysaccharide capsule, which is composed primarily of **Glucuronoxylomannan (GXM)**.

(c) The GXM capsule forms a thick physical barrier around the yeast cell wall. It carries a strong **negative charge** that causes electrostatic repulsion, preventing phagocytes from effectively binding and engulfing the fungus. Additionally, shed GXM polysaccharide diffuses into surrounding tissues, where it interferes with leukocyte migration and dampens local immune responses.

Final Answer: Glucuronoxylomannan (GXM) capsular polysaccharide matrix

Answer: (B)

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Q17.

Solution

Concept: *Schistosoma haematobium* is a trematode (blood fluke) endemic to parts of Africa and the Middle East that infects the venous plexuses surrounding the urinary bladder.

Solution:

Let's evaluate the lifecycle and transmission route of *Schistosoma haematobium*:

(a) The patient's clinical signs—painless hematuria, terminal dysuria, and bladder mucosal "sandy patches" (granulomas around trapped eggs)—along with urine sediment showing large, non-operculated eggs with a sharp **terminal spine** are diagnostic for *S. haematobium*.

(b) Eggs excreted in human urine hatch in fresh water to release miracidia, which infect specific aquatic snail intermediate hosts.

(c) Inside the snail, the parasites multiply and are released back into the water as free-swimming, fork-tailed **cercariae**. Transmission occurs when a human comes into contact with contaminated freshwater, allowing these cercariae to **actively penetrate the unbroken skin**, shed their tails to become schistosomulae, and migrate through the circulation to the vesical venous plexuses.

Final Answer: Active transdermal penetration of human skin by free-swimming fork-tailed cercariae in contaminated water

Answer: (B)

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Q18.

Solution

Concept: The lactose (*lac*) operon in *Escherichia coli* uses dual genetic controls to ensure metabolic efficiency, matching structural gene expression to the availability of glucose and lactose.

Solution:

Let's analyze the occupancy of the regulatory sites in the diagram:

(a) **Operator Site Status:** The diagram shows the active **Repressor protein tightly bound to the Operator site**. The repressor only binds the operator when its inducer (allolactose, a derivative of lactose) is absent. Therefore, the presence of a bound repressor indicates that there is **Zero/No Lactose** in the environment.

(b) **Promoter / CAP Site Status:** The diagram shows the **Catabolite Activator Protein (CAP) binding site is empty**. CAP requires cyclic AMP (cAMP) to bind to this site and recruit RNA polymerase. When environmental glucose concentrations are high, adenylyl cyclase is inhibited, dropping intracellular cAMP levels and leaving the CAP site empty. Therefore, an empty CAP site indicates a **High Glucose** environment.

(c) Combining these observations, this specific molecular configuration occurs when the bacterium is grown in an environment containing high glucose and no lactose.

Final Answer: High Glucose concentration + Zero/No Lactose concentration

Answer: (B)

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Q19.

Solution

Concept: Methicillin resistance in staphylococci, including coagulase-negative species like *Staphylococcus epidermidis*, is driven by horizontal acquisition of specialized penicillin-binding proteins.

Solution:

Let's analyze the molecular mechanism behind methicillin resistance:

(a) The presentation describes prosthetic valve endocarditis caused by a methicillin-resistant, coagulase-negative staphylococcus (likely *Staphylococcus epidermidis*).

(b) Methicillin resistance in staphylococci is mediated by the acquisition of a mobile genetic element called the **Staphylococcal Cassette Chromosome mec (SCCmec)**.

(c) This cassette carries the **mecA gene**, which encodes an alternative transpeptidase known as **Penicillin-Binding Protein 2a (PBP2a)**. Unlike native PBPs, PBP2a has a very low binding affinity for β-lactam antibiotics, including penicillins, cephalosporins, and carbapenems. This allows the bacterium to continue cross-linking its peptidoglycan cell wall even in the presence of these drugs.

Final Answer: Acquisition of the mobile genetic cassette SCCmec carrying the mecA gene encoding PBP2a

Answer: (B)

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Q20.

Solution

Concept: *Legionella pneumophila* is a facultative intracellular pathogen that causes Legionnaires' disease, an atypical pneumonia often linked to contaminated water systems in large buildings.

Solution:

Let's break down the intracellular survival strategy of *Legionella*:

(a) The clinical description—an outbreak of atypical pneumonia among convention attendees linked to a hotel water system, paired with culture growth on Buffered Charcoal Yeast Extract (BCYE) agar requiring L-cysteine and iron—is classic for *Legionella pneumophila*.

(b) Once inside an alveolar macrophage via phagocytosis, the bacterium must avoid being destroyed by lysosomal enzymes.

(c) *Legionella* achieves this using a specialized **Type IV Secretion System** known as the **Dot/Icm** (Defective organelle trafficking/Intracellular multiplication) complex. This system injects effector proteins into the host cell cytoplasm that alter vesicle trafficking, **directly inhibiting phagosome-lysosome fusion**. Instead, the phagosome is remodeled into a protective vesicle surrounded by host endoplasmic reticulum, allowing the bacterium to safely multiply inside the cell.

Final Answer: Direct inhibition of phagosome-lysosome fusion mediated by a Type IV secretion system (Dot/Icm)

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	A	5	C
6	B	7	B	8	A	9	C	10	B
11	B	12	B	13	B	14	B	15	B
16	B	17	B	18	B	19	B	20	B