NEET PG Microbiology Sample Paper-4

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 42-year-old male presenting with spiking fevers and progressive aortic valve insufficiency undergoes an urgent valve replacement. Histopathological analysis of the resected vegetation reveals dense aggregates of small, pleomorphic Gram-negative coccobacilli that are highly fastidious. Molecular analysis confirms an organism that requires exogenous hemin (X-factor) but is completely independent of V-factor (β-nicotinamide adenine dinucleotide) for growth. Which of the following species is the most likely causative pathogen?

(A) Aggregatibacter actinomycetemcomitans

(B) Haemophilus aphrophilus

(C) Haemophilus ducreyi

(D) Haemophilus parainfluenzae

Q2. A multi-drug resistant clinical isolate of *Pseudomonas aeruginosa* from a deep burn wound is analyzed for active resistance mechanics. The strain demonstrates hyper-resistance to both cefepime and meropenem. Genetic sequencing reveals a chromosomal mutation causing constitutive upregulation of a tripartite resistance-nodulation-division (RND) family efflux pump. Which specific protein system is primarily implicated in this phenotype?

(A) MexAB-OprM

(B) MacAB-TolC

(C) AcrAB-TolC

(D) AdeABC

Q3. A 33-year-old female patient diagnosed with advanced pulmonary tuberculosis fails standard first-line therapy. Phenotypic drug susceptibility testing indicates high-level resistance to isoniazid ($> 1.0\,\mu g/mL$). Molecular testing reveals a specific missense mutation in the regulatory promoter region of the *inhA* gene rather than a deletion in the *katG* gene. What clinical or pharmacological consequence does this specific mechanism entail?

(A) Complete cross-resistance to ethionamide

(B) High-level cross-resistance to pyrazinamide

(C) Total preservation of susceptibility to ethionamide

(D) Absolute resistance to high-dose isoniazid monotherapy

Q4. During a laboratory validation process, a technician isolates a Gram-positive, anaerobic, non-spore-forming bacillus from a pelvic abscess secondary to an intrauterine device. The bacterium exhibits a characteristic "sulfur granule" appearance in tissue, but under specialized microscopic inspection, shows true branching filaments. Biochemically, it is catalase-negative and does not reduce nitrate. Which pathogen matches this metabolic profiling?

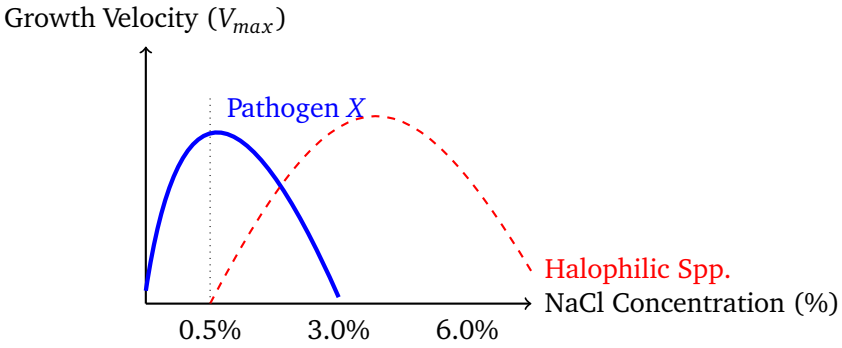
(A) Actinomyces israelii

(B) Actinomyces gerencseriae

(C) Nocardia asteroides

(D) Propionibacterium acnes

Q5. A complex clinical investigation of an outbreak of atypical, water-borne enteritis reveals a Gram-negative bacillus with a curved morphology. The diagram below represents the metabolic response curves of this pathogen under varying sodium chloride (NaCl) concentrations and alkaline conditions. Identify the specific pathogen corresponding to this biochemical footprint:



(A) Vibrio vulnificus

(B) Vibrio cholerae

(C) Vibrio parahaemolyticus

(D) Campylobacter jejuni

Q6. An infant presenting with severe bronchiolitis is diagnosed with Human Respiratory Syncytial Virus (RSV). The structural assembly of the RSV virion depends on various surface glycoproteins. A new therapeutic monoclonal antibody targets the highly conserved prefusion conformation of the viral protein responsible for membrane fusion. Which structural element must remain locked in this specific state to neutralize viral entry effectively?

(A) Glycoprotein G

(B) Glycoprotein F

(C) Matrix Protein M

(D) Small Hydrophobic Protein SH

Q7. A 29-year-old HIV-positive individual with a CD4 count of 45 cells/μL presents with progressive neurological decline, ataxia, and cognitive deficits. Cognitive testing and an MRI reveal asymmetric, non-enhancing white matter demelinating lesions without mass effect. A lumbar puncture confirms the presence of John Cunningham (JC) virus DNA. What specific viral structural or non-structural regulatory gene mutation transforms the latent archetypal JC virus into the neurotropic, pathogenic form causing Progressive Multifocal Leukoencephalopathy (PML)?

(A) Rearrangement in the Non-Coding Control Region (NCCR)

(B) Missense mutation in the VP1 major capsid gene

(C) Frame-shift deletion within the Small t-antigen gene

(D) Promoter duplication within the VP2 structural gene

Q8. A chronic Hepatitis B patient undergoes quantitative serological profiling. The lab reports high levels of serum HBsAg, HBeAg, and HBV DNA ($\approx 10^8$ copies/mL), yet transaminase levels (ALT/AST) remain completely within the normal physiological range. Histological biopsy shows minimal lobular inflammation. Which immunopathogenic phase of chronic Hepatitis B infection does this clinical presentation capture?

(A) Immune Tolerance Phase

(B) Immune Clearance (Reactivation) Phase

(C) Inactive HBsAg Carrier State

(D) Occult HBV Infection Phase

Q9. An outbreak of acute hemorrhagic conjunctivitis is tracked across an ophthalmology clinic. Patients exhibit subconjunctival hemorrhages, periorbital pain, and transient corneal involvement. The causative agent is an unenvloped, positive-sense single-stranded RNA virus belonging to the *Picornaviridae* family. Which specific serotype is responsible for this aggressive presentation?

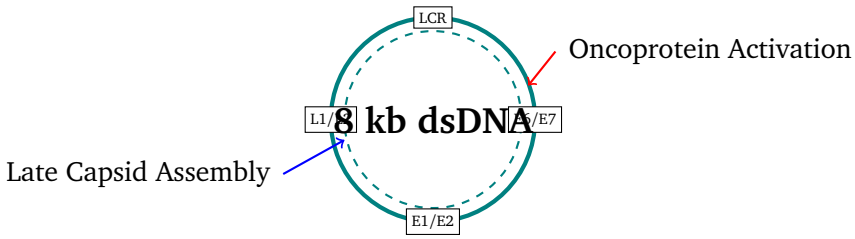
(A) Coxsackievirus A24 variant

(B) Human Adenovirus Serotype 8

(C) Enterovirus D68

(D) Echovirus 11

Q10. The structural map below outlines the replication kinetics and transcription architecture of a major human viral pathogen inside a host cell nucleus, utilizing an intermediate circularized episome structure with differential early and late promoter activation pathways. Identify the virus that corresponds to this lifecycle architecture:



(A) Human Papillomavirus (HPV)

(B) Epstein-Barr Virus (EBV)

(C) Hepatitis B Virus (HBV)

(D) Molluscum Contrigiosum Virus (MCV)

Q11. A bone marrow transplant recipient develops a severe, angioinvasive pulmonary fungal infection. A bronchoalveolar lavage demonstrates septate, acute-angle (45°) branching hyphae. Culture yields colonies that turn smoky blue-green within 48 hours. Molecular screening indicates a mutated *cyp51A* gene. What is the immediate therapeutic implication of this specific molecular finding?

(A) High-level resistance to Voriconazole and Posaconazole

(B) Complete loss of susceptibility to Amphotericin B

(C) Enhanced synergistic response to Echinocandins

(D) Resistance to Flucytosine monotherapy

Q12. A 38-year-old HIV patient from North-East India presents with low-grade fever, weight loss, generalized lymphadenopathy, and prominent umbilicated papular skin lesions across the face. A skin scrap biopsy stained with Wright-Giemsa reveals numerous intracellular oval yeast cells with a clear transverse central septum, replicating by fission rather than budding. What is the most appropriate primary therapy for this condition?

(A) Liposomal Amphotericin B followed by Itraconazole

(B) Oral Fluconazole high dose monotherapy

(C) Intravenous Caspofungin standard course

(D) Topical Ketoconazole combined with oral Griseofulvin

Q13. A 26-year-old traveler returning from an extended trekking expedition in a remote rainforest region presents with a chronic, non-healing cutaneous ulcer on his left forearm with raised indurated borders. A leishmanin skin test is positive. Amastigotes are identified inside tissue macrophages. Species identification reveals *Leishmania braziliensis*. What severe secondary clinical sequela must the physician monitor this patient for in the future?

(A) Destructive mucocutaneous relapse involving the nasopharynx

(B) Visceralization leading to severe hepatosplenomegaly

(C) Post-kala-azar dermal leishmaniasis (PKDL)

(D) Hyper-reactive malarial splenomegaly syndromic complex

Q14. An epidemiological study in a coastal region analyzes the transmission dynamics of a parasitic infection causing profound lymphatic filariasis. Microscopic investigation of nocturnal blood films confirms the presence of sheathed microfilariae with distinct, separate terminal nuclei at the tip of the tail. Which vector species is predominantly responsible for transmitting this specific parasite?

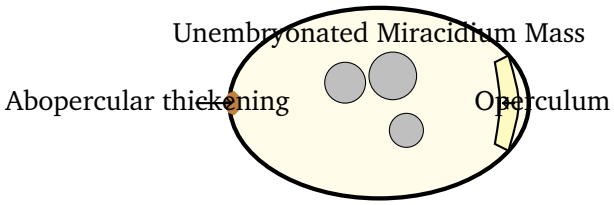
(A) Mansonia uniformis

(B) Culex quinquefasciatus

(C) Anopheles gambiae

(D) Simulium damnosum

Q15. The diagnostic schematic below illustrates the structural configuration and internal morphologic features of an operculated helminthic ovum recovered from the sputum sample of a patient presenting with hemoptysis and chronic cough mistaken for tuberculosis. Identify the parasite species characterized by this egg structure:



(A) Paragonimus westermani

(B) Clonorchis sinensis

(C) Fasciola hepatica

(D) Schistosoma haematobium

Q16. A bench research assay assesses the exact molecular interaction between T-cell receptors (TCR) and superantigens like Toxic Shock Syndrome Toxin-1 (TSST-1). In contrast to conventional peptide antigens, superantigens sidestep classical processing pathways. Which exact structural region of the immunological synapse components do these toxins bind to trigger massive cytokine release?

(A) The variable β-chain ($V\beta$) of the TCR and the outer alpha-1 domain of the MHC Class II molecule

(B) The hypervariable CDR3 region of both α and β chains of the TCR complex

(C) The β_2-microglobulin invariant domain of the MHC Class I molecule

(D) The costimulatory CD28 receptor complex present on naive cytotoxic T lymphocytes

Q17. A neonate presenting with severe recurrent pyogenic bacterial infections, delayed detachment of the umbilical cord ($>$ 6 weeks), and profound leukocytosis with marked neutrophilia is evaluated for a suspected primary immunodeficiency. Flow cytometry reveals a complete absence of CD18 surface expression. What molecular interaction is directly disrupted by this defect?

(A) Stable arrest and adhesion of neutrophils to endothelial ICAM-1

(B) Initial rolling of neutrophils mediated by E-selectin ligands

(C) Transendothelial migration driven by PECAM-1 gradients

(D) Intracellular oxidative burst cascade within phagolysosomes

Q18. A 24-year-old female with systemic lupus erythematosus (SLE) presents with acute nephritic features. Laboratory panels demonstrate a severe drop in total

serum hemolytic complement levels (CH_{50}), specifically with undetectable C4 and C2 titers, while factor B and properdin levels remain within standard physiological limits. What specific activation profile does this complement distribution signify?

(A) Exclusive activation of the Classical pathway

(B) Selective activation of the Alternative pathway amplification loop

(C) Isolated activation of the Mannose-Binding Lectin (MBL) pathway

(D) Terminal complement pathway dysregulation

Q19. An in-vitro study analyzes the differentiation kinetics of naive CD4+ T helper cells. A cell line is cultured in an environment strictly enriched with Interleukin-12 (IL-12) and Interferon-gamma (IFN-γ). Which master transcription factor is upregulated under these conditions, directing the commitment towards the Th1 lineage?

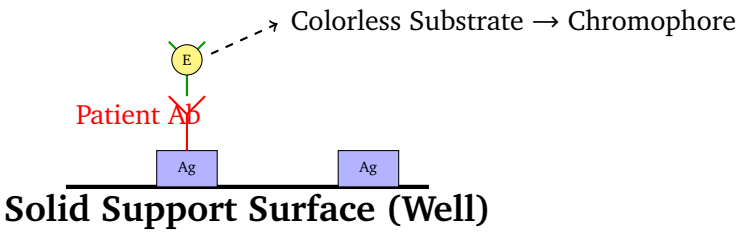
(A) T-bet

(B) GATA-3

(C) RORγt

(D) FoxP3

Q20. The schematic below maps out the core molecular mechanism of a specialized laboratory diagnostic assay used to detect specific serum autoantibodies. Based on the structural design and phase sequence shown below, identify this immunological configuration:



(A) Indirect ELISA

(B) Sandwich ELISA

(C) Direct Immunofluorescence Assay

(D) Competitive Radioimmunoassay

Detailed Solutions

Q1.

Solution

Concept: The genus *Haemophilus* contains fastidious Gram-negative bacilli whose phenotypic differentiation relies on their variable requirements for growth factors: X-factor (hemin) and V-factor (β-nicotinamide adenine dinucleotide, or NAD).

Solution:

Let's analyze the growth factor criteria for the given options:

(a) The question describes a fastidious, pleomorphic Gram-negative coccobacillus causing infective endocarditis. The pathogen requires **X-factor** but is **V-factor independent**.

(b) Let's review the requirements of the major candidate species:

- *Haemophilus influenzae* requires **both** X and V factors.
- *Haemophilus parainfluenzae* and *Haemophilus aphrophilus* (now reclassified as *Aggregatibacter aphrophilus*) require **V-factor only** or are entirely independent of X-factor.
- *Haemophilus ducreyi* (the causative agent of the sexually transmitted disease chancroid) is distinct because it requires **exogenous X-factor (hemin) only** and does not require V-factor for growth.

(c) While endocarditis is traditionally linked to HACEK organisms (like *Aggregatibacter actinomycetemcomitans*), the precise metabolic footprint specified—requiring X-factor while remaining completely independent of V-factor—uniquely characterizes *Haemophilus ducreyi*.

Final Answer: Haemophilus ducreyi

Answer: (C)

Go Back to Question 1

Q2.

Solution

Concept: Multidrug resistance in *Pseudomonas aeruginosa* is frequently driven by mutations that upregulate tripartite efflux systems belonging to the Resistance-Nodulation-Division (RND) superfamily.

Solution:

Let's analyze the primary active RND efflux systems in Gram-negative pathogens:

(a) RND pumps are tripartite systems crossing both the inner and outer membranes along with the periplasmic space. They actively extrude antibiotics using a proton-motive force gradient.

(b) In *Pseudomonas aeruginosa*, the most clinically relevant and well-characterized constitutively expressed system is the **MexAB-OprM** efflux pump.

(c) Chromosomal mutations in regulatory genes (such as *mexR*) lead to overactivation/constitutive upregulation of MexAB-OprM. This overactivation results in simultaneous hyper-resistance to a broad range of antipseudomonal agents, notably including both fourth-generation cephalosporins (**cefepime**) and carbapenems (**meropenem**).

(d) *AcrAB-TolC* is the predominant pump found in *Enterobacteriaceae* (e.g., *E. coli*), while *AdeABC* belongs to *Acinetobacter baumannii*.

Final Answer: MexAB-OprM

Answer: (A)

Go Back to Question 2

Q3.

Solution

Concept: Isoniazid (INH) and ethionamide are structural analog antimicrobials used against *Mycobacterium tuberculosis* that share a common enzymatic intracellular target, but diverge in their pathways of metabolic activation.

Solution:

Let's trace the molecular pharmacology of resistance in these overlapping systems:

(a) Both isoniazid and ethionamide are prodrugs that must be converted into their active forms to inhibit **InhA** (enoyl-acyl carrier protein reductase), an enzyme critical for mycolic acid cell wall synthesis.

(b) Isoniazid requires the catalase-peroxidase enzyme **KatG** for activation, whereas ethionamide is activated by the monooxygenase **EthA**.

(c) High-level resistance to isoniazid typically stems from *katG* deletions or mutations, preventing prodrug activation. However, because ethionamide utilizes EthA, *katG* mutants remain completely susceptible to ethionamide.

(d) Conversely, the clinical isolate in this case has a missense mutation in the **regulatory promoter region of the *inhA* gene**. This causes overproduction (upregulation) of the target enzyme InhA. Because the pool of InhA is greatly amplified, it overwhelms the therapeutic concentrations of both drugs, conferring **complete cross-resistance to ethionamide** along with low-to-moderate level resistance to isoniazid.

Final Answer: Complete cross-resistance to ethionamide

Answer: (A)

Go Back to Question 3

Q4.

Solution

Concept: Pelvic actinomycosis is an indolent, opportunistic infection associated with long-term intrauterine device (IUD) usage, caused by slow-growing, anaerobic, filamentous Gram-positive bacteria.

Solution:

Let's differentiate the metabolic profiles of the filamentous pathogens:

(a) The clinical picture describes a pelvic abscess associated with an IUD showing microscopic true branching filaments and macroscopic "sulfur granules." This strongly suggests a pathogen within the genus *Actinomyces*.

(b) While *Actinomyces israelii* is the classic and most frequent cause of human actinomycosis, it biochemically exhibits **nitrate reductase activity** (it reduces nitrate to nitrite).

(c) In contrast, ***Actinomyces gerencseriae*** (previously classified as *A. israelii* serotype II) is a morphologically identical, companion pathogen found in similar pelvic and cervicofacial lesions but is distinguished biochemically by being **catalase-negative and nitrate reduction-negative**.

(d) *Nocardia asteroides* is ruled out as it is an obligate aerobe and strongly acid-fast.

Final Answer: Actinomyces gerencseriae

Answer: (B)

Go Back to Question 4

Q5.

Solution

Concept: Members of the genus *Vibrio* are curved, Gram-negative rods that can be biochemically separated based on their dependence on sodium chloride (NaCl) concentrations for growth.

Solution:

Let's analyze the metabolic response curve shown in the diagram:

(a) The diagram depicts **Pathogen X**, which displays optimal growth velocity at very low salt parameters ($\approx$ 0.5% NaCl) and can grow in the complete absence of salt (0%). However, its growth ceases entirely as NaCl concentrations approach or exceed 3.0%.

(b) Most *Vibrio* species are obligate halophiles, meaning they require elevated salt levels (typically 1% to 6%) to stabilize their cell membranes and metabolic systems, as shown by the dashed curve (*V. parahaemolyticus* and *V. vulnificus*).

(c) ***Vibrio cholerae*** is the notable exception. It is non-halophilic and capable of robust replication in nutrient-rich fresh water or under conditions with minimal salinity (0%–0.5%). This matches the metabolic footprint of Pathogen X.

Final Answer: Vibrio cholerae

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept: Human Respiratory Syncytial Virus (RSV) utilizes specific surface envelope glycoproteins to orchestrate viral attachment and host cell membrane fusion.

Solution:

Let's evaluate the structural roles of the RSV surface proteins:

(a) RSV possesses two primary target surface projections: Glycoprotein G (mediates attachment) and **Glycoprotein F** (mediates fusion).

(b) The fusion (F) protein is synthesized as an unstable **prefusion** trimer conformation. Upon contact with the host cell membrane, it undergoes a major structural rearrangement into a highly stable **postfusion** conformation. This conformational change drives the insertion of the fusion peptide into the host membrane, enabling viral entry.

(c) Advanced monoclonal antibodies (such as nirsevimab and palivizumab) target highly conserved epitopes accessible only on the **prefusal state of Glycoprotein F**. Locking the F protein in this prefusion configuration neutralizes the virus by preventing the structural transitions required for membrane fusion.

Final Answer: Glycoprotein F

Answer: (B)

Go Back to Question 6

Q7.

Solution

Concept: The transformation of the benign, latent polyomavirus JCV into a neurotropic, highly lytic pathogen is driven by specific structural remodeling within its non-coding regulatory elements.

Solution:

Let's isolate the molecular pathophysiology of Progressive Multifocal Leukoencephalopathy (PML):

(a) In healthy individuals, the John Cunningham (JC) virus persists latently in the kidneys and lymphoid organs in its **"archetypal"** form. The archetypal form contains a highly stable, conserved transcription control region.

(b) In the setting of severe immunosuppression (e.g., advanced HIV/AIDS with a low CD4 count), the virus undergoes mutations characterized by deletion and duplication **rearrangements within the Non-Coding Control Region (NCCR)**.

(c) The altered NCCR structure acts as an enhanced promoter/enhancer element, shifting viral tropism from the kidneys to central nervous system glial cells (oligodendrocytes and astrocytes). This drives high-rate, lytic viral replication that results in asymmetric demyelination and PML.

Final Answer: Rearrangement in the Non-Coding Control Region (NCCR)

Answer: (A)

Go Back to Question 7

Q8.

Solution

Concept: Chronic Hepatitis B virus (HBV) infection transitions through distinct immunopathological phases defined by viral replication activity and the host immune response.

Solution:

Let's analyze the serological and clinical markers described:

(a) The patient has a high viral load (HBV DNA $\approx 10^8$ copies/mL), secretes active replication markers ($HBeAg^+$), but has **normal transaminase levels (ALT/AST)** and minimal liver inflammation on biopsy.

(b) This mismatch—high viral replication alongside a lack of liver inflammation—occurs because HBV is a non-cytopathic virus. Liver injury is mediated by host $CD8^+$ T-cell destruction of infected hepatocytes rather than direct viral toxicity.

(c) In the **Immune Tolerance Phase** (commonly seen in perinatally acquired infections), the host immune system does not attack infected hepatocytes. This leads to high viral replication markers without biochemical or histological signs of hepatitis.

Final Answer: Immune Tolerance Phase

Answer: (A)

Go Back to Question 8

Q9.

Solution

Concept: Acute hemorrhagic conjunctivitis (AHC) is a highly contagious ocular infection primarily caused by specific, unenveloped enterovirus serotypes.

Solution:

Let's match the taxonomy and clinical signs to find the causative serotype:

(a) The question describes an unenveloped, positive-sense single-stranded RNA virus belonging to the *Picornaviridae* family. This rules out Adenoviruses, which are double-stranded DNA viruses (ruling out Option B).

(b) The primary picornaviruses responsible for large epidemic outbreaks of acute hemorrhagic conjunctivitis are **Enterovirus 70** and the **Coxsackievirus A24 variant (CV-A24v)**.

(c) These pathogens replicate rapidly within the conjunctival epithelium, causing sudden eye pain, periorbital edema, and prominent subconjunctival hemorrhages.

Final Answer: Coxsackievirus A24 variant

Answer: (A)

Go Back to Question 9

Q10.

Solution

Concept: Human Papillomaviruses (HPV) are small, unenveloped, double-stranded DNA viruses that replicate as circular episomes within stratified squamous epithelia.

Solution:

Let's break down the genetic schematic provided:

(a) The diagram depicts a circular double-stranded DNA genome of approximately **8 kb** containing specific early (*E*) and late (*L*) genes.

(b) Let's analyze the functional regions shown:

- The **LCR** (Long Control Region) serves as the non-coding regulatory segment containing promoter regions.
- **E6 and E7** are early oncogenes. E6 targets p53 for proteasomal degradation, while E7 binds and inactivates the retinoblastoma (Rb) tumor suppressor protein, promoting cell cycle progression.
- **L1 and L2** code for the major and minor structural capsid proteins synthesized during the late phases of the viral life cycle.

(c) This genomic architecture is characteristic of **Human Papillomavirus (HPV)**.

Final Answer: Human Papillomavirus (HPV)

Answer: (A)

Go Back to Question 10

Q11.

Solution

Concept: Resistance to triazole antifungals in *Aspergillus fumigatus* is primarily driven by specific amino acid substitutions or promoter alterations in the gene encoding the target enzyme.

Solution:

Let's analyze the molecular pharmacology of azole resistance:

(a) The clinical vignette describes invasive pulmonary aspergillosis, confirmed by acute-angle (45°) branching hyphae and smoky blue-green colonies (*Aspergillus fumigatus*).

(b) Triazole antifungals (such as voriconazole, posaconazole, and isavuconazole) inhibit the enzyme **lanosterol 14α-demethylase**, which is encoded by the ***cyp51A* gene**. This enzyme is essential for synthesizing ergosterol, a key component of the fungal cell membrane.

(c) Mutations within the *cyp51A* gene (often paired with tandem repeats in its promoter region, like TR_{34}/L98H) alter the configuration of the enzyme's binding pocket. This prevents triazoles from binding effectively, conferring **high-level resistance to Voriconazole and Posaconazole**. It does not cause cross-resistance to amphotericin B (which binds ergosterol directly) or echinocandins (which inhibit cell wall β-glucan synthesis).

Final Answer: High-level resistance to Voriconazole and Posaconazole

Answer: (A)

Go Back to Question 11

Q12.

Solution

Concept: Talaromycosis (formerly penicilliosis) is an opportunistic dimorphic fungal infection endemic to Southeast and Northeast Asia, presenting with disseminated cutaneous and systemic manifestations in immunocompromised individuals.

Solution:

Let's identify the pathogen and its established treatment protocol:

(a) The clinical presentation outlines an HIV-positive patient from Northeast India with generalized lymphadenopathy and umbilicated skin lesions. Biopsy reveals intracellular oval yeast cells that divide by **fission along a distinct central transverse septum**.

(b) This specific replication pattern (fission along a transverse septum rather than budding) is pathognomonic for ***Talaromyces marneffei*** (formerly *Penicillium marneffei*). This features helps differentiate it from *Histoplasma capsulatum*, which replicates via narrow-based budding.

(c) Disseminated talaromycosis is severe and carries a high mortality rate if untreated. The standard first-line therapeutic regimen mirrors that of other severe systemic mycoses: initial induction therapy with **Liposomal Amphotericin B**, followed by long-term consolidation and maintenance therapy with oral **Itraconazole**.

Final Answer: Liposomal Amphotericin B followed by Itraconazole

Answer: (A)

Go Back to Question 12

Q13.

Solution

Concept: Infections caused by the *Leishmania Viannia* subgenus are prone to late metastatic dissemination from the primary cutaneous lesion to the mucosal surfaces of the upper respiratory tract.

Solution:

Let's evaluate the long-term clinical path of the specified pathogen:

(a) The patient has a chronic cutaneous ulcer containing intracellular amastigotes, and species identification confirms ***Leishmania braziliensis****.

(b) Unlike *Leishmania tropica* or *Leishmania major*, which cause restricted cutaneous leishmaniasis, *Leishmania braziliensis* belongs to the *Viannia* subgenus.

(c) This species can disseminate via the lymphatic system and bloodstream, persisting latently in host tissues. Years or decades after the primary skin ulcer has healed, it can cause a **destructive mucocutaneous relapse involving the nasopharynx** (espundia). This leads to severe ulceration and destruction of the nasal septum, palate, and surrounding facial structures.

Final Answer: Destructive mucocutaneous relapse involving the nasopharynx

Answer: (A)

Go Back to Question 13

Q14.

Solution

Concept: The identification of lymphatic filariasis vectors depends on matching the specific morphological traits of the microfilariae to their primary insect hosts.

Solution:

Let's analyze the structural markers of the microfilariae:

(a) Lymphatic filariasis is primarily caused by three filarial nematodes: *Wuchereria bancrofti*, *Brugia malayi*, and *Brugia timori*.

(b) The question notes that the microfilariae are **sheathed** and feature **distinct, separate terminal nuclei at the tip of the tail**. This morphological pattern is characteristic of ***Brugia malayi***. (In contrast, *Wuchereria bancrofti* microfilariae do not have nuclei extending to the tip of the tail).

(c) The primary arthropod vector responsible for transmitting *Brugia malayi* in coastal, swampy forest regions is the ***Mansonia*** mosquito genus (e.g., *Mansonia uniformis* or *Mansonia indiana*), whose larvae adapt to obtain oxygen by piercing the root systems of aquatic plants.

Final Answer: Mansonia uniformis

Answer: (A)

Go Back to Question 14

Q15.

Solution

Concept: The lung fluke *Paragonimus westermani* produces distinct operculated ova that can be recovered from sputum or stool samples, often mimicking the clinical presentation of pulmonary tuberculosis.

Solution:

Let's analyze the morphological features in the schematic:

(a) The diagnostic diagram illustrates an asymmetric, thick-shelled, oval egg.

(b) Key structural features include a distinct **operculum** cap at one pole, a **thickened abopercular end** at the opposite pole, and an internal unembryonated miracidial cell mass.

(c) This structural layout is characteristic of ***Paragonimus westermani***. The consumption of undercooked freshwater crabs or crayfish containing metacercariae leads to infection. The adult flukes live in fibrous cysts within the lung parenchyma, causing a chronic cough and hemoptysis that can be misdiagnosed as tuberculosis.

Final Answer: Paragonimus westermani

Answer: (A)

Go Back to Question 15

Q16.

Solution

Concept: Superantigens bypass conventional intracellular antigen processing and presentation by physically cross-linking specific outer domains of the immunological synapse.

Solution:

Let's analyze the binding mechanism of superantigens like TSST-1:

(a) Standard peptide antigens are processed inside APCs and presented within the central antigen-binding groove formed by the α and β chains of MHC molecules. They are then recognized by the hypervariable CDR3 loops of the T-cell receptor (TCR).

(b) Superantigens bypass this restrictive system. They bind directly to the **outer alpha-1 domain of the MHC Class II molecule** on the APC and cross-link it to the **variable β-chain ($V\beta$) of the TCR**.

(c) Because this binding occurs outside the peptide-binding groove, it is independent of peptide specificity. This structural cross-linking activates up to 20% of all naive T cells simultaneously, triggering a massive release of pro-inflammatory cytokines (IL-1, IL-2, TNF-α, IFN-γ) that leads to toxic shock syndrome.

Final Answer: The variable β-chain ($V\beta$) region of the T-cell receptor (TCR) and the outer α_1 domain of the MHC class II molecule.

Answer: (A)

Go Back to Question 16

Q17.

Solution

Concept: Leukocyte Adhesion Deficiency Type 1 (LAD-1) is an autosomal recessive immunodeficiency caused by mutations in the ITGB2 gene, which encodes the β_2-integrin component common to several adhesion molecules.

Solution:

Let's evaluate the cellular migration defect caused by a lack of CD18:

(a) Leukocyte migration into tissues involves sequential steps: rolling, activation, tight adhesion/arrest, and diapedesis.

(b) **CD18** forms the common β_2 subunit of integrins like LFA-1 (CD11a/CD18) and Mac-1 (CD11b/CD18). These integrins must interact with **ICAM-1** on endothelial cells to mediate the **stable arrest and tight adhesion** phase of leukocyte migration.

(c) Without CD18, neutrophils roll along the endothelium but cannot adhere firmly or extravasate into tissues. This leads to an absence of pus formation, delayed separation of the umbilical cord, and a marked buildup of neutrophils in the bloodstream (leukocytosis).

Final Answer: Stable arrest and adhesion of neutrophils to endothelial ICAM-1

Answer: (A)

Go Back to Question 17

Q18.

Solution

Concept: The classical, alternative, and lectin pathways of the complement system consume distinct components during activation, creating unique consumption profiles.

Solution:

Let's analyze the complement profile provided:

(a) The patient has active lupus nephritis with a drop in total hemolytic complement (CH_{50}) and **undetectable levels of C4 and C2**, while Factor B and properdin remain within normal limits.

(b) The **Classical pathway** is triggered by immune complexes (such as anti-dsDNA/IgG complexes in SLE) binding to C1. Activated C1 sequentially cleaves **C4** and **C2** to form the C3 convertase (C4b2a). This targeted activation leads to a selective drop in C4 and C2 titers.

(c) Because alternative pathway components (Factor B, properdin) are normal, this profile indicates classical pathway consumption. The MBL pathway also utilizes C4 and C2, but it is triggered by microbial surface carbohydrates rather than autoimmune immune complexes.

Final Answer: Exclusive activation of the Classical pathway

Answer: (A)

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Q19.

Solution

Concept: The lineage commitment of naive $CD4^+$ T helper cells into specialized effector subsets is driven by specific extracellular cytokine environments that upregulate master transcription factors.

Solution:

Let's review the master transcription factors for T-helper cell lineages:

(a) Culturing naive $CD4^+$ T cells in an environment enriched with **IL-12** and **IFN-γ** promotes differentiation along the **Th1 lineage**.

(b) The master transcription factor that drives the Th1 differentiation program is **T-bet**. T-bet upregulates the expression of IFN-γ and IL-12 receptors, forming a positive feedback loop that solidifies the cell's commitment to cell-mediated immunity.

(c) For comparison: *GATA-3* directs Th2 differentiation; *RORγt* directs Th17 differentiation; and *FoxP3* regulates Treg development.

Final Answer: T-bet

Answer: (A)

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Q20.

Solution

Concept: Enzyme-Linked Immunosorbent Assays (ELISA) utilize specialized solid-phase antibody or antigen configurations to detect target analytes in clinical samples.

Solution:

Let's analyze the structural layers shown in the diagram:

(a) The solid support surface (well) is pre-coated with a known **Target Antigen (Ag)**.

(b) The next layer consists of the **Patient's Primary Antibody (Ab)**, which binds specifically to the immobilized antigen.

(c) This is followed by an **Enzyme-Linked Secondary Antibody** (labeled with enzyme 'E') that recognizes the Fc portion of the human antibody. Adding a colorless substrate triggers an enzymatic reaction that produces a measurable color change.

(d) This specific configuration—where an immobilized antigen binds a primary antibody, which is then detected by an enzyme-linked secondary antibody—defines an **Indirect ELISA**. In a Sandwich ELISA, the well is instead pre-coated with a capture antibody rather than an antigen.

Final Answer: Indirect ELISA

Answer: (A)

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Answer Key

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