Questions 2003

1. **LIST ALL THE BACTERIAL CAUSES OF MENINGITIS**
* Neisseria Meningitidis
* Strept. Pneumonia
* H. Influenza type b
* Strept Agalactiae
* Staph. Aureus
* E. Coli
* Listeria Monocytogenes
* Mycobacterium tuberculosis
* Staph. Epidermidis
* Pseudomonas aureginosa
	1. **DESCRIBE THE LAB PROCEDURES FOR MICROBIOLOGICAL DIAGNOSIS AND IDENTIFICATION OF ONE OF THE ORGANISMS YOU LISTED**
* Strept pneumonia – Specimen: sputum, nasal
* Neisseria meningitidis: Smear and culture of blood and spinal fluid specimens
	+ Microscopy of specimen yield gram negative cocci.
	+ Culture in CBA anaerobic at 37 degrees and 5 % CO2.
	+ Colonial morphology grayish white, non pigmented convex shaped colonies
	+ Biochemical test: Carbohydrate fermentation tests. Ferments maltose and glucose
	+ Catalase positive
	+ Oxidase positive colonies confirm.
	+ Serological tests: Latex agglutionation test for capsular antigen
	+ ELISA test for antibody to
	+ Immunofluoresence assays
1. **Write short notes on**
	1. **Shigella dysentriae**
* Gram negative rod
* Causative agent of bacillary diarrhea: Bloody diarrhoea
* Non spore forming
* Facultative anaerobe
* Non – motile
* A member of the enterobacteriacea
* Non – lactose fermenter
* TSI: Alkaline slant, acid butt no gas no H2S
* Mode of transmission fecal - oral
	1. **Dermatophyte infections**
* Are infections of the skin: superficial infections
* Clinical manifestations: Tinea capitis, tinea corporis, tinea pedis, tinea cruris, tinea vesicolor, Malasezia furfur, tinea unguium, tinea barbae, tinea manuum, tinea favosa
* Causative agents include: epidermophyton, trichophyton and microsporum species
* May be classified as zoophilic, anthropophilic or geophilic based on the source.
* Treatement via topical azole drugs such as ketoconazole, or sulphur ointment, or whitefields oitment
	1. **Pathogenesis of polio virus**
* Mode of transmission fecal – oral.
* Replication in the GIT
* Primary viremia in the blood stream.
* Spreads via circulation to the CNS
* Further replication within the neurons at the ventral spinal horn.
* Contributes to the death of the neurons resulting in Descending paralysis to the muscles innervated by affected motor neurons
* Also may affect the brain stem resulting in bulbar poliomyelitis thus may contribute towards respiratory failure.
* Also may cause meningitis
* May also cause asymptomatic infection
	1. **Insertional mutagenesis in relation to viruses**
* Occurs when viral DNA is inserted into host DNA causing either over expression of genes or under expression of host genes based on the point of viral DNA insertion
* It normally occurs within some DNA viruses, however also RNA Retroviruses could cause Insertional mutagenesis when their DNA copy is integrated into host DNA
* If the viral DNA is inserted next to a repressor gene, then the adjacent gene may be over expressed, while if it is inserted next to an enhancer gene, then the adjacent genes may be under expressed
* In viral oncogenesis, viral oncogenes may be inserted next to a cellular myc gene, that was prior inactive, activating it, cause an indiscriminate continuity in the cell cycle with the result being transformation and tumorigenesis
* Avian leucocytosis virus is an example of virus associated with Insertional mutagenesis. HIV virus incorporates its DNA copy into host and so thought to induce Kaposi sarcoma.
1. **Describe life cycle, clinical presentation, diagnosis and treatment of wuchereria bancrofti**
* **Life cycle**. Vector transmitter is the anopheles and the culex mosquitoes.
* Bite of anopheles containing the filariform larva of W. bancrofti. The larva spread to the lymphatic vessels especially of the lower limbs and the urogenital area where they mature into adults.
* Adults show ovoviviparity ,reproduce to give out the microfilariae that move into the blood circulation
* The microfilaria show nocturnal periodicity. During the day circulate within the deep veins while at night they circulate within the superficial veins
* During the next blood meal of the mosquito, the microfilaria are picked up and differentiate within the mosquito to the infective filariform larva that may be transmitted during the next bite
* This whole cycle takes 10 -12 months
* **Clinical presentation**: categorized into asymptomatic phase: Here no disease symptoms are visible
* The acute phase: Manifests as fever, lympadenitis, lymphgenitis, orchitis, epididimitis
* Chronic phase: Manifests as elephantiasis, Hydrocele testis, lymphaedema of legs and arms, Tropical pulmonary Eosinophilia, Chyluria (whitish urine) rupture of lymphatics within the urogenital area
* The symptoms are mainly due to the adults in the lymphatics that cause obstruction that contributes to the edema in addition, inflammatory reactions as well contribute towards the acute phase.
* **Diagnosis**:
* History of residing within endemic areas or where the vectors are found.
* Microscopic observation of the peripheral blood smear. Both thick and thin Giemsa stained smears are used for the identification of microfilaria
* The smears are taken during the night from the superficial veins as the microfilaria show nocturnal periodicity
* Hetrazan induced method
* DEC (Diethyl carbamazine) provocation test (DEC 50mg then blood after 30 min)
* ultrasound of the scrotum – filarial dance sign (adults seen, appear to be dancing)
* Serological tests – filarial specific antibodies e.g IgG; circulating filarial antigens (CFA)
* **Treatment**: Ivermectin, Diethyl Carbamazine effective in killing the microfilaria
* albendazole
* Surgical removal of worms causing lymphatic obstruction
* Reconstructive surgery for the elephantiasis and hydrocele testes patients
1. **WRITE SHORT NOTE ON THE FOLLOWING**
	1. **Complications of E. hystolitica**
* Liver abscesses
* Flask - shaped ulcers in intestinal mucosa
* Ameboma in the rectal and sigmoid colon. These are granulomatous lesions
* Lung disease as a result of penetration of diaphragm by right lobe liver abscess.
* Bacteremia a result of escape of colonic bacteria via the ulcers generated by the amoeba
	1. **Medical importance of ticks**
* Ticks serve as vectors in the transmission of a number of disease causing microorganisms as shown below.
* Borrelia Burgdoferi, cause of borreliosis or lyme disease, is transmitted by a tick, Ixodes scapularis
* Tick typhus, causative organism rickettsia rickettsii ,Rocky mountain spot fever, is transmitted by a tick, Dermacentor variabilis
* Colorado tick fever virus, is transmitted by a tick, Dermacentor andersoni
* [**Babesiosis**](http://www.cdc.gov/babesiosis/) is transmitted by tick (Ixodes scapularis)
* [**Ehrlichiosis**](http://www.cdc.gov/ehrlichiosis/) is transmitted to humans tick (Ambylomma americanum)
* Tularemia, transmitted to humans by tick, dermacentor variabilis
* Anaplasmosis, transmitted by the tick ixodes scapularis
	1. **Control of malaria**
* Sleeping under mosquito nets
* Using window screens
* Advice wearing of protective clothing
* Use of mosquito repellants
* Clearing of bushes and draining of stagnant waters
* Use of insecticides to kill mosquitoes anopheles that transmit plasmodium species
* Prophylaxis using quinine and chloroquine, doxycycline, mefloquine, atovaquone, and proguanil for travelers to endemic areas
	1. **Pathology of T. Solium infection**
* Pathological stages include the adults and the cycticerci
* Adults may contribute towards food spoilage, abdominal pain, discormfort, diarrhea, anorexia, vomiting. These symptoms are due to the attachement of the adults within the intestinal mucosa and invasive properties of the aadults
* The cysticerci during autoinfection, where the proglottids disintegrate within the host, releasing oncopheric eggs that hatch into cysticerci or infection through ingestion of T. solium eggs,that hatch into cysticerci and penetrate the GIT into the blood circulation where they are transported to various organs in the body which include the brain, eyes, muscle causing neurocysticercosis, ophthalmocysticercosis, or myocysticercosis respectively
* Here the cysticerci may cause hypersensitivity reactions
* The cysticerci may then calcify and contribute towards space occupying lesion which may be fatal in case of brain involvement causing neurological conditions such as convulsions, paralysis and may contribute to blindness in the case of eye involvement.
1. **Giving specific examples, give 5 mechanism by which parasitic organisms evade host immune attack**
* Through secretion of IgA proteases that cleave mucosal IgA responsible for mucosal immunity
* Through antigen masking using host antigens such as making its own glycolipids as it travels using host glycoproteins. Entamoeba hystolitica masks its surface antigens using host glycoproteins
* Through cyst formation by various parasites such as helminthes. These cysts are resistant to immune responces. Hydatid cyst formed by echinococcus granulosus
* Residing in GIT lumen where they are sheltered from host Cell mediated immune response. Adults ascaris lumbricoides are located within the GIT lumen where they are safe from Cell Mediated immune responce
* Antigen variation that renders preformed antibodies ineffective towards the new surface antigenic mutations in the parasites. In plasmodium for instance, different stages express different antigens thus antibodies for a certain stage are ineffective for a subsequent stage. Giardia lamblia also exhibits antigenic variation at the various stages of its life cycle.
* Anatomical seclusion of the parasites in areas where they are sheltered from host immune defences, such as in the case of plasmodium species residing within red blood cells. Leishmania species and trypanosomes reside within macrophages.
* Induction of immune suppression by the parasites rendering the immune response absent is strategy that plasmodium species employs in evasion of immune response
* Prevention of fusion of the phagolysome (Phagosome and lysosome fusion) is prevented by lesishmania species thus escaping the destructive lysosomal enzymes.
* Production of parasitic enzymes that have anti immune effect. Filarial worms produce glutathione oxidase and superoxide dismutase that enables them escape antibody dependent cellular cytotoxicity and oxidative stress.

Questions 2004

* 1. List the spirochetes that cause disease in humans and the diseases they cause. (10mks)
* Leptospira species: leptospira interrogans causes leptospirosis
* Borrelia species: Borrelia Burgdoferi causes Lyme disease
* Borrelia recurrentis: louse – borne recurrent fever
* Treponema pallidum pallidum causes syphilis
* Treponema pallidum carateum causes pinta
* Treponema pallidum endemicum cause bejel or non venereal syphilis
* Treponema pallidum pertenue causes yaws
	1. Discuss the transmission, clinical manifestations and laboratory diagnosis of one the diseases listed.
* Treponema pallidum pallidum
	+ Transmitted from spirochete containing lesions on the skin and mucous membrane via sexual contact, blood transfusion, via transplacental, that is from mother to her fetus.
	+ Clinical manifestations:
		- Include the following stages
		- Primary syphilis
			* Multiplication at the site of inoculation. Presents as a non tender chancre that occurs within 2 – 3 weeks.
			* Bacteremia, occurs in this stage
		- Secondary syphilis
			* Occurs within 3 – 6 months after infection
			* Characterized by lesion on skin and maculopapular rash on the palms and soles.
			* Condylomata lata, lesions on the genital area are present at this stage
		- Tertiary syphilis
			* Occurs within 3 – 10 years after infection
			* Presence of Gummas on skin and mucous membrane. treponemes are absent in the lesions at this stage
		- Late syphilis:
			* Occurs within 10 -20 years
			* Shows involvement of the cardiovascular systems: abornamilties in the aorta and heart valves manifest.
			* Also CNS involvement. Neuro syphilis
		- Latent syphilis
			* Dormant syphilis that can only be detected via serological studies.
		- Congenital syphilis
			* Can be transmitted as early as the 1o the week of pregnancy and any time from there
			* Associated with septicemia and may cause still birth
	+ Lab diagnosis:
		- Non – Treponemal Tests: Using cardiolipin derived from beef heart muscle to agglutinate with Treponemal antibodies in the serum. Detects presence of reagin antibody
		- Test for presence of Treponemal reagin antibodies
		- Include: VDRL – venereal disease research laboratory test, Wassermann’s test, Kahn’s Test, RPR – Rapid plasma reagin test.
		- These test are positive during secondary syphilis, may be negative during tertiary syphilis
		- These test require confirmatory test
		- Treponemal Tests: Test for the antigen presence
		- Include Fluorescent Treponemal Antibody absorption test, treponema pallidum haemagglutination assay
		- Treponema pallidum immobilization test
		- Elisa test for Treponemal antigens
	1. Short essay on biological properties of pseudomonas aureginosa.
* Gram negative bacillus
* Are strict aerobes
* Non - fermenters
* Oxidase positive
* Are motile with polar flagella
* Has an antiphagocytic polysaccharide slimy layer
* Has a LPS like any other gram negative rod
* Produces pyocyanin, a blue – green pigment hence appearance of its colonies cyanin like
* Produces toxin A and exoenzyme S
	1. Short essay on diseases caused by aspergillus species
* The main pathogenic species include: Aspergillus flavus, and aspergillus fumigatus
* Aspergillus species causes aspergillosis.
* Aflatoxin of aspergillus flavus may cause hepatitis, hepatocellular carcinoma and immunosuppression
* Aspergillus fumigatus causes infections of the eye, ear, and other organs;
* ‘fungus ball’ in the lungs (aspergilloma)
* Allergic bronchopulmonary aspergillosis
	1. Discuss the laboratory diagnosis of Hepatitis B
* Detection of Serological markers of Hepatitis B infection which include;
	+ Hepatitis B surface antigen (HBsAg)
	+ Antibody to Hepatitis B surface antigen( antibody to HBsAg)
	+ Hepatitis B core antigen (HBcAg)
	+ Antibody to Hepatitis b Core antigen (Antibody to HBcAg )
	+ Hepatitis B e antigen (HBeAg)
	+ Antibody to hepatitis B e antigen (Antibody to HBeAg)
	1. Discuss pathogenesis of Human Herpes Virus 3
* Herpes Zoster
	+ Mode of transmission via respiratory droplets
	+ Infects the mucosa of the respiratory system then spreads to blood
	+ In blood primary viremia develops
	+ Spreads to skin via blood circulation
	+ In skin a vesicular rash develops, Multi nucleated giant cells with intracellular inclusions are seen at the base of the lesions.
	+ The virus then infects the sensory neurons and is carried via retrograde axonal transport to the dorsal root ganglia where they remain latent
	+ In latent viral containing cells, the virus is located in the nucleus but is not integrated into the nucleus.
	+ May recur later on in life during immunosuppression causing shingles that presents as vesicular lesions and nerve pain
	+ At recurrences the virus traces down the nerve and affects the dermatome area innervated by the nerve.
1. Describe life cycle, clinical manifestation, complications, diagnosis and treatment of plasmodium falciparum infection in man
* Life cycle
	+ Human are the intermediate host while the female anopheles species of the mosquitoes are vectors as well as definitive hosts.
	+ The cycle shows two phases, a sexual phase; sporogony inside the anopheles and an asexual phase, schizogony inside the human host
	+ Cycle begins during inoculation of the sporozoites into the human host by the female anopheles mosquito saliva during its blood meal.
	+ The sporozoites then enter an exoerythrocytic stage in the liver where they differentiate into merozoites.
	+ The merozoites enter the erythrocytic phase when the infect circulating red blood cells. Red blood cells of all stages are infected by p. falciparum hence the increased percentage of parasitized red blood cells in p. falciparum infections
	+ The merozoites differentiate into ring – shaped trophozoites, which form an amoeboid like structure that different into schizonts containing more merozoites that infect more red blood cells. This cycle repeats every 2 days. Thus fever spikes after every 2days.
	+ Some merozoites differentiate into male and female gametocytes commencing the sexual phase.
	+ These gametocytes are taken up by the female anopheles mosquito during her next blood meal.
	+ The gametocytes fuse while inside the mosquito gut to form a zygote.
	+ The zygote differentiates into an ookinete.
	+ The ookinete develops into an oocyst that contain numerous sporozoites
	+ Sporozoites migrate to the mosquito salivary glands, and ready to be transmitted by the mosquito to another host. And so the life cycle continues
* Clinical manifestation:
	+ Fever, chills, nausea, vomiting and abdominal pain. Fever spike of 42 degrees
	+ Headaches, myalgias, athralgias commencing 2 weeks after bite by mosquito
	+ Anemia and anemic conditions such as parlour of mucous membranes
	+ Drenching sweats may follow fever.
	+ Febrile episodes of feeling well may be experienced.
	+ Splenomegaly
	+ Hepatomegaly in some cases
* Complications
	+ Merozites may infect brain tissue causing cerebral malaria. Extensive Brain tissue damage
	+ Kidney damage causing black water fever
* Diagnosis
	+ Thin and thick giemsa stained smears are obsereved microscopically for presence of ring – shaped trophozoites
	+ Cresent, banana shaped gametocytes are characteristic for p. falciparum
	+ Very high percentage of parasitized red blood cells indicate p. falciparum
* Treatment
	+ Chloroquine: for chloroquine sensitive p. falciparum species
	+ For chloroquine resistant species: administer doxycycline and mefloquine
	+ Atovaquone and proguanil may also be administered
	+ Quinine administered for mefloquine resistant p. falciparum
1. Shot essay on the following
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	1. Lab diagnosis and control of schistosomiasis
* Detailed patient history for epidemiological diagnosis
* Specimen: Stool sample for s. mansonia and s. japonicum species, while urine samples for s. haematobium species identification
* Specimen concentration techniques such as Kato Katz technique
* Wet preparation of the stool samples.
* Observation for eggs. Presence of eggs with prominent terminal spine diagnostic for s. mansoni, prominent terminal spine diagnostic for s. haematobium, rudimentary lateral spine diagnostic for s. japonicum
* Moderate eosinophilia may occur
* Control:
	+ - Proper disposal of human faeces
		- Avoid use of human faeces as fertilizer
		- Chlorination of water sources
		- Molluscicides to eliminate intermediate host
		- Swimming in endemic areas should be avoided
	1. Diphylobothrium Latum
* Referred to as the fish tapeworm
* Is a cestode (Class Cestode)
* Belongs to the order pseudophyllidae
* Its scolex has two sucking grooves for attachment
* Proglottids are wider than they are long. Gravid uterus in the form of a rosette
* Eggs are oval and operculated
* Is the longest of the tape worms, upto 13 meters
	1. Clinical presentation of visceral leishmaniasis (kala azar)
* Intermittent fever, weakness and weight loss
* Massive splenomegaly
* Hyper pigmentation especially of light skinned people hence the name Kala azar (Black sickness)
* Leucopenia, thrombocytopenia and anemia may occur
* Weakness, infections and gastrointestinal bleeding
* May result in death in untreated cases due to secondary infections
1. Short notes on immune responses to extracellular bacterial infections
* Innate immune response include:
* Alpha defensins, lysozymes and proteases that cleave the bacteria
* Complement activation
* Phagocytosis
* Inflammatory responce
* Adaptive immune response include:
* Humoral immune responce
* Neutralization of bacterial toxins by antibodies
* Antibodies attach to bacteria to achieve the following
	+ Opsonization of bacteria
	+ Prevention of attachment of the bacteria to their target surfaces such mucosal epithelium
	+ Complement fixation and activation
	+ Agglutination of bacteria, thus limiting their spread and facilitating their Phagocytosis
	+ Antibody mediated cellular cytotoxicity
* Cell mediated immune response entail
	+ Internalization of the bacteria by antigen presenting cells
	+ Processing and expression specific peptides in associating to MHC class II to T – helper cell. Activation of Th cells set that contribute towards
		- Activation of Phagocytosis by macrophages
		- Antibody production by B cells
		- More inflammation

Questions 2005

1. Discuss the microbiological aetiology, pathogenesis, clinical presentation and lab diagnosis of typhoid fever
* Microbiological aetiology; caused by gram negative rods; salmonella typhi, and salmonella paratyphi A, B, and C. These organisms are obtained via ingestion of food or water contaminated with human faeces or animal wastes. Humans and animals serve as reservoirs for these organism. Human carriers or mechanical vectors such as the housefly, Musca domestica, or the cockroach, are involved in the transmission. Typhoid fever prevails mostly in the developing countries where standard of living is low.
* Pathogenesis:
	+ Infection starts in the small intestines where the organisms multiply within mononuclear cells in the peyers patches. From there they are spread to other phagocytic cells in the liver, gall bladder and spleen. By then Bacteremia develops. Thus marks the hallmark of fever and other symptoms that are associated with the endotoxin. The organisms survive within the phagosome of the phagocytic organism. The organism predilection to infect the gall bladder will in turn imply excretion of the organisms in faeces thus developing a carrier state. Some of the virulence factors that assist the organism in its pathogenesis include: fimbrae for attachment to the microvilli of small intestines, and the polysaccharide capsule that is antiphagocytic.
* Clinical presentation
	+ Fever, with dull-continuous headache, abdominal tenderness, hepatomegally, splenomegaly
	+ Typhoidal ulcers
	+ Diarrhoea is uncommon
	+ Gastrointestinal bleeding and perforation may occur as a complication
	+ Metastatic disease such as osteomyelitis may present as a complication
* Lab diagnosis
	+ Blood culture may reveal organism, bone marrow culture may be positive
	+ Enrichment media: selenite broth
	+ Selective media: Desoxychocolate citrate agar; Xylose lysine Desoxychocolate agar, Salmonella/Shigella agar
	+ Colorless colonies in Mackonky agar, and Eosin methylene blue agar
	+ Slide agglutination positive for the outer Somatic O antigen
	+ Sub culture on peptone water for flagella H antigen identification
	+ Gram stain: gram negative rods
	+ Are motile
	+ Biochemical characteristics: Oxidase negative; Urease negative
	+ TSI: Alkaline slant, acid butt, no gas but with H2S production
	+ Serological tests: Widal test
1. Write short notes on
	1. H. Influenza
* Is a gram negative coccobacillus with a polysaccharide capsule
* It is among the leading cause of bacterial meningitis.
* Serotype b is associated with invasive disease such as meningitis
* Also causes infections within the respiratory system such as pneumonia
* Requires factor V (NAD) and factor X (Heme) for laboratory growth
* Cultured in Chocolate blood agar
	1. Lab diagnosis of dermatophyte infections
* Specimen to be used: Skin scrapings, nail clippings
* Treatment of specimen using 10 % KOH that dissolves most tissues
* Direct Microscopic observation for athrospores and athroconidia
* Culture in SDA – Saboraud Dextrose Agar usually 10 – 14 days
* Lactophenol blue staining of a portion of culture, identification of microconidia for microsporum species, and macroconidia for M. canis
	1. Characteristics of measles virus
* Is a member of paramyxoviridae
* Has a negative sense RNA that is enveloped
* Has a helical nucleocapsid
* Has a virion polymerase
* Its envelope spike has a haemagglutinin and a fusion protein
* It cause giant cell formation and synctia formation
* Has a size of 150nm
* Has one serotype
	1. Pathogenesis of rabies
* Mode of transmission is via a bite from a rabid dog or any other wild animal such as a fox
* Virus replication within the site of inoculation
* Virus infects the sensory neurons of the skin around around the bite would
* Virus is carried via a retrograde axonal transport system to the CNS
* In the CNS infects the neurons and manifests as encephalitis, convulsions as a result of death of neurons and demyelination.
* From CNS it spreads via the peripheral nerves to the salivary glands and other organs.
* In the salivary glands the virus can then be transmitted via the next bite
* Infected neurons contain negri bodies
* May lead to death if untreated
1. Describe life cycle, clinical manifestations, complications, diagnosis and treatment of Necator Americanus infection in man
* Infective stage is the filariform larva.Filariform larva present in the soil penetrate skin of barefoot person. Then enters circulation and migrates from entry site to the heart, lungs, bronchi, trachea, coughed out and swallowed in sputum, entry into the GIT and finally to the small intestines where developed into adult. Adult lays eggs approximately 9, 000 eggs per day. These are excreted in feaces. The eggs take 2 weeks to embryonate in the soil hatch out to give the L1 larva that develops in the soil to the L3 larva which is ready to penetrate skin of susceptible host. Diagnostic stage is the eggs in faeces.
* Clinical manifestations: Ground itch: at site of penetration
	+ Eosinophilia especially during larval migration phase
	+ May cause pneumonia while in the lung
	+ Laryngitis and wheezing
	+ Alveolar invasion
	+ Adults cause anemia (Such 0.03 ml per day per worm)
	+ Abdominal pains and diarrhea
	+ Anorexia and vomiting may follow
* Comlications include: pneumonia by the larva stages
	+ Jaundice caused by adult worm biliary tract obstruction
* Diagnosis
	+ Detection of oval eggs in stool via kato katz technique
	+ Eosinophilia
	+ Peripheral bood film examination for hypochromic microcytic anemia
	+ Pallor of skin and mucous membrane
* Treatment:
	+ Albendazole, mebendazole, thiabendazole
	+ Transfusion to manage anemia
	+ Iron and folate supplementation
	+ Protein therapy
1. Write short notes on
	1. T. vaginalis
* It is a pear shaped organism
* Is a flagellated protozoa belonging to the class mastigophora
* Exists only as a trophozite
* Has an undulating membrane about 2/3 of its length
* Has two pairs of anterior flagella
* Has a single prominent nucleus
* Its implicated for causing sexual transmitted disease trichomoniasis
	1. Medical importance of mosquitoes
* Implicated for transmitting a number of diseases that include
	+ Malaria caused by plasmodium species and is transmitted by the mosquito anopheles
	+ Rift valley fever virus is transmitted by aedes agypti and aedes albopictus
	+ Dengue fever virus is transmitted by aedes agypti
	+ Wuchereria bancrofti, brugia malayi and brugia timori, these filarial worms that cause elephantiasis are transmitted by the anopheles species, culex species as well
	+ Rift valley fever virus by aedes species
	+ St. Louis Encephalitis transmitted by culex species
	1. Diagnosis and control of E. granulosus
* Diagnosis
	+ History
	+ Physical examination
	+ Ultrasound of liver for presence of Hydatid cysts
	+ Serological tests for worm antigen
	+ X rays for presence of cysts in tissues, lung as well
	+ Radiological examination
	+ CT scan of brain for cysts in brain
	+ Skin test: cansoni skin test
* Control
	+ Proper disposal of consumable meat viscera
	+ Regular deworming of dogs
	+ Avoid feeding of dogs the entrails of sheep
	+ Fencing of slaughter house from dogs
1. Write short notes on
	1. Types of vaccines. Give examples of each type
* whole organism: Natural: Vaccinia for small pos
	+ Attenuated organism: sabin vaccine, BCG vaccine, pertussis
* whole organism but Killed: polio virus salk vaccine
* Sub cellular (subunit) vaccine: capsular polysaccharide antigen used: Pneumococcal vaccine
	+ Surface antigen: Hepatitis B vaccine
* Conjugate vaccine: H. influenza type B vaccine
* Toxoid: Tetanus, Diphtheria
* Recombinant DNA vaccine: Hepatitis B virus vaccine
	1. Complications associated with immunoprophylaxis (immunizations)
* Reversion of attenuated organisms in attenuated vaccines to pathogenic species thus causing disease
* Hypersensitivity reactions towards the vaccines as they contain foreign antigen: Urticaria, eczema, erythematic dermatitis may occur after administration of vaccine intramuscularly.
* Serum sickness is an instance of hypersensitivity towards the vaccine
* In vaccination of live attenuated vaccines to immunocompromised individual it will cause disease
* Vaccination of pregnant women with live attenuated organisms may cause disease to fetusQuestions 2006
1. List the bacterial causative agents of meningitis. Select one organism from the list and describe its biological characteristics, transmission, lab diagnosis, and treatment
* Neisseria Meningitidis
* Strept. Pneumonia
* H. Influenza type b
* Strept Agalactiae
* Staph. Aureus
* E. Coli
* Listeria Monocytogenes
* Mycobacterium tuberculosis
* Staph. Epidermidis
* Pseudomonas aureginosa
* Neisseria meningitidis:
	+ Biological characteristics
		- Gram negative diplococci
		- Have a polysaccharide capsule
		- Are facultative anaerobes
		- Have an endotoxin that is a LPS
		- Has 13 serological groups
		- Are Oxidase positive, catalase positive
		- Are non – motile, non spore
	+ Transmission: via respiratory droplets
	+ Lab diagnosis
		- Smear and culture of blood and spinal fluid specimens
		- Microscopy of specimen yield gram negative cocci.
		- Culture in CBA anaerobic at 37 degrees and 5 % CO2.
		- Colonial morphology grayish white, non pigmented convex shaped colonies
		- Biochemical test: Carbohydrate fermentation tests. Ferments maltose and glucose
		- Catalase positive
		- Oxidase positive colonies confirm.
		- Serological tests: Latex agglutionation test for capsular antigen
		- ELISA test for antibody to
		- Immunofluoresence assays
	+ Treatment:
		- Third generation cephalosporins: ceftriaxone plus a fluoroquinolone such as doxycycline
		- Penicillin G, sulfonamides and ciprofloxacin
1. Using
	1. A specific viral agent, distinguish between antigenic drift and shift
		* Antigenic shift entails reassortment of viral genome with the end result being emergence of new strains of virus, for Influenza virus for instance, antigenic shift is possible only in Influenza A, and is thought to be possible due to the segmentation of viral genome
		* It occurs when different strains of Influenza virus mix, for instance a human strain of influenza infects chicken that already has its own avian strain still present would cause reassortment of the RNA segments to yield a new strain
		* Antigenic drift on the other hand entails mutations of the RNA, causing minor variations in strains. Mutation occurs in type A and B, rarely in C, and mostly affects Haemagglutinin
	2. Compare and contrast the virology and pathogenesis of measles and rubella viral agents

|  |  |
| --- | --- |
| Measles | Rubella |
| * Belongs to paramyxoviridae family
* Belongs to genus morbilivirus
* Has a negative sense RNA genome
* Has a viral RNA polymerase
* Has a helical nucleocapsid
* Surface spike has Haemagglutinin and fusion protein
* More likely to cause a still than congenital malformations
* Causes Cytopathic effect such as multinucleated Giant cell formation
* Involvement of the CNS causing encephalitis
* Has a longer period of onset about 3 weeks
 | * Belong to Togaviridae family
* Belongs to genus rubivirus
* Has a positive sense RNA genome
* Has no virion polymerase
* Has an icosahedral nucleocapsid
* Surface spike has only Haemagglutinin
* May cause congenital malformations rarely a miscarriage
* Cause little Cytopathic effect
* Has a shorter milder disease on set of 2 weeks
* Rarely involves the CNS
 |

* Similarities
	+ Both are RNA viruses
	+ Both have a envelope
	+ Both cause a maculopapular rash
	+ Both are transmitted via respiratory droplets
	1. Short notes on the lab diagnosis of dermatophyte infections
* Specimen to be used: Skin scrapings, nail clippings
* Treatment of specimen using 10 % KOH that dissolves most tissues
* Direct Microscopic observation for athrospores and athroconidia
* Culture in SDA – Saboraud Dextrose Agar usually 10 – 14 days
* Lactophenol blue staining of a portion of culture, identification of microconidia for microsporum species, and macroconidia for M. canis
	1. Short notes on pseudomonas aureginosa
* Gram negative bacillus
* Are strict aerobes
* Non - fermenters
* Oxidase positive
* Are motile with polar flagella
* Has an antiphagocytic polysaccharide slimy layer
* Has a LPS like any other gram negative rod
* Produces pyocyanin, a blue – green pigment hence appearance of its colonies cyanin like
* Produces toxin A and exoenzyme S
1. Describe the life cycle, clinical presentation, diagnosis and control of schistosoma mansonia infection in man
* Life cycle:
	+ Has snails of the Biomphalaria species as the intermediate host and humans as the definitive host.
	+ Cycle begins with skin penetration of free swimming cerceriae in water into a susceptible host during swimming for instance.
	+ As the cerceriae penetrate, they lose their tail and become a schistosomule
	+ The schistosomule enter into the circulation, transported via into arterial circulation, transported via the superior mesenteric artery into portal circulation to liver where they mature into adults. S. mansoni adult species migrate against portal circulation to the inferior mesenteric veins . Adults located in these vessels lay eggs. The eggs penetrate the vessels into the lumen of the large intestines where they are passed out with faeces
	+ Eggs hatch out in water to form miracidium that are picked up by water snails of the biomphalaria species. Miracidium develop into rediae finally to mature cercaeria. These cerceriae are released into the water where they would infect another susceptible host
* Clinical presentation
	+ Acute stage: commences after cerceriae penetration and include swimmers itch “ dermatitis” pruritic papules
	+ Lypmphadenopathy, lymphaganitis
	+ hepatomegaly
	+ Eosinophilia
	+ Chronic stage: cause more morbidity and mortality : presents as gastrointestinal hemorrhages, exsanguinations within the esophagus, massive splenomegaly
* Lab diagnosis
	+ Detailed patient history for epidemiological diagnosis
	+ Specimen: Stool sample for s. mansonia
	+ Specimen concentration techniques such as Kato Katz technique
	+ Wet preparation of the stool samples.
	+ Observation for eggs. Presence of eggs with prominent terminal spine diagnostic for s. mansoni
	+ Moderate eosinophilia may occur
* Control:
	+ - Proper disposal of human faeces
		- Avoid use of human faeces as fertilizer
		- Chlorination of water sources
		- Molluscicides to eliminate intermediate host
		- Swimming in endemic areas should be avoided
1. Short essays
	1. T. vaginalis
* A pear shaped trophozoite. Exist only in the trophozoite stage, has no cyst stage
* Belongs to the family zoomastigophora (mastigophora)
* Has 4 anterior flagellas
* Has a single centrally located nucleus
* Has an undulating membrane that extends about 2/3 of its length
* Implicated as a cause of the sexually transmitted disease trichomoniasis
	1. Medical importance of ticks
* Ticks serve as vectors in the transmission of a number of disease causing microorganisms as shown below.
* Borrelia Burgdoferi, cause of borreliosis or lyme disease, is transmitted by a tick, Ixodes scapularis
* Tick typhus, causative organism rickettsia rickettsii ,Rocky mountain spot fever, is transmitted by a tick, Dermacentor variabilis
* Colorado tick fever virus, is transmitted by a tick, Dermacentor andersoni
* [**Babesiosis**](http://www.cdc.gov/babesiosis/) is transmitted by tick (Ixodes scapularis)
* [**Ehrlichiosis**](http://www.cdc.gov/ehrlichiosis/) is transmitted to humans tick (Ambylomma americanum)
* Tularemia, transmitted to humans by tick, dermacentor variabilis
* Anaplasmosis, transmitted by the tick ixodes scapularis
	1. Life cycle, clinical presentation, lab diagnosis and treatment of trichuris trichiura
* Life cycle:
	+ Humans are the definitve host. Begins with ingestion of food or water contaminated with embryonated eggs.
	+ Eggs hatch within the small intestines into immature adults
	+ The immature forms migrate to the colon where
	+ Adults attach onto the mucosa of the colon.
	+ Male and female mate and female produces eggs
	+ Eggs are passed out in faeces
	+ These eggs embryonate in soil and during the next contamination of food or water sources, they are transmitted.
	+ The cycle from eggs to adults takes 3 months
* Clinical presentation
	+ A light infection maybe asymptomatic
	+ Heavy infestation cause;
	+ Abdominal discomfort, diarrhea, vomiting with anorexia
	+ Abdominal obstruction
	+ Complications include:
		- Appendicitis
		- Rectal prolapse
	+ Eosinophilia
	+ Vitamin A deficiency
* Lab diagnosis
	+ Examination of faeces for typical barrel shaped eggs with bipolar plugs
	+ Sigmoidoscopy to exams adults in the colon
	+ Ultrasonography of the intestines to detect the worms
	+ Serological tests for the trichuris antigen
* Treatment
	+ Albendazole
	+ Mebendazole
	1. Lab diagnosis of visceral leishmaniasis
* Identification of amastigotes in macrophages following spleen, liver, bone marrow or lymph node biopsy
* Organism could be cultured.
* Indirect inoculation of patients serum to mice and examination for disease symptoms
* Serological tests include a fourfold rise in IgG titre
* Indirect immunofluoresence may be positive
* Leishmanin skin test
1. List mechanisms used by pathogens to escape host immune response. For each of the following pathogens briefly describe the unique mechanism the used to evade destruction by the host immune mechanisms
* Through antigen masking using host antigens such as making its own glycolipids as it travels using host glycoproteins. Entamoeba hystolitica masks its surface antigens using host glycoproteins
* Through cyst formation by various parasites such as helminthes. These cysts are resistant to immune responces. Hydatid cyst formed by echinococcus granulosus
* Residing in GIT lumen where they are sheltered from host Cell mediated immune response. Adults ascaris lumbricoides are located within the GIT lumen where they are safe from Cell Mediated immune responce
* Antigen variation that renders preformed antibodies ineffective towards the new surface antigenic mutations in the parasites. In plasmodium for instance, different stages express different antigens thus antibodies for a certain stage are ineffective for a subsequent stage. Giardia lamblia also exhibits antigenic variation at the various stages of its life cycle.
* Anatomical seclusion of the parasites in areas where they are sheltered from host immune defences, such as in the case of plasmodium species residing within red blood cells. Leishmania species and trypanosomes reside within macrophages.
* Induction of immune suppression by the parasites rendering the immune response absent is strategy that plasmodium species employs in evasion of immune response
* Prevention of fusion of the phagolysome (Phagosome and lysosome fusion) is prevented by lesishmania species thus escaping the destructive lysosomal enzymes.
* Production of parasitic enzymes that have anti immune effect. Filarial worms produce glutathione oxidase and superoxide dismutase that enables them escape antibody dependent cellular cytotoxicity and oxidative stress.

Questions 2007

1. List
	1. 3 viral agents associated with childhood fevers
* Measles virus
* mumps virus
* Rubella virus
	1. Pick one of the viral agents listed above and discuss the viral agent under the following topics
* Measles virus
	+ 1. Viral classification
* Belongs to group V ( - sense RNA )
* A member of paramyxoviridae
* Belongs to genus morbillivirus
* Species: Measles virus
	+ 1. Virology
* Are negative sense RNA genome viruses
* Has a linear genome
* Has a helical nucleocapsid
* Possess a virion RNA polymerase
* Has an envelop
* Has Haemagglutinin and fusion proteins on the same envelope spike
* Has a single serotype
	+ 1. Pathogenesis
* Mode of transmission is via respiratory droplets
* Replication within the oropharynx
* Spreads to the lymphatics and into the blood stream to cause viremia
* Virus infects the reticuloendothelial cells weher it replicates
* Are transmitted to the skin via the blood
* The typical rash is due cytotoxic T lymphocytes attacking the virus infected vascular endothelial cells on the skin
* The fusion protein is responsible for its Cytopathic effect of multinucleated giant cell formation
	+ 1. Lab diagnosis
* Physical examination
* Isolation of virus on cell culture
* Serological tests: a rise IgG antibody titre by fourfold is indicative of infection
	+ 1. Prevention
* Through vaccination; a live attenuated virus is used
* Mass vaccination programmes of children and should be made a prerequisite
* Booster should be given after some time as immunity wades
1. Write short notes on the following
	1. Methods of controlling sterilization by heat
* Biological methods:
	+ Entail using the spores of bacillus streatothermophilus. Spores bacillus subtilis are used to test effectiveness of dry hot air ovens
	+ Spores of clostridium could also be used to test effectiveness of autoclave in sterilization
* Chemical methods
	+ Browne’s tubes use of glass tube that is labeled with dye which changes with temperature. Used to monitor autoclaving technique
	+ Bowie dick tape: Appearance of dark brown stripes would indicate satisfactory of the process
	1. Yersinia pestis
* Is a gram negative bacillus
* Exhibits bipolar staining
* safety pin like appearance
* Has a capsule that is easily lost during sample collection for lab diagnosis
* Has a very low infectious dose of about 1-10 organism
* Causative agent of plague
	1. Isolation and identification of M. Tuberculosis
* Specimen isolated include sputum, nasal washing, gastric washing, throat swabs, CSF, Blood
* Staining of specimen using carbolfuchsin (Ziehl Neelsen Acid fast staining)
* Also flouroscent staining to visualize organism and microscopy
* Auramine staining
* Treatment of specimen using NaOH and later concentrated via centrifugation.
* Culture in Lowenstein Jensen medium at 35 -37 degrees at 5% CO2 for 3 - 4 weeks
* Rapid culture broth used include BACTEC medium, grows within 5 days
* Acid fast organisms that present as red rods on a blue background indicative of M. tuberculosis
* Serological tests include: PPD skin test
	1. Cryptococcus neoformans
* It is an oval, budding yeast
* Surrounded by a wide polysaccharide capsule
* Has a narrow based bud
* It is not dimorphic, exist only in the yeast form
* Causative agent of cryptococcosis, and cryptococcal meningitis
* Causes opportunistic infection in immunocompromised individuals
1. Describe the life cycle, clinical presentation, diagnosis, treatment and control of E. Granulosus infection (Hydatid cyst)
* In the life cycle; dogs are the definitive host while sheep are the intermediate host, humans are normally dead end hosts
* Adults in the dog lay eggs that are excreted in feaces, the dog faeces contaminate the environment which the sheep feed on. Humans acquire organism via accidentally eating dogs faeces.
* In the intestines of sheep, the oncospheric embryo penetrate and enter circulation where they are transported to the lungs, bones, liver and brain
* The embryos develop into large fluid filled sacs called Hydatid cysts. The inner germinal layer of the cyst generated protoscolices.
* The cycle is completed when the entrails of the sheep are fed to the dog.
* Clinical presentation
	+ Swelling
	+ Hepatomegally
	+ Enlargement of the abdomen
	+ Symptoms of space occupying lesions such as paralysis, jaundice if cysts cause biliary obstruction
	+ Cysts in liver can cause hepatic dysfunction
	+ Rapture of cysts can induce a serious anaphylactic reaction
* Diagnosis
	+ History
	+ Physical examination
	+ Ultrasound of liver for presence of Hydatid cysts
	+ Serological tests for worm antigen
	+ X rays for presence of cysts in tissues, lung as well
	+ Radiological examination
	+ CT scan of brain for cysts in brain
	+ Skin test: cansoni skin test
* Treatment
	+ Surgical removal of the cysts
	+ Administration of albendazole
* Control
	+ Proper disposal of consumable meat viscera
	+ Regular deworming of dogs
	+ Avoid feeding of dogs the entrails of sheep
	+ Fencing of slaughter house from dogs
1. Write short essays on
	1. Complications of ascaris lumbricoides infection
* Appendicitis
* Intestinal obstruction
* Cholecystitis:
* biliary ascaris
* Intestinal perforation
* Peritonitis
* Pancreatitis
	1. Diagnosis of visceral leishmaniasis (Kala azar)
* Identification of amastigotes in macrophages following spleen, liver, bone marrow or lymph node biopsy
* Organism could be cultured.
* Indirect inoculation of patients serum to mice and examination for disease symptoms
* Serological tests include a fourfold rise in IgG titre
* Indirect immunofluoresence may be positive
* Leishmanin skin test
	1. Medical importance of mosquitoes
* Implicated for transmitting a number of diseases that include
	+ Malaria caused by plasmodium species and is transmitted by the mosquito anopheles
	+ Rift valley fever virus is transmitted by aedes agypti and aedes albopictus
	+ Dengue fever virus is transmitted by aedes agypti
	+ Wuchereria bancrofti, brugia malayi and brugia timori, these filarial worms that cause elephantiasis are transmitted by the anopheles species, culex species as well
	+ Rift valley fever virus by aedes species
	+ St. Louis Encephalitis transmitted by culex species
	1. Control of African trypanosomiasis
* Wearing of protective clothing and sleeping under nets to avoid bite of the glossina species of fly
* Use of insecticides to kill glossina species that serve as vectors
* Clearing of bushes and draining of stagnant water that serve as breeding grounds for the fly
* Sterile insect technique, involving the mating of female with sterile male rendering discontinuity of generations of the fly.
* Use of targets and traps to weaken the tsetsefly and so contribute towards control.
1. Discuss
	1. List both innate and specific host defence mechanisms which are involved during a viral infection
* Innate components include:
	+ Inteferons and Natural killer cells
	+ Interferons are secreted by virally infected cells as well as inflammatory cells
	+ Mode of action of interferons include, prevention of replication of the virus within cell
	+ And is involved in the activation of NK cells that kill virally infected cells
* Specific components include
	+ Humoral immunituy
		- Antibodies
			* Neutralize free virus particles
			* Prevent attachment of virus to its receptors on the cells
			* Cause agglutiniation of virus particles
			* Opsonize the virus particles
			* Complement mediated lysis
		- Cell mediated immunity
			* Mediated by the T helper cells that recognizes viral antigens and secretes cytokines that activate macrophages and cytotoxic T lymphocytes
			* cytotoxic T cells kill virally infected cell based on the presentation of viral antigens on MHC class I
			* Macrophages are activated to phagocytose virally infected cells
			* Natural Killer cells are activated to kill virus infected cell
			* Antibody dependent cellular cytotoxicity depends on antibody to bind to virus and then facilitate its removal via Phagocytosis via macrophages, NK cells or polymorphonuclear leucocytes
	1. Discuss the methods viruses have therefore adopted to evade these innate and specific host defence mechanisms
* Antigenic drift
	+ Entails random mutation in the viral genes rendering preformed antibodies ineffective. An instance is minor changed in the HA and NA proteins in influenza virus
* Antigenic shift
	+ Entails reassortment of viral genome, following mixing of genes that result in emergence of new viral strains to which the body has no immunity to
* Interference with interferon synthesis
	+ Viruses may block interferon induced transcription responses
* Inhibition and modulation of cytokines and chemokines
	+ Cytokines required for activation of effector immune cells may be inhibited by virus
* Inhibition of apoptosis
	+ Virus infected cells programmed for cell death are prevented from achieving the goal
* Immunomodulation of the lymphocytes and other immune cells
	+ Viruses such as CMV induces cell mediated immunosuppression
* Viruses may suppress expression of the MHC class I on virally infected cells hence host surveillance system is rendered blind to see virally infected cells
* Inhibition of inflammation. Without inflammation, the virus escapes detection by the host immune system.