NEET PG Microbiology Sample Paper-1

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 43-year-old structural construction worker presents with a deeply tracking puncture wound on his right lower extremity. Within 48 hours, he develops severe localized pain, marked crepitus, and a serosanguinous discharge with a foul odor. Anaerobic cultivation yields Lecithinase-positive, double-zone hemolytic colonies. What is the precise enzymatic mechanism of the primary lethal toxin driving this rapid necrotizing soft tissue destruction?

(A) Cleavage of synaptobrevin-2 in motor neurons

(B) ADP-ribosylation of elongation factor-2 (EF-2)

(C) Phospholipase C activity splitting lecithin into diglycerides

(D) Pore formation via cholesterol-dependent cytolysin activation

Q2. An isolates profile from an outbreak of acute dysentery in a pediatric ward reveals a Gram-negative bacillus that is non-motile, non-lactose fermenting, and catalase-negative. The toxin produced by this organism premium-targets the microvascular endothelial cells of the kidney. What is the exact intracellular target of this toxin's enzymatic subunit?

(A) Adenylate cyclase regulatory G-protein couples

(B) The 28S rRNA component of the 60S ribosomal subunit

(C) Elongation Factor 1-alpha binding domain

(D) Cleavage of the 16S rRNA component of the 30S subunit

Q3. A 34-year-old male presenting with high fever, relative bradycardia, and abdominal rose spots is suspected of having enteric fever. Blood cultures on Day 6 grow Gram-negative rods. Molecular analysis confirms the virulence of the organism is augmented by a distinct capsular polysaccharide that prevents complement-mediated opsonization. Which specific surface antigen corresponds to this description?

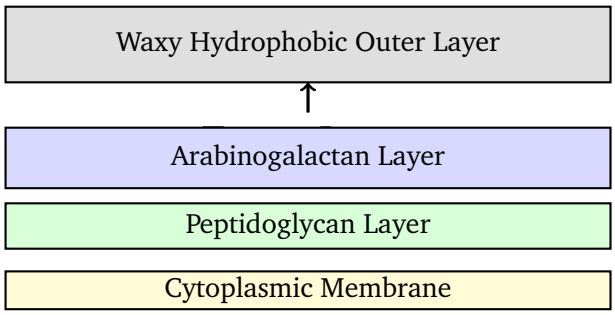
(A) Somatic O antigen

(B) Flagellar H antigen

(C) Vi capsular antigen

(D) K lipopolysaccharide core

Q4. A clinical specimen from a patient with a treatment-refractory cavitary pulmonary lesion demonstrates acid-fast bacilli. The remarkable resistance of this organism to desiccation, disinfectants, and intracellular killing is largely attributable to a specialized lipid-rich cell wall component. Examine the schematic representation of the cell envelope shown below and identify the structure labeled as **Target Layer**.



(A) Lipopolysaccharide (LPS) endotoxin layer

(B) Teichoic acid polymer network

(C) Mycolic acid-rich outer layer

(D) Phosphatidylinositol mannoside (PIM) anchor complex

Q5. A 29-year-old female presents with a painless, indurated genital ulcer with clean margins and firm borders. Dark-field microscopy of the exudate shows

actively motile, corkscrew-shaped spirochetes. What is the fundamental mechanism of motility utilized by this pathogen to navigate through dense extracellular matrices?

(A) Peritrichous flagella spinning external to the outer membrane

(B) Axial filaments (endoflagella) located in the periplasmic space

(C) Actin-based comet tail polymerization inside host cytoplasm

(D) Type IV pilus-mediated twitching mechanism

Q6. A 62-year-old intensive care patient on mechanical ventilation develops nosocomial pneumonia. Sputum culture on MacConkey agar demonstrates pale, non-lactose fermenting colonies that produce a distinct sweet, grape-like odor and a blue-green pigment. This organism possesses an outer membrane porin channel configuration that confers intrinsic resistance to a vast array of beta-lactam antibiotics. Name the organism.

(A) *Klebsiella pneumoniae*

(B) *Acinetobacter baumannii*

(C) *Pseudomonas aeruginosa*

(D) *Serratia marcescens*

Q7. A 22-year-old student presents with acute pharyngitis. A throat swab is cultured on blood agar and demonstrates beta-hemolytic colonies that are highly sensitive to bacitracin. The major virulence factor of this pathogen is a surface protein that prevents phagocytosis by binding plasma fibrinogen and inhibiting the alternative complement pathway. Which structural protein is responsible?

(A) Protein A

(B) M Protein

(C) F Protein

(D) Lipoteichoic acid

Q8. A 38-year-old HIV-positive patient with a CD4+ count of 45 cells/μL presents with progressive visual loss. Ophthalmoscopy reveals extensive retinal necrosis with perivascular hemorrhages ("pizza-pie retinopathy"). The causative double-stranded DNA virus relies on an absolute structural viral encoded protein kinase for its initial intracellular phosphorylation step during therapeutic activation of ganciclovir. Which viral gene encodes this kinase?

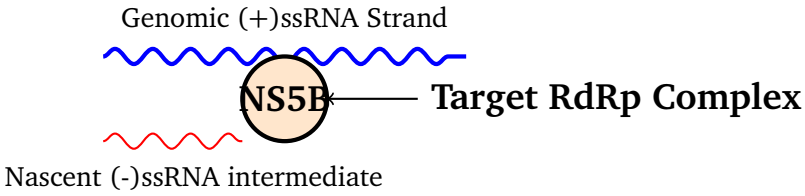
(A) UL97

(B) UL54

(C) US11

(D) UL83

Q9. A 48-year-old intravenous drug user with chronic fatigue is diagnosed with Hepatitis C virus (HCV) infection, genotype 1a. During intracellular viral replication, the genomic positive-sense single-stranded RNA serves directly as a template to produce a full-length negative-sense strand replication intermediate. Look at the structural scheme of the replication replication hub below. Identify the specialized viral non-structural protein designated as the **Target RdRp Complex** executing this template-directed transcription:



(A) NS3/4A Protease

(B) NS5A Phosphoprotein

(C) NS5B RNA-dependent RNA polymerase

(D) NS2-3 Autoprotease

Q10. An infant presenting with severe winter-associated watery diarrhea, vomiting, and dehydration is found to be infected with a segmented, double-stranded RNA virus. The major enterotoxin produced by this pathogen alters membrane permeability, inducing a phospholipase C-dependent calcium influx that

disrupts the tight junctions of enterocytes. What is this viral non-structural toxin?

(A) NSP4

(B) VP4

(C) VP7

(D) NSP1

Q11. A 26-year-old pregnant female at 12 weeks gestation presents with a mild maculopapular rash, low-grade fever, and prominent retroauricular lymphadenopathy. Serology confirms acute infection with a positive-sense, enveloped, single-stranded RNA virus belonging to the Matonaviridae family. What is the classic structural cardiovascular malformation seen in infants born with congenital syndrome from this virus?

(A) Transposition of the great arteries

(B) Tetralogy of Fallot

(C) Patent ductus arteriosus

(D) Coarctation of the aorta

Q12. A bone marrow biopsy from a patient with advanced AIDS presenting with hepato-splenomegaly and pancytopenia reveals small, oval intracellular yeast cells measuring 2-4 micrometers within macrophages, featuring a prominent narrow base of budding. The patient lives in the Ohio-Mississippi River Valley region. What is the most definitive primary morphological description of this systemic dimorphic fungus in its environmental phase?

(A) Thick-walled spherules filled with numerous endospores

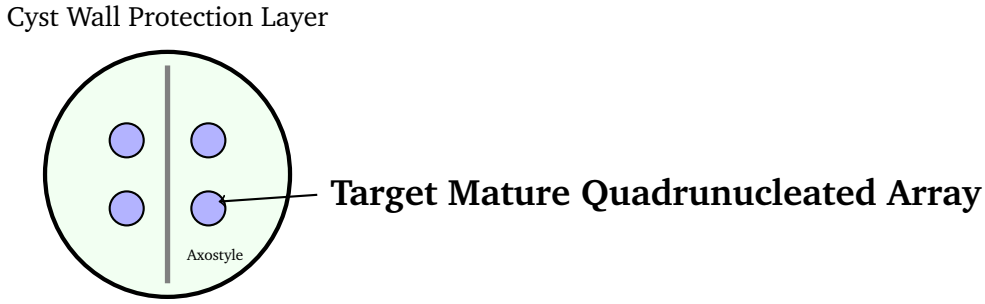
(B) Septate hyphae with large, thick-walled, tuberculate macroconidia

(C) Arthroconidia alternating with empty disjunctor cells

(D) Captain's wheel configuration of multiple budding yeasts

Q13. A 25-year-old traveler returning from an extended rural expedition presents with chronic, foul-smelling, fatty diarrhea, flatulence, and significant weight

loss. Duodenal aspirate analysis shows an active trophozoite form. Examine the architectural diagram below detailing the cross-section of the diagnostic environmental survival stage of this parasite. Identify the internal target element labeled as the **Target Mature Quadrunucleated Array**:



(A) Entamoeba histolytica chromatoid body

(B) Giardia duodenalis mature four-nuclei cluster

(C) Cryptosporidium parvum four-sporozoite packet

(D) Balantidium coli macro-and-micronucleus core

Q14. A 35-year-old male neutropenic patient undergoing remission induction chemotherapy for acute myeloid leukemia develops acute pleuritic chest pain and hemoptysis. A high-resolution CT of the chest demonstrates pulmonary nodules surrounded by a perimeter of ground-glass opacity (the "halo sign"). Tissue biopsy reveals acute-angle (45°) dichotomously branching septate hyphae invading blood vessel walls. What is the fundamental mechanism of angioinvasion used by this fungal pathogen?

(A) Elastase and serine protease degradation of structural matrix

(B) Melanin deposition in cell walls neutralizing host oxidative burst

(C) Massive production of toxic lipopolysaccharide-like complexes

(D) Pseudohyphal transition driven by thermal upshifting

Q15. A 28-year-old female presents with an exquisite, intensely pruritic, serpiginous skin lesion on the dorsum of her right foot after walking barefoot on a tropical sandy beach. The lesion advances a few millimeters per day. This clinical condition, cutaneous larva migrans, is caused by the infective filariform larvae of which parasite?

(A) *Necator americanus*

(B) *Ancylostoma braziliense*

(C) *Strongyloides stercoralis*

(D) *Toxocara canis*

Q16. A 2-year-old boy presenting with recurrent severe pyogenic skin infections and delayed separation of the umbilical cord is found to have a profound leukocytosis with neutrophils failing to accumulate at sites of inflammation. Flow cytometry confirms the absence of CD18. Which structural molecule's failure to form prevents firm adhesion of leukocytes to the activated endothelium?

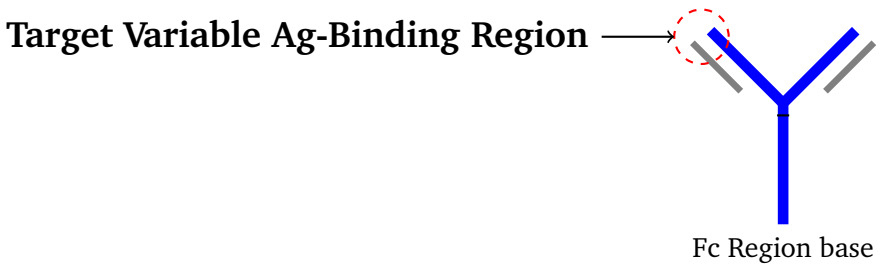
(A) Sialyl-Lewis X ligands

(B) L-selectin receptors

(C) β_2-integrins (LFA-1, Mac-1)

(D) GlyCAM-1 adhesion molecules

Q17. A 6-month-old male infant presents with recurrent sinopulmonary bacterial infections. Laboratory evaluation reveals a complete absence of circulating B cells and pan-hypogammaglobulinemia, confirming X-linked agammaglobulinemia (XLA). Look at the basic structural blueprint of a monomeric immunoglobulin molecule shown below. Identify the region labeled as the **Target Variable Ag-Binding Region** that is entirely missing or unexpressed due to the upstream failure of heavy-chain gene rearrangement:



(A) Constant heavy domain (C_H2)

(B) Variable heavy and light domains ($V_H + V_L$)

(C) Hinge sequence link region

(D) Ch2-Ch3 complement-binding pocket

Q18. A 45-year-old male with long-standing end-stage renal disease receives a living-unrelated kidney transplant. Within minutes of establishing vascular anastomosis, the graft becomes soft, cyanotic, and mottled, with widespread thrombotic occlusion of the glomerular capillaries. This hyperacute rejection episode is fundamentally driven by which pre-existing immune component?

(A) Donor-specific pre-formed circulating IgG antibodies

(B) Host de novo generated CD8+ cytotoxic T lymphocytes

(C) Host NK cell-mediated antibody-dependent cytotoxicity

(D) Host CD4+ Th1 cytokine-mediated macrophage activation

Q19. During an elegant in vitro immunology experiment, naive CD4+ T helper cells are cultivated in an environment containing high concentrations of IL-12 and IFN-γ. This cytokine profile switches on a key lineage-specific master transcription factor that drives the selective differentiation of these cells into the Th1 subset. Which transcription factor is induced?

(A) GATA-3

(B) RORγt

(C) T-bet

(D) FoxP3

Q20. A 4-year-old child presents with partial albinism, recurrent severe staphylococcal skin infections, and progressive peripheral neuropathy. Peripheral blood smear reveals pathognomonic massive, giant lysosomal granules within neutrophils and platelets. This condition arises from a defect in a specific cytosolic transport protein that regulates vesicle trafficking. Name the condition.

(A) Chronic Granulomatous Disease

(B) Chediak-Higashi Syndrome

(C) Job Syndrome (Hyper-IgE)

(D) Wiskott-Aldrich Syndrome

Detailed Solutions

Q1.

Solution

Concept: Gas gangrene (myonecrosis) is a rapidly progressive, life-threatening destructive soft tissue infection primarily caused by the anaerobic, spore-forming rod *Clostridium perfringens*.

Solution:

Let's break down the clinical markers and virulence mechanisms of this infection:

(a) The combination of a deep tracking puncture wound, sudden severe pain, crepitus (gas production in tissue), and foul-smelling serosanguinous discharge is classic for gas gangrene.

(b) On blood agar, *C. perfringens* is identified by its characteristic double zone of hemolysis and a positive Nagler reaction confirming lecithinase activity.

(c) The primary virulence factor driving this pathogenesis is the **alpha-toxin**, a lethal zinc-dependent metalloenzyme. Biochemically, alpha-toxin functions as a **Phospholipase C (lecithinase)** that splits lecithin (phosphatidylcholine) into diglycerides and phosphorylcholine. This breaks down host cell membranes, causing widespread myonecrosis, hemolysis, and vascular damage.

Final Answer: Phospholipase C activity splitting lecithin into diglycerides

Answer: (C)

Go Back to Question 1

Q2.

Solution

Concept: The isolated organism is *Shigella dysenteriae* type 1, an aggressive Gram-negative enteric bacillus that produces Shiga toxin (Stx) and causes severe, epidemic bacillary dysentery.

Solution:

Let's map out the microbiological traits and specific toxin targeting:

(a) Gram-negative, non-motile, non-lactose fermenting, and catalase-negative features distinguish *Shigella* from other enterics. Shiga toxin targets renal microvascular endothelial cells, which can lead to Hemolytic Uremic Syndrome (HUS).

(b) Shiga toxin is an AB_5-subunit exotoxin. The B subunits bind to host cell surface glycolipids (Gb_3), facilitating endocytosis.

(c) Once internalized, the enzymatically active A subunit is cleaved into an A_1 fragment. This fragment functions as an N-glycosidase that cleaves a single adenine residue from the **28S rRNA component of the 60S ribosomal subunit**. This modification blocks aminoacyl-tRNA binding, shutting down protein synthesis and causing cell death.

Final Answer: The 28S rRNA component of the 60S ribosomal subunit

Answer: (B)

Go Back to Question 2

Q3.

Solution

Concept: Enteric (typhoid) fever is caused by the systemic pathogen *Salmonella enterica* serovar Typhi, which avoids early host immune clearance via specialized surface structures.

Solution:

Let's analyze the virulence antigens of *Salmonella* Typhi:

(a) The clinical picture—characterized by a high step-ladder fever, relative bradycardia (Faget sign), and abdominal rose spots—is pathognomonic for typhoid fever.

(b) The specific virulence factor described is an outer capsular polysaccharide known as the **Vi (Virulence) capsular antigen**.

(c) The Vi capsule forms a physical barrier over the underlying somatic O antigen. This layer shields the bacterium from host immune recognition, preventing complement fixation, opsonization, and neutrophil-mediated killing. This allows the pathogen to survive and replicate inside macrophages.

Final Answer: Vi capsular antigen

Answer: (C)

Go Back to Question 3

Q4.

Solution

Concept: *Mycobacterium tuberculosis* has a unique, lipid-rich cell wall architecture that makes it resistant to destruction by macrophages and standard staining techniques.

Solution:

Let's identify the structural layers of the mycobacterial cell envelope shown in the diagram:

(a) The inner layer consists of the plasma membrane, which is covered by a peptidoglycan layer linked to an arabinogalactan sugar matrix.

(b) The outermost layer (labeled as the Target Hydrophobic Mycolate Layer) is composed of a dense, protective arrangement of long-chain fatty acids.

(c) These long-chain β-hydroxy fatty acids are **mycolic acids**, which are covalently bound to the underlying arabinogalactan matrix. This thick, waxy layer creates a hydrophobic shield that blocks lysosomal enzymes and chemical insults, giving the organism its characteristic acid-fast staining property.

Final Answer: Mycolic Acid Covalently Bound Array

Answer: (C)

Go Back to Question 4

Q5.

Solution

Concept: The patient presents with a hard chancre, the classic lesion of primary syphilis, which is caused by the spirochete *Treponema pallidum*.

Solution:

Let's look at the unique structural locomotion system of spirochetes:

(a) *Treponema pallidum* is a thin, helical bacterium with a distinctive corkscrew-like motility that allows it to penetrate mucous membranes and move through viscous extracellular matrix environments.

(b) Unlike most bacteria, which rely on external flagella, spirochetes utilize specialized **axial filaments (endoflagella)**.

(c) These endoflagella are anchored at the cell poles and reside entirely within the **periplasmic space**, between the peptidoglycan cell wall and the outer membrane layer. Rotation of these axial filaments causes the entire outer cell body to twist in a corkscrew motion, driving locomotion without exposing the flagellar proteins to host antibodies.

Final Answer: Axial filaments (endoflagella) located in the periplasmic space

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept: *Pseudomonas aeruginosa* is an opportunistic, Gram-negative, non-lactose fermenting rod that is a major cause of healthcare-associated infections, particularly in intensive care units.

Solution:

Let's evaluate the specific diagnostic and resistance features of the pathogen:

(a) The organism grows as pale, non-lactose fermenting colonies on MacConkey agar, produces a distinct fruity, grape-like sweet odor, and synthesizes pyocyanin/pyoverdine pigments that impart a characteristic blue-green color.

(b) A key factor behind its multi-drug resistance profile is its highly selective outer membrane structure.

(c) ****Pseudomonas aeruginosa**** possesses an outer membrane porin network with a very low baseline permeability. It specifically lacks or down-regulates ****OprD**** porin channels, which are normally required for carbapenems and other β-lactams to enter. Combined with active efflux pumps, this structural barrier confers intrinsic resistance to a broad range of antibiotics.

Final Answer: *Pseudomonas aeruginosa*

Answer: (C)

Go Back to Question 6

Q7.

Solution

Concept: Acute pharyngitis presenting with bacitracin-sensitive, β-hemolytic colonies on blood agar is caused by *Streptococcus pyogenes* (Group A Streptococcus, GAS).

Solution:

Let's trace the molecular pathogenesis of the primary virulence factor of GAS:

(a) The major virulence factor embedded in the cell wall of *Streptococcus pyogenes* is the **M Protein**.

(b) The M protein extends from the cell surface as a coiled-coil heterodimer. It acts as an anti-phagocytic factor by binding host plasma fibrinogen, which forms a surface coat that hides underlying bacterial antigens.

(c) Additionally, the M protein binds host complement regulatory proteins (such as Factor H). This accelerates the decay of the C3 convertase, inhibiting the alternative complement pathway and blocking opsonization by C3b.

Final Answer: M Protein

Answer: (B)

Go Back to Question 7

Q8.

Solution

Concept: The patient is suffering from Cytomegalovirus (CMV) retinitis, a classic opportunistic infection seen in advanced HIV/AIDS when CD4+ counts fall below 50 cells/μL.

Solution:

Let's examine the metabolic activation pathway of the first-line antiviral drug ganciclovir:

(a) Ganciclovir is a nucleoside analogue that inhibits viral DNA polymerase, but it must first undergo a three-step phosphorylation sequence inside the host cell to become active.

(b) The initial, rate-limiting monophosphorylation step cannot be performed by host cell kinases; it requires a specific enzyme encoded by the virus.

(c) This enzyme is a protein kinase encoded by the **UL97 gene** of Cytomegalovirus. Once UL97 performs the initial monophosphorylation, cellular enzymes add two more phosphate groups to form ganciclovir triphosphate, which selectively inhibits the viral DNA polymerase (encoded by UL54). Mutations in the *UL97* gene are the primary cause of clinical ganciclovir resistance.

Final Answer: UL97

Answer: (A)

Go Back to Question 8

Q9.

Solution

Concept: Hepatitis C Virus (HCV) is an enveloped, positive-sense single-stranded RNA virus. It relies on non-structural proteins (NS) to assemble its replication complex on host intracellular membranes.

Solution:

Let's identify the enzymatic function of the proteins involved in the HCV replication hub:

(a) The diagram illustrates template-directed transcription, where the incoming positive-sense genomic RNA serves as a template to synthesize a complementary negative-sense RNA intermediate strand.

(b) This transcription step is catalyzed exclusively by an RNA-dependent RNA polymerase (RdRp).

(c) The **NS5B** gene product is the specialized **RNA-dependent RNA polymerase** that serves as the catalytic core of the replication complex. Because human cells lack an engine to transcribe RNA from an RNA template, NS5B is a primary target for direct-acting antiviral medications (such as Sofosbuvir).

Final Answer: NS5B RNA-dependent RNA polymerase

Answer: (C)

Go Back to Question 9

Q10.

Solution

Concept: Rotavirus (a segmented Reovirus) is a major cause of severe, dehydrating infantile diarrhea worldwide, typically peaking during the winter months.

Solution:

Let's isolate the mechanisms of the distinct viral enterotoxin responsible for this presentation:

(a) Rotavirus induces diarrhea through both malabsorptive changes and secretory pathways, driven by a specific viral non-structural protein that functions as an enterotoxin.

(b) This viral enterotoxin is **NSP4** (Non-Structural Protein 4).

(c) NSP4 acts via a phospholipase C-dependent pathway to trigger the release of calcium ions from the endoplasmic reticulum into the host cell cytosol. This sudden calcium influx disrupts the tight junctions (claudins/occludins) between enterocytes, increasing paracellular permeability and stimulating chloride ion secretion. This leads to profound secretory diarrhea.

Final Answer: NSP4

Answer: (A)

Go Back to Question 10

Q11.

Solution

Concept: The patient is presenting with Rubella (German measles), which is caused by the Rubella virus (a member of the Matonaviridae family). Maternal infection during the first trimester can lead to Congenital Rubella Syndrome (CRS).

Solution:

Let's review the classic teratogenic manifestations of Congenital Rubella Syndrome:

(a) The classic clinical triad of CRS consists of sensorineural deafness, ocular abnormalities (cataracts, microphthalmia), and congenital cardiac anomalies.

(b) The most characteristic structural cardiovascular malformation associated with CRS is a **Patent Ductus Arteriosus (PDA)** (often accompanied by pulmonary artery branch stenosis).

(c) The virus infects the vascular endothelium during development, inhibiting cellular mitosis and causing structural hypoplasia of the vascular wall. This prevents the normal muscular constriction and closure of the ductus after birth.

Final Answer: Patent ductus arteriosus

Answer: (C)

Go Back to Question 11

Q12.

Solution

Concept: The biopsy shows intracellular yeasts within macrophages, which is diagnostic for *Histoplasma capsulatum*, a systemic dimorphic fungus endemic to the Ohio-Mississippi River Valley.

Solution:

Let's describe the morphological appearance of this dimorphic fungus in its different phases:

(a) In host tissues at 37°C, *Histoplasma capsulatum* exists as small, oval yeast cells (2-4 μM) found inside macrophages.

(b) In its environmental phase (saprophytic mold form grown on Sabouraud agar at 25°C), it develops a different morphology.

(c) The environmental form produces **septate hyphae with large, thick-walled, round macroconidia covered in finger-like projections (tuberculate macroconidia)**, along with smaller, infectious microconidia.

Final Answer: Septate hyphae with large, thick-walled, tuberculate macroconidia

Answer: (B)

Go Back to Question 12

Q13.

Solution

Concept: The patient's presentation—chronic, foul-smelling, fatty diarrhea (steatorrhea) following outdoor travel—points to a infection with the flagellated protozoan *Giardia duodenalis* (also known as *G. lamblia* or *G. intestinalis*).

Solution:

Let's analyze the parasite structure shown in the diagnostic diagram:

(a) The diagram depicts the oval, resilient environmental survival stage (cyst stage) of the parasite, which is transmitted via contaminated water.

(b) Internally, this structure contains longitudinal axostyles and a characteristic cluster of nuclei labeled as the Target Mature Quadrunucleated Array.

(c) A mature, infective ***Giardia duodenalis*** cyst typically contains a cluster of four nuclei**. When ingested, it undergoes excystation in the acidic environment of the stomach, releasing two binucleated, pear-shaped trophozoites that attach to the duodenal mucosa via ventral sucking disks.

Final Answer: Giardia duodenalis mature four-nuclei cluster

Answer: (B)

Go Back to Question 13

Q14.

Solution

Concept: Invasive Aspergillosis is a severe, life-threatening infection seen in profoundly immunocompromised patients, particularly those with prolonged neutropenia following chemotherapy.

Solution:

Let's evaluate the virulence factors that drive vascular invasion by *Aspergillus fumigatus*:

(a) The combination of a pulmonary "halo sign" on a CT scan and tissue biopsy showing septate hyphae that branch at acute 45-degree angles is pathognomonic for *Aspergillus*.

(b) *Aspergillus* hyphae are highly angioinvasive; they track into, breach, and occlude blood vessel walls, leading to tissue infarction, necrosis, and hemoptysis.

(c) This invasive migration is driven by the secretion of fungal proteases, specifically **elastases, serine proteases, and metalloproteinases**. These enzymes break down the structural elastin and collagen matrix within the internal elastic lamina of host blood vessels, allowing the hyphae to penetrate the vascular wall.

Final Answer: Elastase and serine protease degradation of structural matrix

Answer: (A)

Go Back to Question 14

Q15.

Solution

Concept: Cutaneous Larva Migrans (creeping eruption) is a zoonotic parasitic skin infection characterized by a progressive, intensely itchy, serpiginous erythematous track.

Solution:

Let's isolate the specific etiologic agent behind this clinical presentation:

(a) The disease occurs when a person walks barefoot on sandy soil or beaches contaminated with animal feces containing hookworm larvae.

(b) The primary pathogen responsible for this condition is ****Ancylostoma braziliense****, a hookworm found in dogs and cats.

(c) The infective filariform larvae penetrate the unprotected human skin. Because humans are incidental, non-definitive hosts, the larvae lack the collagenase enzymes required to breach the basement membrane and enter the systemic circulation. As a result, they remain confined to the epidermis, migrating aimlessly at a rate of a few millimeters per day and provoking a local hypersensitivity reaction.

Final Answer: *Ancylostoma braziliense*

Answer: (B)

Go Back to Question 15

Q16.

Solution

Concept: Leukocyte Adhesion Deficiency Type 1 (LAD-1) is an autosomal recessive immunodeficiency caused by mutations in the *ITGB2* gene, which encodes CD18.

Solution:

Let's map out the molecular consequences of a CD18 deficiency:

(a) The clinical hallmarks—recurrent bacterial infections without pus formation, delayed umbilical cord separation, and high circulating neutrophilia—point directly to LAD-1.

(b) CD18 is the common β_2 integrin subunit. It must non-covalently pair with different α subunits (CD11a, CD11b, CD11c) to form functional surface integrin complexes like LFA-1 (CD11a/CD18) and Mac-1 (CD11b/CD18).

(c) Without functional **β_2-integrins**, leukocytes can roll along the endothelium (a step mediated by selectins), but they cannot establish the high-affinity binding required for **firm adhesion and stable arrest**. This prevents them from migrating out of blood vessels into inflamed tissues.

Final Answer: β_2-integrins (LFA-1, Mac-1)

Answer: (C)

Go Back to Question 16

Q17.

Solution

Concept: X-linked Agammaglobulinemia (XLA) is caused by a mutation in the *BTK* (Bruton Tyrosine Kinase) gene, which arrests B-cell development at the pre-B-cell stage.

Solution:

Let's identify the immunoglobulin domain highlighted in the structural diagram:

(a) In XLA, the absence of functional Bruton Tyrosine Kinase halts signaling through the pre-B-cell receptor. This prevents heavy-chain VDJ gene rearrangement, leading to a failure of B-cell maturation and an absence of circulating antibodies.

(b) The highlighted target zone (enclosed by the red dashed circle) marks the terminal tips of the Fab arms of the monomeric antibody structure.

(c) This specific structural domain is the **Variable heavy and light domains ($V_H + V_L$)**, which form the unique antigen-binding cleft (paratope). Without proper gene rearrangement, these variable antigen-binding regions cannot be expressed.

Final Answer: Variable heavy and light domains ($V_H + V_L$)

Answer: (B)

Go Back to Question 17

Q18.

Solution

Concept: Hyperacute transplant rejection occurs within minutes to hours after vascular anastomosis is completed during organ transplantation.

Solution:

Let's analyze the immunological triggers behind hyperacute rejection:

(a) The rapid onset of graft failure—where the kidney becomes soft, cyanotic, and mottled right after vascular connection—is a classic manifestation of hyperacute rejection.

(b) This reaction is mediated by **pre-formed donor-specific IgG antibodies** present in the recipient's circulation (e.g., anti-ABO blood group or anti-HLA antibodies).

(c) When blood flow is established, these circulating antibodies immediately bind to matching antigens on the donor organ's vascular endothelial cells. This triggers complement activation, recruitment of neutrophils, and diffuse platelet thrombi formation within the glomerular capillaries, leading to ischemic necrosis of the graft.

Final Answer: Donor-specific pre-formed circulating IgG antibodies

Answer: (A)

Go Back to Question 18

Q19.

Solution

Concept: The differentiation of naive CD4+ T helper cells into distinct functional subsets (T_H1, T_H2, T_H17, or T_{reg}) is regulated by the cytokine profile of their immediate microenvironment.

Solution:

Let's map out the master transcription factors linked with each T-helper subset:

(a) Exposure to Interleukin-12 (IL-12) and Interferon-gamma (IFN-γ) activates the STAT1 and STAT4 signaling pathways within naive T cells.

(b) This signaling cascade induces the expression of the master lineage-specific transcription factor **T-bet** (encoded by the *TBX21* gene).

(c) **T-bet** commits the cell to the T_H1 lineage, driving the production of IFN-γ and IL-2 to support cell-mediated immunity.

(d) (Note: GATA-3 regulates T_H2 differentiation, RORγt controls T_H17, and FoxP3 guides regulatory T cells).

Final Answer: T-bet

Answer: (C)

Go Back to Question 19

Q20.

Solution

Concept: Chediak-Higashi Syndrome is a rare autosomal recessive disorder characterized by a defect in intracellular vesicular trafficking.

Solution:

Let's evaluate the clinical triad and morphological hallmarks of this syndrome:

(a) The syndrome is caused by mutations in the **LYST** (Lysosomal Trafficking Regulator) gene, which regulates intracellular protein transport and vesicle fusion.

(b) This defect leads to the formation of **giant, dysfunctional lysosomal granules** inside neutrophils, platelets, and melanocytes, because smaller vesicles fail to separate or fuse properly.

(c) The clinical manifestations follow this structural defect: partial albinism (due to abnormal melanosome distribution), recurrent pyogenic staphylococcal infections (due to impaired neutrophil degranulation), and progressive peripheral neuropathy (due to defective axonal transport).

Final Answer: Chediak-Higashi Syndrome

Answer: (B)

Go Back to Question 20

Answer Key

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