FMGE Microbiology Sample Paper – 4

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

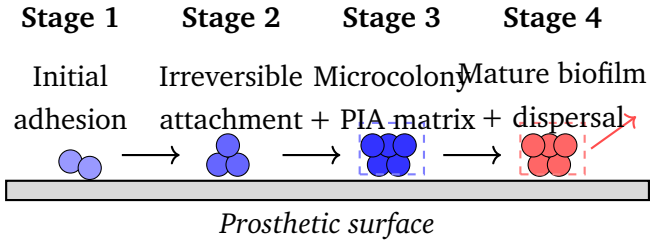
Q1. A 68-year-old man with a prosthetic hip joint presents with chronic, low-grade joint pain and fever 18 months after arthroplasty. Synovial fluid culture grows coagulase-negative staphylococci resistant to multiple antibiotics. The organism's ability to cause this indolent infection is primarily attributed to which of the following?

(A) Production of exotoxins such as TSST-1

(B) Intracellular survival within macrophages

(C) Capsule formation preventing opsonisation

(D) Biofilm formation on prosthetic surfaces



Q2. A 12-hour-old neonate is brought to the NICU with respiratory distress, bulging fontanelle, and temperature instability. CSF analysis reveals a turbid fluid with elevated protein, low glucose, and gram-positive cocci in pairs and chains. Blood cultures also grow the same organism. Which of the following characteristics is most consistent with the causative pathogen?

(A) CAMP test positive, hydrolyses hippurate, group B Lancefield antigen

(B) Optochin sensitive, bile soluble, alpha-haemolytic

(C) Bacitracin sensitive, group A Lancefield antigen

(D) Bile-esculin positive, grows in 6.5% NaCl broth

Q3. A 22-year-old student returning from South Asia presents with stepwise fever for 10 days, relative bradycardia, and rose spots on the trunk. Widal test results: anti-O titre 1:160, anti-H titre 1:320. The physician plans to repeat the test after 7 days. Which statement best describes the diagnostic utility of the Widal test?

(A) Anti-H antibodies are more specific than anti-O antibodies for active typhoid

(B) A rising titre in serial samples is more diagnostic than a single elevated reading

(C) Anti-O titre of 1:40 is sufficient for diagnosis in endemic regions

(D) Widal test remains positive for life and cannot indicate active disease

Q4. A 35-year-old woman develops mild, self-limited bloody diarrhoea after eating at a daycare facility. Stool culture grows a non-motile, lactose-non-fermenting, gram-negative rod that does not produce H_2S. Biochemical testing shows it belongs to serogroup D with a single antigen (phase I only). Which species is most likely responsible?

(A) *Shigella dysenteriae*

(B) *Shigella flexneri*

(C) *Shigella sonnei*

(D) *Shigella boydii*

Q5. A 38-year-old man with HIV infection and a CD4 count of 35 cells/μL presents with three weeks of fever, night sweats, weight loss, and abdominal pain. Blood cultures on Middlebrook 7H10 medium grow acid-fast rods after 2 weeks. Bone marrow biopsy shows foamy macrophages packed with acid-fast organisms. Which of the following is the most appropriate prophylactic agent to prevent this infection?

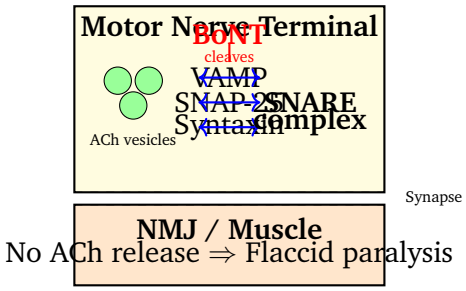
(A) Fluconazole

(B) Isoniazid

(C) TMP-SMX

(D) Azithromycin

Q6. A 6-month-old infant presents with 2 days of poor feeding, weak cry, hypotonia, and progressive descending paralysis. The mother reports that honey was added to the infant's food at 5 months. Cranial nerve palsies are noted. Which of the following best explains the mechanism of action of the toxin responsible?



(A) Cleavage of SNARE proteins (VAMP/SNAP-25/syntaxin) blocking acetylcholine vesicle fusion

(B) Inhibition of acetylcholinesterase at the neuromuscular junction

(C) Blockade of nicotinic acetylcholine receptors on muscle

(D) ADP-ribosylation of Gs protein causing excess cAMP

Q7. A 28-year-old man develops profuse watery diarrhoea, nausea, and abdominal cramps 12 hours after eating raw oysters at a coastal restaurant. He is afebrile and recovers without antibiotics within 3 days. Stool

culture on TCBS agar grows blue-green colonies. The Kanagawa phenomenon is positive. Which organism is responsible?

(A) *Vibrio cholerae* O1

(B) *Vibrio parahaemolyticus*

(C) *Vibrio vulnificus*

(D) *Aeromonas hydrophila*

Q8. A 45-year-old woman is screened for syphilis during routine antenatal care. Her VDRL is reactive at 1:4. The clinician orders a confirmatory treponemal test. The patient provides a history of adequately treated syphilis 12 years ago. Which of the following statements best describes the expected serological finding?

(A) TPHA will be non-reactive since the infection was treated

(B) VDRL is more specific than TPHA for confirming syphilis

(C) TPHA (treponemal test) remains positive for life even after successful treatment

(D) Both VDRL and TPHA will return to non-reactive within 2 years of treatment

Q9. A 25-year-old sexually active man presents with a painful, soft genital ulcer with ragged undermined edges and unilateral tender inguinal lymphadenopathy (bubo) for 1 week. There is no induration. Gram stain of ulcer exudate shows gram-negative coccobacilli arranged in parallel chains ("school of fish" pattern). What is the most likely diagnosis?

(A) Primary syphilis (Treponema pallidum)

(B) Lymphogranuloma venereum (Chlamydia trachomatis L1-L3)

(C) Granuloma inguinale (Klebsiella granulomatis)

(D) Chancroid (Haemophilus ducreyi)

Q10. A 14-year-old boy presents with unilateral tender axillary lymphadenopathy for 3 weeks. He has a papule at the site of a cat scratch on his forearm

that occurred 4 weeks ago. Temperature is 38.1°C. Lymph node biopsy with Warthin-Starry silver stain reveals pleomorphic gram-negative bacilli within the lymph node parenchyma. Which organism is responsible?

(A) *Bartonella henselae*

(B) *Yersinia pestis*

(C) *Francisella tularensis*

(D) *Brucella melitensis*

Q11. A 52-year-old hiker returns from a forested area in the southeastern United States with fever, myalgia, headache, leukopenia, thrombocytopenia, and elevated liver enzymes. Blood smear shows intracytoplasmic inclusions within monocytes. He does not have a rash. Serology confirms the diagnosis. Which of the following is the most appropriate treatment?

(A) Ciprofloxacin

(B) Doxycycline

(C) Azithromycin

(D) Ceftriaxone

Q12. A 67-year-old man on a ventilator in the ICU for 3 weeks develops new fever, purulent secretions, and worsening oxygenation. Bronchoalveolar lavage culture grows a glucose non-fermenting gram-negative rod that is resistant to carbapenems, β-lactams, fluoroquinolones, and aminoglycosides. VITEK identification is *Stenotrophomonas maltophilia*. Which antibiotic is most reliably active?

(A) Meropenem

(B) Colistin

(C) Trimethoprim-sulfamethoxazole (TMP-SMX)

(D) Cefepime

Q13. A 55-year-old man who underwent splenectomy 5 years ago presents to the emergency department in septic shock 48 hours after a dog bite on his hand. He develops purpura fulminans and DIC. Blood cultures grow long, fusiform gram-negative rods that are oxidase-positive and catalase-positive, growing best in a CO_2-enriched environment. Mortality approaches 30% without treatment. Which organism is the most likely causative agent?

(A) *Pasteurella multocida*

(B) *Eikenella corrodens*

(C) *Bacteroides fragilis*

(D) *Capnocytophaga canimorsus*

Q14. A 17-year-old boy with severe localised periodontitis is referred to a cardiologist after developing fever and a new heart murmur. Echocardiography confirms aortic valve vegetation. Blood cultures after 5 days grow a fastidious gram-negative coccobacillus that requires CO_2 and produces leukotoxin targeting neutrophils. This organism also causes localised aggressive periodontitis (formerly juvenile periodontitis). Which organism is responsible?

(A) *Aggregatibacter actinomycetemcomitans*

(B) *Haemophilus aphrophilus*

(C) *Cardiobacterium hominis*

(D) *Eikenella corrodens*

Q15. A researcher is studying the structure of IgG antibodies. She digests an IgG molecule with papain and isolates the Fab fragments. Which of the following best describes the functional significance of the Fab region compared with the Fc region?

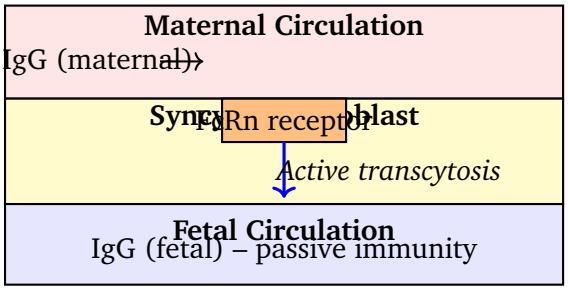
(A) Fc region determines antigen specificity through its hypervariable loops

(B) Fab region (VH + VL) forms the antigen-binding site; Fc region binds C1q and Fc receptors

(C) Fab region mediates ADCC by binding FcγRIII on NK cells

(D) Fc region determines antibody isotype switching in the germinal centre

Q16. A newborn delivered at 36 weeks is found to have detectable maternal IgG antibodies in cord blood. The neonatologist explains that active transcytosis of IgG across the placenta is mediated by a specific receptor. Which receptor mediates this transfer, and on which cell type does it function?



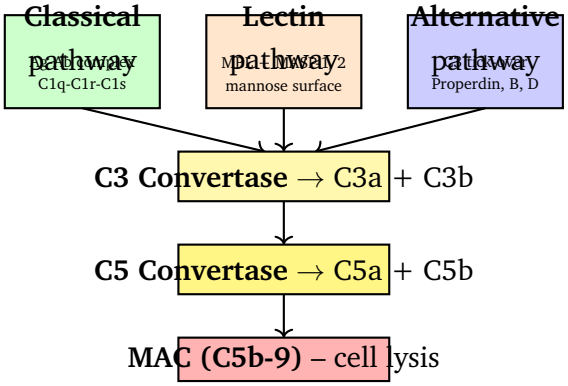
(A) FcγRIII on trophoblast dendritic cells

(B) Poly-Ig receptor on columnar epithelium

(C) FcRn (neonatal Fc receptor) on syncytiotrophoblast

(D) FcγRI (CD64) on placental macrophages

Q17. A 3-year-old child with recurrent encapsulated bacterial infections is found to have low MBL (mannose-binding lectin) levels. The complement pathway that is deficient in this child activates which of the following sequence of proteins?



(A) C1q → C1r → C1s → C4 → C2

(B) C3 → Factor B → Factor D → Properdin

(C) C5 $\rightarrow$ C6 $\rightarrow$ C7 $\rightarrow$ C8 $\rightarrow$ C9

(D) MBL $\rightarrow$ MASP-1/MASP-2 $\rightarrow$ C4 $\rightarrow$ C2 $\rightarrow$ C3 convertase (C4b2a)

Q18. A researcher studying innate immunity observes that NK cells can kill certain tumour cells that have downregulated MHC class I expression to evade cytotoxic T cells. Which of the following best explains the mechanism by which NK cells recognise and kill these MHC class I-deficient tumour cells?

(A) Loss of inhibitory KIR signal ("missing self") combined with activating NKG2D binding to stress ligands (MICA/MICB) triggers NK killing

(B) NK cells require prior sensitisation with specific antigen to kill target cells

(C) Tumour cells are killed only through antibody-dependent cellular cytotoxicity (ADCC)

(D) MHC class I downregulation directly activates CD8+ T cells to help NK cells

Q19. A 55-year-old woman with severe rheumatoid arthritis fails methotrexate therapy. Her physician plans to start a biologic agent targeting a cytokine that is primarily produced by activated macrophages and causes fever, cachexia, and septic shock in excess. Antibodies against this cytokine are also used in Crohn disease and plaque psoriasis. Which cytokine is being targeted?

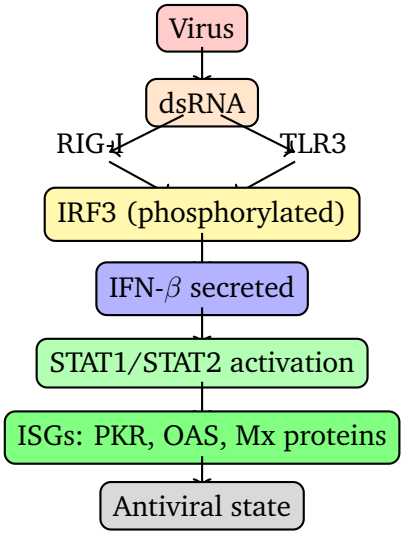
(A) IL-6

(B) TNF-α

(C) IL-1β

(D) IL-17A

Q20. A patient with recurrent viral encephalitis is found to have a loss-of-function mutation in IRF3 (interferon regulatory factor 3). Which antiviral defence pathway is most directly impaired in this patient?



(A) Activation of the complement classical pathway

(B) TNF-α production by macrophages

(C) Type I interferon (IFN-α/β) production via RIG-I/TLR3 $\rightarrow$ IRF3 pathway

(D) IL-12 driven Th1 differentiation

Q21. A 62-year-old man with severe COVID-19 pneumonia develops rapidly worsening hypoxia, elevated CRP (280 mg/L), serum ferritin >10,000 μg/L, and fibrinogen 8.5 g/L. The intensivist considers adding tocilizumab (anti-IL-6 receptor antibody) to his treatment. Which of the following best describes the role of the targeted cytokine?

(A) Direct viral cytopathic effect amplified by IL-6

(B) IL-6 promotes CD8+ cytotoxic T-cell exhaustion

(C) IL-6 inhibits fever and acute phase protein synthesis

(D) IL-6 drives the acute phase response (CRP, fibrinogen), fever, and plasma cell differentiation; dysregulated IL-6 causes cytokine storm

Q22. A mouse model is created in which the thymic cortex is ablated while the medulla is preserved. Which of the following processes will be most significantly impaired as a result?

(A) Positive selection of T cells that recognise self-MHC (occurs in the cortex)

(B) Negative selection of autoreactive T cells (clonal deletion in the medulla)

(C) B-cell class switching in germinal centres

(D) NK cell maturation in the bone marrow

Q23. A patient receives a tumour-specific peptide vaccine. Researchers observe that exogenous tumour antigens presented by dendritic cells activate CD8+ cytotoxic T lymphocytes. This process involves loading exogenous peptides onto which MHC molecule, and what is the term for this mechanism?

(A) MHC class II; conventional antigen presentation

(B) MHC class I; cross-presentation (cross-priming)

(C) MHC class II; superantigen stimulation

(D) MHC class I; direct allorecognition

Q24. A vaccinologist is developing a new subunit vaccine. She adds aluminium hydroxide (alum) to improve immunogenicity. Which of the following best explains the mechanism by which alum acts as an adjuvant?

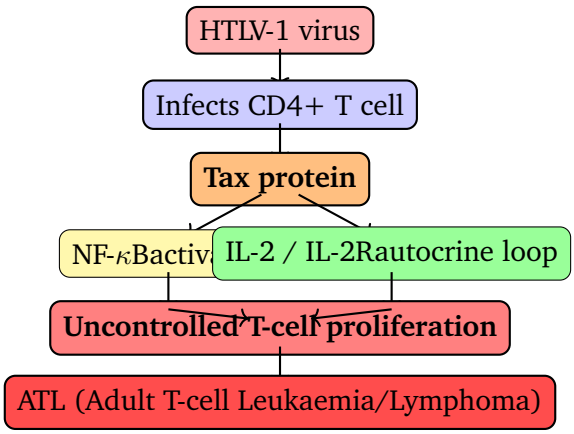
(A) Alum directly binds TLR4 on B cells, inducing polyclonal activation

(B) Alum promotes Th1 responses and IL-12 production by macrophages

(C) Alum creates a depot effect and activates the NLRP3 inflammasome, promoting Th2/IgE responses

(D) Alum inhibits regulatory T cells, preventing immune tolerance

Q25. A 45-year-old Jamaican immigrant presents with skin lesions, hypercalcaemia, lymphadenopathy, and abnormal lymphocytes with “flower cell” morphology on peripheral blood smear. He also reports progressive lower limb weakness and bladder dysfunction (tropical spastic paraparesis). HTLV-1 serology is positive. Which viral protein is responsible for dysregulating the cell cycle leading to T-cell malignancy?



(A) Env (envelope protein) binds CD4 receptor

(B) Rex protein promotes nuclear export of unspliced mRNA

(C) Gag polyprotein mediates viral budding

(D) Tax protein activates NF-κB and drives IL-2/IL-2R autocrine loop

Q26. A 30-year-old farmer in New Mexico develops fever, myalgia, and rapidly progressive non-cardiogenic pulmonary oedema requiring ICU admission. He recently cleaned a barn with rodent droppings. There is no history of animal bites. Serology confirms hantavirus pulmonary syndrome. Which of the following best characterises this infection?

(A) Sin Nombre virus carried by deer mice; transmission via inhalation of rodent excreta; no person-to-person spread

(B) Andes virus; transmitted by tick bite in South America; person-to-person transmission common

(C) Seoul virus; transmitted by rat bite; causes haemorrhagic fever with renal syndrome globally

(D) Hantaan virus; transmitted by person-to-person contact in Asia

Q27. A 28-year-old healthcare worker is evacuated from West Africa after developing haemorrhagic fever. She presents with high fever, vomiting, bloody diarrhoea, conjunctival injection, and petechiae. Coagulation studies show DIC. An experimental monoclonal antibody cocktail is initiated. Which of the following best describes the virological characteristics of the causative agent?

(A) Flavivirus with positive-sense RNA; transmitted by Aedes mosquito

(B) Filovirus with negative-sense ssRNA; VP35 protein blocks interferon response; Zaire species is most virulent

(C) Arenavirus with ambisense RNA; rodent reservoir; causes Lassa fever

(D) Bunyavirus; transmitted by sandfly; causes Crimean-Congo haemorrhagic fever

Q28. An outbreak of severe encephalitis with respiratory involvement occurs among pig farmers in a South Asian country. Several cases have occurred in healthcare workers caring for the patients, suggesting person-to-person spread. Pteropus fruit bats are identified as the reservoir. Which pathogen is responsible?

(A) Japanese encephalitis virus

(B) Hendra virus

(C) Nipah virus

(D) Rabies virus

Q29. A 25-year-old woman in her second trimester develops a mild febrile illness with rash, conjunctivitis, and joint pain after returning from Brazil. Fetal ultrasound 4 weeks later shows microcephaly and intracranial calcifications. Which of the following best characterises the causative virus?

(A) Rubella virus; togavirus; causes periventricular calcifications; prevented by MMR vaccination

(B) Cytomegalovirus; causes periventricular calcifications; transmitted via urine/saliva

(C) Herpes simplex virus type 2; transmitted intrapartum; causes neonatal encephalitis

(D) Zika virus; Aedes aegypti; flavivirus; causes microcephaly and congenital Zika syndrome; sexual transmission possible

Q30. A 38-year-old woman returning from Kerala, India presents with sudden-onset high fever, severe symmetric polyarthralgia affecting small joints of

both hands and feet, and a maculopapular rash. Joint pain is so severe she adopts a bent-over posture. She remains febrile for 5 days. There is no effective antiviral or vaccine currently available. Which virus is responsible?

(A) Chikungunya virus (Alphavirus, Togaviridae)

(B) Dengue virus (Flavivirus)

(C) Ross River virus (Alphavirus, Australasia)

(D) Sindbis virus (Alphavirus)

Q31. An outbreak of acute hepatitis affects 150 residents of a flood-affected village with contaminated drinking water. Jaundice, anorexia, and right upper quadrant pain are the main features. Serology is negative for HAV IgM, HBsAg, and anti-HCV. Three pregnant women in the third trimester develop acute liver failure with 25% mortality. Which hepatitis virus is responsible?

(A) Hepatitis A virus (HAV)

(B) Hepatitis E virus (HEV)

(C) Hepatitis B virus (HBV)

(D) Hepatitis C virus (HCV)

Q32. A 35-year-old intravenous drug user who is a known chronic HBV carrier (HBsAg positive for 4 years) suddenly deteriorates with severe acute hepatitis. Anti-HDV IgM is detected. Which of the following statements best explains why HDV requires HBV for its life cycle?

(A) HDV uses HBV polymerase for RNA replication

(B) HDV requires HBV core antigen (HBcAg) for nucleocapsid formation

(C) HDV requires HBsAg to form its envelope for viral assembly and release

(D) HDV integrates into the host genome using HBV reverse transcriptase

Q33. A 32-year-old aid worker returning from West Africa develops cyclical fever every 48 hours, 8 months after completing chloroquine prophylaxis. Blood smear shows oval/fimbriated erythrocytes with prominent Schüffner's dots. The parasitised RBCs are slightly enlarged and oval. He has had two previous episodes that resolved spontaneously. Which species is the most likely cause?

(A) *Plasmodium falciparum*

(B) *Plasmodium malariae*

(C) *Plasmodium vivax*

(D) *Plasmodium ovale*

Q34. A 40-year-old man returning from India presents with right upper quadrant pain and fever. Ultrasound shows a single 8 cm liver abscess. Aspiration yields "anchovy sauce" reddish-brown pus that is sterile on bacterial culture. Stool examination reveals trophozoites with ingested red blood cells. Which feature of this trophozoite definitively distinguishes the pathogenic species from *Entamoeba dispar*?

(A) Erythrophagocytosis (engulfed RBCs visible in trophozoites)

(B) Presence of four nuclei in the cyst form

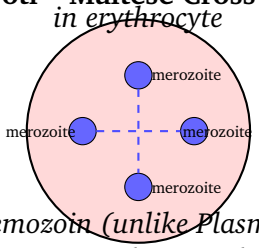
(C) Peripheral chromatin margination on nuclear membrane

(D) Production of galactose-inhibitable lectin surface protein

Q35. A 65-year-old asplenic man develops fever, haemolytic anaemia, and haemoglobinuria 2 weeks after a tick bite in coastal New England. Peripheral blood smear shows intraerythrocytic ring forms including a tetrad arrangement ("Maltese cross"). No malarial pigment (haemozoin) is seen. Which organism is responsible?

Babesia microti – Maltese Cross (Tetrad form)

in erythrocyte



No haemozoin (unlike Plasmodium)

Vector: *Ixodes scapularis*

(A) *Plasmodium vivax*

(B) *Babesia microti*

(C) *Plasmodium falciparum*

(D) *Theileria species*

Q36. A 52-year-old Latin American immigrant is found to have cardiomegaly, an apical aneurysm, and right bundle branch block on ECG. Colonoscopy reveals dilated megacolon. He grew up in a rural area of Brazil. Serology for *Trypanosoma cruzi* is positive. Which of the following best describes the pathogenesis and vector of this disease?

(A) Tsetse fly transmits trypomastigotes causing sleeping sickness (HAT)

(B) Reduviid (kissing) bug transmits trypomastigotes; amastigotes destroy cardiac and smooth muscle

(C) Sandfly transmits promastigotes causing cutaneous or visceral leishmaniasis

(D) *Trypanosoma cruzi* is transmitted by blood transfusion only; no insect vector

Q37. A 45-year-old Polish man develops fever, periorbital oedema, severe myalgia, and marked eosinophilia (35%) after eating homemade pork sausage at a village gathering. Muscle biopsy shows encysted larvae within skeletal muscle fibres surrounded by a collagen capsule (nurse cell). Which organism is responsible?

(A) *Toxocara canis*

(B) *Dracunculus medinensis*

(C) *Wuchereria bancrofti*

(D) *Trichinella spiralis*

Q38. A 58-year-old man with polymyalgia rheumatica on long-term prednisone (40 mg/day) presents with abdominal pain, diarrhoea, and gram-negative bacteraemia. CT abdomen shows thickened small bowel loops. Stool examination reveals rhabditiform and filariform (L3) larvae. Which of the following best explains the mechanism of this complication?

(A) Immunosuppression allows rapid autoinfection cycle of *Strongyloides stercoralis*, with hyperinfection and larvae carrying gut bacteria into the bloodstream

(B) *Ascaris lumbricoides* migrates to the biliary tree in immunocompromised patients

(C) *Hookworm* disseminates haematogenously in steroid-treated patients

(D) *Strongyloides stercoralis* secretes immunosuppressive factors that mimic corticosteroids

Q39. A 35-year-old gardener presents with a painless nodular ulcer on the right index finger that developed 3 weeks after a rose thorn prick. Over the next 2 weeks, additional nodular lesions have appeared in a linear fashion along the lymphatics of the forearm (nodular lymphangitis, "sporotrichoid spread"). Biopsy grows a dimorphic fungus at 25°C showing conidia arranged in a "daisy" (rosette) pattern. What is the treatment of choice for localised cutaneous sporotrichosis?

(A) Fluconazole

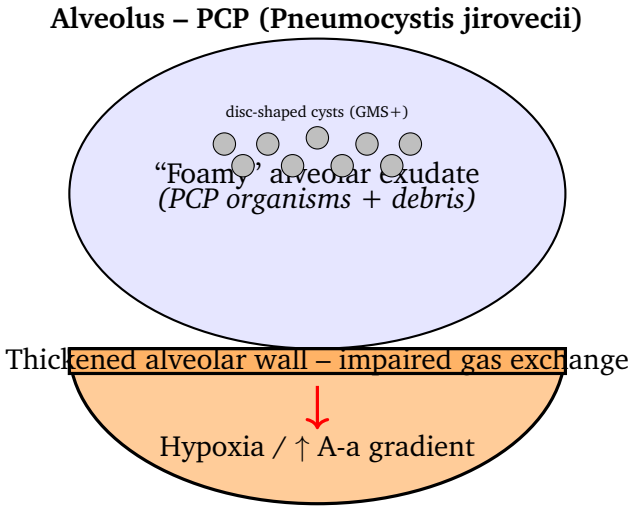
(B) Saturated potassium iodide solution (SSKI) or itraconazole

(C) Amphotericin B

(D) Voriconazole

Q40. A 34-year-old HIV-positive man with a CD4 count of 45 cells/μL presents with 3 weeks of progressive exertional dyspnoea, dry cough, and low-grade fever. Chest X-ray shows bilateral perihilar ground-glass infiltrates.

LDH is 820 IU/L. Bronchoscopic BAL with Gomori methenamine silver stain shows disc-shaped cysts with intracystic bodies. What is the first-line treatment and the CD4 threshold for primary prophylaxis?



(A) Pentamidine IV; CD4 <100 for prophylaxis

(B) Atovaquone; CD4 <150 for prophylaxis

(C) TMP-SMX (trimethoprim-sulfamethoxazole); CD4 <200 for prophylaxis

(D) Clindamycin + primaquine; CD4 <100 for prophylaxis

Detailed Solutions

Q1.

Solution

Concept – Biofilm and Prosthetic Device Infections: *Staphylococcus epidermidis* is the leading cause of prosthetic joint and implant infections. Its primary virulence mechanism is **biofilm formation** — a structured community of bacteria encased in polysaccharide intercellular adhesin (PIA). Biofilm renders organisms resistant to antibiotics (altered metabolism, diffusion barrier) and protects against host immune clearance.

Key Point: Biofilm formation occurs in four stages: (1) initial reversible adhesion via hydrophobic interactions; (2) irreversible attachment through surface proteins (AtlE, Aap); (3) microcolony formation and PIA matrix production (icaADBC operon); (4) mature biofilm with dispersal of organisms to seed new sites. Bacteria in mature biofilm are metabolically quiescent, evading antibiotics that target active cell division.

Why other options are wrong:

- Option A: *S. epidermidis* does not produce significant exotoxins (unlike *S. aureus*: TSST-1, exfoliatin, Panton-Valentine leukocidin).
- Option B: Intracellular survival is characteristic of *Mycobacterium*, *Salmonella*, *Listeria*, *Brucella* — not coagulase-negative staphylococci.
- Option C: Capsule is a virulence factor for *S. pneumoniae*, *Klebsiella*, and *H. influenzae* type b; not the primary mechanism in prosthetic device infection.

Final Answer: Coagulase-negative staph + prosthetic joint + chronic indolent infection + multidrug resistance $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q1

Q2.

Solution

Concept – Group B Streptococcus (GBS) Neonatal Sepsis: *Streptococcus agalactiae* (Group B Strep, GBS) is the most common cause of neonatal meningitis and early-onset sepsis (within 7 days of birth). It colonises the maternal vaginal and rectal flora in 10–30% of women; vertical transmission occurs during delivery.

Key identifying features of GBS:

- **CAMP test positive**: GBS secretes CAMP factor, which enhances the lysis of

sheep RBCs by *S. aureus* beta-haemolysin, producing a characteristic arrowhead haemolysis zone.

- **Hippurate hydrolysis positive**: GBS hydrolyses sodium hippurate (distinguishes it from other beta-haemolytic streptococci).
- **Lancefield Group B antigen**: due to surface polysaccharide; detected by latex agglutination.
- Beta-haemolytic on blood agar; bacitracin resistant (unlike Group A strep).

Why other options are wrong:

- Option B: Optochin sensitive + bile soluble + alpha-haemolysis = *S. pneumoniae*.
- Option C: Bacitracin sensitive + Group A = *S. pyogenes* (causes scarlet fever, pharyngitis — not neonatal meningitis classically).
- Option D: Bile-esculin positive + 6.5% NaCl growth = *Enterococcus* spp.

Final Answer: Neonatal meningitis/sepsis + CAMP positive + hippurate hydrolysis + Group B $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – Widal Test Interpretation in Typhoid: The Widal test detects agglutinating antibodies against *Salmonella typhi* O (somatic) and H (flagellar) antigens. It is an indirect test with significant limitations.

Key interpretive rules:

- **Anti-O antibodies** appear first, indicate active infection, and fall with recovery — more specific.
- **Anti-H antibodies** appear later and persist longer; are elevated post-vaccination and post-prior infection (anamnestic/booster effect) — less specific for active disease.
- **Single reading cut-offs** (O $\geq$ 1:80–1:160, H $\geq$ 1:160) are unreliable in endemic areas due to background positivity.
- **A fourfold rise in titre** in paired sera (7–10 days apart) is the most diagnostically significant finding.

Why other options are wrong:

- Option A: Anti-H is less specific (persists long, boosted by prior vaccination/infection).
- Option C: 1:40 is sub-threshold and non-diagnostic even in endemic areas.
- Option D: Widal positivity does not last for life; it wanes — unlike treponemal tests.

Final Answer: Rising serial Widal titre is most diagnostically significant $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – Shigella Species and Serogroups: The four species of *Shigella* are classified by serogroup: A (*S. dysenteriae*), B (*S. flexneri*), C (*S. boydii*), D (*S. sonnei*). All are non-motile (no H antigen) and lactose non-fermenters.

Shigella sonnei (Group D) distinctive features:

- **Most common** *Shigella* species in **developed countries** (USA, UK, Europe).
- Causes mild, self-limited dysentery; rarely requires antibiotics in immunocompetent hosts.
- Has only **one phase antigen (phase I only)** — single antigen structure (unlike other Shigella species).
- ONPG test positive (late lactose fermenter); produces β-galactosidase.
- Does not produce Shiga toxin (unlike *S. dysenteriae* type 1).

Why other options are wrong:

- Option A: *S. dysenteriae* (Group A): produces Shiga toxin; causes most severe dysentery; common in Asia/Africa.
- Option B: *S. flexneri* (Group B): most common in developing countries; multiple serotypes.
- Option D: *S. boydii* (Group C): rare; found mainly in the Indian subcontinent.

Final Answer: Developed country + mild dysentery + single antigen (non-motile) + serogroup D $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q4

Q5.

Solution

Concept – Disseminated Mycobacterium avium Complex (MAC) in AIDS: MAC (comprising *M. avium* and *M. intracellulare*) is an environmental atypical mycobacterium that causes disseminated infection when CD4 count falls below 50 cells/μL. It is ubiquitous in water and soil; infection is by inhalation/ingestion.

Clinical features of disseminated MAC: Fever, night sweats, weight loss ("B symptoms"), abdominal pain, hepatosplenomegaly, anaemia, elevated ALP. Blood culture on Middlebrook 7H10 or BACTEC system grows acid-fast rods. Bone marrow/liver biopsy shows foamy macrophages laden with acid-fast bacilli.

Prophylaxis and treatment:

- **Primary prophylaxis**: **Azithromycin** 1200 mg weekly (or clarithromycin 500 mg BD) when CD4 $<$ 50 cells/μL.
- **Treatment**: Clarithromycin + ethambutol $\pm$ rifabutin.

Why other options are wrong:

- Option A: Fluconazole prevents cryptococcal meningitis and candidiasis.
- Option B: Isoniazid is used for latent TB prophylaxis; not MAC.
- Option C: TMP-SMX prevents *Pneumocystis jirovecii* pneumonia (PCP) and toxoplasmosis; not MAC.

Final Answer: HIV + CD4 $<$ 50 + disseminated MAC $\Rightarrow$ prophylaxis: $\boxed{D}$ (Azithromycin)

Answer: (D) Go Back to Q5

Q6.

Solution

Concept – Botulinum Toxin Mechanism: *Clostridium botulinum* produces botulinum neurotoxin (BoNT), a zinc-dependent metalloprotease. It is the most potent biological toxin known. In infant botulism (the most common form in developed countries), ingested spores germinate in the immature gut, producing toxin *in situ*.

Mechanism of action – SNARE cleavage:

- BoNT is taken up at the neuromuscular junction via endocytosis.
- The light chain (metalloprotease) cleaves **SNARE proteins**: VAMP (synapto-

brevin), SNAP-25, or syntaxin — depending on serotype (A, E cleave SNAP-25; B, D, F, G cleave VAMP; C cleaves both syntaxin and SNAP-25).
- Cleaved SNARE proteins cannot form the fusion complex, blocking acetylcholine vesicle docking and exocytosis.
- Result: **flaccid paralysis** (descending, beginning with cranial nerves: diplopia, dysarthria, dysphagia) with autonomic dysfunction; **no fever, sensory intact**.

Why other options are wrong:

- Option B: AChE inhibition causes spastic/cholinergic crisis (organophosphate poisoning).
- Option C: Nicotinic receptor blockade = non-depolarising neuromuscular blockers (curare); different mechanism.
- Option D: ADP-ribosylation of Gs = cholera toxin / pertussis toxin mechanism.

Final Answer: Infant botulism + honey + descending flaccid paralysis + SNARE cleavage $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q6

Q7.

Solution

Concept – Vibrio parahaemolyticus Seafood Gastroenteritis: *V. parahaemolyticus* is the most common cause of seafood-associated gastroenteritis worldwide, particularly associated with raw or undercooked shellfish, oysters, and sushi. It is a marine organism found in warm coastal waters.

Key features:

- **TCBS agar**: produces **blue-green colonies** (sucrose non-fermenter; unlike *V. cholerae* which produces yellow colonies on TCBS).
- **Kanagawa phenomenon**: production of thermostable direct haemolysin (TDH) causing beta-haemolysis on special Wagatsuma agar; marker of pathogenicity.
- Incubation: 12–24 hours; self-limited watery or bloody diarrhoea; no treatment usually required.
- Halophilic (requires NaCl for growth).

Why other options are wrong:

- Option A: *V. cholerae* O1 produces rice-water diarrhoea via cholera toxin; yellow colonies on TCBS.
- Option C: *V. vulnificus*: causes wound infections and primary septicaemia; high mortality in liver disease patients; not classic food poisoning.
- Option D: *Aeromonas hydrophila*: freshwater organism, not marine/seafood-associated.

Final Answer: Oysters/sushi + blue-green TCBS colonies + Kanagawa+ + self-limited $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q7

Q8.

Solution

Concept – Syphilis Serology: Treponemal vs Non-Treponemal Tests: Syphilis serology uses two categories of tests: non-treponemal (VDRL, RPR) and treponemal (TPHA, FTA-ABS, TPPA).

Key distinctions:

- **Non-treponemal (VDRL/RPR)**: detect cardiolipin antibodies; titres correlate with disease activity; fall and become non-reactive with successful treatment; used to monitor treatment response.
- **Treponemal (TPHA/FTA-ABS)**: detect antibodies specifically against *T. pallidum* antigens; **remain positive for life** even after successful treatment ("serofast"); used for confirmation, not monitoring.

Clinical interpretation: A reactive TPHA in a patient with a past history of treated syphilis is expected and *does not indicate* active disease — it is the VDRL titre and clinical context that guide management.

Why other options are wrong:

- Option A: Incorrect — TPHA remains positive lifelong regardless of treatment.
- Option B: TPHA is more specific; VDRL has false positives (SLE, pregnancy, EBV, malaria).
- Option D: VDRL returns to non-reactive; TPHA does not.

Final Answer: TPHA stays positive for life even after treatment $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q8

Q9.

Solution

Concept – Chancroid (Haemophilus ducreyi): *H. ducreyi* causes chancroid, a sexually transmitted infection characterised by a painful genital ulcer — in contrast to syphilitic chancre which is painless.

Classic triad of chancroid:

- **Painful** soft genital ulcer with ragged undermined edges ("soft sore")
- **Tender unilateral inguinal lymphadenopathy (bubo)** — may suppurate
- **No induration** (unlike primary syphilis)

Gram stain of ulcer discharge: gram-negative coccobacilli arranged in chains, like a "**school of fish**" or "railroad track" pattern. Culture on chocolate agar with vancomycin (requires X factor, not V factor; fastidious).

Why other options are wrong:

- Option A: Primary syphilis = painless, indurated chancre; spirochaete detected by dark-field microscopy.
- Option B: LGV (*C. trachomatis* L1-L3): small painless papule; massive tender lymphadenopathy; "groove sign"; no exudate for Gram stain.
- Option C: Granuloma inguinale (donovanosis): painless, beefy red ulcer; Donovan bodies (bipolar staining); no lymphadenopathy.

Final Answer: Painful ulcer + tender bubo + school-of-fish Gram stain $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – Cat Scratch Disease (Bartonella henselae): *Bartonella henselae* is transmitted via scratch or bite from bacteraemic cats (especially kittens); cat fleas (Ctenocephalides felis) maintain the bacteraemic cycle among cats.

Clinical features:

- **Primary lesion**: papule/pustule at scratch site (3–10 days post-scratch)
- **Regional lymphadenopathy**: tender, unilateral; develops 1–3 weeks after primary lesion; may suppurate
- Mild fever, malaise
- **In AIDS**: *bacillary angiomatosis* (vascular proliferative lesions in skin/liver)

— treated with azithromycin or doxycycline

Staining: **Warthin-Starry silver stain** reveals pleomorphic gram-negative bacilli within lymph node. Serology (IFA) or PCR confirms diagnosis.

Why other options are wrong:

- Option B: *Y. pestis*: bubonic plague; flea bite; rapidly fatal without treatment; different clinical picture.
- Option C: *F. tularensis*: rabbit/tick exposure; ulceroglandular tularaemia; Biosafety Level 3 organism.
- Option D: *Brucella*: livestock/dairy product exposure; undulant fever; osteoarticular infection.

Final Answer: Cat scratch + regional lymphadenopathy + Warthin-Starry silver stain $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – Human Monocytic Ehrlichiosis (HME) and Anaplasmosis: *Ehrlichia chaffeensis* causes HME; *Anaplasma phagocytophilum* causes human granulocytic anaplasmosis (HGA). Both are obligate intracellular bacteria transmitted by *Ixodes* ticks in overlapping geographic areas.

Key features of HME:

- Incubation: 1–2 weeks after tick bite
- **Fever, headache, myalgia** + leukopenia, thrombocytopenia, elevated liver enzymes
- Rash in ~30% (unlike RMSF where rash is prominent)
- Blood smear: **morulae** (intracytoplasmic inclusions) in monocytes (HME) or neutrophils (HGA)
- **No rash** is a useful distinguishing feature from Rocky Mountain spotted fever

Treatment: **Doxycycline** is the drug of choice for all rickettsial/ehrlichial/anaplasmid infections. It should be started empirically without waiting for serology confirmation. Rifampicin is an alternative in pregnancy.

Why other options are wrong:

- Option A: Ciprofloxacin — not effective against obligate intracellular organisms lacking peptidoglycan.
- Option C: Azithromycin — second-line for *Bartonella*; not first-line for ehrlichiosis.
- Option D: Ceftriaxone — not active against *Ehrlichia* (no cell wall target in obligate intracellulars).

Final Answer: Tick bite + monocyte morulae + leukopenia + thrombocytopenia $\Rightarrow \boxed{B}$ (Doxycycline)

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – Stenotrophomonas maltophilia (MDR Hospital Pathogen): *S. maltophilia* is an environmental, non-fermenting gram-negative rod that is intrinsically resistant to carbapenems (produces L1 metallo-beta-lactamase and L2 serine beta-lactamase), beta-lactams, fluoroquinolones, and aminoglycosides. It is an emerging opportunistic pathogen in ICU/ventilated patients.

Key microbiological features:

- Oxidase negative (unusual for non-fermenters — helps distinguish from *Pseudomonas*)
- DNase positive; produces yellow-green pigment on some media
- Grows at $\leq$ 35°C; prefers moist hospital environments (sinks, ventilators, water)

Treatment:

- **TMP-SMX**: the only consistently active antibiotic; acts on folate synthesis pathway, bypassing the beta-lactamase/carbapenemase resistance.
- Alternatives: ticarcillin-clavulanate, minocycline, fluoroquinolones (variable), ceftazidime (variable).

Why other options are wrong:

- Option A: Meropenem — *S. maltophilia* has intrinsic carbapenem resistance via L1 metallo-beta-lactamase.
- Option B: Colistin — variable activity; not first-line for *S. maltophilia*.
- Option D: Cefepime — generally not reliable against *S. maltophilia*.

Final Answer: MDR non-fermenter + carbapenem resistant + ICU ventilator ⇒ $\boxed{C}$ (TMP-SMX)

Answer: (C) Go Back to Q12

Q13.

Solution

Concept – Capnocytophaga canimorsus Dog Bite Sepsis in Asplenic Patients: *C. canimorsus* is part of normal oral flora of dogs and cats. In immunocompetent individuals, bites rarely cause serious illness. However, in **asplenic or immunocompromised patients**, it causes fulminant sepsis with purpura fulminans and DIC.

Microbiological features:

- **Gram-negative fusiform (spindle-shaped) rods** with tapered ends
- Slow-growing; requires **CO_2-enriched** environment (capnophilic — hence "capno-")
- Oxidase positive, catalase positive
- Blood cultures may take 5–7 days to grow

Clinical triad: asplenic patient + dog bite + rapidly progressive sepsis with purpura fulminans = *C. canimorsus* until proven otherwise. Treatment: penicillin, amoxicillin-clavulanate, or beta-lactams.

Why other options are wrong:

- Option A: *Pasteurella multocida*: common dog/cat bite pathogen in immunocompetent; gram-negative coccobacillus; rapid onset cellulitis (24 hrs); doesn't cause the fulminant picture in asplenic as classically as *C. canimorsus*.
- Option B: *Eikenella corrodens*: human bite pathogen; oxidase positive; associated with fist-fight injuries.
- Option C: *Bacteroides fragilis*: anaerobic; not associated with dog bites; causes intra-abdominal infections.

Final Answer: Asplenic + dog bite + fusiform Gram-negative rods + DIC ⇒ $\boxed{D}$

Answer: (D) Go Back to Q13

Q14.

Solution

Concept – Aggregatibacter actinomycetemcomitans (HACEK Endocarditis): *A. actinomycetemcomitans* (formerly *Actinobacillus actinomycetemcomitans*) is a member of the **HACEK group** (*Haemophilus aphrophilus*, *Aggregatibacter actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens*, *Kingella kingae*) — fastidious gram-negative rods causing culture-negative endocarditis.

Distinctive features of *A. actinomycetemcomitans*:

- Principal cause of **localised aggressive periodontitis (LAP)**, formerly juvenile periodontitis — in young patients with rapid alveolar bone loss disproportionate to oral hygiene.
- Produces **leukotoxin** (RTX toxin) that kills neutrophils and macrophages.
- Requires **CO_2** for growth; grows as "star-shaped" colonies with internal structure.
- Treatment of HACEK endocarditis: ceftriaxone or ampicillin-sulbactam.

Why other options are wrong:

- Option B: *H. aphrophilus*: also HACEK; not associated with aggressive periodontitis.
- Option C: *Cardiobacterium hominis*: HACEK; normal oropharyngeal flora; endocarditis only.
- Option D: *Eikenella corrodens*: HACEK; associated with human bite wounds, not periodontitis.

Final Answer: Localised aggressive periodontitis + CO_2-requiring Gram-negative coccobacillus + leukotoxin $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q14

Q15.

Solution

Concept – Antibody Structure and Functional Domains: IgG is composed of two heavy chains and two light chains joined by disulphide bonds. Papain digestion yields 2 Fab fragments + 1 Fc fragment; pepsin digestion yields F(ab')$_2$ + pFc'.

Functional regions:

- **Fab region** (Fragment antigen-binding): contains **variable domains** (V_H +

V_L) with three hypervariable loops (CDRs — complementarity-determining regions) that form the **antigen-binding site (paratope)**.

- **Fc region** (Fragment crystallisable): constant domains (C_H2 + C_H3):
  - C_H2 binds **C1q** (complement activation — classical pathway)
  - Binds **Fc receptors** (FcγR on macrophages/NK cells for ADCC; FcRn for half-life/transcytosis)
  - Determines isotype (IgG, IgA, IgM, etc.)

Why other options are wrong:

- Option A: Fc region does NOT determine antigen specificity — that is the function of the Fab/CDRs.
- Option C: ADCC via FcγRIII (CD16) on NK cells is mediated by the **Fc** region, not the Fab.
- Option D: Isotype switching is determined by the constant heavy chain gene segment; Fc region reflects the isotype but doesn't cause switching.

Final Answer: Fab (V_H+V_L) = antigen binding; Fc = complement + Fc receptors $\Rightarrow \boxed{B}$

Answer: (B) Go Back to Q15

Q16.

Solution

Concept – IgG Placental Transfer via FcRn: Of all immunoglobulin isotypes, **only IgG** crosses the placenta. This provides passive immunity to the neonate for the first 3–6 months of life, protecting against encapsulated bacteria, tetanus, etc. The transfer begins at approximately 12–16 weeks of gestation and increases significantly in the third trimester.

Mechanism:

- **FcRn (neonatal Fc receptor)** is expressed on the **syncytiotrophoblast** (outer layer of the placental villi directly in contact with maternal blood).
- IgG binds FcRn at acidic pH within endosomes (pH 6.0); the complex undergoes **transcytosis** across the syncytiotrophoblast to the fetal circulation.
- At physiological pH (7.4) in fetal blood, IgG is released from FcRn.
- FcRn also prolongs IgG half-life in adults by the same recycling mechanism in endothelial cells.

Why other options are wrong:

- Option A: FcγRIII (CD16): on NK cells and macrophages; mediates ADCC — not placental transfer.
- Option B: Poly-Ig receptor: mediates IgA transcytosis across mucosal epithelia (gut, respiratory tract) into secretions — not placental IgG transfer.
- Option D: FcγRI (CD64): high-affinity receptor on macrophages and monocytes; not involved in placental IgG transfer.

Final Answer: IgG across placenta via FcRn on syncytiotrophoblast $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q16

Q17.

Solution

Concept – Lectin/MBL Complement Pathway: The complement system has three activation pathways that converge at C3 convertase. The **lectin pathway (MBL pathway)** is a major arm of innate immunity against encapsulated bacteria with mannose-rich surfaces.

Lectin pathway sequence:

(a) **MBL (mannose-binding lectin)** binds mannose/N-acetylglucosamine on microbial surfaces (structurally similar to C1q).
(b) MBL-associated serine proteases **MASP-1** and **MASP-2** are activated.
(c) MASP-2 cleaves **C4** (to C4a + C4b) and **C2** (to C2a + C2b).
(d) C4b + C2a form the **C3 convertase (C4b2a)**.
(e) C3 convertase cleaves C3 $\rightarrow$ C3a (anaphylatoxin) + C3b (opsonin).
(f) C3b + C4b2a = C5 convertase $\rightarrow$ C5a + C5b $\rightarrow$ MAC (C5b-6-7-8-9).

Why other options are wrong:

- Option A: C1q $\rightarrow$ C1r $\rightarrow$ C1s $\rightarrow$ C4 $\rightarrow$ C2 = the **classical pathway** (activated by antigen-antibody complexes).
- Option B: C3 $\rightarrow$ Factor B $\rightarrow$ Factor D $\rightarrow$ Properdin = the **alternative pathway**.
- Option C: C5b $\rightarrow$ C6 $\rightarrow$ C7 $\rightarrow$ C8 $\rightarrow$ C9 = the **terminal/membrane attack complex (MAC)** — common final pathway.

Final Answer: Low MBL + recurrent encapsulated bacterial infections + lectin pathway $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q17

Q18.

Solution

Concept – NK Cell Activation and Missing Self: Natural killer (NK) cells patrol for cells that lack MHC class I expression — a common tumour/viral immune evasion strategy. NK cells integrate signals from inhibitory and activating receptors.

Key receptors:

- **Inhibitory KIR receptors** (Killer cell Immunoglobulin-like Receptors): recognise self-MHC class I (HLA-A, B, C); when MHC-I is present, KIR delivers an inhibitory signal (ITIM motif) — NK cell does NOT kill.
- **"Missing self" hypothesis** (Klas Kärre): if a cell lacks MHC-I, the inhibitory KIR signal is absent $\Rightarrow$ NK cell becomes activated and kills the target.
- **Activating receptors** (NKG2D, NKp44, NKp46): bind stress ligands **MICA/MICB** (expressed on tumour cells, virus-infected cells, cells under genotoxic stress) — provide activating (ITAM) signals.
- Both arms work together: loss of inhibitory KIR signal + gain of activating NKG2D signal = NK cell killing.

Why other options are wrong:

- Option B: NK cells do NOT require prior antigen sensitisation — they are innate immune cells with non-specific killing.
- Option C: ADCC also contributes to NK killing but is not the primary mechanism for MHC-I-deficient tumour killing.
- Option D: MHC-I downregulation activates NK cells, not CD8+ T cells (CD8+ T cells require MHC-I to be present for antigen recognition).

Final Answer: MHC-I downregulation + missing self + NKG2D + stress ligands $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q18

Q19.

Solution

Concept – TNF-alpha in Inflammation and Anti-TNF Biologics: Tumour necrosis factor-alpha (TNF-α) is a pleiotropic cytokine primarily produced by activated **macrophages** (also T cells, NK cells, mast cells). It is a key mediator of the acute inflammatory response and chronic inflammatory diseases.

Physiological and pathological roles of TNF-α:

- **Fever**: acts on hypothalamic COX-2 to generate prostaglandins
- **Septic shock**: in excess, causes vasodilation, hypotension, DIC
- **Cachexia**: inhibits lipoprotein lipase; induces muscle wasting
- **NF-κB activation**: promotes inflammatory gene transcription
- **Apoptosis**: via TNFR1 (death domain); also promotes survival via NF-κB

Anti-TNF biologics:

- **Infliximab** (chimeric), **adalimumab** (human): anti-TNF-α monoclonal antibodies — used in RA, Crohn disease, UC, plaque psoriasis, ankylosing spondylitis.
- Risk: reactivation of latent TB (TNF-α essential for granuloma maintenance); screen with TST/IGRA before starting.

Why other options are wrong:

- Option A: IL-6 — tocilizumab (anti-IL-6R); used in RA and COVID-19 cytokine storm.
- Option C: IL-1β — anakinra, canakinumab; used in gout, autoinflammatory syndromes.
- Option D: IL-17A — secukinumab; used in psoriasis, ankylosing spondylitis.

Final Answer: RA failing methotrexate + fever + cachexia + Crohn/psoriasis $\Rightarrow$ $\boxed{B}$ (TNF-α)

Answer: (B) Go Back to Q19

Q20.

Solution

Concept – Type I Interferon Pathway (IFN-α/β): Type I interferons (IFN-α/β) are the first line of antiviral defence. They are produced by virtually all nucleated cells in response to viral RNA, and signal via the JAK-STAT pathway to establish an antiviral state in surrounding cells.

Pathway of IFN-β production (relevant to IRF3 mutation):

(a) Virus enters cell and produces **dsRNA** (intermediate or genomic)
(b) **Cytoplasmic sensors**: **RIG-I** (DDX58) detects short dsRNA; **MDA5** detects long dsRNA
(c) **Endosomal sensor**: **TLR3** detects extracellular/endosomal dsRNA
(d) Both pathways activate **IRF3** (via MAVS for RIG-I/MDA5, via TRIF for TLR3)

(e) Phosphorylated IRF3 homodimerises and translocates to nucleus → transcribes **IFN-β**
(f) IFN-β secreted; binds IFNAR on same/adjacent cells
(g) Activates **JAK1/TYK2** → phosphorylates **STAT1/STAT2** → ISGF3 complex
(h) ISGF3 binds ISRE promoter elements → transcribes **ISGs** (interferon-stimulated genes): PKR, OAS, Mx1, IFIT1
(i) ISG products block viral replication at multiple steps

Why other options are wrong:

- Option A: Complement pathway is not directly impaired by IRF3 mutation.
- Option B: TNF-α production is via NF-κB pathway (not primarily IRF3).
- Option D: IL-12/Th1 differentiation is a T-cell adaptive immunity pathway; not directly IRF3-dependent.

Final Answer: IRF3 mutation + recurrent viral encephalitis = impaired IFN-β production $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q20

Q21.

Solution

Concept – IL-6 in Acute Phase Response and COVID-19 Cytokine Storm: Interleukin-6 (IL-6) is produced primarily by macrophages, monocytes, T cells, and endothelial cells. It is a pleiotropic cytokine with both pro-inflammatory and protective functions.

Key biological actions of IL-6:

- **Acute phase response**: signals hepatocytes to produce CRP, fibrinogen, serum amyloid A, hepcidin; suppresses albumin.
- **Fever**: acts on hypothalamus via PGE2.
- **Plasma cell differentiation**: promotes terminal B-cell differentiation and antibody production.
- **T-cell activation**: promotes Th17 differentiation (with TGF-β); inhibits Treg differentiation.
- **COVID-19 cytokine storm**: massive IL-6 release by alveolar macrophages causes hyperinflammation, endothelial damage, and multi-organ failure — **tocilizumab** (anti-IL-6R, anti-IL-6 receptor monoclonal antibody) reduces mortality in severe COVID-19.

Why other options are wrong:

- Option A: IL-6 amplifies inflammation rather than having direct viral cytopathic synergy.
- Option B: IL-6 drives, not inhibits, immune activation; CD8 exhaustion is mediated by PD-1 ligand signalling.
- Option C: IL-6 promotes (not inhibits) fever and acute phase proteins.

Final Answer: COVID-19 cytokine storm + elevated CRP/ferritin/fibrinogen + tocilizumab $\Rightarrow \boxed{D}$ (IL-6)

Answer: (D) Go Back to Q21

Q22.

Solution

Concept – Thymic Selection of T Cells: The thymus is where T-cell precursors (from bone marrow) mature and are selected. Two critical processes occur in distinct anatomical locations:

Thymic selection:

- **Positive selection** (occurs in the thymic **cortex**): Double-positive (CD4+CD8+) thymocytes interact with cortical thymic epithelial cells (cTECs) presenting self-peptide-MHC. Only T cells whose TCR can recognise self-MHC with low affinity survive (*useful T cells*). Failure to recognise = death by neglect ($\sim$95% of thymocytes).
- **Negative selection** (occurs in the thymic **medulla**): Surviving T cells encounter medullary thymic epithelial cells (mTECs) and dendritic cells presenting self-antigens (including peripheral antigens via AIRE gene expression). T cells that bind self-antigen too strongly $\Rightarrow$ apoptosis (clonal deletion). This prevents autoimmunity.

In this question: Ablation of the cortex eliminates cTECs, so **positive selection** cannot occur. All T cells will fail to develop (death by neglect), and no mature T cells will exit.

Why other options are wrong:

- Option B: Negative selection requires medulla (preserved in this model) — less directly impaired.
- Option C: B-cell class switching occurs in germinal centres (peripheral lymph nodes/spleen) — not thymus.

- Option D: NK cell maturation is in bone marrow — not thymus-dependent.

Final Answer: Thymic cortex ablation $\Rightarrow$ positive selection impaired $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q22

Q23.

Solution

Concept – Cross-Presentation by Dendritic Cells: Conventionally, exogenous antigens (phagocytosed or endocytosed) are processed in lysosomes and presented on **MHC class II** to CD4+ T helper cells. Endogenous antigens (synthesised within the cell, e.g., viral proteins or tumour antigens) are presented on **MHC class I** to CD8+ cytotoxic T cells.

Cross-presentation (cross-priming):

- A specialised function of **dendritic cells (especially CD8α+ / BDCA-3+ DCs in humans)**
- Exogenous antigens (tumour cell debris, dead cells, particulate antigens) are taken up and **routed to the MHC class I presentation pathway**
- Mechanisms: phagosome-to-cytosol leakage; ER-phagosome fusion; retrograde transport to cytosol for proteasomal degradation then TAP loading onto MHC-I
- Allows priming of CD8+ CTLs against tumour antigens and viral antigens in infected cells not themselves antigen-presenting cells

Why other options are wrong:

- Option A: MHC class II + conventional presentation = CD4+ T helper cell activation (not CTL activation).
- Option C: Superantigen stimulation binds MHC-II Vβ outside the peptide groove, causing polyclonal T-cell activation.
- Option D: Direct allorecognition: recipient T cells recognise intact donor MHC-II on donor APCs — relevant to transplant rejection.

Final Answer: Exogenous antigen + MHC class I + CD8+ T-cell activation = **cross-presentation** $\Rightarrow$ $\boxed{B}$

Answer: (B) Go Back to Q23

Q24.

Solution

Concept – Adjuvants and Alum Mechanism: Adjuvants enhance vaccine immunogenicity by mimicking "danger signals" that innate immunity uses to detect infection. Without adjuvant, subunit vaccines are poorly immunogenic.

Mechanisms of alum (aluminium hydroxide/phosphate):

- **Depot effect**: forms a precipitate at the injection site, creating a slow-release reservoir of antigen that prolongs APC exposure.
- **NLRP3 inflammasome activation**: alum crystals activate the NLRP3 inflammasome in macrophages and dendritic cells, leading to IL-1β and IL-18 release.
- **Promotes Th2 responses**: drives IgG1 and IgE production; not ideal for Th1/cellular immunity.
- **Complement activation** (via alternative pathway): C3b deposition on alum-antigen particle enhances B-cell activation.
- **Uric acid release** from necrotic cells acts as danger signal at injection site.

Why other options are wrong:

- Option A: Alum does not directly bind TLR4 (that is the mechanism of LPS/MPL adjuvants).
- Option B: Alum promotes Th2 (not Th1); MF59 or AS01 adjuvants promote stronger CD4+ and Th1 responses.
- Option D: Alum does not selectively inhibit regulatory T cells.

Final Answer: Alum = depot effect + NLRP3 inflammasome + Th2/IgE $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q24

Q25.

Solution

Concept – HTLV-1 and Adult T-Cell Leukaemia/Lymphoma (ATL): Human T-lymphotropic virus type 1 (HTLV-1) is a delta-retrovirus (like HTLV-2) that infects CD4+ T cells. Endemic in Japan (southwestern), Caribbean, South America, West Africa, and the Middle East. Transmission: sexual, vertical (breast milk), and blood transfusion.

HTLV-1 diseases:

- **ATL (Adult T-cell Leukaemia/Lymphoma)**: develops in ∼2–5% of carriers after 20–40 year latency. Features: flower cells (CD4+ lymphocytes with multi-lobulated nuclei), skin lesions, hypercalcaemia, lymphadenopathy, lytic bone lesions.
- **HAM/TSP** (HTLV-1-Associated Myelopathy/Tropical Spastic Paraparesis): demyelinating spinal cord disease; progressive lower limb spasticity and bladder dysfunction.

Tax protein mechanism:

- Transactivates HTLV-1 LTR and host genes
- Activates **NF-κB** pathway $\rightarrow$ transcribes survival and proliferation genes
- Drives **IL-2 and IL-2R (CD25) expression** $\rightarrow$ autocrine proliferation loop
- Inhibits p53 and CDK inhibitors $\rightarrow$ cell cycle dysregulation
- Promotes genomic instability via inhibition of DNA repair

Why other options are wrong:

- Option A: Env binds GLUT-1 receptor (glucose transporter 1) — not CD4 (that is HIV gp120).
- Option B: Rex is an HTLV regulatory protein but drives mRNA export, not malignant transformation.
- Option C: Gag = structural protein for virion assembly; not oncogenic.

Final Answer: HTLV-1 + ATL + HAM/TSP + flower cells = Tax protein (NF-κB + IL-2 autocrine) $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q25

Q26.

Solution

Concept – Hantavirus Pulmonary Syndrome (HPS): Hantaviruses are negative-sense ssRNA viruses (family *Bunyaviridae/Hantaviridae*). Different hantavirus species cause distinct clinical syndromes in different geographic regions.

Key facts about Sin Nombre virus and HPS:

- **Reservoir**: deer mouse (*Peromyscus maniculatus*) in North America
- **Transmission**: inhalation of aerosolised rodent **urine, faeces, or saliva**; no arthropod vector
- **No person-to-person spread** (important distinguishing feature from Andes

virus which does transmit person-to-person in South America)
- **Clinical presentation**: prodrome (fever, myalgia) $\rightarrow$ rapidly progressive non-cardiogenic pulmonary oedema; high mortality (30–40%)
- No specific antiviral; supportive care/ECMO

Why other options are wrong:

- Option B: Andes virus (South America) does have person-to-person spread; tick-bite transmission is not the route for hantaviruses.
- Option C: Seoul virus causes haemorrhagic fever with renal syndrome (HFRS) globally via rats, not pulmonary syndrome.
- Option D: Hantaan virus causes HFRS in Asia; spread is rodent excreta not person-to-person.

Final Answer: New Mexico farmer + barn cleaning + rodent droppings + non-cardiogenic pulmonary oedema $\Rightarrow \boxed{A}$ (Sin Nombre virus, no P2P spread)

Answer: (A) Go Back to Q26

Q27.

Solution

Concept – Ebola Virus (Filoviridae): Ebola virus is a negative-sense single-stranded RNA virus belonging to the family *Filoviridae*, along with Marburg virus. It causes one of the most severe haemorrhagic fevers known.

Virological characteristics:

- **Morphology**: filamentous, pleomorphic, enveloped; characteristic "shepherd's crook" appearance on electron microscopy
- **Genome**: negative-sense ssRNA; 7 genes including NP, VP35, VP40, GP, VP30, VP24, L (RNA-dependent RNA polymerase)
- **VP35**: interferon antagonist — blocks IRF3/IRF7 activation and PKR, preventing IFN-α/β production; critical virulence factor
- **Species**: Zaire (most virulent, CFR 60–90%), Sudan, Bundibugyo, Tai Forest, Reston (non-pathogenic in humans)
- **Transmission**: direct contact with blood/body fluids of symptomatic patients; no airborne spread
- Reservoir: fruit bats (*Pteropus* and other genera suspected)
- Treatment: Atoltivimab/maftivimab/odesivimab (Inmazeb); Ansuvimab

Why other options are wrong:

- Option A: Flavivirus with +ssRNA + Aedes = Dengue, Zika, Yellow Fever — not Ebola.
- Option C: Arenavirus with ambisense RNA + rodent reservoir = Lassa fever (Lassa virus), not Ebola.
- Option D: Nairovirus (Bunyaviridae) causes CCHF; sandfly-borne Bunyavirus causes Rift Valley Fever.

Final Answer: West Africa + haemorrhagic fever + DIC + healthcare worker + VP35 IFN block $\Rightarrow \boxed{B}$ (Filovirus, negative-sense ssRNA)

Answer: (B) Go Back to Q27

Q28.

Solution

Concept – Nipah Virus (Paramyxoviridae): Nipah virus is a Henipavirus (family *Paramyxoviridae*) with negative-sense ssRNA genome. It is a zoonotic pathogen that causes severe encephalitis with high case fatality rates (40–75%).

Key epidemiological and clinical features:

- **Natural reservoir**: *Pteropus* fruit bats ("flying foxes") — asymptomatic carriers
- **First outbreak**: 1998–1999 Malaysia/Singapore — pig farmers; pigs were amplifying hosts
- **Bangladesh/Kerala (India) outbreaks**: direct bat-to-human or human-to-human transmission; smaller cluster sizes but higher CFR
- **Unique feature**: **person-to-person transmission** documented in Bangladesh and Kerala outbreaks (unlike Japanese encephalitis)
- **Clinical**: encephalitis + respiratory involvement; relapsing encephalitis reported
- No licensed vaccine or specific antiviral; ribavirin used empirically

Why other options are wrong:

- Option A: Japanese encephalitis virus (JEV) — Culex mosquito vector; no person-to-person spread; pig/water-bird reservoir; vaccine available.
- Option B: Hendra virus — another Henipavirus; fruit bat reservoir (Australia); horses are intermediate hosts; rare human cases; no documented human-to-human spread.
- Option D: Rabies virus — no person-to-person; Negri bodies; animal bite transmission.

Final Answer: Bat reservoir + pig-farm encephalitis + respiratory involvement + P2P spread + South Asia $\Rightarrow \boxed{C}$ (Nipah virus)

Answer: (C) Go Back to Q28

Q29.

Solution

Concept – Zika Virus and Congenital Zika Syndrome: Zika virus is a **flavivirus** (positive-sense ssRNA, enveloped; related to dengue, West Nile, yellow fever) transmitted primarily by *Aedes aegypti* (also *Aedes albopictus*).

Key features of Zika:

- **Adult infection**: usually mild or asymptomatic; fever, rash, conjunctivitis, arthralgias; rarely GBS (Guillain-Barré syndrome)
- **Congenital Zika syndrome**: virus crosses placenta and has tropism for neural progenitor cells in the developing fetal cortex $\Rightarrow$ **microcephaly**, lissencephaly, intracranial calcifications (cortical, not periventricular like CMV)
- **Sexual transmission**: documented male-to-female and male-to-male; unique among arboviruses; virus persists in semen for months
- No vaccine; no specific treatment

Why other options are wrong:

- Option A: Rubella causes periventricular/subcortical calcifications, cataracts, deafness, cardiac defects (PDA, VSD); togavirus, not flavivirus.
- Option B: CMV causes periventricular calcifications (not cortical); most common congenital infection; acquired via maternal primary infection or reactivation.
- Option C: HSV-2 causes neonatal herpes (intrapartum); encephalitis with temporal lobe involvement; not microcephaly.

Final Answer: Brazil + pregnant + rash/conjunctivitis/arthralgia + microcephaly $\Rightarrow \boxed{D}$ (Zika, Flavivirus, sexual transmission)

Answer: (D) Go Back to Q29

Q30.

Solution

Concept – Chikungunya Virus (Alphavirus, Togaviridae): Chikungunya virus (CHIKV) is an **alphavirus** in the family *Togaviridae* with positive-sense ssRNA genome. It is transmitted by *Aedes aegypti* and *Aedes albopictus* mosquitoes.

Clinical features:

- **Abrupt-onset high fever** (39–40°C)
- **Severe, debilitating symmetric polyarthralgia/arthritis**: predominantly small joints (hands, wrists, feet, ankles); patients adopt a bent-forward/stooped posture ("chikungunya" = "that which bends up" in Makonde language)
- **Maculopapular rash**: trunk and limbs, often pruritic
- **Chronic arthritis**: can persist for months to years (unlike dengue)
- **No specific antiviral; no licensed vaccine**: NSAIDs/analgesics for symptom relief

Why other options are wrong:

- Option B: Dengue causes severe bone/muscle pain ("breakbone fever"), thrombocytopenia, haemorrhage; arthritis is not as prominent; tetravalent vaccine (Dengvaxia) available.
- Option C: Ross River virus (Australia/Pacific) causes similar arthritis syndrome but geographically distinct.
- Option D: Sindbis virus causes arthralgia (Scandinavia/Europe/Africa) but is much rarer and not associated with Kerala outbreaks.

Final Answer: Kerala + abrupt fever + severe polyarthralgia + bent posture + no vaccine $\Rightarrow \boxed{A}$ (Chikungunya, Alphavirus)

Answer: (A) Go Back to Q30

Q31.

Solution

Concept – Hepatitis E Virus (HEV): HEV is a **positive-sense ssRNA** virus, now classified in the family *Hepeviridae* (genus *Orthohepevirus*). Genotypes 1 and 2 infect humans; predominantly waterborne in developing countries.

Key characteristics:

- **Transmission**: faecal-oral route, primarily through **contaminated water**; outbreaks after flooding
- **Self-limited** in immunocompetent patients (like HAV)
- **No chronic infection** with genotypes 1 and 2 (genotypes 3 and 4 can cause chronic hepatitis in immunocompromised)
- **High mortality in pregnancy**: 20–25% case fatality rate in the **third trimester** (mechanism unclear — possibly hormonal, immune tolerance, or fulminant hepatic failure)
- No effective antiviral; ribavirin used in chronic HEV in transplant patients
- HAV IgM negative, HBsAg negative, anti-HCV negative — suggests HEV in the right epidemiological setting

Why other options are wrong:

- Option A: HAV — also waterborne and self-limited, but HAV IgM was stated to be negative in this question.
- Option C: HBV — parenteral/sexual transmission; acute HBV mortality in pregnancy is not as dramatically elevated.
- Option D: HCV — blood-borne; not waterborne; does not cause acute outbreaks.

Final Answer: Flood + contaminated water + acute hepatitis + high mortality in pregnancy + HAV/HBV/HCV serology negative $\Rightarrow \boxed{B}$ (HEV)

Answer: (B) Go Back to Q31

Q32.

Solution

Concept – Hepatitis D Virus (HDV) Dependence on HBV: HDV (Hepatitis Delta virus) is a unique **subviral satellite** — a defective RNA virus that can only infect hepatocytes in the presence of HBV. It has the smallest genome of any animal RNA virus (1.7 kb circular negative-sense ssRNA).

Why HDV requires HBV:

- HDV encodes only its own HDAg (Hepatitis Delta Antigen) and replication machinery (ribozyme).
- HDV **cannot make its own envelope**. It borrows **HBsAg (Hepatitis B surface antigen)** from HBV to form its lipid envelope, which is essential for viral **assembly, budding, and release** from hepatocytes, and for **entry** into new hepatocytes via NTCP receptor.

Co-infection vs Superinfection:

- **Co-infection** (simultaneous HBV + HDV): acute hepatitis, usually self-limited; low risk of chronicity.
- **Superinfection** (HDV in chronic HBV carrier — as in this case): more severe, rapid progression to cirrhosis; worse prognosis; 70–90% develop chronic HDV.

Why other options are wrong:

- Option A: HDV has its own RNA-dependent RNA polymerase (host RNA Pol II actually replicates HDV RNA in nucleus).
- Option B: HBcAg forms HBV nucleocapsid; HDV has its own HDAg core.
- Option D: HBV does use reverse transcriptase; HDV replication does not use it.

Final Answer: HDV borrows HBsAg to form its envelope $\Rightarrow \boxed{C}$

Answer: (C) Go Back to Q32

Q33.

Solution

Concept – Plasmodium ovale (Relapsing Malaria): *P. ovale* is the rarest of the four classic human malaria species, endemic primarily in West Africa. Along with *P. vivax*, it forms dormant liver hypnozoites that cause relapsing malaria.

Distinguishing features of *P. ovale*:

- **Schüffner's dots**: prominent eosinophilic stippling of parasitised RBCs (also seen in *P. vivax*)
- **Oval/fimbriated RBCs**: infected RBCs appear oval with irregular, crenated (fimbriated) edges — distinguishes *P. ovale* from *P. vivax* (round, enlarged RBCs)
- **Slight RBC enlargement** (less than *P. vivax*)
- **48-hour (tertian) fever cycle**
- **Hypnozoites in liver**: cause relapses months to years after primary infection; treated with primaquine (or tafenoquine) after checking G6PD status

Why other options are wrong:

- Option A: *P. falciparum*: no Schüffner's dots; Maurer's clefts; banana-shaped

gametocytes; multiple ring forms per RBC; no relapse (no hypnozoites); most dangerous.
- Option B: *P. malariae*: quartan fever (72 hours); "band-form" trophozoites; no Schüffner's dots; can cause recrudescence (persistent blood-stage infection) but not true relapse.
- Option C: *P. vivax*: similar features but RBCs are more markedly enlarged and round (not oval); also has Schüffner's dots and hypnozoites.

Final Answer: West Africa + 48-hour fever + oval/fimbriated RBCs + Schüffner's dots + relapse $\Rightarrow \boxed{D}$ (*P. ovale*)

Answer: (D) Go Back to Q33

Q34.

Solution

Concept – Entamoeba histolytica vs E. dispar: *E. histolytica* and *E. dispar* are morphologically identical by light microscopy. They can only be definitively distinguished by serology, antigen detection, or PCR. However, one morphological feature on stool examination is diagnostic.

Definitive distinguishing feature:

- **Erythrophagocytosis**: trophozoites of *E. histolytica* (never *E. dispar*) actively ingest red blood cells — ingested RBCs are visible as dark inclusions within the cytoplasm. This finding is **pathognomonic** for *E. histolytica*.
- *E. dispar* trophozoites contain bacteria and food vacuoles but **never RBCs**.

Amoebiasis clinical features:

- Amoebiasis clinical forms: amoebic colitis (bloody diarrhoea, "flask-shaped" ulcers) and extraintestinal amoebiasis (amoebic liver abscess — "anchovy sauce" pus, sterile on bacterial culture).
- Virulence factors: Gal/GalNAc lectin (adhesion), amoebapore (pore-forming protein), cysteine proteases.

Why other options are wrong:

- Option B: Both *E. histolytica* and *E. dispar* have 4-nucleate cysts — cannot distinguish them.
- Option C: Peripheral chromatin margination is seen in both species.

- Option D: Gal/GalNAc lectin is present in both species; though Gal-lectin serology (antibody to specific epitopes) can differentiate them — it is not the same as "galactose-inhibitable lectin surface protein" as a morphological feature on stool microscopy.

Final Answer: Erythrophagocytosis (ingested RBCs in trophozoites) definitively identifies *E. histolytica* $\Rightarrow \boxed{A}$

Answer: (A) Go Back to Q34

Q35.

Solution

Concept – Babesia microti (Tick-borne Haemolytic Infection): *Babesia* species are intraerythrocytic protozoan parasites transmitted by *Ixodes* ticks, sharing the same vector as Lyme disease (*B. burgdorferi*) and anaplasmosis.

Key features of babesiosis:

- **Maltese cross (tetrad form)**: four merozoites arranged in a cross/tetrad pattern within a single RBC — **pathognomonic** for *Babesia* spp.; never seen in *Plasmodium*.
- **No haemozoin (malaria pigment)**: distinguishes from *Plasmodium* (which produces dark haemozoin from haemoglobin digestion).
- **Extracellular merozoites** visible on blood smear (merozoites seen outside RBCs — not seen in malaria).
- **Ring forms similar to *P. falciparum***: multiple ring forms per RBC; appliqué position; may be confused with *P. falciparum* early.
- **Severe disease**: in asplenic patients, elderly, or immunocompromised — haemolytic anaemia, haemoglobinuria, renal failure.
- **Treatment**: atovaquone + azithromycin (mild-moderate); clindamycin + quinine (severe); exchange transfusion if parasitaemia $> 10\%$.

Why other options are wrong:

- Option A: *P. vivax*: Schüffner's dots, oval enlarged RBCs; no Maltese cross.
- Option C: *P. falciparum*: multiple rings, banana gametocytes, Maurer's clefts; produces haemozoin; no Maltese cross.
- Option D: *Theileria*: primarily veterinary; not endemic in North America/New England.

Final Answer: Asplenic + New England tick bite + Maltese cross + no haemozoin $\Rightarrow \boxed{B}$ (*Babesia microti*)

Answer: (B) Go Back to Q35

Q36.

Solution

Concept – Trypanosoma cruzi and Chagas Disease: Chagas disease (American trypanosomiasis) is caused by *T. cruzi*, transmitted by **Triatomine bugs (reduviid bugs, "kissing bugs")** that defaecate near the bite wound during feeding; trypomastigotes in the faeces enter via the bite wound or mucous membranes.

Pathogenesis and clinical features:

- **Acute phase**: Romaña's sign (painless periorbital oedema from conjunctival entry), Chagoma (skin nodule at bite site), fever, myocarditis
- **Chronic phase** (years to decades later): Intracellular **amastigotes** (non-flagellate, non-motile form) destroy cardiac muscle and autonomic ganglia of GI smooth muscle:
  - **Chagasic cardiomyopathy**: dilated cardiomegaly, apical aneurysm, RBBB, arrhythmias, sudden cardiac death
  - **Megacolon/megaoesophagus**: destruction of Auerbach's plexus → dysmotility

Why other options are wrong:

- Option A: Tsetse fly + *T. brucei* = African sleeping sickness (HAT — Human African Trypanosomiasis); CNS involvement; different geography.
- Option C: Sandfly + *Leishmania* promastigotes/amastigotes = leishmaniasis; not cardiac/megacolon syndrome.
- Option D: *T. cruzi* does have an insect vector (reduviid bug) as the primary mode of transmission.

Final Answer: Brazil + cardiomegaly + apical aneurysm + RBBB + megacolon + *T. cruzi* serology ⇒ $\boxed{C}$ (reduviid bug + amastigotes in cardiac/smooth muscle)

Answer: (C) Go Back to Q36

Q37.

Solution

Concept – Trichinellosis (Trichinella spiralis): *Trichinella spiralis* is a nematode transmitted by ingestion of undercooked pork (or bear/wild boar) containing en-

cysted larvae.

Life cycle:

(a) Ingested encysted larvae (in meat) release adults in small intestine
(b) Adults mate; females release newborn larvae directly (viviparous)
(c) Larvae penetrate intestinal wall, disseminate haematogenously
(d) Larvae encyst in striated skeletal muscle (especially diaphragm, tongue, extraocular muscles, intercostals)
(e) Host forms collagen capsule around larvae = **"nurse cell"**

Classic clinical triad of trichinellosis:

- **Periorbital oedema** (larvae in extraocular muscles/periorbital fat)
- **Myalgia** (encysting larvae in skeletal muscle)
- **Eosinophilia** (tissue-invasive helminth = intense eosinophilia 20–50%)

Plus: fever, splinter haemorrhages, elevated CPK, elevated LDH. Diagnosis: serology (ELISA), muscle biopsy showing encysted larvae.

Why other options are wrong:

- Option A: *Toxocara canis* (dog roundworm) causes visceral larva migrans; liver, lung, eye involvement; not "nurse cell" in muscle.
- Option B: *Dracunculus medinensis* (Guinea worm): waterborne; subcutaneous migration; no muscle encystation.
- Option C: *Wuchereria bancrofti*: filarial; lymphoedema/elephantiasis; no muscle cysts.

Final Answer: Pork + periorbital oedema + myalgia + eosinophilia + nurse cell in muscle $\Rightarrow \boxed{D}$ (*Trichinella spiralis*)

Answer: (D) Go Back to Q37

Q38.

Solution

Concept – Strongyloides stercoralis Hyperinfection Syndrome: *S. stercoralis* is unique among intestinal helminths in its ability to **complete its life cycle within the human host** (autoinfection). This normally occurs at a low level. Immunosuppression — particularly **corticosteroids** — dramatically accelerates autoinfection, leading to hyperinfection/dissemination syndrome.

Mechanism of hyperinfection:

- Normally, rhabditiform (L1) larvae in the gut are excreted in faeces or mature into filariform (L3) larvae in the colon, penetrate colonic mucosa, and re-enter the bloodstream (internal autoinfection).
- Corticosteroids impair eosinophil function and Th2 immunity that normally suppress *Strongyloides*; also, cortisol mimics the worm's ecdysteroid receptor signal, stimulating accelerated larval development.
- **Hyperinfection**: massive numbers of filariform larvae invade the bowel wall, carrying intestinal bacteria (gram-negative bacilli) into the bloodstream $\rightarrow$ **gram-negative bacteraemia and meningitis**.
- **Dissemination**: larvae found in lungs, liver, CNS, heart.

Diagnosis: rhabditiform AND filariform (L3) larvae in stool or duodenal aspirate. Treatment: ivermectin (drug of choice).

Why other options are wrong:

- Option B: *Ascaris* causes biliary disease (wandering); not gram-negative bacteraemia; not immunocompromised-specific.
- Option C: Hookworm does not disseminate haematogenously.
- Option D: *Strongyloides* does not produce immunosuppressive factors — the immunosuppression is host-derived (corticosteroids).

Final Answer: Corticosteroids + rhabditiform/filariform larvae + gram-negative bacteraemia $\Rightarrow \boxed{A}$ (Strongyloides hyperinfection)

Answer: (A) Go Back to Q38

Q39.

Solution

Concept – Sporotrichosis (Sporothrix schenckii): *Sporothrix schenckii* is a **dimorphic fungus** (mould at 25°C, yeast at 37°C/body temperature). It causes subcutaneous mycosis following traumatic inoculation via plant material.

Key clinical features:

- **Classic exposure**: rose thorns, barberry, sphagnum moss, timber, hay
- **Lymphocutaneous (nodular lymphangitis / sporotrichoid spread)**: primary nodule/ulcer at inoculation site, followed by painless nodular lesions along draining lymphatics — classic "ascending" pattern

- **Other forms**: fixed cutaneous, pulmonary (rare, immunocompromised), disseminated (very rare — AIDS)
- **Morphology at 25°C**: hyphae with **conidia in a "daisy head" (rosette) or "sleeve" arrangement** along conidiophores
- **Asteroid bodies**: PAS-positive yeast cells with radiating eosinophilic extensions in tissue (not always seen)

Treatment:

- **Localised cutaneous/lymphocutaneous**: **Saturated potassium iodide solution (SSKI)** (traditional, cheap, effective) or **itraconazole** (preferred in modern practice, fewer side effects)
- **Pulmonary/disseminated**: itraconazole; severe cases: amphotericin B

Why other options are wrong:

- Option A: Fluconazole — less effective against *Sporothrix*; not first-line.
- Option C: Amphotericin B — reserved for severe disseminated disease; not first-line for localised cutaneous disease.
- Option D: Voriconazole — not recommended for sporotrichosis; used for Aspergillus and some Candida.

Final Answer: Rose thorn + nodular lymphangitis + dimorphic fungus + rosette conidia at 25°C $\Rightarrow \boxed{B}$ (SSKI or itraconazole)

Answer: (B) Go Back to Q39

Q40.

Solution

Concept – Pneumocystis jirovecii Pneumonia (PCP): *Pneumocystis jirovecii* (formerly *P. carinii*) is a fungus (formerly classified as a protozoan; now placed in *Ascomycota*) that causes life-threatening pneumonia in immunocompromised patients, especially those with HIV and CD4 count < 200 cells/μL.

Clinical and diagnostic features:

- **Insidious onset**: progressive exertional dyspnoea, dry cough, low-grade fever
- **CXR/CT**: bilateral perihilar ground-glass opacities ("bat-wing"); may be normal early
- **Elevated LDH**: sensitive but non-specific marker; correlates with extent of

lung damage

- **Widened A-a gradient**: disproportionate to severity on CXR
- **BAL staining**: **Gomori methenamine silver (GMS)**: stains cyst walls black; disc-shaped cysts (5–8 μm) with up to 8 intracystic bodies. Also Giemsa (trophic forms), Calcofluor white, DIF (direct immunofluorescence – most sensitive).
- **Serum beta-D-glucan**: elevated; helpful adjunct (but not specific).

Treatment and prophylaxis:

- **First-line treatment**: **TMP-SMX (high-dose)** – 15–20 mg/kg/day trimethoprim component for 21 days
- **Adjunctive corticosteroids**: if $PaO_2 < 70$ mmHg or A-a gradient > 35 (reduces inflammation and mortality)
- **Primary prophylaxis**: TMP-SMX when CD4 $<$ **200 cells/μL** (or $<$14%) in HIV; can be discontinued when CD4 rises > 200 on ART
- Alternatives: dapsone, atovaquone, pentamidine (inhaled for prophylaxis)

Why other options are wrong:

- Option A: Pentamidine IV is second-line for treatment; CD4 < 100 threshold is incorrect (threshold is < 200).
- Option B: Atovaquone is an alternative agent for mild-moderate PCP; CD4 $<$ 150 is not the standard threshold.
- Option D: Clindamycin + primaquine is a second-line treatment option; CD4 < 100 threshold is incorrect.

Final Answer: HIV + CD4 45 + bilateral GGO + LDH elevated + GMS disc-shaped cysts $\Rightarrow$ TMP-SMX; prophylaxis at CD4 $< 200 \Rightarrow \boxed{C}$

Answer: (C) Go Back to Q40

Answer Key – FMGE Microbiology Sample Paper 4

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