FMGE Microbiology Sample Paper – 1

Duration: 60 Minutes **Maximum Marks: 40**

Instructions

- This paper contains **40 Multiple Choice Questions** modelled on the **Microbiology** section of FMGE (Bacteriology, Immunology, Virology, Parasitology, and Mycology).
- Each correct answer carries **+1 mark**. There is **no negative marking** – attempt all questions.
- Questions follow the clinically integrated pattern of the FMGE examination.
- Click a question number in the answer key to navigate to its solution.

FMGE Microbiology – Q1 to Q40

Q1. A 25-year-old male presents to the outpatient department with a painful, erythematous swelling on the back of his neck for 3 days. On examination, a fluctuant furuncle is noted. Gram stain of the aspirated pus reveals gram-positive cocci arranged in clusters. The laboratory performs a tube coagulase test at 37°C for 4 hours. Which of the following organisms consistently yields a positive tube coagulase test?

(A) Staphylococcus aureus

(B) Staphylococcus epidermidis

(C) Staphylococcus saprophyticus

(D) Streptococcus pyogenes

Q2. A 10-year-old boy is brought with fever, migratory arthritis, and a new cardiac murmur 3 weeks after an episode of sore throat that was treated inadequately. His antistreptolysin O (ASO) titre is markedly elevated. Which organism is responsible for the preceding pharyngitis that led to this complication?

(A) Streptococcus pneumoniae

(B) Streptococcus pyogenes

(C) Staphylococcus aureus

(D) Viridans streptococci

Q3. A 5-year-old girl is admitted with a 4-day history of bloody diarrhoea after eating undercooked beef at a barbecue. On day 5, she develops oliguria, pallor, and her blood film shows schistocytes. Renal function tests reveal blood urea nitrogen of 68 mg/dL and creatinine 3.2 mg/dL. Which organism is the most likely cause of her current condition?

(A) Salmonella enteritidis

(B) Shigella dysenteriae type 1

(C) Escherichia coli O157:H7

(D) Campylobacter jejuni

Q4. A 22-year-old college student from a hostel returns from a rural area with a 2-week history of stepladder-pattern fever, headache, relative bradycardia, and faint rose-coloured spots on his abdomen. Stool culture is ordered, and the Widal test shows elevated O and H agglutinin titres. The organism responsible possesses a surface polysaccharide antigen that inhibits phagocytosis and complement activation. Which antigen is this?

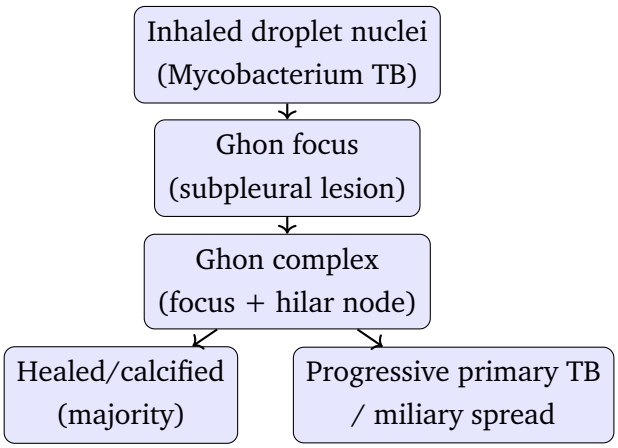
(A) H antigen

(B) O antigen

(C) M protein

(D) Vi antigen

Q5. An 8-year-old child presents with low-grade fever, night sweats, and failure to thrive for 6 weeks. The Mantoux test shows 18 mm induration. Chest X-ray reveals a peripheral mid-zone opacity with ipsilateral hilar lymphadenopathy. The physician explains the progression of primary tuberculosis to the parents. Which of the following correctly represents the sequence of events in primary pulmonary tuberculosis?



Which of the following is the hallmark lesion of primary TB that forms at the site of initial bacterial implantation in the lung?

(A) Ghon focus

(B) Simon focus

(C) Puhl's lesion

(D) Assmann focus

Q6. A 35-year-old farmer punctures his foot on a rusty nail while working in the field. He had not received any immunisation in adulthood. Seven days later, he presents with trismus (lockjaw), risus sardonicus, and opisthotonos. The toxin produced by the causative organism acts by blocking which inhibitory neurotransmitter pathway?

(A) Acetylcholine release at the neuromuscular junction

(B) Glycine and GABA release from Renshaw cells and interneurones

(C) Acetylcholinesterase degradation of acetylcholine

(D) NMDA receptor activation in the anterior horn cells

Q7. During a cholera outbreak in a flood-affected district, a 30-year-old man presents with sudden onset profuse watery diarrhoea described as rice-water stools, painless abdominal cramps, and severe dehydration with a blood pressure of 80/50 mmHg. Stool dark-field microscopy shows organisms with rapid darting motility. The El Tor biotype responsible for the current pandemic produces which toxin subunit responsible for adenylate cyclase activation?

(A) B subunit

(B) A2 subunit

(C) A1 subunit

(D) Both A and B subunits together

Q8. A 24-year-old sexually active male presents with a 3-day history of dysuria and a profuse purulent yellowish-green urethral discharge. Gram stain of the discharge shows gram-negative diplococci within polymorphonuclear neutrophils. The organism responsible for this condition primarily adheres to the urethral columnar epithelium using which surface structure?

(A) Lipopolysaccharide (LPS)

(B) Outer membrane proteins (Opa proteins)

(C) IgA protease

(D) Pili (fimbriae)

Q9. A 14-month-old unvaccinated child is admitted with a 2-day history of high fever, irritability, neck stiffness, and bulging fontanelle. CSF analysis shows turbid fluid, polymorphonuclear pleocytosis, elevated protein, and decreased glucose. Gram stain of CSF reveals small gram-negative coccobacilli. The capsular polysaccharide of the causative organism, used in conjugate vaccine formulation, is composed of which carbohydrate?

(A) Polyribosylribitol phosphate (PRP)

(B) N-acetylneuraminic acid

(C) Polyglutamic acid

(D) Capsular polysaccharide type 4

Q10. A 40-year-old wool-sorter presents with a painless, pruritic papule on his right forearm that evolved into a vesicle and then a black eschar surrounded by non-pitting oedema over 5 days. Blood culture grows gram-

positive, spore-forming bacilli arranged in long chains with a bamboo-stick appearance. This organism's tripartite exotoxin comprises protective antigen (PA) plus which two additional components?

(A) Haemolysin and lecithinase

(B) Lethal factor (LF) and oedema factor (EF)

(C) Alpha toxin and beta toxin

(D) Exotoxin A and exotoxin S

Q11. A 22-year-old patient with cystic fibrosis is admitted with worsening dyspnoea, productive cough with greenish sputum, and fever. Sputum culture on blood agar produces a characteristic blue-green pigment and a fruity grape-like odour. The organism forms a mucoid biofilm within the airways, making eradication difficult. Which of the following pigments is responsible for the characteristic blue-green colour of this organism's colonies?

(A) Pyoverdin

(B) Pyorubin

(C) Pyocyanin

(D) Melanin

Q12. A 28-year-old pregnant woman at 36 weeks gestation who is on immunosuppressant therapy for a renal transplant develops fever and flu-like illness after consuming soft cheese and deli meat. She delivers prematurely; the neonate develops meningitis on day 2 of life. CSF Gram stain shows gram-positive, tumbling motility rods. This organism is best isolated from food samples by which method?

(A) Alkaline peptone water enrichment

(B) Selenite F broth enrichment

(C) Mannitol salt agar selective culture

(D) Cold enrichment at 4°C in tryptose broth

Q13. A 7-year-old unvaccinated child presents with low-grade fever, sore throat, and a greyish-white membrane on the tonsils and soft palate that bleeds on attempted removal. Neck examination reveals cervical lymphadenopathy producing a "bull neck" appearance. To confirm toxin production by the isolated organism, which laboratory test is the gold standard?

(A) Elek's gel immunodiffusion test

(B) Nagler's reaction

(C) CAMP test

(D) Optochin sensitivity test

Q14. A 38-year-old veterinarian presents with a 6-week history of recurrent fever that waxes and wanes (undulant pattern), drenching night sweats, arthralgia, and malaise. He regularly handles cattle and assists in calving. Blood culture on Castaneda medium at 37°C in 5–10% CO_2 is positive. The causative organism is a small, gram-negative coccobacillus. Which species of Brucella primarily infects cattle?

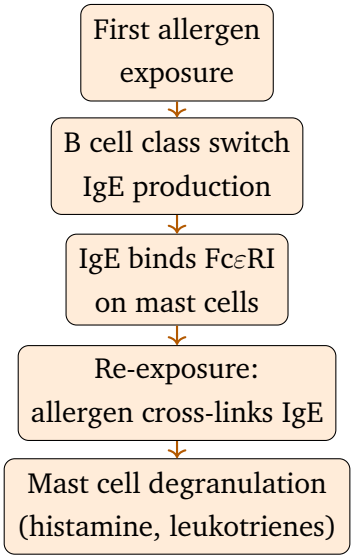
(A) Brucella melitensis

(B) Brucella abortus

(C) Brucella suis

(D) Brucella canis

Q15. A 19-year-old female with a history of atopy develops urticaria, bronchospasm, and hypotension within minutes of receiving a penicillin injection. Her serum IgE is significantly elevated. The sequence of events in this reaction is depicted below. Which antibody class is responsible for mediating this type of hypersensitivity reaction?



(A) IgG

(B) IgM

(C) IgE

(D) IgA

Q16. A 34-year-old male who is HIV positive presents with recurrent oral candidiasis, weight loss, and night sweats. His CD4+ T-cell count is 160 cells/μL. The HIV virus primarily infects which cell type because gp120 has high affinity for the CD4 molecule expressed on its surface?

(A) CD8+ cytotoxic T lymphocytes

(B) B lymphocytes

(C) Natural killer cells

(D) CD4+ T helper lymphocytes

Q17. During an immunology practical, a student is studying antigen presentation. An antigen-presenting cell (APC) internalises a bacterial protein by endocytosis, processes it in endosomes, and presents the resulting peptide fragments to T lymphocytes. The peptide-MHC complex formed activates CD4+ T helper cells. Which MHC molecule is responsible for presenting exogenous (extracellular) antigens to CD4+ T cells?

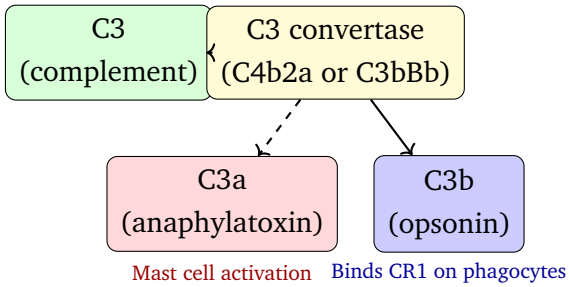
(A) MHC class II

(B) MHC class I

(C) MHC class III

(D) Beta-2 microglobulin

Q18. A patient with a recurrent pyogenic bacterial infection is found to have a deficiency in a complement component. Investigations reveal that phagocytes fail to recognise and engulf opsonised bacteria efficiently. The diagram below depicts the cleavage of complement component C3. Which cleavage product acts as the principal opsonin, binding to CR1 receptors on phagocytes?



(A) C3a

(B) C3b

(C) C5a

(D) C1q

Q19. A researcher is studying a patient with recurrent viral infections and a history of one episode of lymphoma. Flow cytometry shows a profound deficiency of $CD3^{-}$, $CD16^{+}$, $CD56^{+}$ lymphocytes. These cells normally kill tumour cells and virally infected cells without prior sensitisation by recognising the absence of which surface molecule?

(A) B7 (CD80/CD86) co-stimulatory molecule

(B) CD4 receptor

(C) MHC class I molecule

(D) T cell receptor (TCR)

Q20. A microbiologist is explaining affinity maturation of antibodies following repeated antigen exposure. During a germinal centre reaction within secondary lymphoid follicles, B cells undergo a process that introduces point mutations in the variable region genes of immunoglobulin heavy and light chains at a rate approximately one million times higher than the background somatic mutation rate. This process is called:

(A) V(D)J recombination

(B) Class switch recombination

(C) Allelic exclusion

(D) Somatic hypermutation

Q21. A 50-year-old male develops high fever, rigors, and hypotension after a urinary catheter procedure. Blood cultures grow gram-negative bacilli. The initial innate immune response is triggered when macrophages detect the lipopolysaccharide (LPS) component of the gram-negative bacterial cell wall through pattern recognition receptors. Which receptor specifically recognises LPS and initiates the innate immune response via the MyD88-NF-κB signalling pathway?

(A) Toll-like receptor 4 (TLR4)

(B) NOD1 receptor

(C) RIG-I receptor

(D) NLRP3 inflammasome

Q22. A 5-year-old boy is evaluated for a syndrome characterised by immune dysregulation, polyendocrinopathy (insulin-dependent diabetes mellitus, thyroiditis), enteropathy, and eczema (IPEX syndrome). Genetic analysis reveals a mutation in the FOXP3 gene. Loss of FOXP3 expression impairs the development and function of which lymphocyte subset that is critical for maintaining peripheral self-tolerance?

(A) CD8+ cytotoxic T cells

(B) Regulatory T cells (CD4+CD25+FoxP3+)

(C) Th17 cells

(D) Natural killer T (NKT) cells

Q23. During an immune response to a viral infection, virus-infected cells are coated with IgG antibodies directed against viral surface proteins. $CD3^{-}$, $CD16^{+}$, $CD56^{+}$ lymphocytes are then recruited and kill these antibody-coated target cells by recognising the Fc region of the bound IgG through an Fc receptor. Which Fc receptor expressed on these lymphocytes mediates this antibody-dependent cellular cytotoxicity (ADCC)?

(A) FcαRI (CD89)

(B) FcεRI

(C) FcγRIII (CD16)

(D) FcγRI (CD64)

Q24. A medical student is asked to compare the primary and secondary immune responses during a pharmacology viva. She correctly identifies key differences: the secondary response occurs after re-exposure to the same antigen, has a shorter lag phase, produces a higher antibody titre, and is predominantly of which immunoglobulin class compared to the IgM-dominant primary response?

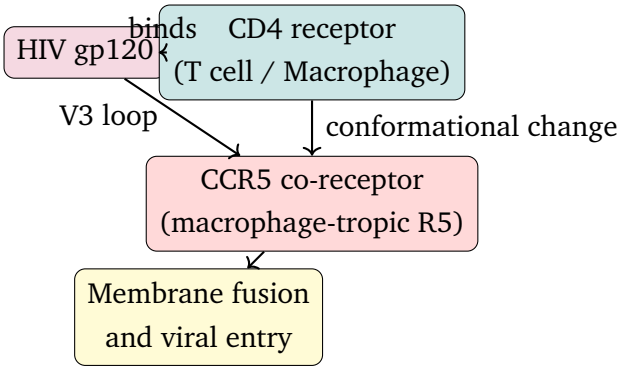
(A) IgA

(B) IgE

(C) IgM

(D) IgG

Q25. A virologist is studying the early stages of HIV infection. HIV-1 gp120 first binds CD4 on the host cell, which induces a conformational change exposing the V3 loop. For macrophage-tropic (R5) strains of HIV that predominate during early infection, the V3 loop then binds a second co-receptor on the cell surface before fusion occurs. This interaction is illustrated below. Which co-receptor do R5 strains of HIV preferentially use?



(A) CCR5

(B) CXCR4

(C) CCR3

(D) CXCR1

Q26. A 32-year-old healthcare worker sustains a needlestick injury from a known hepatitis B patient. At 4 weeks post-exposure, his serology is checked. He is in the "window period" of acute hepatitis B infection where both HBsAg and anti-HBs are undetectable. Which serological marker is the MOST USEFUL to diagnose acute hepatitis B infection during this window period?

(A) HBeAg

(B) Anti-HBc IgM

(C) Anti-HBs

(D) HBsAg

Q27. A 28-year-old woman presents with recurrent painful vesicular lesions on her lips that reactivate during periods of stress or fever. She was first infected as a child. After primary infection, the causative virus travels retrogradely along sensory axons and establishes latency in which anatomical location, from which it reactivates to cause recurrent disease?

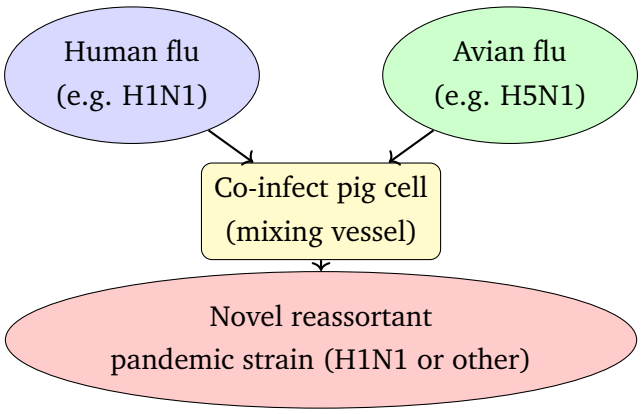
(A) Parotid gland acinar cells

(B) Liver hepatocytes

(C) Trigeminal ganglion (dorsal root ganglia)

(D) Regional lymph nodes

Q28. An epidemiologist is investigating a novel influenza A pandemic strain. She explains that the segmented, negative-sense RNA genome of influenza A allows for the simultaneous infection of a single host cell (often a pig, which serves as a "mixing vessel") by two different influenza A strains. This results in the exchange of entire genome segments, producing a novel strain with a new haemagglutinin or neuraminidase subtype. The diagram below illustrates this process. This mechanism is called:



(A) Antigenic drift

(B) Genetic recombination

(C) Phenotypic mixing

(D) Antigenic shift

Q29. A 14-year-old boy from a rural district is brought to casualty with a 3-day history of hydrophobia, aerophobia, excessive salivation, and agitation following a bat bite 45 days earlier for which he did not seek post-exposure prophylaxis. The patient dies despite intensive care. Post-mortem examination of the brain reveals eosinophilic intracytoplasmic inclusion bodies in the hippocampal pyramidal neurons and cerebellar Purkinje cells. What are these inclusion bodies called?

(A) Negri bodies

(B) Guarnieri bodies

(C) Cowdry type A inclusions

(D) Aschoff bodies

Q30. A 20-year-old male resident of a tropical city presents on day 3 of fever with severe myalgia, retro-orbital pain, and a maculopapular rash. His platelet count is 78,000/μL and haematocrit has risen by 22%. IgM and IgG dengue serology is sent. Investigations reveal high IgG titre, suggesting this is a secondary dengue infection with a different serotype, increasing risk of dengue haemorrhagic fever (DHF). Which antigen test, positive in the first 5 days of illness, helps in early virological diagnosis of dengue?

(A) Dengue IgM antibody

(B) Dengue NS1 antigen

(C) Dengue E protein ELISA

(D) Dengue IgG antibody

Q31. A 42-year-old woman undergoes cervical Pap smear screening that reveals high-grade squamous intraepithelial lesion (HSIL). HPV DNA testing is positive for HPV type 16. The oncogenic potential of high-risk HPV types is primarily mediated by two early proteins that inactivate key tumour suppressor pathways. Which pair of viral proteins is responsible for inactivating p53 and Rb tumour suppressors respectively?

(A) E5 (p53) and E4 (Rb)

(B) E7 (p53) and E6 (Rb)

(C) E6 (p53) and E7 (Rb)

(D) E2 (p53) and E1 (Rb)

Q32. A 4-year-old unvaccinated child presents with a 3-day prodrome of high fever, coryza, conjunctivitis, and cough. On examination, the physician notices tiny white spots resembling grains of sand on a red base on the buccal mucosa opposite the lower molars, before the appearance of the

characteristic morbilliform rash. These pathognomonic mucosal lesions of measles are called:

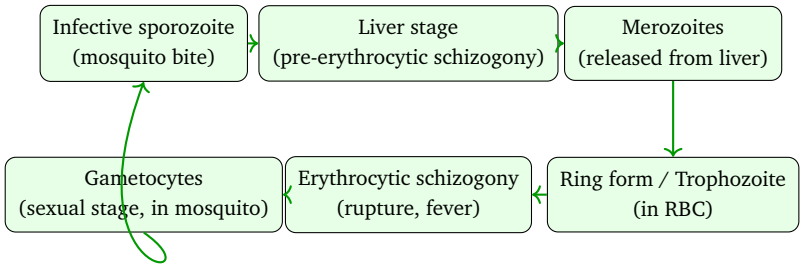
(A) Fordyce spots

(B) Wickham's striae

(C) Rose spots

(D) Koplik spots

Q33. A 5-year-old child from a malaria-endemic region of Africa is brought in with high fever, seizures, and impaired consciousness (Glasgow Coma Scale 8). A thick blood film stained with Giemsa shows ring-form trophozoites and banana-shaped gametocytes. The life cycle of the causative parasite is illustrated below. Which unique pathological mechanism of this Plasmodium species causes obstruction of cerebral microvasculature, leading to cerebral malaria?



(A) Knob formation on infected RBCs causing cytoadherence to endothelium

(B) Double infection of a single RBC

(C) Formation of Schüffner's dots on RBC surface

(D) Preferential invasion of reticulocytes only

Q34. A 32-year-old male returning from a trip to India presents with a 10-day history of bloody mucoid diarrhoea (8–10 stools per day), lower abdominal cramps, and tenesmus. Stool microscopy reveals large trophozoites with a single nucleus containing a centrally placed karyosome. The cytoplasm of these trophozoites contains engulfed erythrocytes. Colonoscopy shows flask-shaped ulcers with undermined edges. Which feature of Entamoeba histolytica trophozoites is most diagnostic on stool microscopy?

(A) Trophozoites with multiple nuclei

(B) Ingested red blood cells (erythrophagocytosis) in trophozoite cytoplasm

(C) Excystation of quadrinucleate cysts in the small intestine

(D) Trophozoites with two nuclei and axostyle

Q35. A 28-year-old backpacker who drank unfiltered stream water presents with 3 weeks of foul-smelling, greasy (steatorrhoeic) diarrhoea, bloating, and significant weight loss. There is no blood in the stool. Duodenal aspirate on microscopy shows pear-shaped, binucleate trophozoites with four pairs of flagella and a characteristic sucking disc. Fresh stool microscopy shows oval cysts with 4 nuclei. Which of the following clinical features best differentiates Giardia infection from amoebic dysentery?

(A) Presence of blood in stool

(B) Perianal pruritus

(C) Steatorrhoea (fatty, foul-smelling stools without blood)

(D) Liver abscess

Q36. A newborn is evaluated at birth for a triad of abnormalities found on antenatal ultrasound and confirmed postnatally: hydrocephalus, bilateral chorioretinitis, and intracranial periventricular calcifications. The mother had been a cat owner and reported a flu-like illness in the first trimester for which she sought no medical attention. Serological testing of the mother reveals high IgM anti-Toxoplasma antibody titre. The congenital infection is caused by transplacental transmission of which stage of the parasite?

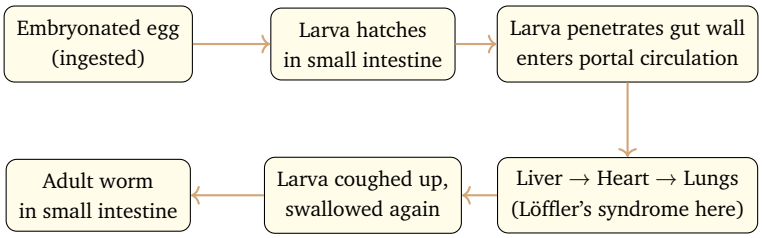
(A) Oocysts from contaminated soil

(B) Bradyzoites in tissue cysts

(C) Sporozoites from cat faeces

(D) Tachyzoites (actively dividing form during acute infection)

Q37. A 9-year-old child from a rural area presents with dry cough, fever, and mild breathlessness of 5 days duration. Chest X-ray shows transient bilateral pulmonary infiltrates. Blood count reveals eosinophil count of 2,800 cells/μL. Three weeks later, the child passes a large, pinkish-white worm in stool. The life cycle of the responsible parasite is shown below. The pulmonary symptoms occurred during which phase of the parasite's life cycle?



(A) Pulmonary larval migration phase (Löffler's syndrome)

(B) Adult worm intestinal phase

(C) Liver schizogony phase

(D) Encysted larval phase in muscle

Q38. A 45-year-old farmer from a coastal district of India presents with massive non-pitting oedema of both lower limbs (elephantiasis) and scrotal lymphoedema. A peripheral blood smear taken at midnight reveals sheathed microfilariae. Lymphatic obstruction by adult worms causes this chronic presentation. Which organism is responsible, and what is the characteristic feature of its microfilariae in blood films?

(A) Brugia timori — diurnal periodicity

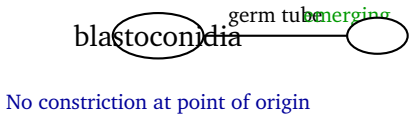
(B) Wuchereria bancrofti — nocturnal periodicity (peak in blood at midnight)

(C) Loa loa — nocturnal periodicity

(D) Mansonella perstans — non-periodic

Q39. A 38-year-old female on prolonged broad-spectrum antibiotics and corticosteroids develops thick, white, curd-like plaques on the buccal mucosa and oropharynx that bleed on scraping. KOH mount shows budding

yeast cells with pseudohyphae. A germ tube test is performed by incubating the yeast in human serum at 37°C for 2.5–3 hours. The appearance after incubation is illustrated below. A positive germ tube test rapidly differentiates the likely pathogen from other Candida species. Which organism is identified?



(A) Candida tropicalis

(B) Candida glabrata

(C) Candida albicans

(D) Candida krusei

Q40. A 55-year-old male who received an allogeneic bone marrow transplant 6 weeks ago presents with persistent fever unresponsive to antibacterial antibiotics, dry cough, and pleuritic chest pain. HRCT chest reveals a nodular opacity with a surrounding halo sign and an air-crescent sign. Bronchoalveolar lavage is performed and a serum galactomannan assay is sent. Which organism is the most likely cause of this invasive pulmonary fungal infection in the immunocompromised host, and which test is used for early non-invasive diagnosis?

(A) Candida albicans — (1,3)-β-D-glucan assay

(B) Cryptococcus neoformans — India ink preparation

(C) Mucor species — KOH mount of BAL

(D) Aspergillus fumigatus — serum galactomannan assay

Detailed Solutions

Q1.

Solution

Concept – Coagulase Test: The tube coagulase test detects free coagulase (staphylocoagulase), an enzyme that converts fibrinogen to fibrin, clotting plasma in a test tube. **Staphylococcus aureus** is the only clinically important coagulase-positive staphylococcus; it remains positive after 4 hours of incubation at 37°C.

Key Point: S. aureus causes a wide spectrum of infections including furuncles, carbuncles, cellulitis, osteomyelitis, bacteraemia, infective endocarditis, toxic shock syndrome, and food poisoning (via preformed enterotoxin). Free coagulase production is its primary virulence-distinguishing feature from other staphylococci.

Why other options are wrong:

- Option B: S. epidermidis is coagulase-negative; causes biofilm-related prosthetic device and catheter infections; novobiocin sensitive.
- Option C: S. saprophyticus is coagulase-negative; causes community-acquired UTIs in sexually active young women; novobiocin resistant.
- Option D: S. pyogenes is a streptococcus (not a staphylococcus); identified by beta-haemolysis, Lancefield group A, and bacitracin sensitivity.

Final Answer: Gram-positive cocci in clusters + positive tube coagulase $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – ASO Titre and Rheumatic Fever: Antistreptolysin O (ASO) titre measures antibodies produced against streptolysin O, a haemolysin produced by **Streptococcus pyogenes** (Group A beta-haemolytic streptococcus). A rising or elevated ASO titre confirms a preceding streptococcal pharyngeal infection. Rheumatic fever is an autoimmune sequela occurring 2–4 weeks after untreated pharyngitis with S. pyogenes.

Key Point: S. pyogenes causes pharyngitis, scarlet fever, impetigo, erysipelas, necrotising fasciitis, and toxic shock syndrome. Non-suppurative sequelae include acute rheumatic fever (ARF) and post-streptococcal glomerulonephritis (PSGN). M protein is the major virulence factor and target of molecular mimicry in ARF.

Why other options are wrong:

- Option A: S. pneumoniae causes pneumonia, meningitis, and otitis media; does not cause rheumatic fever; diagnosed by optochin sensitivity.
- Option C: S. aureus causes skin and soft tissue infections, endocarditis; does not cause rheumatic fever; ASO negative.
- Option D: Viridans streptococci cause infective endocarditis and dental caries; do not cause ARF.

Final Answer: Rheumatic fever after pharyngitis + elevated ASO titre $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q2

Q3.

Solution

Concept – Haemolytic Uraemic Syndrome (HUS): E. coli O157:H7 (STEC, Shiga toxin-producing E. coli) produces Shiga toxin (verotoxin) types 1 and 2. Shiga toxin is absorbed from the colon, enters the bloodstream, and damages glomerular endothelial cells, causing the triad of HUS: microangiopathic haemolytic anaemia (MAHA), thrombocytopaenia, and acute renal failure. MAHA is evidenced by schistocytes on blood film.

Key Point: E. coli O157:H7 is transmitted via undercooked ground beef, unpasteurised dairy, and contaminated produce. It does NOT ferment sorbitol (unlike most E. coli), which is used on sorbitol-MacConkey agar. Antibiotics are contraindicated as they increase Shiga toxin release and risk of HUS.

Why other options are wrong:

- Option A: S. enteritidis causes non-bloody diarrhoea and septicaemia; rarely causes HUS.
- Option B: Shigella dysenteriae type 1 also produces Shiga toxin and can cause HUS; however, in this clinical context (undercooked beef, not bloody dysentery with tenesmus), E. coli O157:H7 is the primary choice.
- Option D: C. jejuni causes bloody diarrhoea but is more commonly associated with Guillain-Barré syndrome, not HUS.

Final Answer: Bloody diarrhoea after undercooked beef + HUS triad + schistocytes $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q3

Q4.

Solution

Concept – Vi Antigen of Salmonella typhi: The Vi (virulence) antigen is a capsular polysaccharide (poly-N-acetyl galactosaminuronic acid) found on **Salmonella typhi**. It inhibits phagocytosis and blocks complement activation (by preventing C3b deposition), allowing the organism to survive intracellularly. The Widal test detects antibodies against O (somatic) and H (flagellar) antigens of S. typhi.

Key Point: Typhoid fever presents with stepladder-pattern fever, relative bradycardia, rose spots (salmon-coloured blanching macules on abdomen), hepatosplenomegaly, and "pea-soup" diarrhoea. Blood culture is the gold standard in week 1; Widal test is used from week 2. The Vi antigen is used in the Vi polysaccharide typhoid vaccine.

Why other options are wrong:

- Option A: H antigen is the flagellar antigen of Salmonella; it is not a capsular antigen and does not inhibit phagocytosis.
- Option B: O antigen is the lipopolysaccharide (LPS) somatic antigen; it contributes to endotoxin activity but is not the phagocytosis-inhibiting capsule.
- Option C: M protein is a virulence factor of Streptococcus pyogenes (not Salmonella).

Final Answer: Typhoid fever + capsular antigen inhibiting phagocytosis $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q4

Q5.

Solution

Concept – Primary Tuberculosis and Ghon Focus: In primary tuberculosis, inhaled droplet nuclei containing **Mycobacterium tuberculosis** implant in the subpleural region of the lower part of the upper lobe or upper part of the lower lobe. Here, macrophages engulf the bacteria but fail to kill them, forming an initial parenchymal granuloma called the **Ghon focus**. The Ghon focus, together with the draining ipsilateral hilar lymph node enlargement, constitutes the **Ghon complex** (primary complex or Ranke complex).

Key Point: In the majority of immunocompetent individuals, the Ghon complex heals with fibrosis and calcification (visible on X-ray). In immunocompromised individuals (malnutrition, HIV), primary progressive TB or miliary TB may ensue. Tuberculin sensitivity (Mantoux test) develops 4–8 weeks after primary infection.

Why other options are wrong:

- Option B: Simon focus refers to apical lung foci of haematogenous seeding during primary TB, not the initial lesion.
- Option C: Puhl's lesion refers to subapical reactivation TB lesions; this is a post-primary (secondary TB) concept.
- Option D: Assmann focus (infraclavicular focus) is a radiological description of early post-primary TB.

Final Answer: Site of initial bacterial implantation in primary TB lung $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – Tetanospasmin Mechanism: Clostridium tetani produces tetanospasmin (a zinc metalloprotease), which travels retrogradely along motor neurons to the spinal cord and brainstem. There, it cleaves synaptobrevin (VAMP), blocking exocytosis of the inhibitory neurotransmitters **glycine** (from Renshaw cells) and **GABA** (from interneurones). Loss of inhibitory control over alpha motor neurons causes sustained muscle spasms (spastic paralysis).

Key Point: Clinical features: trismus (masseter spasm), risus sardonicus (facial muscle spasm), opisthotonos (back arching), dysphagia, and autonomic instability. Tetanospasmin is an A-B toxin: B fragment mediates neurospecific binding, A fragment (light chain) is the zinc endopeptidase. Treatment includes wound debridement, human tetanus immunoglobulin (HTIG), metronidazole, and sedation.

Why other options are wrong:

- Option A: Blocking ACh at the NMJ causes flaccid paralysis (as in botulinum toxin) — the opposite of tetanus.
- Option C: Acetylcholinesterase inhibition by organophosphates causes cholinergic syndrome; not tetanospasmin's mechanism.
- Option D: NMDA receptor activation is irrelevant to tetanospasmin's mechanism of action.

Final Answer: Trismus + opisthotonos + rusty nail + blocks inhibitory neurotransmitters $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q6

Q7.

Solution

Concept – Cholera Toxin Mechanism: Vibrio cholerae cholera toxin (CT) is an A-B toxin. The B pentamer binds GM1 ganglioside on intestinal epithelial cells. The A subunit consists of A1 (enzymatically active) and A2 (linker) fragments. **The A1 subunit** is the catalytic domain that ADP-ribosylates the αs subunit of Gs protein, permanently activating adenylate cyclase. This raises intracellular cAMP, activating protein kinase A, which phosphorylates CFTR causing massive Cl^- secretion and water loss — producing rice-water stools.

Key Point: V. cholerae is a gram-negative comma-shaped bacillus with darting motility (like shooting star). It grows on TCBS agar (yellow colonies). El Tor biotype is responsible for the 7th (current) pandemic and produces haemolysin. Treatment is rehydration (ORS/IV fluids) and doxycycline.

Why other options are wrong:

- Option A: B subunit mediates binding to GM1 ganglioside; it has no enzymatic activity.
- Option B: A2 subunit is merely the linker peptide connecting A1 to the B pentamer; it has no adenylate cyclase activity.
- Option D: Both subunits together are not the single answer; specifically A1 subunit is the enzymatic active portion.

Final Answer: Cholera toxin adenylate cyclase activation $\Rightarrow$ A1 subunit $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q7

Q8.

Solution

Concept – Gonococcal Adherence via Pili: Neisseria gonorrhoeae possesses type IV pili (fimbriae) as its primary adhesin for attachment to non-ciliated columnar and transitional epithelial cells of the urethra, cervix, rectum, and conjunctiva. Pili mediate the initial reversible attachment; Opa (opacity) proteins then mediate tight, irreversible adhesion and invasion.

Key Point: N. gonorrhoeae is a gram-negative diplococcus (coffee-bean shaped), oxidase positive, ferments only glucose (not maltose — distinguishes from N. meningitidis). Key virulence factors: pili (adhesion), Opa proteins (invasion), Por proteins (survival), and IgA protease (cleaves secretory IgA). Complications: PID, Fitz-Hugh-Curtis syndrome, disseminated gonococcal infection (DGI).

Why other options are wrong:

- Option A: LPS (endotoxin) of gonococci induces local inflammation but is not the primary adhesin.
- Option B: Opa proteins mediate tight adhesion and invasion (secondary step) after pili-mediated initial attachment.
- Option C: IgA protease neutralises mucosal IgA immunity but is not an adhesin for epithelial attachment.

Final Answer: Primary adherence to urethral epithelium $\Rightarrow \boxed{D}$ Pili (fimbriae)

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – Haemophilus influenzae type b PRP Capsule: Haemophilus influenzae type b (Hib) possesses a polysaccharide capsule composed of **polyribosylribitol phosphate (PRP)**. This capsule is the basis of the Hib conjugate vaccine (PRP conjugated to a protein carrier like tetanus toxoid — PRP-T, or meningococcal outer membrane protein — PRP-OMP). The PRP capsule inhibits phagocytosis and complement-mediated killing, making Hib a major cause of meningitis in unvaccinated children under 5 years.

Key Point: H. influenzae requires two growth factors: factor X (haematin) and factor V (NAD+) on blood agar, growing best around S. aureus (satellitism phenomenon). It forms a chocolate agar satellite around S. aureus colonies. Hib causes meningitis (most common in < 5 years), epiglottitis (thumb sign on lateral neck X-ray), pneumonia, and septic arthritis.

Why other options are wrong:

- Option B: N-acetylneuraminic acid (sialic acid) is the capsular component of N. meningitidis group B and some E. coli strains.
- Option C: Polyglutamic acid is the capsule of Bacillus anthracis (the only protein capsule known in bacteria).
- Option D: Capsular polysaccharide type 4 is a description associated with S. pneumoniae capsular types, not H. influenzae.

Final Answer: Hib capsular polysaccharide in conjugate vaccine $\Rightarrow \boxed{A}$ PRP

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – Anthrax Tripartite Toxin: Bacillus anthracis produces a tripartite exotoxin consisting of three proteins encoded on plasmid pXO1: (1) Protective Antigen (PA) – binds host cell receptor and forms a pore for toxin entry; (2) **Lethal Factor (LF)** – a zinc metalloprotease that cleaves MAPKKs causing macrophage lysis and septic shock; (3) **Oedema Factor (EF)** – an adenylate cyclase that raises intracellular cAMP causing massive oedema. PA + LF = Lethal Toxin; PA + EF = Oedema Toxin.

Key Point: B. anthracis is a gram-positive, non-motile, non-haemolytic, spore-forming bacillus with a poly-D-glutamic acid capsule (encoded on plasmid pXO2). Forms spores in soil (persists for decades). Cutaneous anthrax (wool-sorter's disease) presents as painless eschar with oedema. Inhalational anthrax (mediastinal widening on CXR) carries high mortality. Treat with ciprofloxacin or doxycycline.

Why other options are wrong:

- Option A: Haemolysin and lecithinase are produced by Clostridium perfringens (alpha toxin = lecithinase).
- Option C: Alpha toxin and beta toxin are produced by C. perfringens in gas gangrene.
- Option D: Exotoxin A and exotoxin S are Pseudomonas aeruginosa toxins.

Final Answer: Anthrax toxin (PA + LF + EF) $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q10

Q11.

Solution

Concept – Pseudomonas Pigments: Pseudomonas aeruginosa produces multiple pigments. **Pyocyanin** is the blue-green pigment responsible for the characteristic colour of infected wounds and culture media. Pyoverdin (fluorescein) is a yellow-green fluorescent siderophore. Pyorubin is a reddish-brown pigment. Pyomelanin is a dark-brown pigment. Pyocyanin specifically acts as a redox-active molecule that generates reactive oxygen species, contributing to lung damage in cystic fibrosis.

Key Point: P. aeruginosa is a gram-negative, non-lactose-fermenting, oxidase-positive bacillus. It is an opportunistic pathogen causing infections in CF patients (mucoid alginate biofilm), burns, ventilator-associated pneumonia, and neu-

tropenic sepsis. Produces multiple virulence factors: exotoxin A (ADP-ribosylates EF-2, inhibits protein synthesis, similar to diphtheria toxin), elastase, alkaline protease, and type 3 secretion system.

Why other options are wrong:

- Option A: Pyoverdin is yellow-green fluorescent; it is a siderophore, not responsible for the blue-green colour.
- Option B: Pyorubin is a reddish-brown pigment produced by some P. aeruginosa strains in small amounts.
- Option D: Melanin is produced by Cryptococcus neoformans and some P. aeruginosa strains; not the primary blue-green pigment.

Final Answer: Blue-green pigment of Pseudomonas $\Rightarrow \boxed{C}$ Pyocyanin

Answer: (C) Go Back to Q11

Q12.

Solution

Concept – Listeria Cold Enrichment: **Listeria monocytogenes** is a gram-positive, facultatively intracellular, psychrotrophic bacillus that exhibits characteristic tumbling motility (umbrella-shaped motility at room temperature) and is positive for CAMP test and catalase. It grows optimally at 4°C (cold enrichment), distinguishing it from most pathogens. Cold enrichment in nutrient broth at 4°C for weeks is a classic microbiological technique for isolating Listeria from food samples.

Key Point: Listeriosis occurs in pregnancy (septic abortion, neonatal meningitis), elderly, and immunocompromised individuals. It is transmitted via contaminated soft cheeses, deli meats, raw milk, and ready-to-eat foods. Uses ActA protein to hijack actin polymerisation for intracellular motility ("comet tail"). Treatment: ampicillin + gentamicin (synergy). Not treated with cephalosporins (intrinsically resistant).

Why other options are wrong:

- Option A: Alkaline peptone water enrichment is used for Vibrio cholerae isolation.
- Option B: Selenite F broth enrichment is used for Salmonella and Shigella from stool samples.
- Option C: Mannitol salt agar is a selective medium for Staphylococcus aureus.

Final Answer: Listeria isolation from food $\Rightarrow$ $\boxed{D}$ Cold enrichment at 4°C

Answer: (D) Go Back to Q12

Q13.

Solution

Concept – Elek's Gel Immunodiffusion Test: The **Elek's test** (gel immunodiffusion test) is the gold standard in vitro test for confirming toxin production by **Corynebacterium diphtheriae**. A strip impregnated with diphtheria antitoxin is placed on an agar plate alongside a streak of the test organism. If the organism produces diphtheria toxin, a line of precipitation (precipitin line) forms within 24–48 hours where the toxin and antitoxin meet by diffusion.

Key Point: C. diphtheriae is a gram-positive, pleomorphic, club-shaped (Chinese letter/cuneiform arrangement) bacillus. Toxin is encoded by the β-corynephage (phage-encoded, tox+ gene). Diphtheria toxin ADP-ribosylates EF-2 (elongation factor 2), inhibiting protein synthesis. Clinical: pseudomembrane in throat, bull neck, myocarditis (ECG changes), neuropathy (palatal palsy first). Schick test detects immunity; Tellurite agar (selective) grows black/grey colonies.

Why other options are wrong:

- Option B: Nagler's reaction detects lecithinase (alpha toxin) of Clostridium perfringens on egg-yolk agar.
- Option C: CAMP test identifies Listeria monocytogenes and Group B Streptococcus (S. agalactiae).
- Option D: Optochin sensitivity test is used to identify Streptococcus pneumoniae.

Final Answer: Diphtheria toxin confirmation $\Rightarrow$ $\boxed{A}$ Elek's gel immunodiffusion test

Answer: (A) Go Back to Q13

Q14.

Solution

Concept – Brucella Species and Animal Reservoirs: Brucella abortus primarily infects cattle (bovines) and causes infectious abortion (Bang's disease) in cattle. It is the most common cause of brucellosis in veterinarians and cattle farmers. Different Brucella species infect different hosts: B. melitensis (goats/sheep – most

virulent), B. abortus (cattle), B. suis (pigs), B. canis (dogs).

Key Point: Brucella is a gram-negative coccobacillus, facultative intracellular organism. Grows slowly on Castaneda medium requiring 5–10% CO_2 (except B. canis and B. suis). Undulant fever (relapsing/remitting), night sweats, and arthralgia are hallmarks. Diagnosis: blood culture (gold standard), Brucella agglutination test (standard tube agglutination test, STAT), and Rose Bengal test (screening). Treatment: doxycycline + rifampicin for 6 weeks (or streptomycin for 3 weeks).

Why other options are wrong:

- Option A: B. melitensis primarily infects goats and sheep; it is the most virulent species.
- Option C: B. suis primarily infects pigs.
- Option D: B. canis primarily infects dogs; transmission to humans is uncommon.

Final Answer: Brucella infecting cattle $\Rightarrow \boxed{B}$ Brucella abortus

Answer: (B) Go Back to Q14

Q15.

Solution

Concept – Type I Hypersensitivity (Anaphylaxis) and IgE: Type I (immediate) hypersensitivity is mediated by **IgE** antibodies bound to high-affinity FcεRI receptors on mast cells and basophils. On re-exposure, the allergen cross-links two adjacent IgE molecules on mast cells, triggering degranulation and release of preformed mediators (histamine, tryptase) and newly synthesised mediators (prostaglandins, leukotrienes, cytokines). This results in vasodilation, bronchospasm, urticaria, and angioedema.

Key Point: IgE is produced by B cells after IL-4 and IL-13-driven class switch recombination. The sequence: allergen exposure $\rightarrow$ Th2 activation $\rightarrow$ IL-4 $\rightarrow$ B cell class switch to IgE $\rightarrow$ IgE binds FcεRI on mast cells $\rightarrow$ re-exposure $\rightarrow$ cross-linking $\rightarrow$ degranulation. Anaphylaxis treatment: epinephrine (adrenaline) IM, antihistamines, corticosteroids. Serum IgE levels are elevated in atopy and parasitic infections.

Why other options are wrong:

- Option A: IgG mediates Type II (cytotoxic), Type III (immune complex), and

ADCC; not anaphylaxis.

- Option B: IgM mediates primary immune response and complement activation (Type II/III); not IgE-dependent degranulation.
- Option D: IgA is the primary mucosal antibody in secretions; does not mediate mast cell degranulation.

Final Answer: Type I hypersensitivity (anaphylaxis) $\Rightarrow \boxed{C}$ IgE

Answer: (C) Go Back to Q15

Q16.

Solution

Concept – HIV and CD4+ T Helper Cells: HIV gp120 (surface glycoprotein) has high affinity for the **CD4 molecule** expressed predominantly on **CD4+ T helper lymphocytes**. Binding of gp120 to CD4 triggers a conformational change, allowing gp120 to bind a co-receptor (CCR5 for macrophage-tropic R5 strains, CXCR4 for T-cell-tropic X4 strains). This leads to gp41-mediated membrane fusion and viral entry. Progressive depletion of CD4+ T cells (below 200 cells/μL) leads to AIDS.

Key Point: CD4+ T helper cells are the master regulators of adaptive immunity – they help B cells (antibody production), CD8+ CTLs, and macrophages. Their depletion accounts for susceptibility to opportunistic infections (Pneumocystis jirovecii pneumonia $< 200/\mu$L, CMV retinitis $< 50/\mu$L, toxoplasmosis $< 100/\mu$L). CD4 count guides prophylaxis and treatment initiation.

Why other options are wrong:

- Option A: CD8+ cytotoxic T cells express very little CD4; they are the immune effectors that kill HIV-infected cells.
- Option B: B lymphocytes do not express CD4; they can be indirectly affected through loss of T helper cell support.
- Option C: Natural killer cells are $CD3^{-}$, $CD16^{+}$, $CD56^{+}$; they do not express CD4 as a primary receptor.

Final Answer: HIV primary target (gp120 + CD4) $\Rightarrow \boxed{D}$ CD4+ T helper lymphocytes

Answer: (D) Go Back to Q16

Q17.

Solution

Concept – MHC Class II and Exogenous Antigen Presentation: MHC class II molecules (HLA-DR, HLA-DP, HLA-DQ) are expressed on professional antigen-presenting cells (APCs): dendritic cells, macrophages, and B cells. They present **exogenous antigens** (derived from extracellular pathogens via endocytic pathway) to **CD4+ T helper cells**. The endosomal pathway: protein ingested by endocytosis $\rightarrow$ degraded in acidified endosomes/lysosomes $\rightarrow$ peptides loaded onto MHC class II in MIIC $\rightarrow$ presented on cell surface.

Key Point: Mnemonic: **"E-X-O-genous = class II" and "E-N-DO-genous = class I"**. MHC class I (HLA-A, B, C) presents endogenous antigens (viral/tumour peptides) to CD8+ CTLs. MHC class III genes encode complement proteins (C2, C4, factor B) and cytokines (TNF). Beta-2 microglobulin is associated non-covalently with MHC class I (not class II).

Why other options are wrong:

- Option B: MHC class I presents endogenous (intracellular) antigens to CD8+ cytotoxic T cells; it is expressed on all nucleated cells.
- Option C: MHC class III genes do not encode antigen-presenting molecules; they encode complement and cytokine genes.
- Option D: Beta-2 microglobulin is the invariant light chain of MHC class I; it is not an antigen-presenting molecule itself.

Final Answer: Exogenous antigen + CD4+ T cells $\Rightarrow \boxed{A}$ MHC class II

Answer: (A) Go Back to Q17

Q18.

Solution

Concept – C3b as Principal Opsonin: During complement activation (classical, lectin, or alternative pathway), C3 convertase cleaves C3 into C3a and **C3b**. C3b covalently binds to pathogen surfaces and acts as the principal opsonin of the complement system. Phagocytes (neutrophils, macrophages) express complement receptor 1 (CR1/CD35) that specifically binds C3b, facilitating phagocytosis (opsonophagocytosis). C3a is an anaphylatoxin that activates mast cells.

Key Point: Opsonins enhance phagocytosis: C3b (via CR1) and IgG (via FcγR). C3b also opsonises for NK cell killing. Downstream: C3b + C3 convertase forms C5 convertase, cleaving C5 into C5a (potent anaphylatoxin/chemotactic) and C5b

(initiates MAC). C3 deficiency → recurrent infections with encapsulated bacteria (Streptococcus pneumoniae, H. influenzae, N. meningitidis). DAF (CD55) and CD59 protect host cells from complement.

Why other options are wrong:

- Option A: C3a is an anaphylatoxin; it activates mast cells causing histamine release but does not directly opsonise bacteria for phagocytosis.
- Option C: C5a is the most potent anaphylatoxin and chemotactic factor; it does not bind CR1 for opsonisation.
- Option D: C1q initiates the classical complement pathway by binding IgG/IgM on antigen surfaces; it is not the opsonin for phagocyte recognition.

Final Answer: Principal complement opsonin binding CR1 $\Rightarrow \boxed{B}$ C3b

Answer: (B) Go Back to Q18

Q19.

Solution

Concept – Natural Killer Cells and Missing Self Hypothesis: Natural Killer (NK) cells ($CD3^-$, $CD16^+$, $CD56^+$) are large granular lymphocytes of the innate immune system. They kill target cells (tumour cells, virus-infected cells) without prior sensitisation and without recognising specific antigens. The "**missing self**" hypothesis (proposed by Klas Kärre): NK cells are normally inhibited when they detect **MHC class I** molecules on normal host cells. When MHC class I is down-regulated (as by viruses to evade CTLs, or by tumours), NK cells are activated and kill the target.

Key Point: NK cell activation is a balance between inhibitory signals (via KIR receptors recognising MHC class I — inhibits NK killing) and activating signals (NKG2D recognising stress ligands like MICA/MICB). NK cells kill via perforin-granzyme pathway and FasL/Fas-mediated apoptosis. They also perform ADCC via CD16 (FcγRIII) binding IgG-coated cells.

Why other options are wrong:

- Option A: B7 (CD80/CD86) provides co-stimulatory signals for T cell activation (T cell CD28 binds B7); NK cells do not use CD28 for activation.
- Option B: CD4 is the receptor for HIV and MHC class II; it is not expressed on NK cells as their recognition molecule.
- Option D: TCR is expressed only on T lymphocytes; NK cells do not have a

TCR and do not recognise antigens specifically.

Final Answer: NK cells recognise absence of $\Rightarrow$ $\boxed{C}$ MHC class I molecule

Answer: (C) Go Back to Q19

Q20.

Solution

Concept – Somatic Hypermutation in Germinal Centres: Somatic hypermutation (SHM) is the process by which the enzyme activation-induced cytidine deaminase (AID) introduces point mutations in the variable region (V) genes of immunoglobulin heavy and light chains at a rate $\sim 10^{-3}$ mutations/base pair/generation – approximately one million times higher than the baseline somatic mutation rate. SHM occurs specifically in germinal centres (GCs) of secondary lymphoid follicles after antigen stimulation. B cells with higher-affinity mutations are positively selected (affinity maturation).

Key Point: GC reactions also produce **class switch recombination (CSR)**: switching constant region of heavy chain from IgM to IgG, IgA, or IgE (also AID-dependent). V(D)J recombination occurs in bone marrow during B cell development (produces primary diversity). Allelic exclusion ensures only one heavy and one light chain allele is productively rearranged per B cell.

Why other options are wrong:

- Option A: V(D)J recombination generates primary antibody diversity in bone marrow; it is not the same as somatic hypermutation.
- Option B: Class switch recombination changes antibody isotype (class) but does not introduce point mutations in V regions for affinity maturation.
- Option C: Allelic exclusion ensures monospecificity of B cells; it occurs in bone marrow and does not increase antibody diversity.

Final Answer: Point mutations in V region of Ig in germinal centres $\Rightarrow$ $\boxed{D}$ Somatic hypermutation

Answer: (D) Go Back to Q20

Q21.

Solution

Concept – TLR4 Recognises LPS (Pattern Recognition in Innate Immunity): **Toll-like receptor 4 (TLR4)** is expressed on macrophages, dendritic cells, and neutrophils. It specifically recognises **lipopolysaccharide (LPS)** from gram-negative bacterial outer membranes. LPS is presented to TLR4 by CD14 and MD-2 co-proteins. TLR4 signals via the **MyD88-NF-κB pathway**, leading to production of pro-inflammatory cytokines (TNF-α, IL-1, IL-6, IL-12) that initiate the innate immune response and sepsis syndrome.

Key Point: Key TLR specificities: TLR1/2 (lipoproteins), TLR3 (dsRNA), TLR4 (LPS), TLR5 (flagellin), TLR7/8 (ssRNA), TLR9 (unmethylated CpG DNA). TLRs are pattern recognition receptors (PRRs) of innate immunity. They recognise pathogen-associated molecular patterns (PAMPs) – conserved microbial structures not found on host cells.

Why other options are wrong:

- Option B: NOD1 receptor (intracellular PRR) recognises iE-DAP from gram-negative bacterial peptidoglycan – not LPS.
- Option C: RIG-I (retinoic acid-inducible gene I) is a cytoplasmic PRR recognising viral RNA; does not recognise LPS.
- Option D: NLRP3 inflammasome is activated by danger signals (uric acid crystals, ATP, etc.) and processes IL-1β; not the primary LPS sensor.

Final Answer: LPS recognition via MyD88-NF-κB $\Rightarrow \boxed{A}$ TLR4

Answer: (A) Go Back to Q21

Q22.

Solution

Concept – Regulatory T Cells (Tregs) and FoxP3: **Regulatory T cells (Tregs)** are a subset of CD4+ T cells that express the transcription factor **FoxP3** along with CD25 (IL-2 receptor α chain). FoxP3 is the master regulator of Treg development and function. Tregs maintain peripheral self-tolerance by suppressing self-reactive T cells through: (1) secretion of anti-inflammatory cytokines (IL-10, TGF-β, IL-35); (2) direct cytotoxicity (granzyme B); (3) consumption of IL-2. FoxP3 mutations cause IPEX syndrome – a rare X-linked disorder of immune dysregulation.

Key Point: IPEX syndrome (Immune dysregulation, Polyendocrinopathy, Enteropathy, X-linked) is the hallmark disease of FoxP3/Treg deficiency. Features:

early-onset type 1 DM, thyroiditis, haemolytic anaemia, eczema, and severe enteropathy. Without Tregs, self-reactive T cells escape peripheral tolerance and attack self-tissues (autoimmunity).

Why other options are wrong:

- Option A: CD8+ cytotoxic T cells (CTLs) kill virally infected/tumour cells; they do not require FoxP3 for their function.
- Option C: Th17 cells produce IL-17 and IL-22; they promote inflammation (especially anti-fungal immunity) – the opposite of immune suppression.
- Option D: NKT cells recognise lipid antigens via CD1d; they produce IL-4 and IFN-γ; they do not express FoxP3.

Final Answer: FoxP3 mutation, IPEX, peripheral tolerance $\Rightarrow$ $\boxed{B}$ Regulatory T cells (CD4+CD25+FoxP3+)

Answer: (B) Go Back to Q22

Q23.

Solution

Concept – ADCC and FcγRIII (CD16): Antibody-dependent cellular cytotoxicity (ADCC) occurs when NK cells (and macrophages/neutrophils) recognise IgG-coated target cells via their Fcγ receptors and kill them by perforin-granzyme pathway. NK cells express **FcγRIII (CD16)**, which binds the Fc region of IgG1 and IgG3 antibodies. ADCC is important for eliminating virus-infected cells, antibody-coated tumour cells, and in mediating the effects of therapeutic monoclonal antibodies (e.g., trastuzumab, rituximab).

Key Point: CD16 (FcγRIII) is also a surface marker used to identify NK cells by flow cytometry (CD3$^-$, CD16$^+$, CD56$^+$). The Fcγ receptors: FcγRI (CD64) on macrophages – high affinity; FcγRIII (CD16) on NK cells/macrophages; FcγRII (CD32) on macrophages/neutrophils. FcαRI (CD89) binds IgA and is expressed on neutrophils and macrophages.

Why other options are wrong:

- Option A: FcαRI (CD89) binds IgA, not IgG; expressed on neutrophils and macrophages for IgA-dependent cytotoxicity.
- Option B: FcεRI is the high-affinity IgE receptor on mast cells and basophils mediating Type I hypersensitivity, not ADCC.
- Option D: FcγRI (CD64) is expressed on macrophages and dendritic cells; it binds IgG with high affinity but is not the primary mediator of ADCC by NK

cells.

Final Answer: NK cell + IgG-coated target ⇒ ADCC via $\boxed{C}$ FcγRIII (CD16)

Answer: (C) Go Back to Q23

Q24.

Solution

Concept – Secondary Immune Response Predominantly IgG: The **secondary (anamnestic/booster) immune response** is characterised by: (1) shorter lag phase (1–3 days vs. 7–10 days); (2) much higher antibody titres (10–1000-fold greater); (3) predominantly **IgG** class (due to class switch from IgM to IgG in germinal centres after memory B cell activation); (4) higher affinity antibodies (affinity maturation); (5) longer duration. This response is mediated by long-lived memory B cells and memory T cells established during the primary response.

Key Point: Primary response: IgM produced first (predominant), then some IgG. Secondary response: rapid IgG surge (predominant), minimal IgM. This is the basis of vaccination (priming followed by boosters) and serological diagnosis (IgM = recent/acute infection; IgG = past infection or immunity).

Why other options are wrong:

- Option A: IgA is the predominant mucosal antibody; it is produced during secondary responses at mucosal sites but is not the dominant serum antibody of secondary response.
- Option B: IgE is produced in allergic/atopic responses and parasitic infections; it is not the dominant secondary response immunoglobulin.
- Option C: IgM is the first antibody produced in the primary response; the secondary response switches predominantly to IgG, not IgM.

Final Answer: Secondary immune response predominant antibody ⇒ $\boxed{D}$ IgG

Answer: (D) Go Back to Q24

Q25.

Solution

Concept – HIV CCR5 Co-receptor for R5 (Macrophage-Tropic) Strains: HIV-1 uses CD4 as the primary receptor and a chemokine receptor as a co-receptor for cell entry. **Macrophage-tropic (M-tropic / R5) strains** use **CCR5** (β-chemokine

receptor) as the co-receptor. These R5 strains predominate during early/acute HIV infection (transmitted strains) and target macrophages and CD4+ T cells bearing CCR5. Individuals homozygous for the CCR5-Δ32 deletion are highly resistant to HIV-1 R5 strain infection. This is the basis for maraviroc (CCR5 antagonist antiretroviral drug).

Key Point: T-cell-tropic (T-tropic / X4) strains use CXCR4 as co-receptor; they predominate in late-stage HIV and are associated with accelerated CD4 decline. The co-receptor tropism shift from R5 to X4 is associated with rapid disease progression. Maraviroc requires tropism testing before use.

Why other options are wrong:

- Option B: CXCR4 is the co-receptor for T-cell-tropic (X4) strains, which predominate in late-stage HIV disease, not early infection.
- Option C: CCR3 is a chemokine receptor used by some HIV-1 strains in vitro but is not the primary physiologically relevant co-receptor.
- Option D: CXCR1 (IL-8 receptor) is not a known HIV co-receptor.

Final Answer: HIV R5 (macrophage-tropic) co-receptor $\Rightarrow \boxed{A}$ CCR5

Answer: (A) Go Back to Q25

Q26.

Solution

Concept – Hepatitis B Window Period and Anti-HBc IgM: During the **window period** of acute hepatitis B infection, HBsAg has been cleared from serum but anti-HBs (protective antibody) has not yet appeared. During this serologically silent period, both HBsAg and anti-HBs are negative. **Anti-HBc IgM** (IgM antibody to hepatitis B core antigen) is the only marker that remains positive throughout the window period and is diagnostic of acute hepatitis B. It appears early in acute infection and persists for 6 months.

Key Point: HBV serological sequence: HBsAg (first to appear, > 1 month post-exposure) $\rightarrow$ HBeAg (indicates high replication/infectivity) $\rightarrow$ Anti-HBc IgM $\rightarrow$ Window period (HBsAg$-$/Anti-HBs$-$/Anti-HBc IgM$+$) $\rightarrow$ Anti-HBs (immunity). Anti-HBc IgG persists for life as a marker of past infection or chronic exposure. Anti-HBs alone = vaccination (no anti-HBc).

Why other options are wrong:

- Option A: HBeAg indicates active viral replication and high infectivity; it

is present before the window period but may also become negative before HBsAg; it is not the window period marker.
- Option C: Anti-HBs is the neutralising protective antibody that appears AFTER the window period; its presence indicates recovery or vaccination.
- Option D: HBsAg is the first marker to appear but is negative during the window period by definition.

Final Answer: Window period marker of acute Hepatitis B $\Rightarrow$ $\boxed{B}$ Anti-HBc IgM

Answer: (B) Go Back to Q26

Q27.

Solution

Concept – HSV Latency in Trigeminal Ganglion: Herpes simplex virus type 1 (HSV-1) causes primary oral infection (herpes labialis/cold sores). After primary infection, the virus travels retrogradely along sensory nerve axons to the **trigeminal ganglion** (specifically the trigeminal sensory ganglion, a type of dorsal root ganglion for the face), where it establishes latency. During latency, only latency-associated transcripts (LATs) are produced. Reactivation (by UV exposure, fever, stress, immunosuppression) causes anterograde transport back to the skin/mucosa.

Key Point: HSV-1 latency site: trigeminal ganglion (for orofacial disease). HSV-2 latency site: sacral dorsal root ganglia S2–S4 (for genital herpes). Varicella-Zoster Virus (VZV) also latent in dorsal root/cranial nerve ganglia – reactivates as zoster (shingles). Acyclovir (requires viral thymidine kinase for activation) is the treatment; it does not eliminate latent virus.

Why other options are wrong:

- Option A: Parotid gland acinar cells harbour EBV (Epstein-Barr virus) and also CMV in saliva, not HSV latency.
- Option B: Liver hepatocytes harbour HBV, HCV, and CMV; not the site of HSV neural latency.
- Option D: Regional lymph nodes serve as a site of viral replication during primary immune response; HSV does not establish latency in lymph nodes.

Final Answer: HSV-1 latency site $\Rightarrow$ $\boxed{C}$ Trigeminal ganglion

Answer: (C) Go Back to Q27

Q28.

Solution

Concept – Antigenic Shift in Influenza A: Antigenic shift is the sudden, major change in influenza A surface antigens (haemagglutinin HA and/or neuraminidase NA) due to **reassortment of entire genome segments** when two different influenza A strains co-infect the same host cell. The segmented, negative-sense, single-stranded RNA genome of influenza A (8 segments) allows this exchange. Pigs serve as the main "mixing vessel" because they have receptors for both human (sialic acid α2,6) and avian (sialic acid α2,3) influenza strains. Antigenic shift produces a new subtype (e.g., H1N1 1918, H2N2 1957, H3N2 1968), to which the population has no pre-existing immunity, causing pandemics.

Key Point: Antigenic drift is a continuous, gradual accumulation of point mutations in HA and NA genes due to RNA polymerase errors (no proofreading); it causes seasonal epidemics and requires annual vaccine reformulation. Influenza A subtypes are classified by HA (H1–H18) and NA (N1–N11) combinations. Antiviral treatment: oseltamivir (Tamiflu) inhibits NA, preventing viral release.

Why other options are wrong:

- Option A: Antigenic drift is gradual point mutation accumulation; it does NOT involve exchange of entire genome segments or creation of new subtypes.
- Option B: Genetic recombination involves crossing over within the same strand; influenza uses segmented reassortment, not recombination.
- Option C: Phenotypic mixing involves mixing of surface proteins without genome change; it produces no new viral progeny with altered genotype.

Final Answer: Influenza genome segment exchange causing pandemic $\Rightarrow \boxed{D}$ Antigenic shift

Answer: (D) Go Back to Q28

Q29.

Solution

Concept – Negri Bodies in Rabies: Negri bodies are pathognomonic eosinophilic intracytoplasmic inclusion bodies found in rabies virus-infected neurons. They represent accumulations of viral nucleocapsid material (RNP complexes) within the neuronal perikaryon. They are most reliably found in the **hippocampal pyramidal neurons** (Ammon's horn), cerebellar Purkinje cells, and dorsal root ganglia. Post-mortem brain biopsy with Seller's stain or H&E reveals these inclusions for

diagnosis.

Key Point: Rabies virus (Lyssavirus, ssRNA, enveloped, bullet-shaped) travels centripetally along peripheral nerves to the CNS, then centrifugally to salivary glands. Incubation period: 20–90 days (longer with distant bites). Clinical stages: prodrome → acute neurological phase (furious/dumb) → coma → death. Post-exposure prophylaxis (PEP): wound wash + HRIG + rabies vaccine series. No effective treatment once symptomatic.

Why other options are wrong:

- Option B: Guarnieri bodies are cytoplasmic inclusions seen in smallpox (variola virus) and vaccinia virus-infected cells.
- Option C: Cowdry type A (eosinophilic nuclear inclusions) are seen in HSV, CMV, and yellow fever virus infections.
- Option D: Aschoff bodies are granulomatous lesions in myocardium of rheumatic fever; not intracytoplasmic viral inclusions.

Final Answer: Rabies + eosinophilic intracytoplasmic neuronal inclusions $\Rightarrow \boxed{A}$ Negri bodies

Answer: (A) Go Back to Q29

Q30.

Solution

Concept – Dengue NS1 Antigen for Early Diagnosis: Dengue NS1 (non-structural protein 1) is secreted in high concentrations in blood during the febrile phase (days 1–5 of illness), before the appearance of dengue-specific antibodies. **Dengue NS1 antigen ELISA** is the most reliable early virological test during the acute phase. It is positive in both primary and secondary dengue infection, with higher concentrations in primary infections. After day 5, dengue IgM (primary) or IgG (secondary) antibodies appear and serological tests become useful.

Key Point: Dengue haemorrhagic fever (DHF) occurs more commonly during secondary infection with a different dengue serotype (1–4) due to antibody-dependent enhancement (ADE): non-neutralising heterotypic antibodies from the first infection facilitate uptake of the second serotype into Fc-receptor-bearing cells (macrophages), amplifying viral load. WHO dengue criteria: fever + 2 of: nausea/vomiting, rash, aches, positive tourniquet test, leukopenia.

Why other options are wrong:

- Option A: Dengue IgM appears after day 5 and peaks at 2 weeks; it is not useful for early diagnosis in the first 5 days.
- Option C: Dengue E (envelope) protein ELISA is not a standard clinical diagnostic test; NS1 is the validated early antigen test.
- Option D: Dengue IgG appears late and indicates past infection or secondary infection; elevated IgG alone is not diagnostic of acute early dengue.

Final Answer: Early (day 1–5) dengue diagnosis $\Rightarrow \boxed{B}$ NS1 antigen test

Answer: (B) Go Back to Q30

Q31.

Solution

Concept – HPV E6 and E7 Oncoproteins: High-risk HPV types (16, 18, 31, 33, 45) integrate their DNA into the host genome, producing viral oncoproteins E6 and E7. **E6 protein** binds and targets p53 tumour suppressor for ubiquitin-mediated proteasomal degradation (via E6-AP ubiquitin ligase), preventing apoptosis and DNA repair. **E7 protein** binds and inactivates the retinoblastoma protein (Rb), releasing E2F transcription factor and driving uncontrolled cell cycle progression (G1→S transition).

Key Point: Mnemonic: **"E6 kills p53; E7 kills Rb"**. Both p53 and Rb are tumour suppressors; their inactivation leads to cervical squamous cell carcinoma. HPV types 6 and 11 cause condylomata acuminata (genital warts) – low-risk types. Prevention: Gardasil 9 (nonavalent vaccine against HPV 6, 11, 16, 18, 31, 33, 45, 52, 58). Cervical screening: Pap smear every 3 years, cotesting with HPV DNA every 5 years.

Why other options are wrong:

- Option A: E5 promotes cell proliferation via EGFR signalling but does not directly inactivate p53 or Rb; E4 disrupts keratin networks.
- Option B: This reverses E6 and E7 roles; E7 inactivates Rb (not p53), and E6 degrades p53 (not Rb).
- Option D: E2 is a viral transcription/replication factor; E1 is a helicase for viral DNA replication; neither directly inactivates p53 or Rb.

Final Answer: HPV oncoproteins: E6 degrades p53, E7 inactivates Rb $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q31

Q32.

Solution

Concept – Koplik Spots in Measles: Koplik spots are pathognomonic enanthems of measles (morbilli), caused by Measles morbillivirus (Paramyxoviridae, ssRNA, negative-sense, enveloped). They appear 1–2 days BEFORE the maculopapular rash (exanthem) on the buccal mucosa, typically opposite the lower molar teeth. They appear as tiny white/blue-white spots (salt-and-pepper) on an erythematous base and are pathognomonic for measles.

Key Point: Measles clinical sequence: Incubation (10–14 days) $\rightarrow$ Prodrome (3 C's: Cough, Coryza, Conjunctivitis + fever) $\rightarrow$ Koplik spots $\rightarrow$ Maculopapular rash (starts on face/hairline, spreads cephalocaudally). Warthin-Finkeldey giant cells (multinucleated) in lymphoid tissue. Complications: pneumonia, croup, encephalitis, SSPE (subacute sclerosing panencephalitis, years later). Live attenuated MMR vaccine given at 9 months (first dose in India) and 15–18 months.

Why other options are wrong:

- Option A: Fordyce spots are ectopic sebaceous glands appearing as small yellowish-white spots on the lip vermilion/buccal mucosa; they are benign and not disease-related.
- Option B: Wickham's striae are white lacy lines seen on the buccal mucosa in lichen planus (autoimmune condition).
- Option C: Rose spots are salmon-coloured blanching maculopapular lesions on the abdomen in typhoid fever (S. typhi infection), not measles.

Final Answer: Measles pathognomonic mucosal lesion $\Rightarrow \boxed{D}$ Koplik spots

Answer: (D) Go Back to Q32

Q33.

Solution

Concept – Plasmodium falciparum Knob Formation and Cerebral Malaria: Plasmodium falciparum uniquely causes infected RBCs to develop surface **knobs** – electron-dense protrusions composed of P. falciparum erythrocyte membrane protein 1 (PfEMP1) and other exported proteins. **Knob formation** enables cytoadherence of infected RBCs to the vascular endothelium of deep microvasculature (via ICAM-1, CD36, PECAM receptors), causing sequestration. In cerebral malaria, this sequestration occurs in cerebral microvessels, causing obstruction, ischaemia, local inflammation, and coma.

Key Point: Falciparum malaria features: ring forms and gametocytes (banana/sickle-shaped, pathognomonic) on blood film; NO Schüffner's dots (only P. vivax/P. ovale have these); can infect RBCs of all ages (unlike P. vivax which infects only reticulocytes, P. malariae which infects only mature RBCs); rosetting; high parasitaemia. Treatment: artesunate-based combination therapy (ACT) for uncomplicated; IV artesunate for severe malaria.

Why other options are wrong:

- Option B: Double infection of RBCs occurs in falciparum malaria (multiple ring forms per RBC) but does not directly cause cerebral microvascular obstruction.
- Option C: Schüffner's dots are characteristic of P. vivax and P. ovale (not P. falciparum); they do not cause cytoadherence.
- Option D: P. vivax preferentially infects reticulocytes; P. falciparum infects RBCs of all ages; reticulocyte preference is NOT a P. falciparum feature.

Final Answer: Cerebral malaria mechanism $\Rightarrow \boxed{A}$ Knob formation causing cytoadherence

Answer: (A) Go Back to Q33

Q34.

Solution

Concept – Entamoeba histolytica Erythrophagocytosis: The most pathognomonic finding on stool microscopy for **Entamoeba histolytica** invasion is the presence of **trophozoites containing ingested red blood cells (erythrophagocytosis)** in their cytoplasm. These trophozoites (10–60 μm) have a single nucleus with centrally placed, dot-like karyosome and uniformly distributed peripheral chromatin. Erythrophagocytosis confirms invasive amoebiasis (unlike the non-pathogenic E. dispar, which does not ingest RBCs).

Key Point: E. histolytica life cycle: cyst (infective form, 4 nuclei) $\rightarrow$ small intestine excystation $\rightarrow$ trophozoite (invasive) $\rightarrow$ large intestine $\rightarrow$ flask-shaped ulcers with undermined edges $\rightarrow$ dysentery. Complications: liver abscess (anchovy sauce/chocolate brown pus), lung abscess, brain abscess. Diagnosis: stool microscopy (RBC-containing trophozoites) + serology (ELISA) for liver abscess. Treatment: metronidazole (tissue amoebiasis) + diloxanide furoate (luminal cysts).

Why other options are wrong:

- Option A: Multinucleated trophozoites are NOT a feature of E. histolytica; trophozoites are uninucleate (single nucleus). Multinucleation is a feature of cysts (4 nuclei in mature cysts).
- Option C: Quadrinucleate cyst excystation describes the infection mechanism (ingestion of 4-nucleate cysts), not a microscopic diagnostic finding in trophozoites.
- Option D: Trophozoites with two nuclei and axostyle describe Giardia lamblia (binucleate trophozoites), not E. histolytica.

Final Answer: E. histolytica diagnostic microscopy finding $\Rightarrow$ $\boxed{B}$ Ingested RBCs in trophozoite cytoplasm

Answer: (B) Go Back to Q34

Q35.

Solution

Concept – Giardia Causing Steatorrhoea (Without Blood): Giardia lamblia (G. intestinalis) infects the duodenum and jejunum, causing malabsorption of fat and fat-soluble vitamins (A, D, E, K). The result is **steatorrhoea** – fatty, bulky, foul-smelling, greasy stools. Crucially, Giardia causes **NON-bloody diarrhoea** (no mucosal invasion, no ulceration), distinguishing it from amoebic dysentery (which causes bloody mucoid diarrhoea with tenesmus).

Key Point: Giardia trophozoite: pear-shaped (piriform), bilaterally symmetrical, 2 nuclei (binucleate), 4 pairs of flagella, ventral sucking disc (adhesion to duodenal epithelium). "**Falling leaf" or "tumbling" motility**. Cysts: oval, 4 nuclei, survive in cold water (transmitted via contaminated water/fecal-oral route). Diagnosis: stool microscopy (cysts/trophozoites), ELISA for Giardia antigen (most sensitive). Treatment: metronidazole or tinidazole.

Why other options are wrong:

- Option A: Presence of blood in stool is characteristic of amoebic dysentery (E. histolytica), shigellosis, or STEC; Giardia does NOT cause bloody diarrhoea.
- Option B: Perianal pruritus is characteristic of Enterobius vermicularis (pinworm) infestation, not Giardia.
- Option D: Liver abscess is a complication of E. histolytica, not Giardia lamblia.

Final Answer: Giardia vs amoeba key differentiator $\Rightarrow$ $\boxed{C}$ Steatorrhoea without blood

Answer: (C) Go Back to Q35

Q36.

Solution

Concept – Congenital Toxoplasmosis via Tachyzoites: Congenital toxoplasmosis results from transplacental transmission of **tachyzoites** of Toxoplasma gondii during acute primary maternal infection. During the acute phase, tachyzoites (rapidly dividing, crescent/arc-shaped obligate intracellular forms) circulate in the bloodstream and cross the placenta to infect the foetus. The classic triad of congenital toxoplasmosis: **hydrocephalus, chorioretinitis, and intracranial (periventricular) calcifications**.

Key Point: T. gondii life cycle: Definitive host = cat family (oocysts excreted in faeces). Intermediate hosts = all warm-blooded animals (including humans). Three forms: (1) Tachyzoites (acute/invasive phase – transplacental transmission); (2) Bradyzoites (chronic/latent in tissue cysts in muscle/brain); (3) Sporozoites (in oocysts). Congenital infection risk highest in 3rd trimester (due to placental permeability) but severity greatest in 1st trimester. Maternal IgG antibodies cross placenta (protective), but acute maternal infection allows tachyzoite transmission.

Why other options are wrong:

- Option A: Oocysts are shed in cat faeces into soil; they are the environmental form and do not cross the placenta. Ingestion of oocysts causes infection, not direct transplacental transfer.
- Option B: Bradyzoites are the chronic/latent stage in tissue cysts (muscle, brain); reactivation of bradyzoites causes toxoplasmic encephalitis in AIDS, not congenital transmission from primary infection.
- Option C: Sporozoites are contained within oocysts; they are not a circulating form in maternal blood and do not cross the placenta.

Final Answer: Transplacental toxoplasmosis – transmitted as $\Rightarrow \boxed{D}$ Tachyzoites

Answer: (D) Go Back to Q36

Q37.

Solution

Concept – Löffler's Syndrome (Pulmonary Larval Migration of Ascaris): The pulmonary symptoms (transient pulmonary infiltrates, cough, fever, eosinophilia) in ascariasis occur during the **pulmonary larval migration phase** – also known as **Löffler's syndrome**. After ingestion of embryonated eggs, larvae hatch in the small intestine, penetrate the intestinal wall, enter the portal circulation, travel to the liver → right heart → pulmonary capillaries → break into alveoli → migrate up the respiratory tree → are swallowed → develop into adult worms in the small intestine. The lung phase causes eosinophilic inflammation.

Key Point: Löffler's syndrome (pulmonary eosinophilia): transient bilateral pulmonary infiltrates (on CXR – "fleeting shadows"), blood eosinophilia, dry cough, low-grade fever. Occurs 10–14 days post-ingestion of eggs. Adult worms in intestine cause malnutrition, intestinal obstruction, biliary/pancreatic obstruction (large worm burden). Diagnosis: stool examination (eggs), Charcot-Leyden crystals. Treatment: albendazole or mebendazole.

Why other options are wrong:

- Option B: The adult worm intestinal phase causes malnutrition and mechanical complications (obstruction); pulmonary symptoms do NOT occur at this phase.
- Option C: Liver schizogony is a stage in malaria (Plasmodium spp.) life cycle; Ascaris does not undergo liver schizogony.
- Option D: Encysted larval phase in muscle is characteristic of Trichinella spiralis (trichinosis) after consuming undercooked pork; not Ascaris.

Final Answer: Pulmonary eosinophilia (Löffler's) in ascariasis $\Rightarrow \boxed{A}$ Pulmonary larval migration phase

Answer: (A) Go Back to Q37

Q38.

Solution

Concept – Wuchereria bancrofti and Nocturnal Periodicity: Wuchereria bancrofti is a filarial nematode transmitted by Culex mosquitoes (in tropical/subtropical areas including India). Adult worms reside in lymphatics, causing lymphangitis, lymphadenitis, and eventually lymphoedema (elephantiasis). The microfilariae (larval stage in blood) exhibit **nocturnal periodicity** – they appear in peripheral blood predominantly at night (peak between 10 PM and 2 AM), coinciding with the nocturnal feeding habit of Culex mosquitoes. The microfilariae

are sheathed and lack nuclei at the tail tip.

Key Point: Filariasis periodicity: W. bancrofti (nocturnal, Indian subcontinent), Brugia malayi (nocturnal, South/Southeast Asia), Brugia timori (nocturnal, East Indonesia), Loa loa (diurnal, West Africa – transmitted by Chrysops fly), Mansonella perstans (non-periodic). Blood film timing for diagnosis: midnight for W. bancrofti. Nocturnal diurnal reversal occurs in patients who work at night (reversal of periodicity). Treatment: diethylcarbamazine (DEC).

Why other options are wrong:

- Option A: Brugia timori shows nocturnal periodicity (not diurnal); it is found in East Indonesian islands, not Indian coastal districts.
- Option C: Loa loa shows **diurnal** periodicity (microfilariae in blood during daytime), transmitted by Chrysops fly in West Africa.
- Option D: Mansonella perstans shows non-periodic microfilaraemia and is found in tropical Africa and South America; not associated with elephantiasis.

Final Answer: Elephantiasis + midnight microfilariae + coastal India $\Rightarrow \boxed{B}$ Wuchereria bancrofti – nocturnal periodicity

Answer: (B) Go Back to Q38

Q39.

Solution

Concept – Germ Tube Test for Candida albicans: The **germ tube test** (Reynolds-Braude phenomenon) is a rapid, presumptive test to identify **Candida albicans**. When incubated in human serum at 37°C for 2–3 hours, C. albicans produces short, thin filamentous outgrowths (germ tubes) directly from the yeast cells **without a constriction** at the point of origin (distinguishing it from pseudohyphae). Approximately 95% of C. albicans strains (and C. dubliniensis) produce germ tubes; other Candida species (tropicalis, glabrata, parapsilosis, krusei) are germ tube negative.

Key Point: C. albicans is the most common cause of oral thrush (white curd-like plaques), vaginal candidiasis (cottage-cheese discharge), oesophageal candidiasis (AIDS indicator disease < CD4 200), and invasive candidiasis in neutropenic patients. KOH mount: pseudohyphae + blastoconidia. On Dalmau plate (cornmeal agar): chlamydoconidia (thick-walled, spherical terminal spores) – also pathognomonic. Treatment: fluconazole (susceptible), or echinocandins/amphotericin B

(for azole-resistant/invasive disease).

Why other options are wrong:

- Option A: C. tropicalis is germ tube negative; it forms pseudohyphae and is associated with invasive candidiasis in leukaemia.
- Option B: C. glabrata is germ tube negative; it is intrinsically resistant to fluconazole; causes UTIs and bloodstream infections in elderly/immunocompromised; does not form true hyphae or pseudohyphae.
- Option D: C. krusei is germ tube negative; it is intrinsically resistant to fluconazole; associated with bone marrow transplant patients.

Final Answer: Positive germ tube test (serum, 37°C, 3h) $\Rightarrow$ $\boxed{C}$ Candida albicans

Answer: (C) Go Back to Q39

Q40.

Solution

Concept – Aspergillus fumigatus: Invasive Aspergillosis and Galactomannan: Aspergillus fumigatus is the most common cause of invasive pulmonary aspergillosis in immunocompromised hosts (neutropenia, bone marrow transplant, solid organ transplant, prolonged corticosteroid use). The **galactomannan assay** detects galactomannan, a polysaccharide component of the Aspergillus cell wall released during hyphal invasion, in serum or BAL fluid. It is an early, non-invasive biomarker for invasive aspergillosis (sensitivity 71–80%, specificity 85–90%).

Key Point: Radiology of invasive aspergillosis: **halo sign** (early – consolidation surrounded by ground-glass haze of haemorrhage), **air-crescent sign** (late – crescent of air around a necrotic nodule as neutrophil recovery begins). Aspergillus also causes: allergic bronchopulmonary aspergillosis (ABPA) in asthma/CF, aspergilloma ("fungus ball" in pre-existing lung cavity), chronic pulmonary aspergillosis. A. fumigatus: hyaline, septate hyphae with Y-shaped (dichotomous, 45°) branching on histology (Gomori methenamine silver stain). The (1,3)-β-D-glucan test is less specific (positive in Aspergillus, Candida, Pneumocystis).

Why other options are wrong:

- Option A: Candida albicans rarely causes primary pulmonary infection with halo sign/air-crescent; (1,3)-β-D-glucan is positive but less specific.
- Option B: Cryptococcus neoformans causes meningitis (India ink shows large polysaccharide capsule) and pulmonary cryptococcosis; diagnosed by cryptococcal antigen lateral flow assay – not galactomannan.

- Option C: Mucor species (Mucormycosis/Zygomycosis) cause angioinvasive pulmonary disease (reverse halo/atoll sign), especially in diabetics and haematological malignancy; histology shows non-septate broad ribbon-like hyphae with wide-angle branching; galactomannan is negative in mucormycosis.

Final Answer: Immunocompromised + halo sign + air-crescent + galactomannan positive $\Rightarrow \boxed{D}$ Aspergillus fumigatus

Answer: (D) Go Back to Q40

Answer Key – FMGE Microbiology Sample Paper 1

1	A	2	B	3	C	4	D	5	A
6	B	7	C	8	D	9	A	10	B
11	C	12	D	13	A	14	B	15	C
16	D	17	A	18	B	19	C	20	D
21	A	22	B	23	C	24	D	25	A
26	B	27	C	28	D	29	A	30	B
31	C	32	D	33	A	34	B	35	C
36	D	37	A	38	B	39	C	40	D