NEET PG Microbiology Sample Paper-5

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 34-year-old male presents to the emergency department with a high-grade fever, severe headache, and neck stiffness. A lumbar puncture is performed, and cerebrospinal fluid (CSF) analysis reveals an elevated opening pressure, high protein, low glucose, and marked neutrophilic leukocytosis. Gram staining of the CSF sediment demonstrates Gram-negative intracellular diplococci. Which of the following is the primary virulence factor responsible for the initial nasopharyngeal colonization and adherence of this causative organism?

(A) IgA1 protease

(B) Polysaccharide capsule

(C) Pili (Fimbriae)

(D) Lipooligosaccharide (LOS)

Q2. A 45-year-old intensive care unit patient on mechanical ventilation develops a nosocomial pneumonia. Culture of the bronchoalveolar lavage fluid on nutrient agar yields colonies with a distinct blue-green pigment and a sweet, grape-like odor. The organism is a Gram-negative bacillus, motile, oxidase-positive, and a non-fermenter on MacConkey agar. Which of the following resistance mechanisms represents its most common intrinsic barrier against beta-lactam antibiotics?

(A) Production of plasmid-mediated TEM-1 beta-lactamase

(B) Inducible chromosomal AmpC beta-lactamase and active efflux pumps

(C) Alteration of penicillin-binding protein 2a (PBP2a)

(D) Mutations in the gyrA gene encoding DNA gyrase

Q3. A 28-year-old pregnant woman at 26 weeks of gestation presents with low-grade fever, chills, and generalized myalgia after consuming unpasteurized cheese. Blood cultures are drawn and turn positive for a Gram-positive rod that exhibits characteristic "tumbling motility" at 25°C but is non-motile at 37°C. On sheep blood agar, it produces a narrow zone of beta-hemolysis. Which cellular mechanism allows this pathogen to escape the phagosome of host cells and multiply intracellularly?

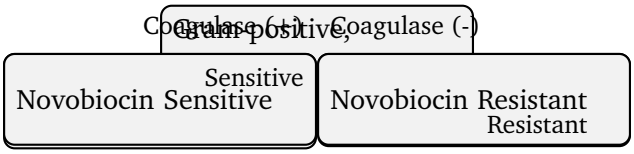
(A) Production of Lecithinase C

(B) Secretion of Listeriolysin O and phospholipases

(C) Expression of Internalin A and B surface proteins

(D) Elaboration of Cord Factor (Trehalose dimycolate)

Q4. Review the biochemical diagnostic workflow outlined below for differentiating Gram-positive, catalase-positive cocci:



A 22-year-old sexually active female presents with acute dysuria, urgency, and suprapubic pain. Her urine culture grows a Gram-positive, catalase-positive, coagulase-negative coccus that corresponds to the “Novobiocin Resistant” endpoint in the provided algorithm. Which organism is the most likely cause of her urinary tract infection?

(A) Staphylococcus saprophyticus

(B) Staphylococcus epidermidis

(C) Staphylococcus lugdunensis

(D) Enterococcus faecalis

Q5. A 52-year-old male concrete worker presents with a deep, contaminated puncture wound on his right foot sustained from a rusty nail 3 days ago. He reports muscle spasms in his jaw and difficulty swallowing. On examination, there is marked trismus and a sardonic smile (risus sardonicus). The toxin responsible for this clinical presentation acts primarily by which of the following molecular neurotoxic pathways?

(A) Inhibiting the release of acetylcholine at the neuromuscular junction

(B) Cleaving Synaptobrevin to block the release of GABA and glycine in spinal interneurons

(C) ADP-ribosylation of elongation factor 2 (EF-2) to arrest protein synthesis

(D) Activating adenylate cyclase via G-protein stimulation to increase cAMP levels

Q6. An active surveillance protocol in a tertiary care neonatal unit identifies an increase in late-onset sepsis cases. Blood cultures grow Gram-negative bacilli that form mucoid pink colonies on MacConkey agar. Biochemical profiling confirms the organism is indole-negative, citrate-positive, and non-motile. Phenotypic screening reveals resistance to third-generation cephalosporins, which is not reversed by clavulanic acid but is inhibited by boronic acid. What is the most likely mechanism of beta-lactam resistance in this strain?

(A) Production of Extended-Spectrum Beta-Lactamase (ESBL)

(B) Hyperproduction of chromosomal AmpC cephalosporinase

(C) Klebsiella pneumoniae Carbapenemase (KPC) production

(D) Metallo-beta-lactamase (MBL) production

Q7. A 40-year-old migrant laborer presents with a 3-month history of chronic productive cough, hemoptysis, night sweats, and significant weight loss. A sputum acid-fast bacilli (AFB) smear is highly positive. A chest X-ray reveals cavitary lesions in the right upper lobe. The virulence of this pathogen, specifically its ability to prevent phagosome-lysosome fusion inside alveolar

macrophages and induce granuloma formation, is strongly associated with which cell wall component?

(A) Lipoarabinomannan (LAM)

(B) Cord factor (Trehalose 6,6'-dimycolate)

(C) Mycolic acid monomers

(D) Sulfatides

Q8. A 9-month-old infant is brought to the pediatric emergency department with a 3-day history of low-grade fever, harsh barking cough, inspiratory stridor, and hoarseness. A frontal cervical radiograph demonstrates subglottic narrowing known as the "steeple sign." The causative agent is an enveloped, non-segmented, negative-sense single-stranded RNA virus. Which viral surface glycoprotein is uniquely responsible for both host cell attachment and the agglutination of erythrocytes?

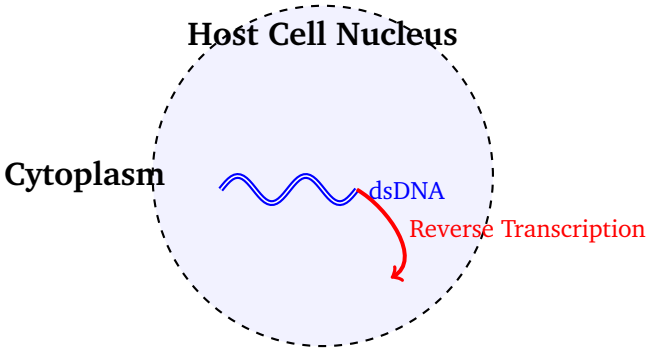
(A) Hemagglutinin-Neuraminidase (HN) protein

(B) Fusion (F) protein

(C) Glycoprotein (G) attachment protein

(D) Matrix (M) protein

Q9. Consider the viral genome structural layout and target site of replication shown below:



A 35-year-old male undergoes a routine pre-employment health screening. His serological profile returns positive for HBsAg, positive for anti-HBc IgM, and negative for anti-HBs. This virus utilizes the unique replication strategy shown in the diagram, where a double-stranded DNA virus replicates via an

RNA intermediate inside the host cell. Which viral enzyme performs this specific reverse transcription step within the nucleocapsid?

(A) Host cellular RNA Polymerase II

(B) Viral RNA-dependent RNA Polymerase

(C) Viral DNA-dependent DNA Polymerase with reverse transcriptase activity

(D) Host cellular DNA Polymerase alpha

Q10. A 62-year-old male recipient of a renal transplant on a maintenance immunosuppressive regimen (tacrolimus, mycophenolate mofetil, and prednisolone) presents with a 2-week history of progressive blurring of vision in his left eye. Fundoscopic examination reveals extensive, cheesy necrotizing retinitis with perivascular hemorrhages ("pizza-pie retinopathy"). Which of the following diagnostic modalities is considered the gold standard for confirming active replication of the most likely viral pathogen in this patient's aqueous or vitreous humor?

(A) Tzanck smear showing multinucleated giant cells

(B) Quantitative Polymerase Chain Reaction (qPCR) for CMV DNA

(C) Shell vial culture assay for Herpes Simplex Virus

(D) Detection of serum anti-CMV IgM antibodies

Q11. An 8-year-old boy living in a rural tropical area is brought to the clinic with an acute onset of high fever, retro-orbital pain, severe generalized myalgia ("breakbone fever"), and a maculopapular rash. Laboratory findings indicate severe leukopenia and thrombocytopenia. Two days later, he develops petechiae, epistaxis, and a positive tourniquet test. What immunological phenomenon explains why a subsequent infection with a different serotype of this virus leads to this severe, life-threatening hemorrhagic manifestation?

(A) Antibody-Dependent Enhancement (ADE) mediated by non-neutralizing antibodies

(B) Molecular mimicry leading to autoantibody production against platelets

(C) Direct cytopathic destruction of vascular endothelial cells by the virus

(D) Continuous activation of the alternative complement pathway by viral NS1 antigen

Q12. A 42-year-old HIV-positive male with a CD4+ T-lymphocyte count of 45 cells/μL presents to the clinic with subacute onset of low-grade fever, chronic non-productive cough, and progressive exertional dyspnea. Chest computed tomography (CT) displays bilateral ground-glass opacities extending from the hila. Silver staining of a bronchoalveolar lavage specimen reveals multiple crushed-cup or crescent-shaped cysts. Which structural component of this opportunistic fungal pathogen's cell wall is completely absent, making it intrinsically resistant to cell wall-active antifungal agents like echinocandins during its trophic form?

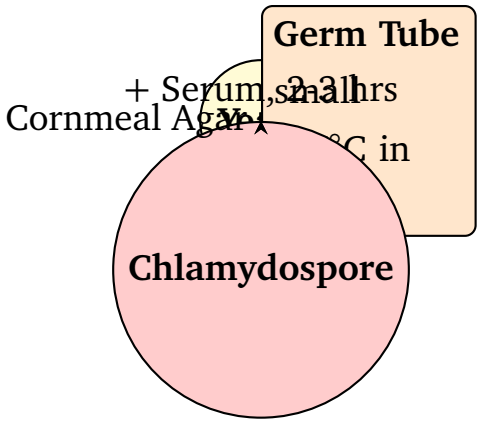
(A) Ergosterol

(B) Chitin

(C) β-(1,3)-D-glucan

(D) Alpha-mannan

Q13. Refer to the morphological differentiation pathway shown below for a common opportunistic fungal pathogen:



A 48-year-old diabetic female presents with dense white, curd-like vaginal discharge accompanied by intense pruritus vulvae. Microscopic evaluation reveals pseudohyphae and budding yeast cells. When incubated in human serum at 37°C for 2.5 hours, parallel-sided hyphal extensions without constriction at their point of origin are observed, matching the positive "Germ

Tube" path above. Which species is definitively identified by this specific phenotypic switch?

(A) Candida glabrata

(B) Candida tropicalis

(C) Candida albicans

(D) Candida krusei

Q14. A 29-year-old backpacker returns from a 3-week trekking trip in the wilderness. He presents with a 10-day history of foul-smelling, explosive, watery diarrhea, abdominal bloating, flatulence, and significant steatorrhea. Stool microscopy reveals flagellated, pear-shaped trophozoites with two nuclei and a large ventral sucking disk, resembling an "old man with glasses." Which anatomical site is the primary zone of colonization and attachment for this parasite?

(A) Cecum and ascending colon

(B) Duodenum and upper jejunum

(C) Terminal ileum

(D) Rectosigmoid colon

Q15. A 38-year-old male from a rural farming community presents with a 1-month history of right upper quadrant abdominal pain, low-grade intermittent fever, and mild jaundice. A routine complete blood count shows marked peripheral eosinophilia. An ultrasound of the liver reveals a solitary, large, well-defined hypoechoic cystic lesion in the right lobe with a double-line outline and fluid-filled internal split membranes ("water-lily sign"). What is the definitive host in the life cycle of the parasite responsible for this condition?

(A) Sheep

(B) Human

(C) Dog (and other canids)

(D) Freshwater snail

Q16. A 4-year-old boy is evaluated for recurrent, severe pyogenic skin abscesses, furunculosis, and deep organ infections with Staphylococcus aureus and Aspergillus fumigatus since infancy. Notably, his wounds heal poorly and exhibit a lack of purulent exudate despite high neutrophil counts in peripheral blood. A nitroblue tetrazolium (NBT) slide test is performed, and the patient's neutrophils fail to reduce the dye to blue formazan. This primary immunodeficiency disorder is caused by an inherited defect in which cellular biochemical entity?

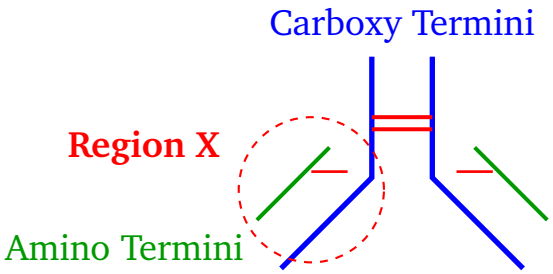
(A) Myeloperoxidase enzyme

(B) NADPH oxidase enzyme complex

(C) Adenosine deaminase (ADA)

(D) LFA-1 integrin (CD11/CD18)

Q17. Examine the simplified structural schematic of the human monomeric immunoglobulin molecule provided below:



During an adaptive immune response, hypervariable loops within the variable domains of both heavy and light chains form the precise antigen-binding site. Which of the following functional designations and proteolytic cleavage fragments correctly corresponds to "Region X" indicated within the dashed circle?

(A) Fc fragment, responsible for complement activation and opsonization

(B) Fab fragment, responsible for antigen binding and idiotypic specificity

(C) F(ab')2 fragment, generated via pepsin digestion containing divalent binding sites

(D) J-chain attachment domain, responsible for mucosal dimeric secretion

Q18. A 24-year-old female diagnosed with systemic lupus erythematosus (SLE) presents with acute worsening of peripheral edema, hematuria, and hypertension. Laboratory testing reveals elevated titers of anti-dsDNA antibodies and a profound reduction in serum C3 and C4 complement levels, indicating severe lupus nephritis. In this type III hypersensitivity reaction, which molecular fragment acts as the most potent anaphylatoxin to recruit and activate neutrophils, leading to local endothelial damage and tissue destruction?

(A) C3b

(B) C4b

(C) C5a

(D) C9 membrane attack complex

Q19. A researcher is studying the mechanism of antigen processing and presentation to T-lymphocytes. She inhibits the function of the Transporter associated with Antigen Processing (TAP1/TAP2) complex in a human cell line using a targeted small-molecule inhibitor. This specific inhibition will selectively impair the processing and presentation of which class of antigens to which lineage of T-lymphocytes?

(A) Endogenous antigens via MHC Class I to CD8+ T-cells

(B) Exogenous antigens via MHC Class II to CD4+ T-cells

(C) Cross-presented lipid antigens via CD1d to NKT cells

(D) Bacterial superantigens directly to the Vβ region of T-cell receptors

Q20. A 30-year-old male undergoes an allogeneic bone marrow transplantation from an HLA-matched sibling donor for acute myeloid leukemia. Three weeks post-transplantation, while his absolute neutrophil count is recovering, he abruptly develops a diffuse maculopapular rash involving his palms and soles, severe watery diarrhea with abdominal cramping, and elevated serum bilirubin. A skin biopsy confirms apoptotic keratinocytes and lymphocytic infiltration. What is the fundamental immunological mechanism driving this clinical condition?

(A) Host residual cytotoxic T-lymphocytes recognizing and destroying donor stem cells

(B) Pre-existing donor-specific antibodies in the host causing hyperacute vascular occlusion

(C) Mature donor T-lymphocytes present in the graft recognizing and attacking recipient histocompatibility antigens

(D) Type I IgE-mediated immediate hypersensitivity reaction to prophylactic antimicrobials

Detailed Solutions

Q1.

Solution

Concept: The initial stage of pathogenesis for *Neisseria meningitidis* involves colonization of the mucosal surfaces of the human nasopharynx. To establish colonization against mechanical clearing mechanisms like ciliary clearance and mucous flow, the bacterium must express highly specialized surface structures that facilitate tight adherence to host epithelial cells before producing systemic invasion.

Solution:

(a) Pili (fimbriae) are long, filamentous surface appendages composed of pilin subunits that extend far beyond the bacterial capsule. They mediate the initial, critical step of attachment to specific CD46 receptors on non-ciliated nasopharyngeal epithelial cells.

(b) While the polysaccharide capsule is a vital virulence factor, its primary role is providing resistance against phagocytosis and complement-mediated lysis in the bloodstream, rather than initial mucosal adherence.

(c) IgA1 protease is an excreted enzyme that cleaves secretory IgA at the hinge region to evade mucosal immunity, but it acts as an enzymatic facilitator rather than the physical primary structural adhesin.

(d) Lipooligosaccharide (LOS) behaves as an endotoxin responsible for inducing a severe inflammatory response, intravascular coagulation, and vascular damage during systemic meningococcemia.

(e) Therefore, mutations that disrupt the assembly of pili completely abolish the capacity of *Neisseria meningitidis* to colonize the nasopharynx, emphasizing their absolute primacy in host-pathogen attachment.

Final Answer: The primary virulence factor responsible for initial adherence is Pili (Fimbriae).

Answer: (C)

Go Back to Question 1

Q2.

Solution

Concept: *Pseudomonas aeruginosa* is a notorious nosocomial pathogen that exhibits high levels of intrinsic resistance to a wide array of antimicrobial agents. This baseline resistance renders it completely impervious to standard narrow-spectrum and broad-spectrum beta-lactams unless specific antipseudomonal formulations are strategically deployed.

Solution:

(a) The baseline intrinsic resistance of *Pseudomonas aeruginosa* to beta-lactam antibiotics is mediated by the synchronous activity of an inducible chromosomal AmpC beta-lactamase enzyme and active multi-drug efflux systems.

(b) The AmpC beta-lactamase hydrolyzes cephalosporins and penicillins, and its expression is dynamically upregulated in response to beta-lactam exposure, leading to rapid treatment failure.

(c) Concurrently, the MexAB-OprM efflux pump system continuously ejects any antibiotic molecules that successfully cross the outer membrane before they can reach their target penicillin-binding proteins.

(d) Plasmid-mediated TEM-1 beta-lactamases represent acquired resistance traits frequently found in Enterobacteriaceae rather than the classic intrinsic profile of *Pseudomonas*.

(e) Alterations in PBP2a are unique to Methicillin-Resistant *Staphylococcus aureus* (MRSA), and mutations in the gyrA gene alter DNA gyrase to confer resistance against fluoroquinolones, not beta-lactam antibiotics.

Final Answer: The primary mechanism is Inducible chromosomal AmpC beta-lactamase and active efflux pumps.

Answer: (B)

Go Back to Question 2

Q3.

Solution

Concept: *Listeria monocytogenes* is a facultative intracellular foodborne pathogen capable of crossing the intestinal, blood-brain, and placental barriers. Once internalized inside a host macrophage or non-professional phagocyte, the bacterium must quickly escape the phagosomal vesicle to avoid destruction by lysosomal enzymes.

Solution:

(a) Following internalization mediated by internalin surface proteins, the lower pH of the maturing phagocytic vacuole activates Listeriolysin O (LLO), a cholesterol-dependent pore-forming cytolysin secreted by the bacterium.

(b) Listeriolysin O, working synergistically with two distinct bacterial phospholipase C enzymes (PlcA and PlcB), disrupts and lyses the phagosomal membrane, releasing the pathogen directly into the host cell cytoplasm.

(c) Once inside the cytoplasm, the bacterium replicates and uses ActA protein to polymerize host cell actin, forming actin tails that propel it into neighboring cells without entering the extracellular environment.

(d) Lecithinase C is a toxin associated with *Clostridium perfringens* that damages cell membranes externally, causing gas gangrene.

(e) Cord factor is an integral cell wall glycolipid characteristic of *Mycobacterium tuberculosis* that prevents phagosome-lysosome fusion but does not lyse the vacuole or release the bacterium into the cytoplasm.

Final Answer: The mechanism is Secretion of Listeriolysin O and phospholipases.

Answer: (B)

Go Back to Question 3

Q4.

Solution

Concept: The practical differentiation of Gram-positive, catalase-positive cocci follows a strict laboratory algorithm based on biochemical testing. Once an organism is confirmed as a coagulase-negative *Staphylococcus* (CoNS), antibiotic susceptibility testing using a novobiocin disk is mandatory to identify uropathogens.

Solution:

(a) The biochemical diagnostic workflow indicates that a Gram-positive, catalase-positive, coagulase-negative coccus which displays resistance to novobiocin is classified as *Staphylococcus saprophyticus*.

(b) *Staphylococcus saprophyticus* is a well-documented cause of community-acquired urinary tract infections in young, sexually active females, trailing only *Escherichia coli* in incidence.

(c) Conversely, *Staphylococcus epidermidis* is a novobiocin-sensitive organism that typically forms biofilms on indwelling medical devices like prosthetic valves and intravenous catheters.

(d) *Staphylococcus lugdunensis* is also sensitive to novobiocin and is known for causing aggressive, destructive infective endocarditis resembling *Staphylococcus aureus* infections.

(e) *Enterococcus faecalis* is a catalase-negative, Gram-positive coccus arranged in pairs or short chains, placing it completely outside this diagnostic algorithm from the initial screening stage.

Final Answer: The organism is Staphylococcus saprophyticus.

Answer: (A)

Go Back to Question 4

Q5.

Solution

Concept: *Clostridium tetani* produces an extremely potent protein exotoxin known as tetanospasmin. This toxin is synthesized as a single polypeptide chain that is subsequently cleaved into a heavy chain and a light chain linked by a disulfide bridge, facilitating target-specific neurotoxicity.

Solution:

(a) Tetanospasmin is released at the site of a contaminated wound, enters peripheral motor nerve terminals via receptor-mediated endocytosis, and travels via retrograde axonal transport to the central nervous system.

(b) Upon reaching the presynaptic terminals of inhibitory interneurons (Renshaw cells) in the spinal cord, the light chain of the toxin acts as a zinc-dependent endopeptidase.

(c) This endopeptidase specifically cleaves synaptobrevin, a crucial Soluble NSF Attachment Protein Receptor (SNARE) protein required for vesicle docking and exocytosis.

(d) The destruction of synaptobrevin blocks the exocytosis of inhibitory neurotransmitters, specifically gamma-aminobutyric acid (GABA) and glycine, which normally dampen motor neuron activity.

(e) The loss of synaptic inhibition results in unchecked, continuous alpha motor neuron firing, manifesting clinically as generalized muscle spasms, trismus, and risus sardonicus.

Final Answer: The neurotoxic pathway is Cleaving Synaptobrevin to block the release of GABA and glycine in spinal interneurons.

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept: The emergence of beta-lactam resistance among nosocomial Enterobacteriaceae poses a severe challenge in neonatal intensive care units. Identifying the specific class of beta-lactamase produced is critical for selecting an appropriate antimicrobial regimen and enforcing infection control measures.

Solution:

(a) The clinical vignette describes *Klebsiella pneumoniae*, an indole-negative, non-motile, lactose-fermenting bacillus exhibiting resistance to third-generation cephalosporins.

(b) Extended-Spectrum Beta-Lactamases (ESBLs) are inhibited by clavulanic acid, whereas AmpC enzymes are typically resistant to clavulanic acid but can be selectively inhibited by boronic acid derivatives or cloxacillin.

(c) Hyperproduction of the chromosomal AmpC beta-lactamase enables the bacterium to hydrolyze penicillins, aztreonam, and third-generation cephalosporins like ceftriaxone and cefotaxime.

(d) Klebsiella pneumoniae Carbapenemase (KPC) and Metallo-beta-lactamase (MBL) production would confer broad resistance to carbapenems (like meropenem and imipenem), which is not indicated by this phenotypic profile.

(e) Therefore, the specific pattern of resistance and enzyme inhibition confirms the presence of hyperproduced chromosomal AmpC cephalosporinase.

Final Answer: The mechanism of resistance is Hyperproduction of chromosomal AmpC cephalosporinase.

Answer: (B)

Go Back to Question 6

Q7.

Solution

Concept: The complex lipid-rich cell wall of *Mycobacterium tuberculosis* accounts for its acid-fast staining characteristics, slow growth rate, resistance to common antibiotics, and capacity to survive inside host macrophages for extended periods.

Solution:

(a) Sulfatides (sulfolipids) are surface glycolipids that directly prevent the fusion of the phagosome containing the mycobacterium with host cell lysosomes, allowing intracellular survival.

(b) Cord factor (trehalose 6,6'-dimycolate) is a separate glycolipid responsible for the characteristic microscopic parallel arrangement of mycobacterial cells and is highly toxic to host tissues, inducing granulomatous inflammation.

(c) Lipoarabinomannan (LAM) is a major immunoregulatory cell wall component that suppresses T-cell activation and downregulates macrophage response to interferon-gamma.

(d) Mycolic acids form the long-chain fatty acid hydrophobic barrier of the cell wall but require specific esterification into functional glycolipids to actively modulate intracellular traffic.

(e) Therefore, the targeted disruption of phagosome-lysosome fusion is mediated primarily by the action of sulfatides within the host macrophage.

Final Answer: The specific cell wall component is Sulfatides.

Answer: (D)

Go Back to Question 7

Q8.

Solution

Concept: Human Parainfluenza Virus Type 1 (HPIV-1) is the most frequent cause of acute laryngotracheobronchitis (croup) in infants. It belongs to the Paramyxoviridae family and possesses a non-segmented, negative-sense, single-stranded RNA genome enclosed in a lipid envelope containing distinct surface spikes.

Solution:

(a) The envelope of Parainfluenza virus contains two major glycoprotein projections: the Hemagglutinin-Neuraminidase (HN) protein and the Fusion (F) protein.

(b) The HN protein is a multifunctional surface molecule that mediates the initial attachment of the virion to sialic acid receptors on the surface of respiratory epithelial cells.

(c) Concurrently, the HN protein binds to receptors on erythrocytes, causing cross-linking and hemagglutination, which serves as a classical laboratory diagnostic feature.

(d) The Fusion (F) protein is responsible for mediating the fusion of the viral envelope with the host cell plasma membrane at neutral pH and inducing syncytia formation.

(e) The Glycoprotein (G) attachment protein is found on Respiratory Syncytial Virus (RSV), which lacks hemagglutinating activity entirely, while the Matrix (M) protein is non-glycosylated and lines the inner viral envelope.

Final Answer: The viral surface glycoprotein is Hemagglutinin-Neuraminidase (HN) protein.

Answer: (A)

Go Back to Question 8

Q9.

Solution

Concept: Hepatitis B Virus (HBV) is a partially double-stranded DNA virus belonging to the Hepadnaviridae family. Its replication cycle is unique among DNA viruses because it requires an RNA intermediate phase, utilizing an encoded viral enzyme with reverse transcriptase activity.

Solution:

(a) Upon entering a hepatocyte, the viral relaxed circular DNA (rcDNA) is transported to the nucleus and repaired by host enzymes into covalently closed circular DNA (cccDNA).

(b) Host cell RNA Polymerase II transcribes the cccDNA into viral messenger RNAs, including a longer-than-genome-length 3.5kb pregenomic RNA (pgRNA) transcript.

(c) The pgRNA is packaged into new nucleocapsids in the cytoplasm, where the viral DNA polymerase acts as a reverse transcriptase to synthesize the negative-strand DNA using the pgRNA template.

(d) The viral polymerase then degrades the template RNA via inherent RNase H activity and synthesizes the complementary positive-strand DNA.

(e) This specialized life cycle makes the viral DNA-dependent DNA Polymerase with reverse transcriptase activity the essential target for nucleoside analog therapies like tenofovir and entecavir.

Final Answer: The viral enzyme is Viral DNA-dependent DNA Polymerase with reverse transcriptase activity.

Answer: (C)

Go Back to Question 9

Q10.

Solution

Concept: Cytomegalovirus (CMV) is a major cause of opportunistic viral disease in solid organ transplant recipients under intense immunosuppression. CMV retinitis can progress rapidly to permanent blindness if not diagnosed quickly and treated with systemic antivirals like ganciclovir.

Solution:

(a) The clinical presentation of full-thickness retinal necrosis with intraretinal hemorrhages is highly characteristic of CMV retinitis in an immunocompromised host.

(b) To confirm active viral replication in localized ocular compartments, detecting and quantifying viral nucleic acids via Polymerase Chain Reaction (PCR) is the most sensitive and rapid modality.

(c) Quantitative PCR (qPCR) performed on aqueous or vitreous humor samples provides definitive, real-time evidence of active CMV replication within the eye.

(d) Serological assays detecting anti-CMV IgM are unreliable in transplant recipients due to suppressed humoral responses and high baseline rates of past exposure.

(e) Tzanck smears are used for Herpes Simplex or Varicella-Zoster virus cutaneous lesions, and shell vial cultures have been superseded by faster, more sensitive molecular diagnostic methods.

Final Answer: The gold standard diagnostic modality is Quantitative Polymerase Chain Reaction (qPCR) for CMV DNA.

Answer: (B)

Go Back to Question 10

Q11.

Solution

Concept: Dengue virus, a member of the Flaviviridae family, has four distinct antigenic serotypes (DENV-1 to DENV-4). Primary infection with one serotype elicits lifelong immunity against that specific serotype but only temporary cross-protective immunity against heterologous serotypes, establishing a major immunological framework for severe disease complications.

Solution:

(a) During a secondary infection with a completely different dengue serotype, pre-existing sub-neutralizing, non-neutralizing IgG antibodies bind to the new viral variant, forming immune complexes.

(b) Instead of neutralizing the virus, the constant region (Fc portion) of these bound antibodies attaches to Fc-gamma receptors present on the surface of circulating monocytes and tissue macrophages.

(c) This antibody-mediated attachment facilitates the rapid entry and internalization of the virus into host phagocytic cells, exponentially increasing viral entry, transcription, replication, and total viral load.

(d) The infected monocytes subsequently trigger a severe systemic inflammatory cascade, releasing massive amounts of vasoactive cytokines such as tumor necrosis factor-alpha and interleukins into the circulation.

(e) This hyper-cytokinemia leads directly to widespread endothelial cell dysfunction, plasma leakage, severe thrombocytopenia, consumptive coagulopathy, and the clinical presentation of Dengue Hemorrhagic Fever.

Final Answer: The phenomenon is Antibody-Dependent Enhancement (ADE) mediated by non-neutralizing antibodies.

Answer: (A)

Go Back to Question 11

Q12.

Solution

Concept: *Pneumocystis jirovecii* is an opportunistic fungal pathogen that causes severe pneumonia in immunocompromised individuals, particularly those with advanced HIV disease. Although classified taxonomically as a fungus based on nucleic acid sequencing, its cell wall characteristics differ markedly from standard molds and yeasts.

Solution:

(a) The life cycle of *Pneumocystis jirovecii* includes morphologically distinct phases, switching between a fragile haploid trophic form and a highly resistant, thick-walled diploid cystic form.

(b) The trophic form is the primary metabolic phase responsible for adhering closely to type I alveolar pneumocytes, allowing the pathogen to extract nutrients and colonize respiratory spaces.

(c) During this trophic stage, the fungal cell wall completely lacks the complex carbohydrate polymer beta-(1,3)-D-glucan, which is only synthesized during cyst wall maturation.

(d) Echinocandin class antifungals, such as caspofungin and micafungin, function by non-competitively inhibiting the enzyme complex responsible for synthesizing beta-(1,3)-D-glucan.

(e) Because the actively replicating trophic forms lack this specific target component, echinocandins are ineffective at halting colonization, rendering the organism intrinsically resistant to cell wall-active agents during this phase.

Final Answer: The absent component is β-(1,3)-D-glucan.

Answer: (C)

Go Back to Question 12

Q13.

Solution

Concept: *Candida albicans* is a dimorphic opportunistic fungus that exists as a normal commensal of the human gastrointestinal, respiratory, and female genitourinary tracts. Pathogenic transition involves a rapid morphogenetic switch from a single-celled yeast phase to an invasive hyphal network.

Solution:

(a) In a laboratory setting, standard diagnostic confirmation of *Candida albicans* relies on testing its capability to undergo rapid phenotypic transformation when exposed to specific environmental triggers.

(b) When round or oval budding yeast cells are incubated in human serum or specialized broth at 37°C for 2 to 3 hours, they project specialized filaments known as germ tubes.

(c) True germ tubes are characterized by parallel-sided walls that demonstrate no constriction or narrowing at their specific site of origin from the parent blastoconidium cell body.

(d) This rapid morphological presentation contrasts with pseudohyphae, which show clear structural pinches or cell wall constrictions at each recurring septal junction along the filament chain.

(e) Production of these true germ tubes under these specific physical parameters provides an accurate, cost-effective confirmation that separates *Candida albicans* from non-albicans species.

Final Answer: The species is Candida albicans.

Answer: (C)

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Q14.

Solution

Concept: *Giardia duodenalis* (also known as *Giardia lamblia*) is a flagellated protozoan parasite that causes waterborne diarrheal illness following the ingestion of mature, hardy infectious cysts from contaminated water sources.

Solution:

(a) Upon ingestion, exposure to the highly acidic environment of the stomach induces excystation, releasing two metabolically active, flagellated vegetative trophozoites into the intestinal lumen.

(b) These released trophozoites migrate directly to the alkaline environment of the duodenum and upper jejunum, which provides optimal biochemical conditions for survival and growth.

(c) The trophozoite uses a large, prominent ventral sucking disk (adhesive disk) to mechanically attach to the microvilli of the mucosal brush border, avoiding clearance by peristaltic movements.

(d) This persistent mechanical attachment over large surface areas causes widespread flattening of intestinal villi, cellular inflammation, and the inhibition of mucosal brush border enzymes.

Final Answer: The primary zone of colonization is Duodenum and upper jejunum.

Answer: (B)

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Q15.

Solution

Concept: Hydatid disease (hydatidosis) is a zoonotic parasitic infection caused by the larval stage of the tapeworm *Echinococcus granulosus*. The lifecycle involves distinct animal hosts required to maintain transmission through specific prey-predator interactions.

Solution:

(a) The definitive host is the animal that harbors the adult, sexually mature stage of the tapeworm within its intestinal tract, shedding infectious eggs into the environment.

(b) Dogs and other wild canids serve as the definitive hosts for *Echinococcus granulosus*, expelling millions of microscopic embryonated eggs in their feces onto pastures and soil.

(c) Intermediate hosts, primarily sheep, cattle, and goats, ingest these eggs while grazing. The eggs hatch into oncospheres, cross the intestinal wall, and migrate to organs to form hydatid cysts.

(d) Humans act as accidental, dead-end intermediate hosts by ingesting food or water contaminated with canine fecal matter, leading to the formation of slow-growing cysts in the liver or lungs.

(e) Because the parasite lifecycle cannot be completed within a human host—as canine predators do not typically consume human tissue—man remains an evolutionary dead end.

Final Answer: The definitive host is Dog (and other canids).

Answer: (C)

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Q16.

Solution

Concept: Chronic Granulomatous Disease (CGD) is a primary immunodeficiency disorder typically inherited as an X-linked recessive trait. It affects phagocytic cells, preventing them from successfully killing certain engulfed microorganisms.

Solution:

(a) Phagocytes from CGD patients can ingest microorganisms normally but are unable to generate the reactive oxygen species required to kill them due to a defect in the NADPH oxidase enzyme complex.

(b) The multi-subunit NADPH oxidase complex transfers electrons from NADPH to molecular oxygen, generating the superoxide anion radical that initiates the oxidative burst inside the phagolysosome.

(c) Genetic mutations affecting key components like gp91phox or p22phox prevent the formation of this functional enzyme complex, abolishing microbicidal superoxide production.

(d) This defect impairs the clearance of catalase-positive pathogens (such as *Staphylococcus aureus* and *Aspergillus*), which degrade their own metabolic hydrogen peroxide, leaving the phagocyte without reactive oxygen radicals.

(e) The Nitroblue Tetrazolium (NBT) slide test assesses this oxidative capacity; normal neutrophils reduce the yellow dye to blue formazan crystals, whereas CGD cells fail to change color.

Final Answer: The inherited defect is in the NADPH oxidase enzyme complex.

Answer: (B)

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Q17.

Solution

Concept: Immunoglobulin molecules can be enzymatically cleaved by specific proteolytic enzymes into distinct fragments. This cleavage isolates their structural components, helping researchers analyze the separate mechanisms of antigen binding and biological effector functions.

Solution:

(a) Papain digestion cleaves the immunoglobulin heavy chains above the hinge region, breaking the interchain disulfide bonds that link the main stems.

(b) This enzymatic cleavage yields three separate fragments: two identical, monovalent Antigen-Binding Fragments (Fab) and one crystallizable Fragment (Fc) that mediates effector functions.

(c) Each Fab fragment contains one complete light chain and the amino-terminal variable and constant domains of one heavy chain, forming a single antigen-binding site.

(d) The variable domains of these paired chains form hypervariable loops (complementarity-determining regions) that bind specific antigenic epitopes, defining the idiotype.

(e) The Fc fragment contains the remaining carboxy-terminal constant domains of the heavy chains, which bind to cellular Fc receptors and initiate the classical complement cascade.

Final Answer: Region X corresponds to Fab fragment, responsible for antigen binding and idiotypic specificity.

Answer: (B)

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Q18.

Solution

Concept: Type III hypersensitivity reactions involve the systemic or localized deposition of circulating antigen-antibody (immune) complexes in tissue basement membranes. This deposition triggers the complement cascade, causing localized tissue damage.

Solution:

(a) In systemic lupus erythematosus, autoantibodies bind to cell-free DNA, and these immune complexes deposit along the glomerular basement membrane in the kidneys, activating the classical complement pathway.

(b) Cleavage of complement proteins during cascade activation generates small soluble glycopeptide fragments known as anaphylatoxins (C3a, C4a, and C5a).

(c) Among these fragments, C5a serves as the most potent anaphylatoxin and chemotactic factor for neutrophils, monocytes, and macrophages in vivo.

(d) C5a binds to specific high-affinity G-protein-coupled receptors on vascular endothelial cells and mast cells, triggering degranulation and increasing vascular permeability.

(e) This process establishes a strong chemotactic gradient that recruits neutrophils to the deposition site, where they release lysosomal enzymes and reactive oxygen species, causing tissue damage.

Final Answer: The most potent anaphylatoxin fragment is C5a.

Answer: (C)

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Q19.

Solution

Concept: Antigen presentation is required for T-lymphocytes to recognize protein antigens. Cells use two distinct intracellular pathways to process and display antigens based on whether the target protein originates inside or outside the cell.

Solution:

(a) The endogenous antigen presentation pathway processes proteins synthesized within the host cytoplasm, such as viral proteins or mutated tumor antigens.

(b) These cytoplasmic proteins are broken down into small peptide fragments by the proteasome complex, then transported across the membrane of the rough endoplasmic reticulum.

(c) This translocation is mediated by the Transporter associated with Antigen Processing (TAP1/TAP2) heterodimer, an ATP-binding cassette transporter.

(d) Inside the endoplasmic reticulum lumen, these transported peptides are loaded onto newly synthesized MHC Class I molecules before traveling through the Golgi apparatus to the cell surface.

(e) Inhibiting the TAP complex prevents endogenous peptides from entering the endoplasmic reticulum, selectively blocking the presentation of surface MHC Class I complexes to CD8+ cytotoxic T-cells.

Final Answer: The inhibition selectively impairs presentation of Endogenous antigens via MHC Class I to CD8+ T-cells.

Answer: (A)

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Q20.

Solution

Concept: Acute Graft-versus-Host Disease (aGVHD) is a major complication of allogeneic hematopoietic stem cell transplantation. It develops when immune cells in the donor graft recognize the recipient's tissues as foreign.

Solution:

(a) The pathogenesis of acute graft-versus-host disease requires three sequential phases: initial tissue damage from the conditioning regimen, donor T-cell activation, and cellular effector destruction.

(b) Mature, immunocompetent donor T-lymphocytes included in the bone marrow graft recognize minor and major histocompatibility antigens on the host cells as foreign.

(c) These donor T-cells proliferate and differentiate into cytotoxic effectors, migrating to target tissues that express high concentrations of these histocompatibility complexes.

(d) The primary target organs affected during this immunological attack include the skin, the gastrointestinal tract mucosa, and the biliary epithelial tree.

(e) Host-versus-graft reactions represent standard transplant rejection where recipient immune cells attack the graft, which is the opposite of the clinical presentation described here.

Final Answer: The mechanism is Mature donor T-lymphocytes present in the graft recognizing and attacking recipient histocompatibility antigens.

Answer: (C)

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Answer Key

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