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By

Associat Professor Dr. Nada Khazal Kadhim Hindi

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The Content

Chapter 4: Mycology (67)

- (definition, morphology and classification of fungi)
- **Cutaneous, subcutaneous**, **systemic**, **opportunistic** mycosis
- Dermatophytoses (**Tinea pedis, Tinea corporis, Tinea capitis, Tinea cruris, Tinea unguium, Tinea Barbae,** Epidemiology, Pathology, Clinical significance, diagnosis and treatment)
- **Subcutaneous** (**Sporotrichosis, Chromomycosis, Mycetoma** Epidemiology, Pathology, Clinical significance, diagnosis and treatment)
- **Systemic Mycoses (Coccidioidomycosis, Histoplasmosis Blastomycosis**, **Paracoccidioidomycosis** Epidemiology, Pathology, Clinical significance, diagnosis and treatment)
- **Opportunistic Mycoses (Candidiasis, Cryptococcosis, Aspergillosis, Mucormycosis**, **Pneumocystis** Epidemiology, Pathology, Clinical significance, diagnosis and treatment)

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- **Introduction to Parasitology:**
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- Classification of parasites.
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- **Intestinal flagellates: *Giardia lamblia*** (Morphology, habitat, epidemiology,pathogenesis, methods of transmission, diagnosis, control and treatment).
- Atrial flagellates: ***Trichomonas vaginalis*** (Morphology, habitat, pathogenesis, methods of transmission, diagnosis and treatment).
- Tissue flagellates: **Leishmania** spp. (Morphology, habitat, epidemiology, and pathogenesis, methods of transmission, diagnosis, control and treatment for each species).
- **Sporozoa: Plasmodium** spp. (Morphology, habitat, epidemiology, pathogenesis, methods of transmission, diagnosis, control and treatment).
- ***Toxoplasma gondii*** (Morphology, transmission, pathogenesis, diagnosis, control and treatment).

Helminthes (Cestoda, Trematode, Nematode)

- **Cestoda** (*Taenia solium, Taenia sagenata, D.latum, Hymenolepis nana and Echinococcus granulosus*) Morphology, habitat, epidemiology, pathogenesis, methods of transmission, diagnosis, control and treatment.
    - **Trematoda**: (*Fasciola hepatica, Fasciolopsis buski Schistosoma haematobium, Schistosoma mansoni, Schistosoma japonicum)* Morphology, habitat, epidemiology, pathogenesis, methods of transmission, diagnosis, control and treatment).
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Chapter 6:Virology and vaccines (117)

- Interoduction (definition, morphology and structure of viruses)
    Pathogenic Viruses (**Measles, Rubella, small pox, mumps, rabies viruses, influenzae A,B, poliomyelitis, herpes viruses, Retero viruses(HIV), Reo viruses, Hepatitis A, BC,D**), and other viruse family
- Vaccines (types of vaccines, definition, type of immunity)
- live, attenuated microorganisms;
- killed microorganisms;
- microbial extracts;
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Chapter:four
Fungi (Mycosis)

Fungi (Mycosis)

They are a diverse group of saprophytic and parasitic eukaryotic organisms. Human fungal diseases (mycoses) are classified by the location on or in the body where the infection occurs. They are called **cutaneous** when limited to the epidermis, **subcutaneous** when the infection penetrates significantly beneath the skin, and **systemic** when the infection is deep within the body or disseminated to internal organs. **Systemic mycoses** can be further divided into those that are caused by **true pathogenic fungi** capable of infecting healthy individuals, and those that are **opportunistic**, infecting primarily those individuals who have predisposing conditions such as immunodeficiency or debilitating diseases. Fungi produce and secrete a variety of unusual metabolic products, some of which, when ingested, are highly toxic to animals, including humans. Thus, fungi can cause poisonings as well as infections. Lastly, fungal spores are important as human allergenic agents.

Characteristics of Major Fungal Groups

Fungi can be distinguished from other infectious organisms such as bacteria or viruses because they are eukaryotes (that is, they have a membrane-enclosed nucleus). Their characteristic structures, habitats, and modes of growth and reproduction are used to distinguish between different groups of fungi.

A. Cell wall and membrane components

The fungal cell wall and cell membrane are fundamentally different from those of bacteria and other eukaryotes. Fungal cell walls are composed largely of chitin, the fungal membrane contains ergosterol, rather than the cholesterol found in mammalian membranes.

B. Habitat and nutrition

All fungi are heterotrophs; that is, they require some preformed organic carbon source for growth. Fungi do not ingest food particles as do organisms such as protozoa, but depend upon transport of soluble nutrients across their cell membranes. To obtain these soluble nutrients, fungi secrete degradative enzymes (for example, cellulases, proteases, nucleases) into their immediate environment. It is this ability that enables fungi to live saprophytically on organic waste. Therefore, the natural habitat of almost all fungi is soil or water containing decaying organic matter. [Note: Some fungi can be parasitic on living organisms. However, these parasitic infections usually originate from the individual's contact with fungus-infested soil, an exception being Candida, which is part of the normal human mucosal flora

C. Modes of fungal growth

Most fungi exist in one of two basic morphologic forms (that is, either as filamentous mold or unicellular yeast). However, some fungi are dimorphic (that is, they switch between these two forms in response to environmental conditions).

1-**Filamentous (mold like) fungi**: The vegetative body, or thallus, of mold-like fungi is typically a mass of threads with many branches. This mass is called a mycelium, which grows by branching and tip elongation. The threads (hyphae) are actually tubular cells that, in some fungi, are partitioned into segments (septate); whereas, in other fungi, the hyphae are uninterrupted by crosswalls (nonseptate). Even in septate fungi, however, the septae are perforated so that the cytoplasm of the hyphae is continuous. When hyphal

filaments become densely packed, the mycelium may have the appearance of a cohesive tissue. An example of this is the body of a mushroom.

2-Yeast-like fungi: These fungi exist as populations of single, unconnected, spheroid cells, not unlike many bacteria, although they are some ten times larger than a typical bacterial cell. Yeast-like fungi generally reproduce by budding.

Some fungal species, especially those that cause systemic mycoses, are dimorphic, being usually yeast-like in one environment and mold-like in another. Examples of conditions that affect the choice of morphology are temperature and carbon dioxide levels.

D. Sporulation

Sporulation is the principal means by which fungi reproduce and spread through the environment. Fungal spores are metabolically dormant, protected cells, released by the mycelium in enormous numbers. They can be borne by air or water to new sites, where they germinate and establish colonies. Spores can be generated either asexually or sexually

Asexual sporulation: Asexual spores (conidia) are formed by mitosis in or on specialized hyphae (conidiophores,). The color of a typical fungal colony seen on bread, fruit, or culture plate is caused by the conidia; conidia can become airborne and, therefore, are a major source of fungal infection.

Sexual sporulation: This process is initiated when a haploid nucleus from each of two compatible strains of the same species fuse to form a transient diploid. The products of meiosis of this transient diploid become sexual spores (ascospores). Spores, especially sexual spores, often have a characteristic shape and surface ornamentation pattern that may serve as the primary or only means of species identification.

E. Laboratory identification

Most fungi can be propagated on any nutrient agar surface. The standard medium is Sabouraud dextrose agar, which, because of its low pH (5.0), inhibits bacterial growth while allowing fungal colonies to form. Various antibacterial antibiotics can also be added to the medium to further inhibit bacterial colony formation. Cultures can be started from spores or hyphal fragments. Clinical samples may be pus, blood, spinal fluid, sputum, tissue biopsies, or skin scrapings. Identification is usually based on the microscopic morphology of conidial structures. Serologic tests and immunofluorescent techniques are also useful in identification of fungi from clinical isolates.

1- Cutaneous Mycoses (Dermatophytes)

Also called dermatophytoses, these common diseases are caused by a group of related fungi, the dermatophytes. Dermatophytes fall into three genera, each with many species: *Trichophyton, Epidermophyton, and Microsporum.*

Epidemiology

The causative organisms of the dermatophytoses are often distinguished according to their natural habitats: anthropophilic (residing on human skin), zoophilic (residing on the skin

of domestic and farm animals), or geophilic (residing in the soil). Most human infections are by anthropophilic and zoophilic organisms. Transmission from human to human or animal to human is by infected skin scales.

Pathology
A defining characteristic of the dermatophytes is the ability to use keratin as a source of nutrition. This ability allows them to infect keratinized tissues and structures, such as skin, hair, and nails. There is some specificity, however. Whereas all three genera attack the skin, Microsporum does not infect nails and Epidermophyton does not infect hair. None invade underlying, nonkeratinized tissue.

Clinical significance
Dermatophytoses are characterized by itching, scaling skin patches that can become inflamed and weeping. Specific diseases are usually identified according to affected tissue (for example, scalp, pubic area, or feet), but a given disease can be caused by any one of several organisms, and some organisms can cause more than one disease depending, for example, on the site of infection or condition of the skin.

The following are the most commonly types of dermatophytoses

A. Tinea pedis (athlete's foot): Organisms most often isolated from infected tissue are Trichophyton rubrum, Trichophyton mentagrophytes, and Epidermophyton floccosum. The infected tissue is initially between the toes, but can spread to the nails, which become yellow and brittle. Skin fissures can lead to secondary bacterial infections, with consequent lymph node inflammation.

B. Tinea corporis (ringworm): Organisms most often isolated are E. floccosum and several species of Trichophyton and Microsporum. Lesions appear as advancing annular rings with scaly centers. The periphery of the ring, which is the site of active fungal growth, is usually inflamed and vesiculated. Although any site on the body can be affected, lesions most often occur on nonhairy areas of the trunk.

C.Tinea capitis (scalp ringworm): Several species of Trichophyton and Microsporum have been isolated from scalp ringworm lesions, the predominant infecting species depending on the geographic location of the patient. In the United States, for example, the predominant infecting species is Trichophyton tonsurans. Disease manifestations range from small, scaling patches, to involvement of the entire scalp with extensive hair loss. The hair shafts can become invaded by Microsporum hyphae, as manifested by their green fluorescence in long-wave ultraviolet light (Wood lamp).

D. Tinea cruris (jock itch): Causative organisms are E. floccosum and T. rubrum. Disease manifestations are similar to ringworm, except that lesions occur in the moist groin area, where they can spread from the upper thighs to the genitals.

E. Tinea unguium (onychomycosis): The causative organism is most often T. rubrum. The nails are thickened, discolored, and brittle. Treatment must be continued for three to four months until all infected portions of the nail grow out and are trimmed off.

F. **Tinea Barbae**: The causative organism is most often Trichophyton. Edematous erythematous lesion in beard hair

Treatment

Removal of infected skin, followed by topical application of antifungal antibiotics such as miconazole or clotrimazole, is the first course of treatment. Refractory infections usually respond well to oral griseofulvin and itraconazole. Infections of the hair and nails usually require systemic (oral) therapy. Terbinafine is the drug of choice for onychomycosis.

Table: types of mycosis

Skin Disease	Location of Lesions	Clinical Features	Fungi Most Frequently Responsible
Tinea corporis (ringworm)	Nonhairy, smooth skin	Circular patches with advancing red, vesiculated border and central scaling. Pruritic	*Trichophyton rubrum, Epidermophyton floccosum*
Tinea pedis (athlete's foot)	Interdigital spaces on feet of persons wearing shoes	Acute: itching, red vesicular. Chronic: itching, scaling, fissures	*Trichophyton rubrum, Trichophyton mentagrophytes, Epidermophyton floccosum*
Tinea cruris (jock itch)	Groin	Erythematous scaling lesion in intertriginous area. Pruritic	*Trichophyton rubrum, Trichophyton mentagrophytes, Epidermophyton floccosum*
Tinea capitis	Scalp hair. Endothrix: fungus inside hair shaft. Ectothrix: fungus on surface of hair	Circular bald patches with short hair stubs or broken hair within hair follicles. Kerion rare. *Microsporum*-infected hairs fluoresce	*Trichophyton mentagrophytes, Microsporum canis, Trichophyton tonsurans*
Tinea barbae	Beard hair	Edematous, erythematous lesion	*Trichophyton mentagrophytes, Trichophyton rubrum, Trichophyton verrucosum*
Tinea unguium (onychomycosis)	Nail	Nails thickened or crumbling distally; discolored; lusterless. Usually associated with tinea pedis	*Trichophyton rubrum, Trichophyton mentagrophytes, Epidermophyton floccosum*
Dermatophytid (id reaction)	Usually sides and flexor aspects of fingers. Palm. Any site on body	Pruritic vesicular to bullous lesions. Most commonly associated with tinea pedis	No fungi present in lesion. May become secondarily infected with bacteria

Table: types of dermatophytes

Category	Mycosis	Causative Fungal Agents
Superficial	Pityriasis versicolor	*Malassezia* species
	Tinea nigra	*Hortaea werneckii*
	White piedra	*Trichosporon* species
	Black piedra	*Piedraia hortae*
Cutaneous	Dermatophytosis	*Microsporum* species, *Trichophyton* species, and *Epidermophyton floccosum*
	Candidiasis of skin, mucosa, or nails	*Candida albicans* and other *Candida* species
Subcutaneous	Sporotrichosis	*Sporothrix schenckii*
	Chromoblastomycosis	*Phialophora verrucosa, Fonsecaea pedrosoi,* and others
	Mycetoma	*Pseudallescheria boydii, Madurella mycetomatis,* and others
	Phaeohyphomycosis	*Exophiala, Bipolaris, Exserohilum,* and other dematiaceous molds
Endemic (primary, systemic)	Coccidioidomycosis	*Coccidioides posadasii* and *Coccidioides immitis*
	Histoplasmosis	*Histoplasma capsulatum*
	Blastomycosis	*Blastomyces dermatitidis*
	Paracoccidioidomycosis	*Paracoccidioides brasiliensis*
Opportunistic	Systemic candidiasis	*Candida albicans* and many other *Candida* species
	Cryptococcosis	*Cryptococcus neoformans* and *Cryptococcus gattii*
	Aspergillosis	*Aspergillus fumigatus* and other *Aspergillus* species
	Hyalohyphomycosis	Species of *Fusarium, Paecilomyces, Trichosporon,* and other hyaline molds
	Phaeohyphomycosis	*Cladophialophora bantiana*; species of *Alternaria, Cladosporium, Bipolaris, Exserohilum* and numerous other dematiaceous molds
	Mucormycosis (zygomycosis)	Species of *Rhizopus, Lichtheimia, Cunninghamella,* and other zygomycetes
	Pneumocystis pneumonia	*Pneumocystis jiroveci*
	Penicilliosis	*Penicillium marneffei*

2-Subcutaneous Mycoses

Subcutaneous mycoses are fungal infections of the dermis, subcutaneous tissue, and bone. Causative organisms reside in the soil and decaying or live vegetation. Subcutaneous fungal infections are almost always acquired through traumatic lacerations or puncture wounds, often acquired from the prick of a thorn. As expected, these infections are more common in individuals who have frequent contact with soil and vegetation and wear little protective clothing. The subcutaneous mycoses are not transmissible from human to human under ordinary conditions.

Clinical Significance

A.Sporotrichosis: The causative organism, Sporothrix schenckii, is a dimorphic fungus that exhibits the yeast form in infected tissue. This infection, characterized by a granulomatous ulcer at the puncture site, may produce secondary lesions along the draining lymphatics. The disease is self-limiting, but may persist in a chronic form. Oral itraconazole is the drug of choice.

B.**Chromomycosis (also called chromoblastomycosis):** This infection is characterized by warty nodules that spread slowly along the lymphatics and develop crusty abscesses Pathogens causing this mycosis include several species of pigmented soil fungi, for

example, Phialophora and Cladosporium, and the infection is most commonly seen in the tropics.

C. Mycetoma (Madura foot): Mycetoma appears as a localized abscess on the feet, & Sinusitis (discharges pus, serum, and blood through sinuses channel). The infection can spread to the underlying bone and results in crippling deformities. The pathogenic agents are various soil fungi or actinomycetes, depending on the climate of the geographic area. Most common are Madurella grisea and Actinomadura madurae. Mycetomas appear similar to the lesions of chromomycosis, but the defining characteristic of mycetoma is the presence of colored grains, composed of compacted hyphae, in the exudate. The color of the grains (black, white, red, or yellow) is characteristic of the causative organism and, therefore, useful in identifying the particular pathogen. There is no effective chemotherapy for fungal mycetoma; the treatment is usually surgical excision.

3.Systemic Mycoses

The organisms responsible for systemic mycoses fall into two general categories: 1) those that infect normal healthy individuals (pathogens), and 2) those that primarily infect debilitated, and/or immunocompromised individuals (opportunistic pathogens). **Coccidioidomycosis, histoplasmosis, and blastomycosis are the most common systemic mycotic infections in the immunocompetent host,** while **paracoccidioidomycosis causes infection in immunocompromised patients**. These infections occur in defined geographic areas where fungal pathogens are found in the soil and can be aerosolized. Clinical manifestations closely resemble those seen in tuberculosis in that asymptomatic primary pulmonary infection is common, whereas chronic pulmonary or disseminated infection is rare. The fungi causing these diseases are uniformly dimorphic, exhibiting the yeast form in infected tissue, and the mycelial form in culture or in their natural environment.

Epidemiology and pathology

Entry into the host is by inhalation of airborne spores, which germinate in the lungs. From the lungs, dissemination can occur to any organ of the body where the fungi can invade and destroy tissue. In spite of the seemingly grave nature of potentially systemic disease, most cases of coccidioidomycosis, histoplasmosis, and paracoccidioidomycosis in otherwise healthy patients present only mild symptoms and are self-limiting. In immunosuppressed patients, however, the same infections can be life-threatening.

Clinical significance

Coccidioidomycosis is caused by Coccidioides immitis, in cases of disseminated disease, lesions occur most often in the bones and the central nervous system (CNS) & causes meningitis. Most cases of coccidioidomycosis occur in the arid areas of southwestern United States and Central and South America. In the soil, the fungus generates spores by septation of hyphal filaments (arthrospores). These spores become readily airborne and enter the lungs, where they germinate and develop into large (twenty to forty µm) spherules filled with many endospores. Rupture of the spherule releases the endospores, each of which can form a new spherule.

A. **Histoplasmosis**: Pulmonary infections is caused by Histoplasma capsulatum. In the soil, the fungus generates conidia, which, when airborne, enter the lungs and germinate into yeast-like cells. These yeast cells become engulfed by macrophages in which they multiply. Pulmonary infections may be acute but relatively benign and self-limiting, or chronic, progressive, and fatal. Dissemination is rare. Disseminated disease results in invasion of cells of the reticuloendothelial system, which distinguishes this organism as the only fungus to exhibit intracellular parasitism. Definitive diagnosis is by isolation and culture of the organism, which is a slow process taking four to six weeks, or by detection of exoantigen, which can be completed in several days. The disease occurs worldwide, but is most prevalent in central North America. Soils that are laden with bird, chicken, or bat droppings are a rich source of H. capsulatum spores.

B. **Blastomycosis** is caused by Blastomyces dermatitidis. Like Histoplasma, the fungus produces microconidia, most often in the soil, which become airborne and enter the lungs. There they germinate into thick-walled yeast cells that often appear with buds. Initial pulmonary infections, rarely disseminate to other sites; however, when dissemination occurs, secondary sites include skin (seventy percent), bone (thirty percent), and genitourinary tract (twenty percent), where they manifest as ulcerated granulomas. Definitive diagnosis is accomplished by isolation and culture of the organism. Identifiable colonies can be obtained in one to three weeks, but identity can be established more rapidly by subjecting the young mycelial colonies to an exoantigen test. Infections are most common in the South Central and South Eastern United States.

C. **Paracoccidioidomycosis** also called South American blastomycosis, is caused by Paracoccidioides brasiliensis. The clinical presentation is much like that of histoplasmosis and blastomycosis except that the most common secondary site of infection is the mucosa of the mouth and nose, where painful, destructive lesions may develop. Like other dimorphic pathogens, morphologic identification via conidia is slow, but the yeast form observed in infected tissue or exudates has a characteristic ship's steering wheel appearance caused by the presence of multiple buds . The disease is restricted to Central and South America, and over ninety percent of patients with symptomatic disease are mature males. It is speculated that female sex hormones may inhibit formation of the yeast form.

Treatment

Systemic mycoses are usually treated with amphotericin B, sometimes in combination with flucytosine. Ketoconazole, fluconazole, and itraconazole are also used, depending on the stage and site of the disease.

4-Opportunistic Mycoses

Opportunistic mycoses afflict debilitated or immunocompromised individuals, and are rare in healthy individuals. The use of immunosuppressive drugs for organ transplantation, widespread use of chemotherapy in cancer treatment, and the high frequency of immunodeficient individuals caused by the AIDS epidemic have resulted in significant

expansion of the immunocompromised population, as well as increasing the spectrum of opportunistic fungal pathogens.

A.Candidiasis (candidosis) is caused by the yeast *Candida albicans*, which are normal body flora found in the skin, mouth, vagina, and intestines. Although considered a yeast, *C. albicans* is dimorphic, and can form a true mycelium. Infections occur when competing bacterial flora are eliminated, for example, by antibacterial antibiotics, allowing the yeast to overgrow. Candida infections have various manifestations depending on the site.

Oral candidiasis (thrush) presents as raised, white plaques on the oral mucosa, tongue, or gums. The plaques can become confluent and ulcerated and spread to the throat. Most HIV-positive individuals eventually develop oral candidiasis, which often spreads to the esophagus

Systemic candidiasis is a potentially life-threatening infection that occurs in debilitated individuals, cancer patients (with neutropenia), individuals on systemic corticosteroids, and patients treated with antibiotics. Systemic candidiasis may involve the gastrointestinal tract, kidneys, liver, and spleen. Candida can causes

Vaginal candidiasis; presents as itching and burning pain of the vulva and vagina, accompanied by a thick or thin white discharge.

Treatment: Both oral and vaginal infections are treated topically with nystatin or clotrimazole. Oral systemic antifungal agents such as ketoconazole, fluconazole, and itraconazole. Amphotericin B by itself or in combination with flucytosine is used in systemic disease.

B.Cryptococcosis is caused by the yeast Cryptococcus neoformans which is found worldwide. The organism is especially abundant in soil containing bird (especially pigeon) droppings, although the birds are not infected. The most common form of cryptococcosis is a mild,
subclinical lung infectionin in immunocompromised patients, the infection often disseminates to the brain and meninges, with fatal consequences. However, about half of patients with cryptococcal meningitis have no obvious immunologic defect.The organism has a characteristic thick capsule that surrounds the budding yeast cell.

C. Aspergillosis is caused by several species of the genus Aspergillus, but primarily by Aspergillus fumigatus. Aspergillus is rarely pathogenic in the normal host, but can produce disease in immunosuppressed individuals and patients treated with broad-spectrum antibiotics. The disease has a worldwide distribution. Aspergilli are ubiquitous, growing only as filamentous molds and producing prodigious numbers of conidiospores. They reside in dust and the soil, decomposing organic matter. In fact, hospital outbreaks affecting neutropenic patients (that is, those with decreased neutrophils in their blood) have been traced to dust from neighboring construction work. Aspergillosis manifests itself in several forms, depending in part on the immunologic state of health of the patient.
Acute aspergillus infections: The most severe, and often fatal, form of aspergillosis is acute invasive infection of the lung, from which the infection can be disseminated to the brain, gastrointestinal tract, and other organs. A less severe, noninvasive lung infection gives rise to a

fungus ball (aspergilloma), a mass of hyphal tissue that can form in lung cavities derived from prior diseases, such as tuberculosis. Although the lung is the most common primary site of infection, the eye, ear, nasal sinuses, and skin can also be primary sites.

D.Mucormycosis is caused most often by Rhizopus oryzae, like the aspergilli. Mucor infections occur worldwide, but are almost entirely restricted to individuals with some underlying predisposing condition, such as burns, leukemias, or acidotic states such as diabetes mellitus.

E. Pneumocystis jiroveci pneumonia is caused by the unicellular eukaryote, P. jiroveci (formerly, P. carinii). Before the use of immunosuppressive drugs and the onset of the AIDS epidemic, infection with this organism was a rare occurrence. It is one of the most common opportunistic diseases of individuals infected with HIV.

Diagnosis and treatment

Definitive diagnosis of an aspergillus infection is afforded by detection of hyphal masses, and isolation of the organism from clinical samples. Aspergillus hyphae characteristically form V-shaped branches (septate hyphae) that are distinguished from Mucor species. Also, septae are present in aspergillus hyphae but absent from mucor hyphae. In culture, the spore-bearing structures of the aspergilli are unmistakable but. Treatment of aspergillus infections is typically by amphotericin B and surgical removal of fungal masses or infected tissue. The antifungal drugs miconazole, ketoconazole, &itraconazole.

Chapter five

Parasitology

Parasitology

Introduction to Parasitology

Parasitology: is the science dealing with the study of protozoa & pathogenic effects.

Parasite: an organism that lives in or on anther organisms (host) and obtains its food from host.

Host: an organism which harbors parasite.
The parasites of medical importance fall into kingdom: Protista and animalia.
The parasites are classified as three phyla into

1. phylum: protozoa
2. phylum: platyhelminthus (cestoda)
3. phylum: nematode
4. phylum: trematode

Protozoa is single organism microscopic (belong to Protista). In contrast**, Helminthes** are multicellular organism or worm, macroscopic (belong to animalia). It possessing well differentiated tissues &organ system. The length of worm vary from less than millimeter to more than meter.

Classes of Protozoa:
Types of locomotion of organelle have been used to divide these into four major classes:

1. **Rhizopods (amoebae):** organelle of locomotion are pseudopodia and the mode of reproduce by binary fission. Such as *E. histolytica*
2. **Ciliaphora**: Organelles of locomotion are cilia and the mode of reproduce by binary fission. Such as *Blantidium coli*
3. **Mastigophora or flagellated**: organelle of locomotion are flagella and the mode of reproduce by binary fission.
4. **Sporozoa**: is non motile and reproduce by sporogony\schizogony.

The types of relationships between parasites and host

1. **Phoresis:** the parasite transport through the host with mechanism. *E. histolytica*
2. **Commensalism:** this relation positive for parasite while neutralized for host.
3. **Mutalism:** positive for parasite and host.
4. **Parasitism:** positive for parasite and negative for host**.**

The infected phases of parasites:
1. ovum. 2. larva. 3. cyst. 4. adult phase (worm).

Transmission of parasitic infection

1. **modes or portals of entry the host:**

Ingestion, inoculation, inhalation, congenital, venereal, and other.

2. **portals of exit from host:**

Respiratory tract, gastrointestinal tract, genital tract, biting insect, and allergy.

1. Class: Rhizopods (amoebae): *Entamoeba histolytica*& *Entamoeba coli*

Morphology

E. histolytica & *E. coli* living in intestinal.The live cycle consists of two stage: trophozoite & cyst. The morphology of cyst & troph. of *E. histolytica*& *E. coli* as shown in following table.

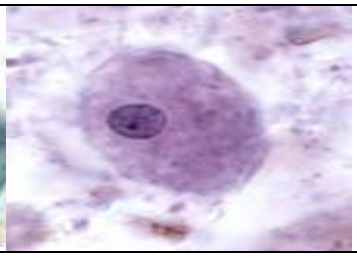
a protozoa, that infects predominantly humans and other mammals such as dogs and cats can become infected (the environmental survival form of the organism) with their feces. The active (trophozoite) stage exists only in the host and in fresh feces; cysts survive outside the host in water and soils and on foods, especially under moist conditions on the latter. When swallowed they cause infections by excysting (to the troph. stage) in the digestive tract.

Amebiasis (or amoebiasis) or amebic dysentery is the name of the infection caused by *E. histolytica*. In addition to infection of the large intestine, the organism may invade other internal organ such as the lung, liver, skin and brain.

Signs and symptoms amebic dysentery:

In severe cases of intestinal amebiasis, the organism invades the lining of the intestine, producing sores (ulcers), bloody diarrhea, severe abdominal cramps, vomiting, chills, and fevers as high (40°C). In addition, a case of acute amebic dysentery3 may cause complications, including inflammation of the appendix, a tear in the intestinal wall (perforation), or a sudden, severe inflammation of the colon (fulminating colitis).

Table: the comparison between trophozoite of *E. histolytica*& *E. coli*

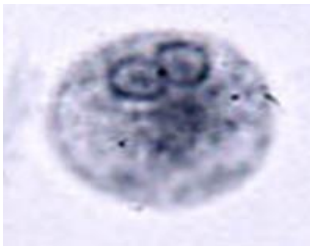
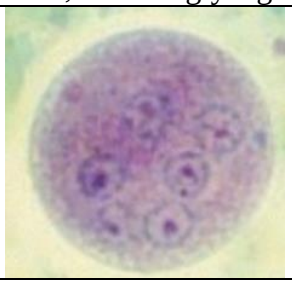
characteristic	**Troph.** of *E. histolytica*	**Troph.** of *E. coli*
Size	8-65µm	12-55µm
No. of nuclei	One	one
Karyosome	Small& centeral	Large irregular shape, eccentric
Peripheral chromatin	Fine& evenly distributed	Coarse& unevenly distributed
Cytoplasm	Finely granular	Coarse& often vacuolated
Cytoplasmic inclusion	Ingested RBC	Bacteria, other debris
Motility	Progressive, finger like pseudopodia	Non Progressive, blunt pseudopodia
Figure		

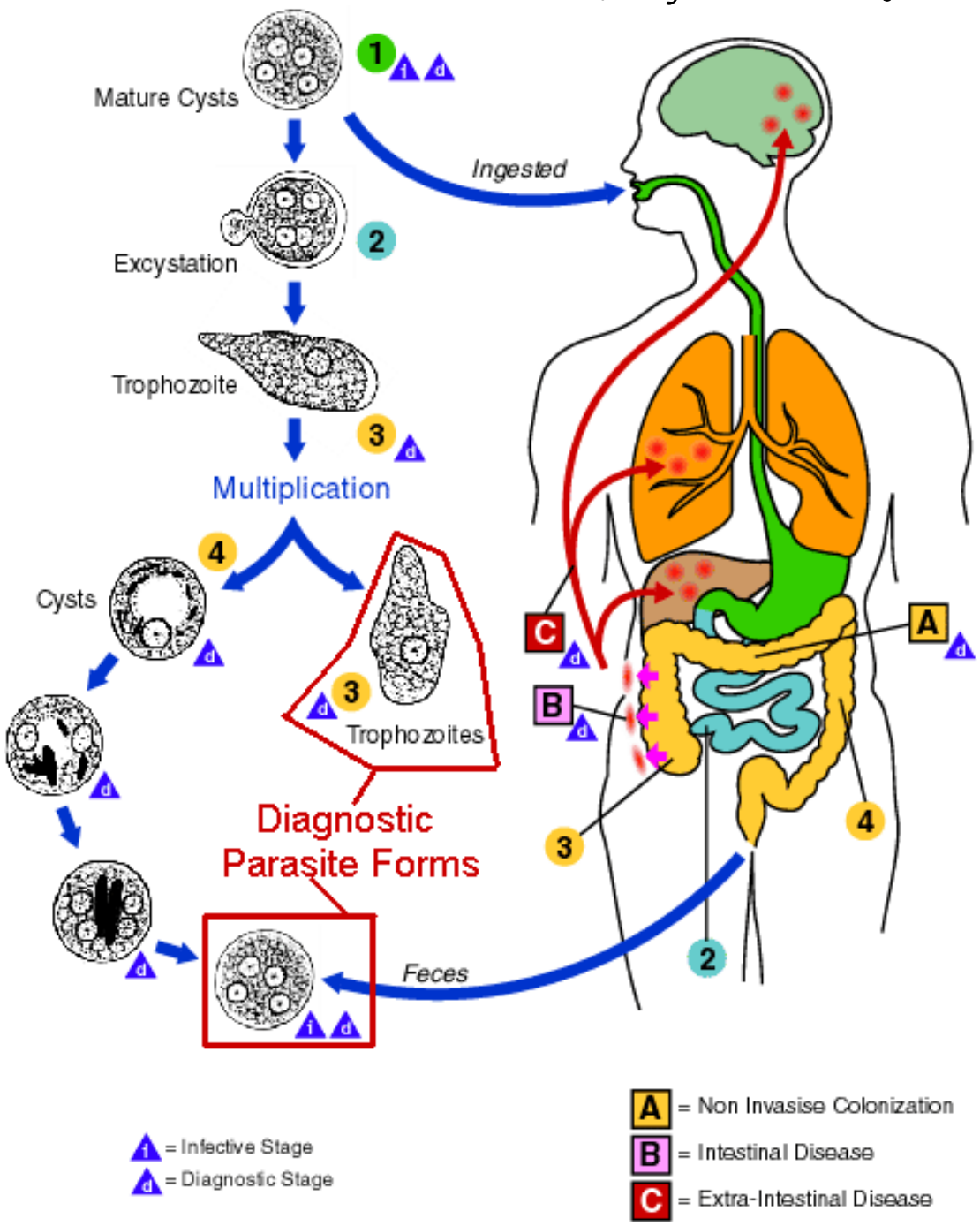
Entamoeba coli

is a non-pathogenic amoeba with worldwide distribution. Its life cycle is similar to that of *E. histolytica* but it does not have an invasive stage and does not ingest red blood cells.

Laboratory diagnosis of amebiasis is made by stool examination. **The diagnostic stages are troph. Or cyst or both in diarrhea stool. The infective stage is cyst.**

Table: the comparison between cyst of *E. histolytica* & *E. coli*

characteristic	**cyst** of *E. histolytica*	**cyst** of *E. coli*
Size	8-22µm	8-35µm
shape	Spherical to round	Spherical to round
No. of nuclei	One to four	One to eight
Karyosome	Small& centeral	Large irregular shape, eccentric
Peripheral chromatin	Fine& evenly distributed	Coarse
Cytoplasm	Finely granular	granular
Cytoplasmic inclusion	Chromatoid bars, rounded ends, diffuse glycogen mass	Chromatoid bars, rounded with pointed ends, diffuse glycogen mass
Figure		



Life cycle of *Entamoeba histolytica*

2. Class: Ciliaphora: *Blantidium coli*

B. coli have two types of nuclei: **macronucleus** that responsible for all activities of parasite except the reproduction, while **micronucleus** that responsible for the reproduction only.

B. coli live in digestive system. It cause blantidiasis similar ameobiasis but differ from *E. histolytica* that invade the liver. It has two phases: troph. & cyst.

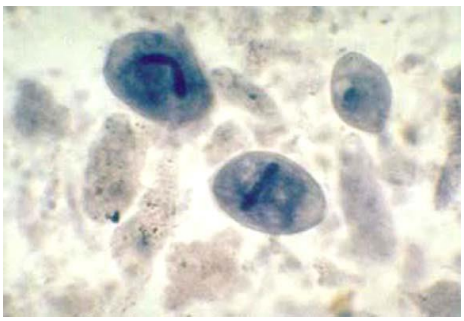
1. Troph.: found in large intestine is consider largest parasite of protozoa, ovule shape, covered with equal long cilia have two nuclei **macronucleus** (kidney shape) & **micronucleus** (vascular shape). It has two contracted vacuoles & many vacuoles contain bacteria or RBC in the acute infection with this parasite.
2. Cyst: spherical shape has thick cell wall but difficult to diagnostic nuclei.

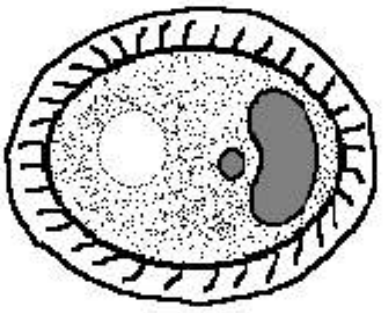
Clinical symptoms

Balantidiasis. Symptomatic patients may experience a variety of discomforts, ranging from mild colitis and diarrhea to full – blown clinical balantidiasis, which may often resemble amebic dysentery. In this case, abscesses and ulcers may form in the mucosa and submucosa of the large intestine followed by secondary bacterial infection. Acute infections are characterized by up to 15 liquid stools per day containing pus mucus, and blood. Patients who suffer from chronic infections may develop a tender colon, anemia, cachexia, and occasional diarrhea, alternating with constipation. *Balantidium coli* has been known to invade areas other than the intestine, such as the liver, lungs, pleura, mesenteric nodes, and urogenital tract.

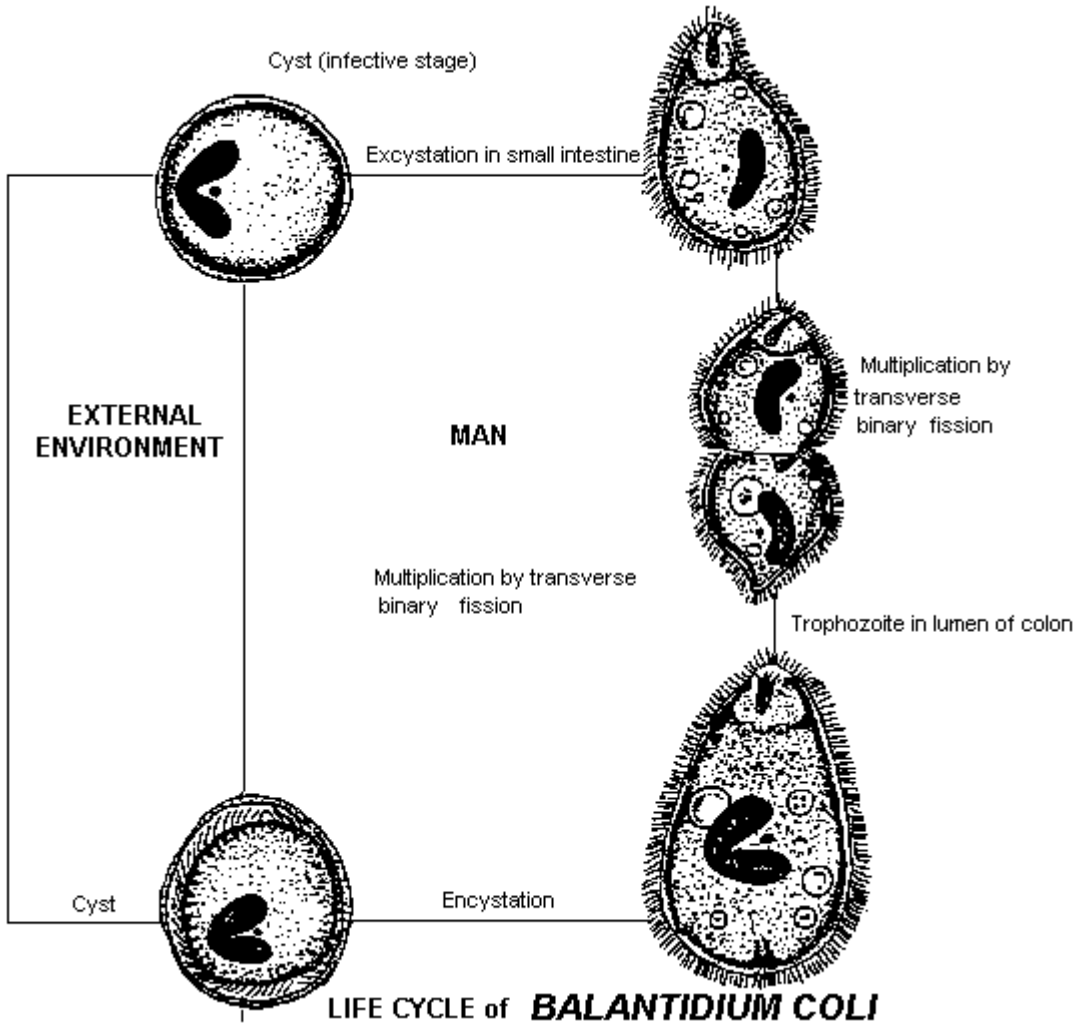
Life cycle

Human infection with *B. coli* is initiated upon ingestion of infective cysts in contaminated food or water, unlike that of *E. histolytica*, multiplication of the *B. coli* nuclei does not occur in the cyst phase, following excystation in the small intestine, the resulting trophozoites take up residence and feed primarily in the cecal region and terminal portion of the ileum, as well as in the lumen, mucosa, and submucosa of the large intestine. The multiplication of each trophozoite occurs by transverse binary fission, from which two young trophozoites emerge. The *B. coli* trophozoites are delicate and do not survive in the outside environment. Encystation occurs in the lumen. The resulting cysts mature and ultimately become the infective form for transmission into a new host. These cysts may survive for weeks in the outside environment.





Balantidium coli troph & cyst



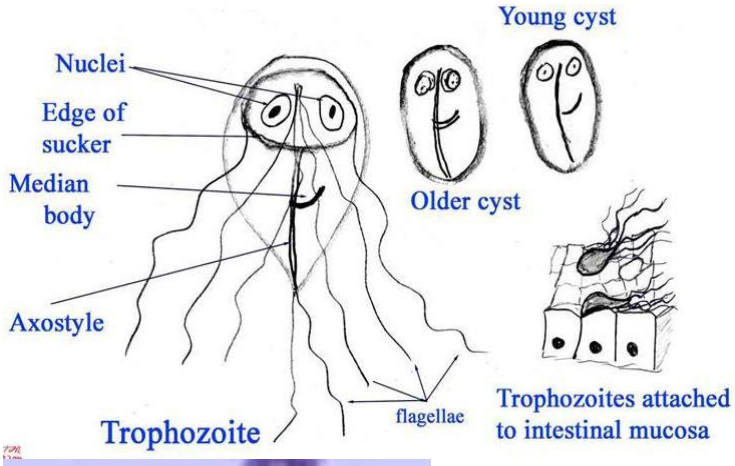
Class: Mastigophara (Flagellates)

1. *Giardia lamblia*

Giardia lamblia causes *giardiasis*, living in duodenum. The live cycle consists of two stages: trophozoite & cysts

1. Trophozoite: is pear-shaped (symmetric organism), length 9-21µ, with two nuclei, four pairs of flagella, two axostylels and a suction disk which it attaches to the intestinal wall.

2. Cyst is ellipsoid or oval cyst is thick walled with four nuclei and several internal fibers, length 8-12 µ. Each cyst gives rise to two troph. During excystation in the intestinal tract.





-Pathogenesis: transmission occurs by ingestion of the cyst in focally contaminated food and water. Excystation takes place in the duodenum. Where the troph. attaches to the gut wall but does not invade. Troph. Causes inflammation of the duodenum mucosa, leading to malabsorption of protein and fat.

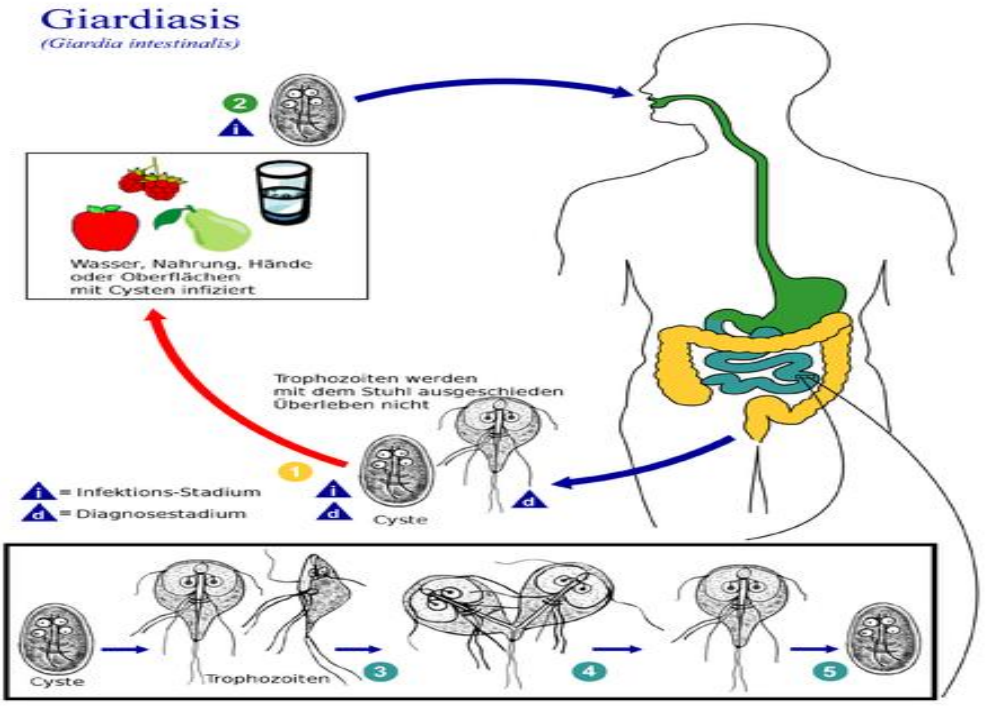
-Clinical finding:

Giardiasis ("Traveler's Diarrhea"). Symptomatic infections with *Giardia lamblia* may be characterized by a wide variety of clinical symptoms, ranging from mild diarrhea (watery, non bloody, foul smelling diarrhea (semi solid and greasy), abdominal cramps, anorexia, and flatulence to tenderness of the epigastric region steatorrhea, and malabsorption syndrome. Patients suffering from a severe case of giardiasis produce light – colored stools with a light fat content that may be caused by secretions produced by the irritated mucosal lining. Fat soluble vitamin deficiencies, folic acid deficiencies, hypoproteinemia with hypogammaglobulinemia, and structural changes of the intestinal villa may also be observed in such cases.

Diagnosis

1. By finding troph, cyst in stool. **2**. Using ELISA test. **3**. String test.

Diagnostic stages are troph. Or cyst or both in diarrhea stool. The **infective stage** is cyst**.**



Life cycle of *Giardia lamblia*

2.Trichomonas vaginalis

1. Pathogenic to human &causes vaginitis (trichomoniasis).

2.**troph**. Is round or pear like in shape, contains 4-6 flagella, all originating from anterior end & only one extend posteriorly. The motility is rapid & jerky. **No cyst is seen**

3.The undulating membrane extending half of the body length. Prominent axostyle that often curves around the nucleus & granules may be seen along in the axostyle. The nucleus is oval shape & only one.

Clinical symptoms

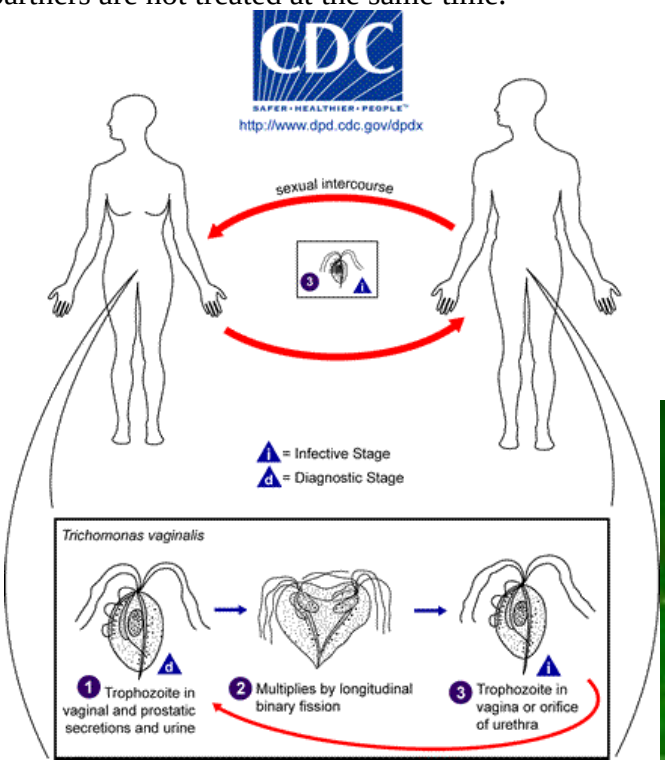
1. *T. vaginalis* reside on the mucosal surface of the vagina in infected women. The most common sites in male are the prostate gland region & the epithelium of the urethra.

2. Vaginitis may be found in infected women. It is characterized by foul smelling, greenish- yellow, vaginal discharge, burning &itching may also present. Red punctuate lesions may be seen in vaginal mucosa. Urethral involvement, dysuria & increased frequency of urination are among the most commonly symptoms. Cystitis is rare occur.

Life cycle

Trichomonas vaginalis trophzoites reside on the mucosal surface of the vagina in infected women. The growing trophozoites multiply by longitudinal binary fission and feed on local bacteria and leukocytes. The *Trichomonas vaginalis* trophozoites thrive in a slightly alkaline or slightly acid PH environment, such as that commonly seen in an unhealthy vagina. The most common infection site of *T. vaginalis* in males is the prostate gland region and the epithelium of the urethra. The detailed life cycle in the male host is unknown.

Diagnosis is by microscopic examination of fresh substances released through the vagina.**Treatment** is by metronidazole given by mouth. Reinfection is common if sexual partners are not treated at the same time.





Life cycle of *Trichomonas vaginalis* **trophzoites**

3.Trichomonas tenax

Trophzoite: Oval to pear in Shape. Have one nuclei, vesicular filled with chromatin granules. Have five flagella, all originating anteriorly, four extends anteriorly, one extends posteriorly. Undulating membrane extending 2/3 of body length. Thick axostyle and Small anterior cytosome opposite undulating membrane.

Life cycle

Trichomonas tenax trophozoites survive in the body as mouth scavengers that feed primarily on local microorganisms. Located in the tartar between the teeth, tonsillar crypts

pyorrheal pockets, and gingival margin around the gums, *T. tenax* trophozoites multiply by lonitudinal binary fission. These trophozoites are unable to survive the digestive process.

Clinical symptoms

The typical *Trichomonas tenax* infection does not produce any notable symptoms. On a rare occasion, *T. tenax* has been known to invade the respiratory tract, but this appears to have mainly occurred in patients with underlying thoracic or lung abscesses of pleural exudates.

3. **B:Class Haemoflagellates:** ***Leishmania & Trypanosoma***

Blood and Tissue Protozoal Infections

The major protozoal diseases that involve the blood and internal organs are malaria (Plasmodium species), toxoplasmosis (Toxoplasma species), trypanosomiasis (Trypanosoma species), and leishmaniasis (Leishmania species). Plasmodium and Toxoplasma are sporozoans, whereas Trypanosoma and Leishmania are flagellates, sometimes referred to as hemoflagellates. These parasites are unicellular with flagellum in the beginning of the parasite which helping it in motile.

1.These parasites invading the blood, tissues,& endothelial layer of organs&the tissues of skin.

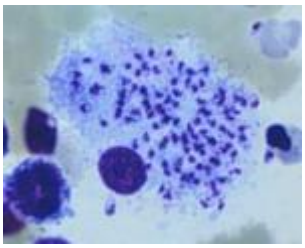
2.These flagellates including two genus that important to human:

- ***Leishmania:*** This genus is circular or ovum in shape with one nucleus located at the center of the cell and in front of the nucleus presents the motile generator which is short.

- ***Trypanosoma:*** The cell of this genus is tall with one central nucleus and the motile generator located in the terminal of parasite.

1. The flagellate belong to these two genus pass through the life cycle; the live cycle between two host: vertebrate host(terminal host like human)and arthropod host (mediated host like the fly) in many stages with different shapes, through the shape of the body, presence of flagellate or absent, the shape locate of motile generator and the presence of waved membrane or absent, as following:

A.Amastigote: the parasite circular or ovum in shape, the nucleus lies near the center and it in front of it present motile generator which extend short flagella from it & have not waved membrane.



A. Promastigote: the body is spindle with nucleus in the center and motile generator located near the beginning of the body arise a flagellate from generator extend out of the body and have not waved membrane.

C. Epimastigote: the body is tall and motile generator lies in front of the nucleus that move little away from the center of the body and arise on it flagellate that connect with the body and with the waved membrane therefore extend with free end.

D.Trypomastigote: The body is spindle and the nucleus lies in the center, the motile generator lies in the last part of the parasite and arise from it a flagellate extend along the external adage to the waved membrane and the end of flagella is free.

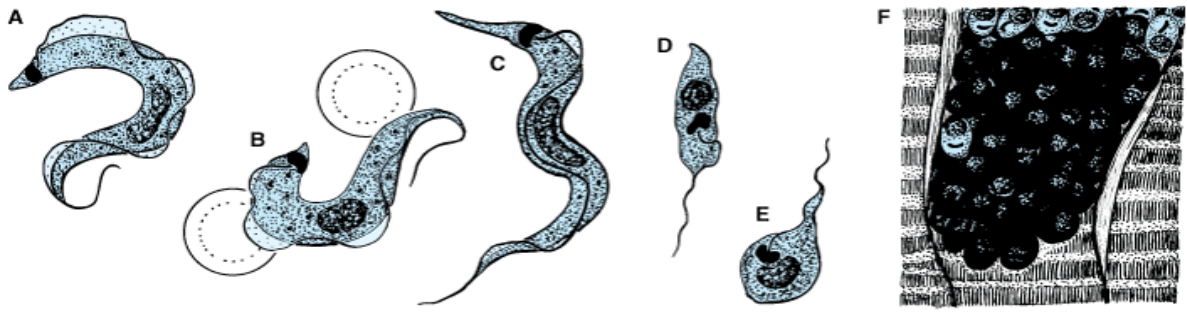


Figure: A, B, C: Trypomastigotes in blood;
D: epimastigote**,**
E: promastigote,
F: amastigote colony in heart muscle.

Note:the parasite that belong to *Leishmania* pass through the life cycle in amastigote and promastigote forms while *Trypanosoma* pass through the life cycle with all forms.

Leishmania:

A. L.donovani: causes visceral Leishmianiasis, Kalaazar and Dum Dum fever. Spleenomegaly & hepatomegaly. Parasite in human present in amastigote form, while in the insect (Sand fly) promastigote.

1.Visceral leishmaniasis (local name, kala-azar): This disease is caused by Leishmania donovani in India, East Africa, and China. In the visceral disease, the parasite initially infects macrophages, which, in turn, migrate to the spleen, liver, and bone marrow, where the parasite rapidly multiplies. The spleen and liver enlarge, and jaundice may develop. Most individuals have only minor symptoms, and the disease may resolve spontaneously. However, in some cases, complications resulting from secondary infection and emaciation result in death.

B. ***L.tropica :*** causes tropic sore or Baghdad boil, oriental sore and cutenaeous Leishmianiasis, the insect transport ***L.tropica*** is sand fly.

1.Cutaneous leishmaniasis (local name, oriental sore): This disease is caused by Leishmania tropica in north and west Africa, Iran, and Iraq. The cutaneous form of the disease is characterized by ulcerating single or multiple skin sores. Most cases spontaneously heal, but the ulcers leave unsightly scars. In Mexico and Guatemala, the cutaneous form is due to Leishmania mexicana, which produces single lesions that rapidly heal.

C. L. braziliensis : **causes Mucocutaneous leishmaniasis.**

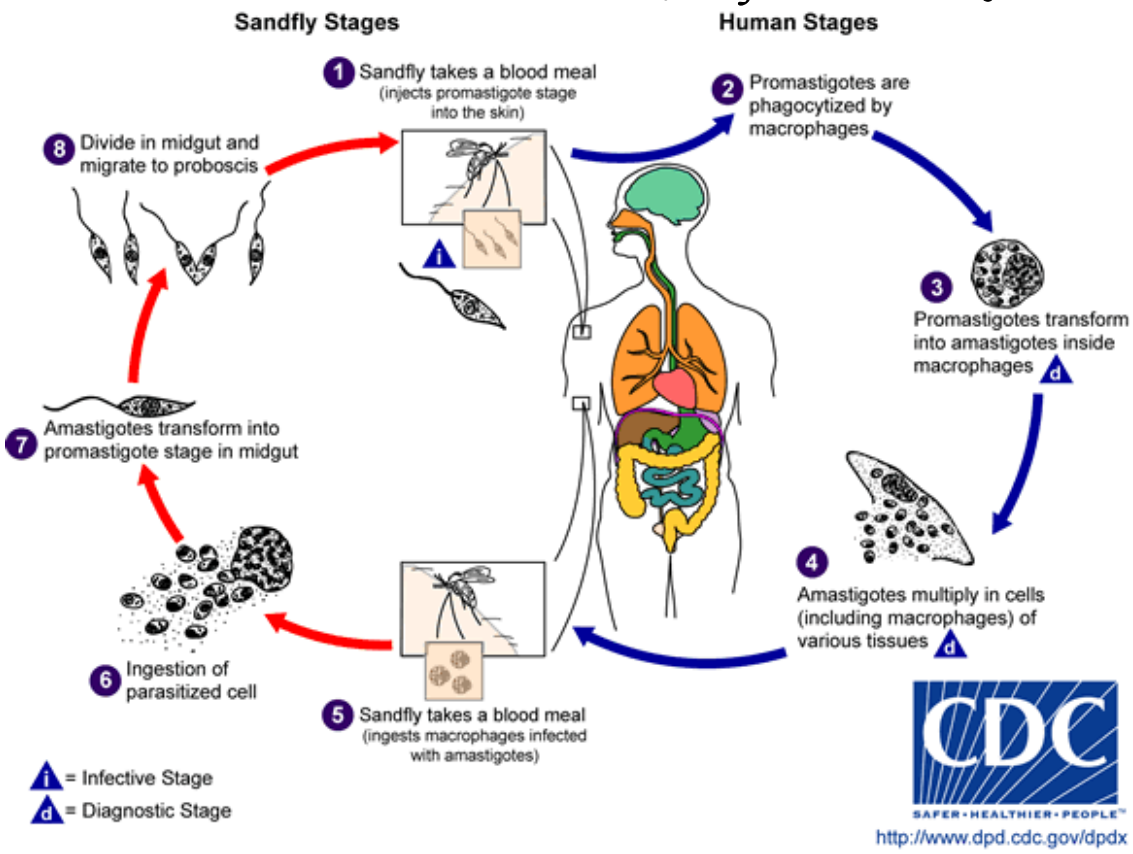
1. **Mucocutaneous leishmaniasis** (local name, espundia): This disease is caused by Leishmania brasiliensis in Central and South America, especially the Amazon regions. In this form of the disease, the parasite attacks tissue at the mucosal-dermal junctions of the nose and mouth, producing multiple lesions. Extensive spreading into mucosal tissue can obliterate the nasal septum and the buccal cavity, ending in death from secondary infection.

•Trypanosoma:

T.gambiense: causing sleeping disease to human and the mediated host is Tse_Tse fly.
T.cruzi: cause chagas disease, American trypanosomiasis.

Diagnosis of *L.donovani*

1. Thick blood film (amastigot). 2. skin test: is used to measure delyed hypersensitivity.
3. Detection of antibody by ELISA. 4. can be cultured on NNN media (Novy Macneel Nicolle)



Life cycle of Leishmania

Class: Sporozoa

- Sporozoa: Plasmodium spp. (Morphology, habitat, epidemiology, pathogenesis, methods of transmission, diagnosis, control and treatment).
- *Toxoplasma gondii* (Morphology, transmission, pathogenesis, diagnosis, control and treatment).

A:Malaria (Plasmodium species)

a. *Plasmodium falciparum* causes malignant tertian malaria
b. *P. malariae*: causes Quartean malaria
c. *P. vivax*: causes benign tertian malaria
d. *P. ovale*: causes benign tertian malaria

Malaria is an acute infectious disease of the blood, caused by one of four species of the protozoal genus,. P. falciparum accounts for some fifteen percent of all malaria cases, and P. vivax for Geighty percent of malarial cases.

The plasmodial parasite is transmitted to humans through the bite of a female Anopheles mosquito, or by an infected, blood-contaminated, needle.

Sporozoans reproduce asexually in human cells by a process called schizogony, in which multiple nuclear divisions are followed by envelopment of the nuclei by cell walls DE3WSXreproduction occurs in the mosquito, where new spores (sporozoites) are formed process called sporogony. **Schizogony (asexually in human; intermediated host), sporogony (Sexual reproduction in the mosquito; definitive host).**

Table : *Plasmodium vivax* :Typical characteristics (based on Giemsa stain).	
Appearance of infected RBCs	Enlarged , distroted
Ring form	Delicate cytoplasmic ring measuring 1/3 RBC diameter Single chromatin dot, Ring surrounds a vacuole
Developing trophozoite	Irregular ameboid appearance Ring remnants common Brown pigment
Immature schizont	Multiple chromatin bodies Brown pigment
Mature schizont	12 to 24 merozoites occupying majority of the RBCs Merozoites surrounded by cytoplasmic material Brown pigment may be present
Microgametocyte	Large pink to purple chromatin mass surrounded by colorless to pale halo
Macrogametocyte	Round to oval cytoplasm , Eccentric chromatin mass Delicate light – brown pigment present throughout cell

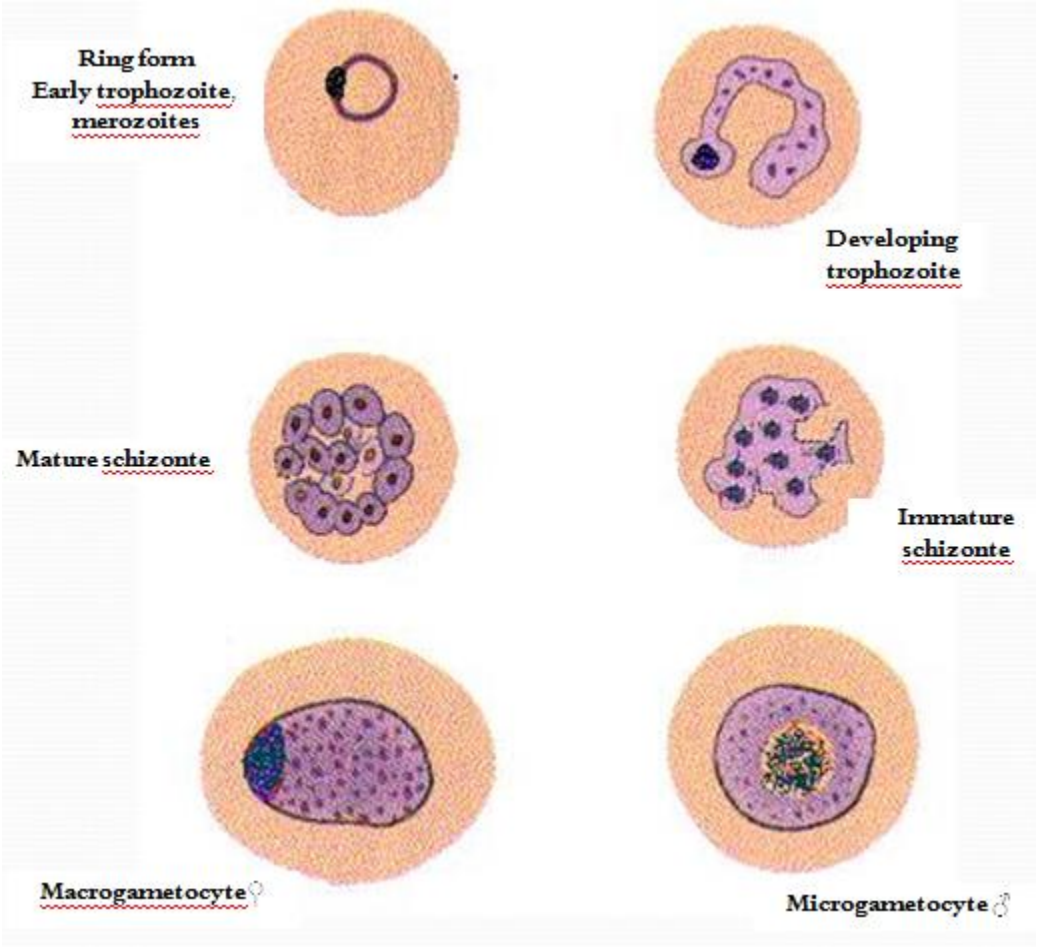


Figure: :Typical characteristics of *Plasmodium vivax* (based on Giemsa stain)

Pathology and clinical significance:

Plasmodium sporozoites are injected into the bloodstream, where they rapidly migrate to the liver. There they form cyst-like structures containing thousands of merozoites. Upon release, the merozoites invade red blood cells, using hemoglobin as a nutrient. Eventually, the infected red cells rupture, releasing merozoites that can invade other erythrocytes. If large numbers of red cells rupture at roughly the same time, a paroxysm (sudden onset) of fever can result from the massive release of toxic substances. **Plasmodium falciparum** is the most dangerous plasmodial species. It can cause a rapidly fulminating disease, characterized by persistent high fever and orthostatic hypotension. Infection can lead to capillary obstruction and death if treatment is not prompt. P. malariae, P. vivax, and P. ovale cause milder forms of the disease, probably because they invade either young or old red cells, but not both. This is in contrast to P. falciparum, which invades cells of all ages. Even today, malarial infection is a common and serious disease, causing some 300 million cases per year, with a death rate of about one percent.

Clinical symptoms

Benign Tertian Malaria. Patients infected with *P. vivax* typically begin to develop symptoms following a 10 to 17 day incubation period following exposure. These vague symptoms mimic those usually seen in cases of the flu, including nausea, vomiting, headache, muscle pains, and photophobia . As infected RBCs begin to rupture, the resulting merozoites, hemoglobin, and toxic cellular was products initiate the first in a series of paroxysms. These paroxysms typically occur every 48 hours.

Life cycle

Members of the mosquito genus *Anopheles* are responsible for the tranismission of malaria to humans via a blood meal. These vector transfers malarial **sporozoites** form its salivary gland into the human wound. Following entrance into the body, the sporozoites are carried through the preipheral blood to the parenchymal cells of the liver. It is here where **schizogony** occurs. This **exoerythrocytic cycle** of growth and reproduction lasts from 8 to 25 days, depending on the specific *Plasmodium* species involved. The infected liver cells eventually rupture and introduce merozoites into the circulating blood.
These migrating merozoites target age and – size – specific RBCs to invade, and upon doing so initiate the **erythrocytic cycle** of growth .it is in this asexual phase that the plasmodia feed on hemoglobin and pass through the numerous stages of growth, including the six morphologic forms previosly described. Upon formation of the merozoites, one of three paths may take place. Some of the RBCs infected with merozoites rupture, releasing these forms to target and infect new RBCs, and this part of the cycle repeats itself. Numerous erythrocytic cycles may occur. However, other infected RBCs containing merozoites develop into microgametocytes and macrogametocytes. Still others are destroyed by the immune system of otherwise healthy individual.

Transmission of malaria back into the vector occur when the mosquito ingests mature male (micro) and female (macro) **gametocytes** during a blood meal, thus initiating the sexual cycle of growth. a male and female gametocyte unite in the mosquito's stomach and form a zygote matures into an oocyst . upon complete maturation , the oocyst ruptures and releases numerous sporozoites , which migrate into salivary gland of the mosquito and are ready to infect another unsuspecting human . Thus the cycle repeats itself.

Drug treatment depends on the stage of infection. Primaquine is effective against the exoerythrocytic forms in the liver and bloodstream and also against the gametocytic form, but inactive against parasites in red blood cells. Therefore, for the erythrocytic form, primaquine is administered in conjunction with a blood schizontocide such as chloroquine,

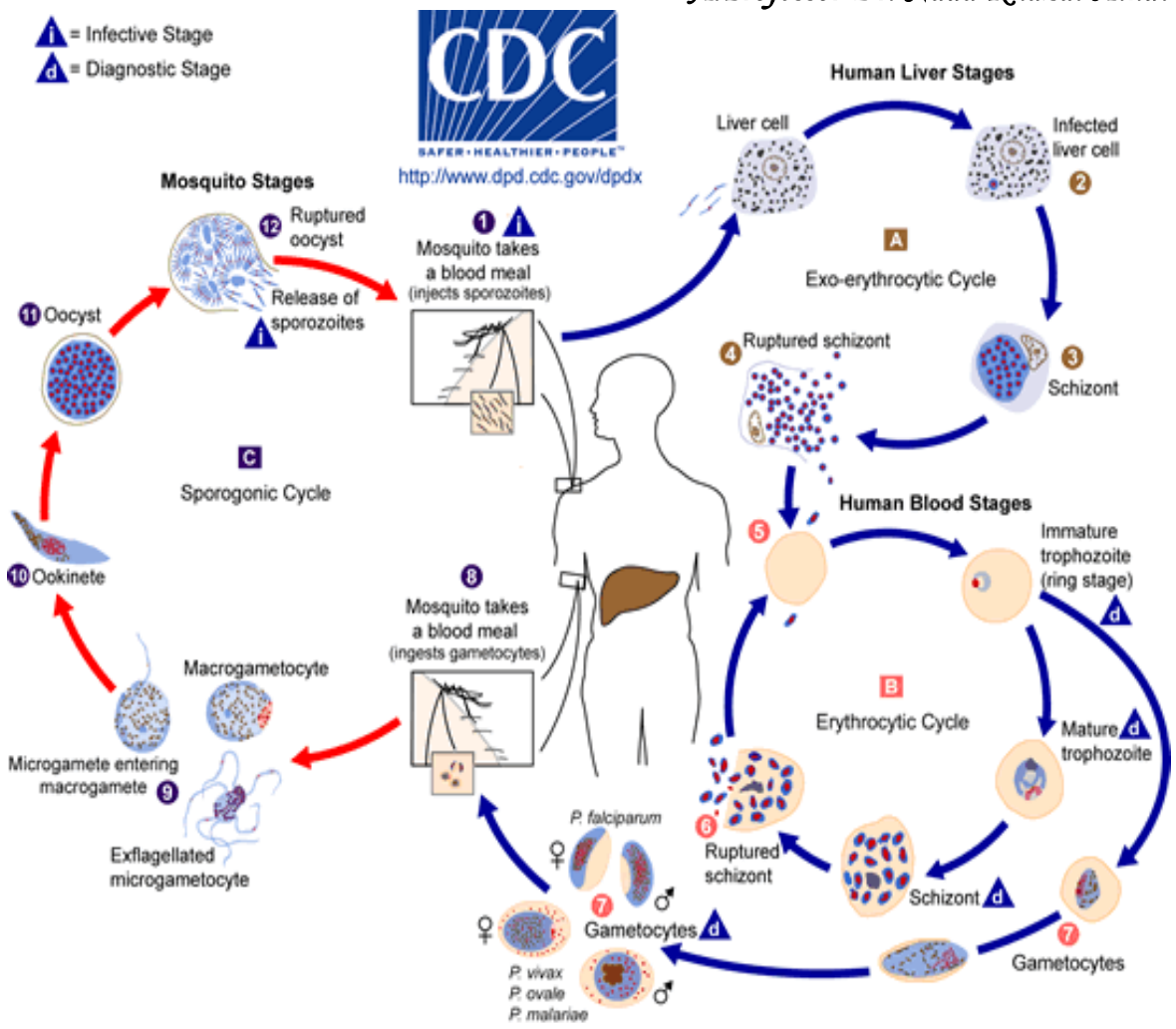
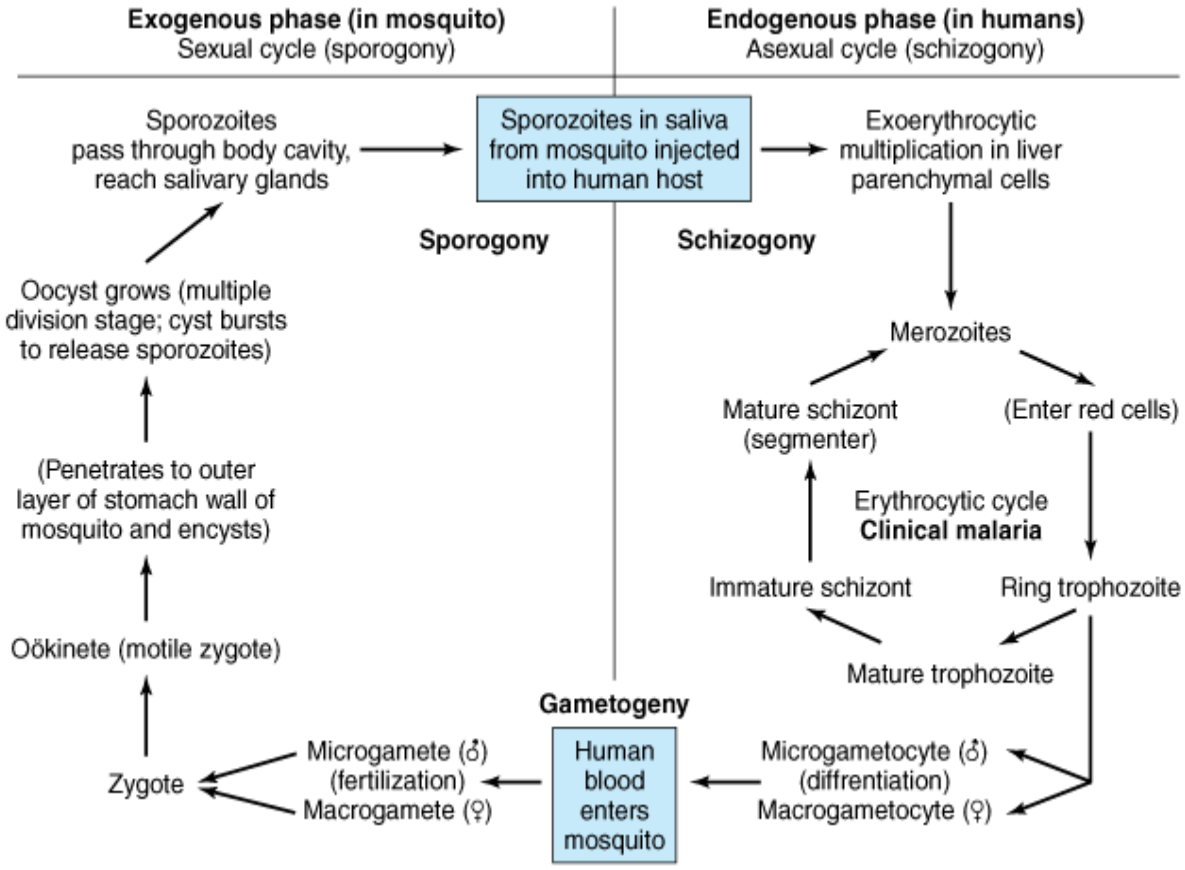


Figure: *Plasmodium vivax* life cycle .



Life cycle of Plasmodium (sexual and a sexual cycle)

B: Toxoplasmosis (*Toxoplasma gondii)*

Toxoplasma gondii is a sporozoan, distributed worldwide, that infects all vertebrate species, although the definitive host is the cat. Humans can become infected by the accidental ingestion of oocysts present in cat feces, by eating raw or undercooked meat, congenitally from an infected mother, or from a blood transfusion.

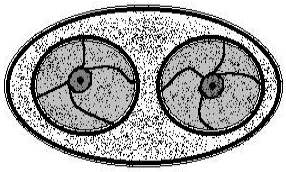
Table:***Toxoplasma gondii*** **oocyst : Typical characteristics**	
Size range	25 to 35 μm long , 10 to 15 μm wide
Appearance	Transparent
Shape	Oval
Other features	**Young oocyst** contains two sporoblasts. **Mature oocyst** contains two sporocysts, each containing four sporozoites.
Figure	 

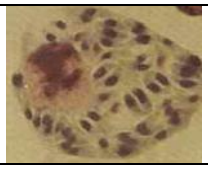
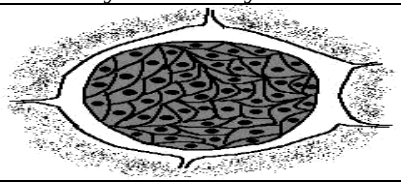
Table:***Toxoplasma gondii*** **tachyzoite : Typical characteristics**	
General comment	Actively multiplying morphologic form
Size	3 to 7 μm by 2 to 4 μm
Shape	Crescent shaped , often more rounded one end
Number of nucleui	One
Other features	Contains a variety of organelles that are not readily visible
Figure	 

Table:***Toxoplasma gondii*** **Bradyzoite : Typical characteristics**	
General comment	Slow - growing morphologic form
Size	Smaller than tachyzoites
Physical appearance	Similar to that of the tachyzoites
Number of nucleui	One
Other features	Hundreds to thousands of bradyzoites enclose themselves to form a cyst that may measure 12 to 100 μm in diameter
Figure	

Pathology and clinical significance:

There are two kinds of Toxoplasma trophozoites found in human infections: rapidly growing tachyzoites (tachy = rapid) that are seen in body fluids in early, acute infections, and slowly growing bradyzoites (brady = slow) that are contained in cysts in muscle and brain tissue and in the eye. Tachyzoites directly destroy cells, particularly parenchymal and reticuloendothelial cells, whereas bradyzoites released from ruptured tissue cysts cause local inflammation with blockage of blood vessels and necrosis. Infections of normal human hosts are common and usually asymptomatic. However, they can be very severe in immunocompromised individuals, who may also suffer recrudescence (relapse) of the infection. Congenital infections can also be severe, resulting in stillbirths, brain lesions, and hydrocephaly and they are a major cause of blindness in newborns.

Life cycle

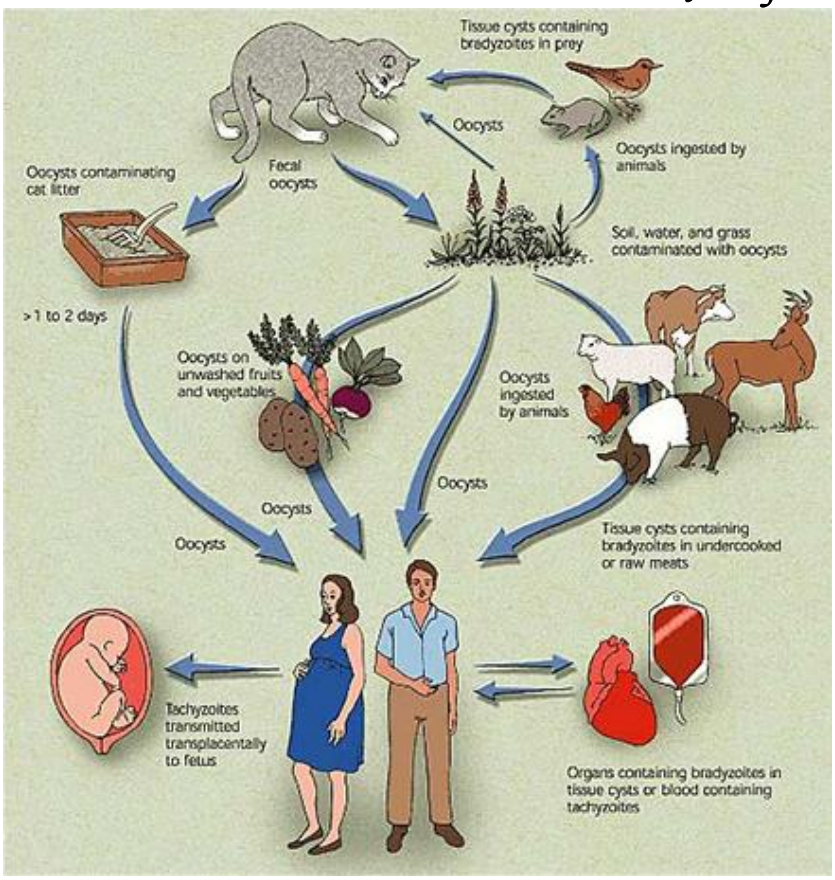
The definitive host in the *Toxoplasma* life cycle is the cat (or other felines). Upon ingestion of *Toxoplasma* cysts present in the brain or muscle tissue of contaminated mice or rats, the enclosed bradyzoites are released in the cat and quickly transform into tachyzoites. Both sexual and asexual reproduction occurs in the gut of the cat. The sexual cycle results in the production of immature oocysts, which are ultimately shed in the stool. The oocysts complete their maturation in the outside environment, a process that typically takes from 1 to 5 days. Rodents, particularly mice and rats, serve as the intermediate hosts, ingesting the infected mature *Toxoplasma* oocysts while foraging for food. The sporozoites emerge from the mature oocyst and rapidly convert into actively growing tachyzoites in the intestinal epithelium of the rodent.

Clinical symptoms

Toxoplasmosis a common infection with the protozoan intracellular parasite *Toxoplasma gondii*. It is marked in its inborn form by liver and brain involvement with calcium in the brain (cerebral calcification), convulsions, blindness, too-small head and fluid on the brain (microcephaly and hydrocephaly), and mental retardation. The acquired form is marked by rash, disease of the lymph nodes (lymphadenopathy), fever, malaise, central nervous system disorders, swelling of the heart wall (myocarditis), and swelling of lung tissue (pneumonitis). Cats acquire the organism by eating infected birds and mice. Lumps (cysts) of the organism are carried from cat feces to humans or by human ingestion of inadequately cooked meat containing the lumps. Infection through the placenta occurs only during acute infection of the mother. The other route of human infection occurs when contaminated blood is transfused into an uninfected person.

Diagnosis and treatment:

The initial diagnostic approach involves detection of parasites in tissue specimens, but this may often be inconclusive. With the recent availability of commercial diagnostic kits, serologic tests to identify toxoplasma are now routinely used. These include tests for Toxoplasma-specific IgG and IgM. The treatment of choice for this infection is the antifolate drug pyrimethamine, given in combination with sulfadiazine.



Life cycle of *Toxoplasma*

Helminthes (Worm)

A. Cestodaes (Tape Worm)
B. Trematodes (Flukes).
C. Nematodes (Roundworms)

Cestodaes (Tape Worm)

1.*Taenia saginata*=Beef Tape Warm
2.*Taenia solium*=Pork TW
3.*Diphyllobotherium latum* =Broad fish TW
4.*Echinococcus granulosis*=Dog TW
5.*Hymenolepis nana* =Dwarf TW

General properties of Helminthes

1.Helminthes:thes organisms differ from protozoa in the fact that there are multicellular and contain internal organ system.
2.cestodes segmented Worm that primarily intestinal parasite.
3. They lack a digestive system and absorb soluble nutrient directly through their cuticle, causing mechanical blockage of the intestine.

4. Adult worm consist of :
a. head or scolex, with hooks and sucker that function to attach the worm to the intestinal wall.
B.neck is very short after scolex.
C. body segment (proglottids); each segment contain set of male and female sexual organs (immature &mature & gravid segment). Gravid segment contain fertile eggs, these pass out of the body in the stool.
5.cestodes utilize more than one host.
6.The infection occur in human during ingestion the larva in mediated host in most parasites ingestion the egg may be occur.

Table: types of cestoda

Organism	Insect Vector	Stage That Infects Humans	Stage(s) in Humans Most Associated with Disease	Important Stage(s) Outside of Humans
Taenia solium	None	1. Larvae in undercooked pork	Adult tapeworm in intestine	Larvae in muscle of pig
		2. Eggs in food or water contaminated with human feces	Cysticercus, especially in brain	None
Taenia saginata	None	Larvae in undercooked beef	Adult tapeworm in intestine	Larvae in muscle of pig
Diphyllobothrium latum	None	Larvae in undercooked fish	Adult tapeworm in intestine can cause vitamin B12 deficiency	Larvae in muscle of freshwater fish
Echinococcus granulosus	None	Eggs in food or water contaminated with dog feces	Hydatid cysts, especially in liver and lung	Adult tapeworm in dog intestine produces eggs

1.*Taenia saginata*=Beef Tape Warm

Adult worm develop in the small intestine, in human measure about 5 m and the body consist of the following : **scolex** which is rounded in shape &have 4 sucker, neck, immature segment which the genital organ are not develop, mature contain full set of sexual male &female organ, **gravid segment** contain uterus with lateral arms about (15-30 arm) &fertile eggs. **Eggs** of this worm are spherical in shape & consist of 3 hexacanth embryo and has three paris of hooklet.

T. saginata causes **Taeniasis**; this disease caused by the larval form of *T.saginata.* this disease is transmitted by larvae in undercooked or raw beef (mediated host is beef),most infected individual are asymptomatic. **Infective stage is cysticercus bovis** .Taeniasis dignosed by detection of proglottides or eggs in stool. It is treated with niclosamide.

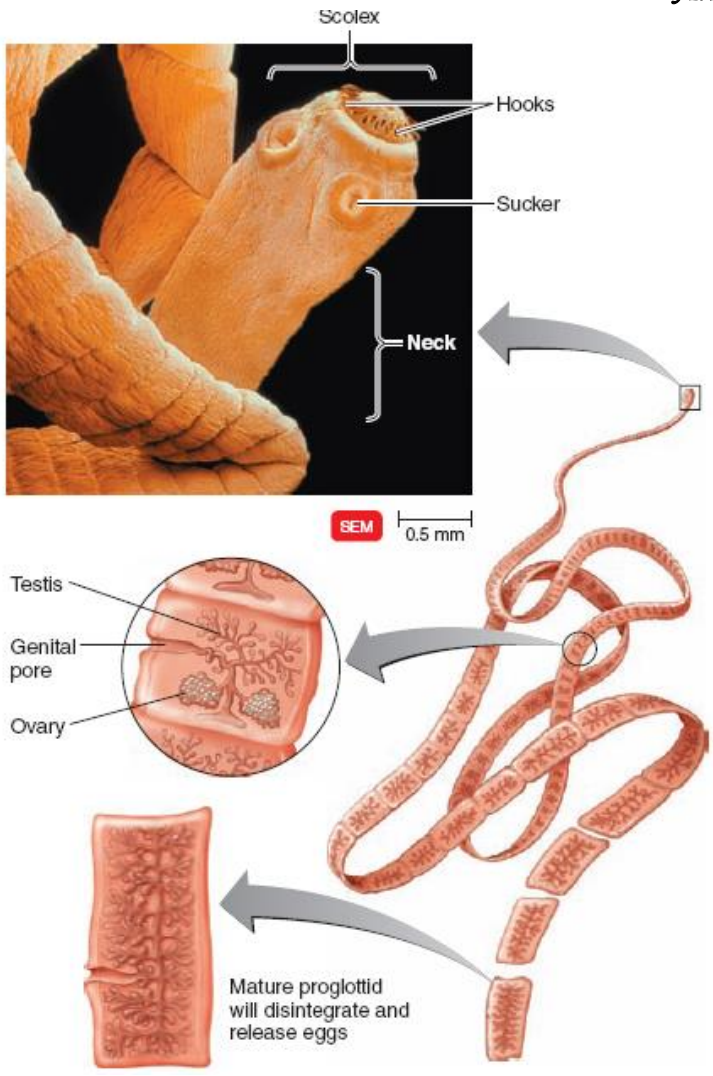
Life cycle

Infection with *Taenia saginata* occurs following ingestion of raw / undercooked pork meat contaminated with a **cysticercus larva**. Scolex attachment to the intestinal mucosa occurs in the small intestine where maturation into an adult worm occurs. The resulting adult multiplies , producing numerous eggs, some of which may be passed into the feces. These eggs are then consumed by the proper animal species (beef), where the onchosphere hatches. The onchosphere then migrates via the blood to the animal tissue and converts into the infective cysticercus larva stage. A new cycle is initiated upon human ingestion of the infected animal meat.

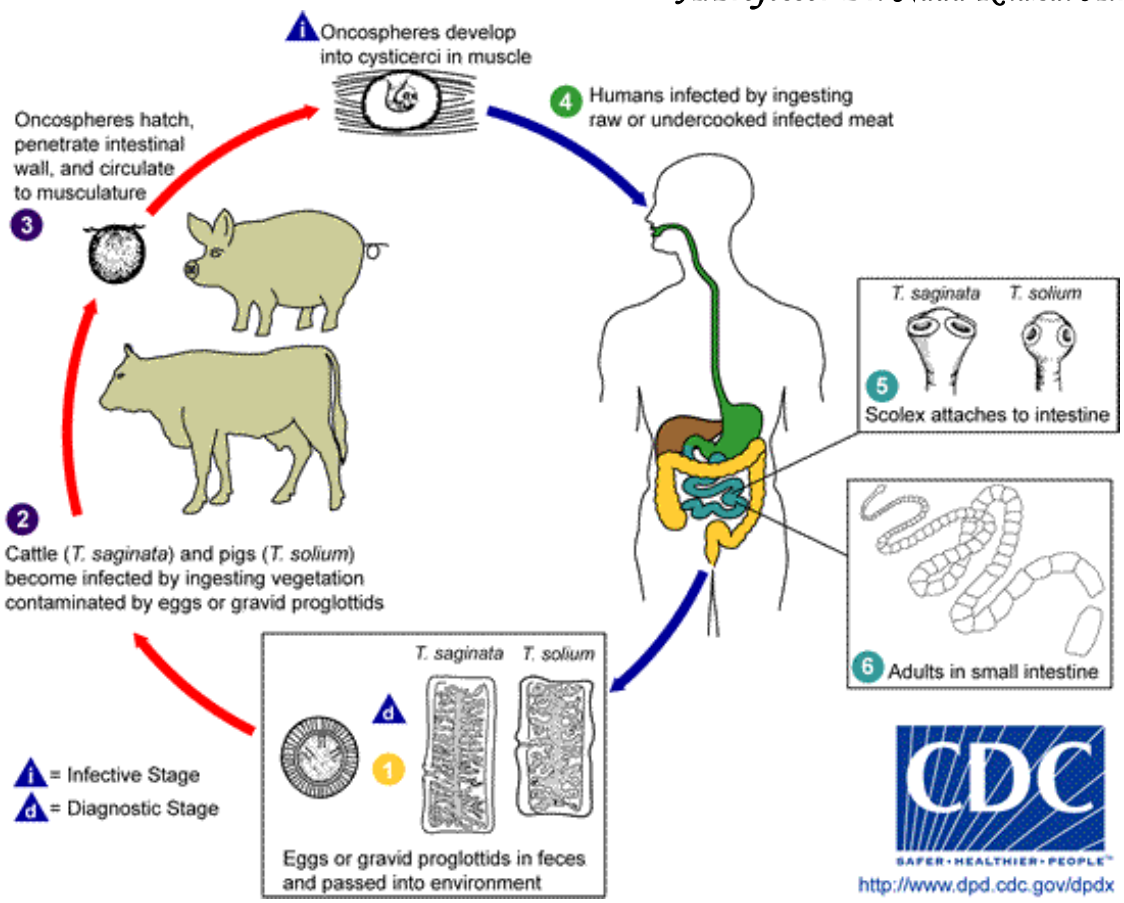
Clinical symptoms

Taeniasis / Beef Tape worm infection .

Nondescript symptoms, such as diarrhea, abdominal pain, change in appetite, and slight weight loss, may be experienced by *Taenia*- infected patients. In addition, symptoms including dizziness, vomiting, and nausea may also develop in such patients. Laboratory tests often reveal the presence of a moderate Eosinophilia . The prognosis is usually good.



Beef Tape worm



***Taenia saginata* life cycle**

2. *Taenia solium*=Pork TW

Adult worm develop in the middle of small intestine, in human measure about 3 m and the body consist of the following: **scolex** which is rounded in shape &have 5 suckers, also has rostellum and contain double circle and small hooks.

Neck, immature, mature segment, & eggs are same in *T. saginata*, while **gravid segment** contain uterus with lateral arms about (7-15arm) &fertile eggs.

***T. solium* causes :**

A: Taeniasis; this disease caused by the larval form of *T. solium*. This disease is transmitted by larvae in undercooked or raw pork (mediated host) this worm causes diarrhea, most infected individual are asymptomatic.

Infective stage is cysticercus cellulosae. Taeniasis dignosed by detection of proglottides or egg in stool.

B:cysticercosis this disease follows by ingestion of *T. solium* eggs from human feces & produces infection in the brain and eyes. It is treated with niclosamide.

Life cycle

Infection with taenia solium occurs following ingestion of raw / undercooked pork meat contaminated with a **cysticercus larva**. Scolex attachment to the intestinal mucosa occurs in the small intestine where maturation into an adult worm occurs. The resulting adult

multiplies, producing numerous eggs, some of which may be passed into the feces. These eggs are then consumed by the proper animal species (pig), where the onchosphere hatches. The onchosphere then migrates via the blood to the animal tissue and converts into the infective cysticercus larva stage. a new cycle is initiated upon human ingestion of the infected animal meat.

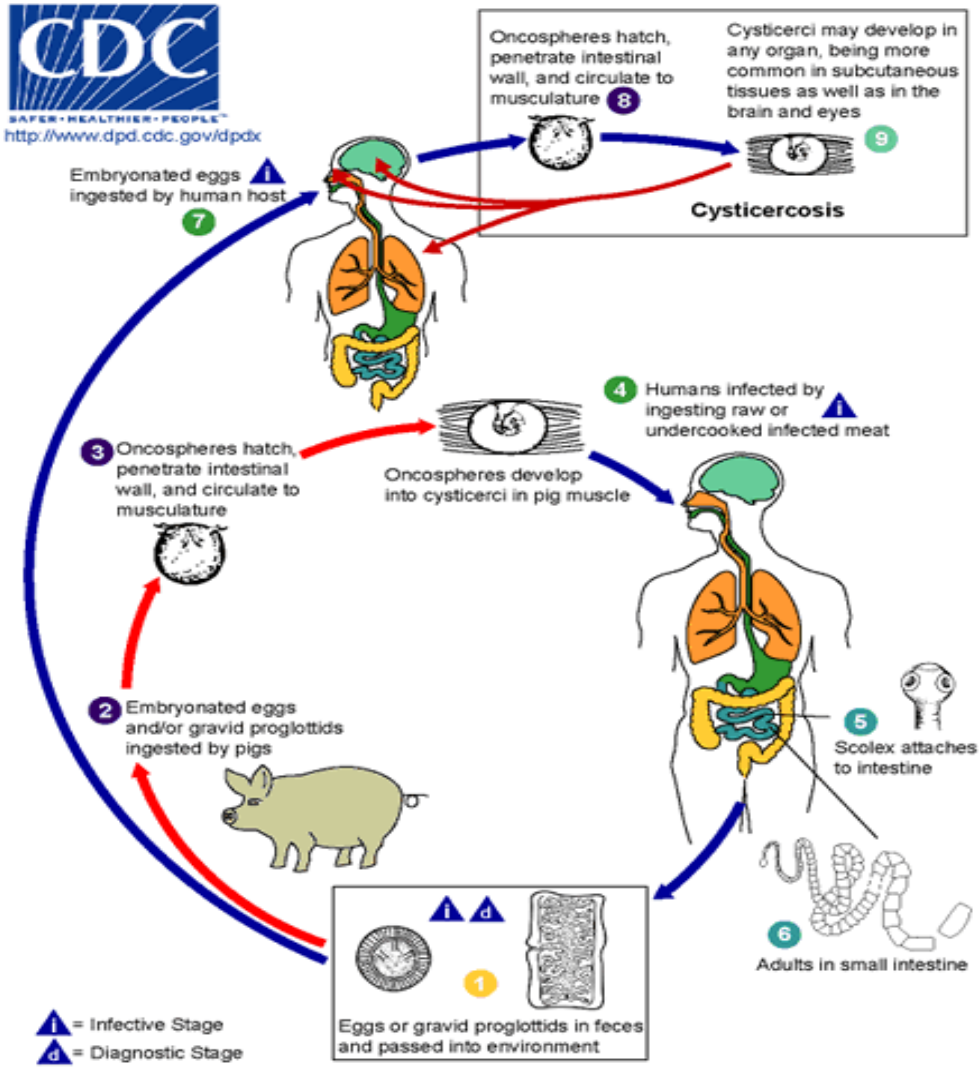


***Figure: Taenia solium* life cycle**

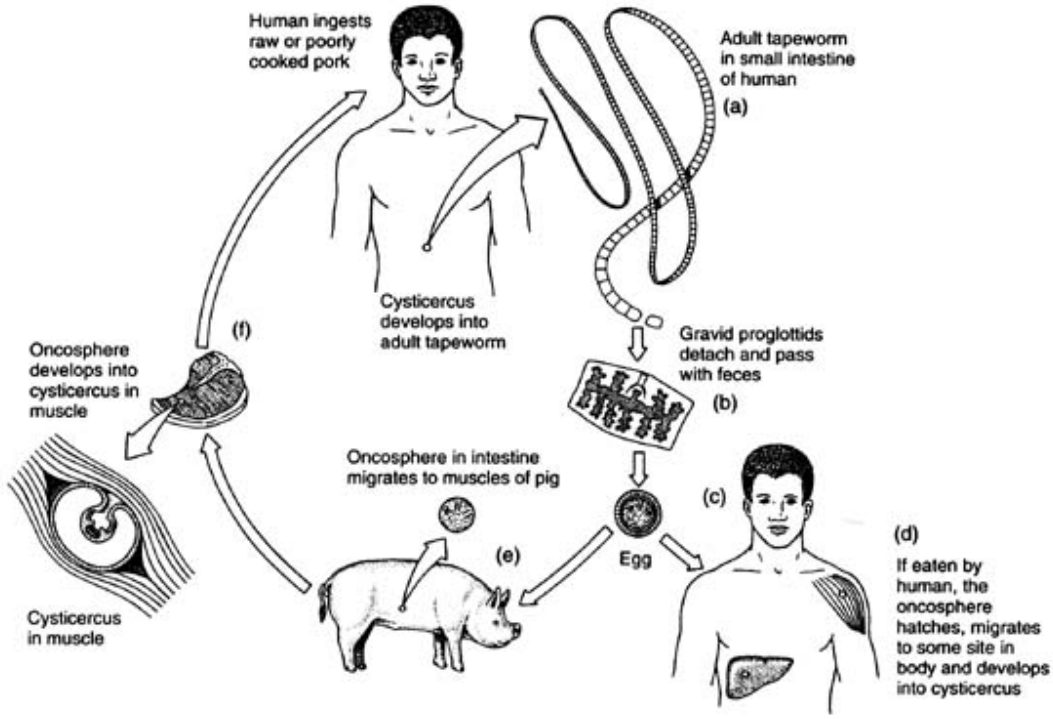


Figure 21.17

Life cycle of *Taenia solium*. (*a*) Adult tapeworm in the small intestine of a human. (*b*) Gravid proglottids detach from the strobila and migrate out of the anus or pass with feces. (*c*) Shelled oncosphere. (*d*) If eaten by a human, the oncosphere hatches, migrates to some site in the body, and develops into a cysticercus. (*e*) Cysticerci will also develop if the eggs are eaten by a pig. (*f*) The life cycle is completed when a person eats pork containing live cysticerci.

Drawing by William Ober and Claire Garrison.

Clinical symptoms

Taeniasis / pork Tape worm infection

Nondescript symptoms, such as diarrhea, abdominal pain, change in appetite, and slight weight loss , may be experienced by *Taenia*- infected patients. In addition, symptoms including dizziness, vomiting, and nausea may also develop in such patients. Laboratory tests often reveal the presence of a moderate Eosinophilia. The prognosis is usually good.

3. *Diphyllobotherium latum* =Broad fish TW

The length of *D.latum* about 10m-15m . **scolex** is spatulate shape ,it have contain 2 long sucking groove .**eggs** of *D.latum* are avol shape &have opericulum at anterior end &have knobe in posterior end. *D.latum* causes **Diphyllobotheriasis,** that transmitted by larvae in undercooked or raw fish. **Diphyllobotheriasis** is diagnosed by detection of characteristic eggs in the stool. It is treated with niclosamide.

4.*Echinococcus granulosis*=Dog TW

E. granulosis causes **Echinococcos**is, infection produce large **hydatid cysts** in liver, lung, brain. This disease follows ingestion of egg in dog feces. Sheep often serve as an intermediate host. Anaphylactic reaction to worm antigens can occur if the cyst ruptures.

The disease follows ingestion of eggs in dog feces. Sheep often serve as an intermediate host. **Echinococcos**is is diagnosed by CT scan or biopsy of infected tissue and is treated with albendazole and surgical excision of cysts.

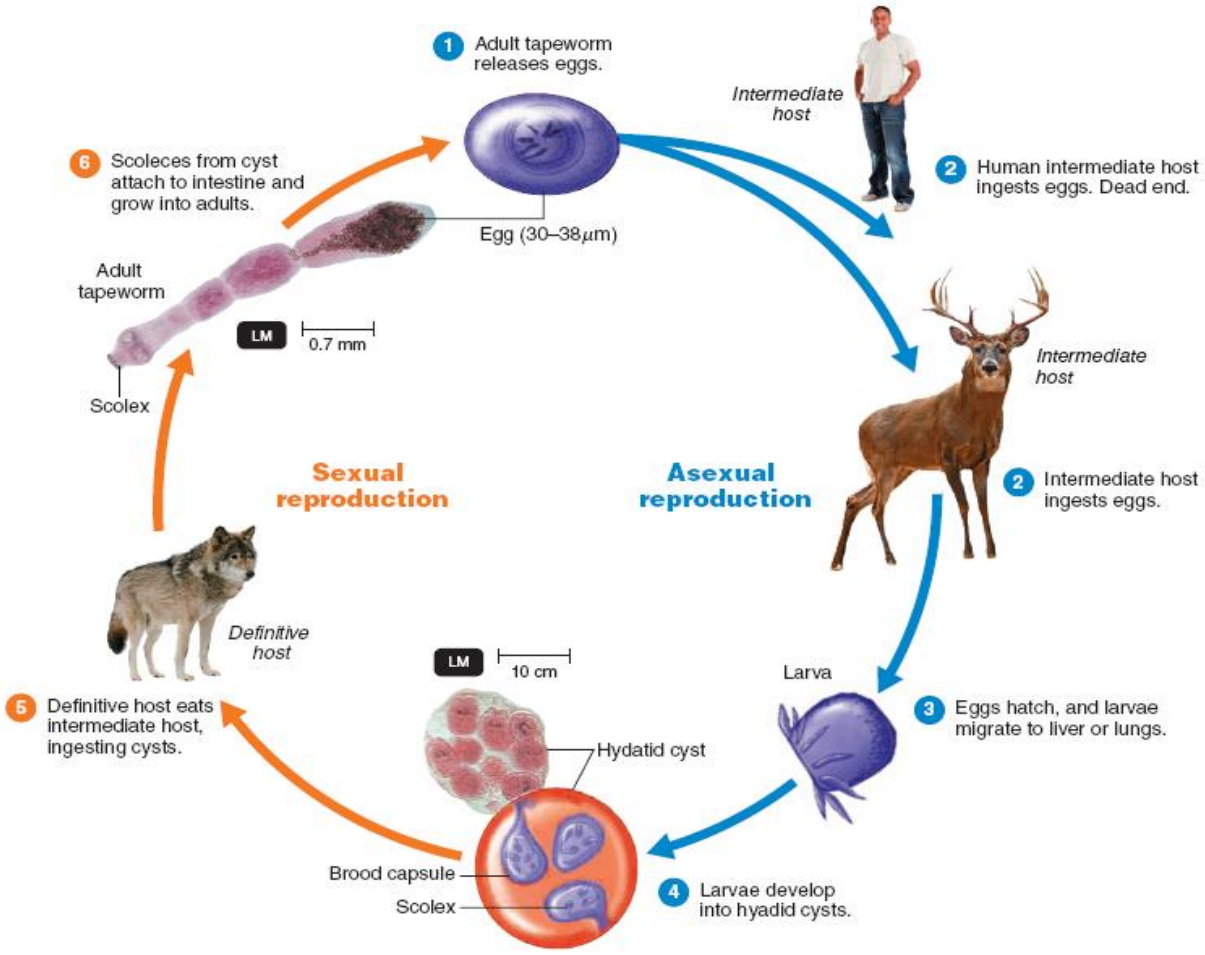


Figure: life cycle of *Echinococcus granulosis*

5.*Hymenolepis nana* =Dwarf TW

Adult worm is smallest tape worm of man it has length about 25-40 mm. **Scolex** contain 4 sucker with short rostellum armed with circle of hook. **Eggs** are oval in shape with hexanth embryo ,3 pairs of hooklet and has polar thinking protect the embryo. ***H. nana*** causes ***Hymenolepiasis***



Eggs of cestoda

Trematodes (flukes)

Trematodes are small (about 1 cm), flat, leaf-like worms that, depending on the species, infest various organs of the human host (for example, intestinal veins, urinary bladder, liver, or lung). All parasitic trematodes use freshwater snails as an intermediate host.

1. **schistosomes (Blood flukes)**
2. ***Fasciolopsis buski* (giant intestinal fluke)** causes Fasciolopsis. Live in Small intestine
3. ***Fasciola hepatica* (sheep liver fluke)** causes Fasciolosis. live in Liver (bile ducts, after migration through parenchyma)

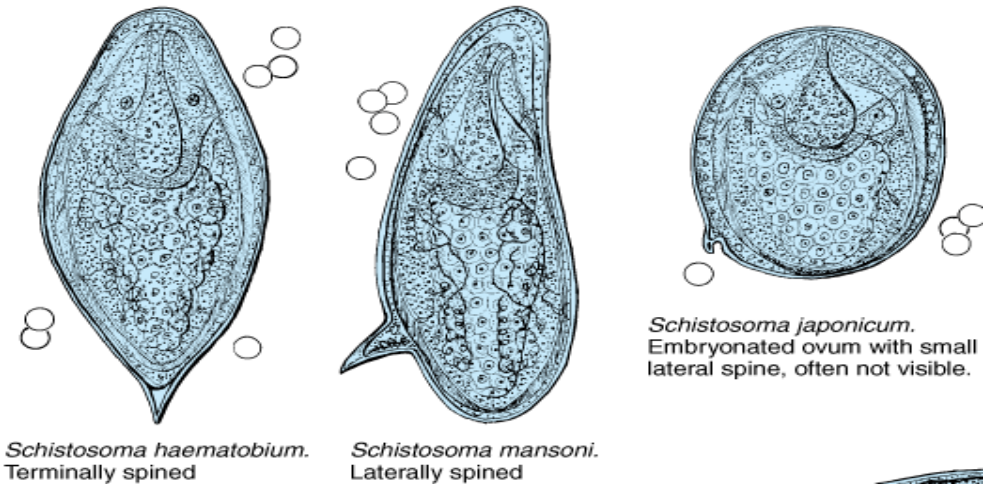
1. Schistosomes (Blood flukes) has three sp.

A.* Schistosomes *haematobium: Shape of egg: oval, contain terminal spine. Resident the vein surrounds the urinary bladder. causes **urinary bilhariziasis** and hematuria, fibrosis, granulomas. The disease is transmitted by direct skin penetration. Diagnosis (egg in urine). It is treated with praziquantel.

B.* Schistosomes *msnsoni: Shape of egg: large, oval, contain lateral spine. Resident the vein surround large intestine. causes **intestinal Schistosomiasis**.

C.* Schistosomes *japonicum: Shape of egg: oval, contain lateral blunt projection spine. Resident the vein surround small intestine. causes **intestinal Schistosomiasis**.
The eggs of ***S. msnsoni*** & ***S. japonicum*** causes damage in intestine and liver, gastrointestinal tract (GIT) bleeding and diarrhea. Damage to the intestinal wall is caused by the host's inflammatory response to eggs deposited at that site. The eggs also secrete proteolytic enzymes that further damage the tissue. The disease is transmitted by direct skin penetration. Diagnosis (egg in stool). It is treated with praziquantel.

Life cycle of schistosomes
schistosomes have only one intermediate host (the snail). schistosome cercaria acquired directly penetrating the skin of swimmers in contaminated rivers and lakes. After dissemination and development in the human host, adult schistosomes take up residence in various abdominal veins, depending on the species. schistosomes have separate, distinctive sexes. male in which the smaller female resides and continuously mates with the male. This mating takes place in the human liver. Fertilized eggs penetrate the human host's vascular walls and enter the intestine or bladder, emerging from the body in feces or urine. In fresh water, the organisms infect snails in which they multiply, producing cercaria (the final, free-swimming larval stage), which are released into the fresh water to complete the cycle.



Eggs of schistosomes

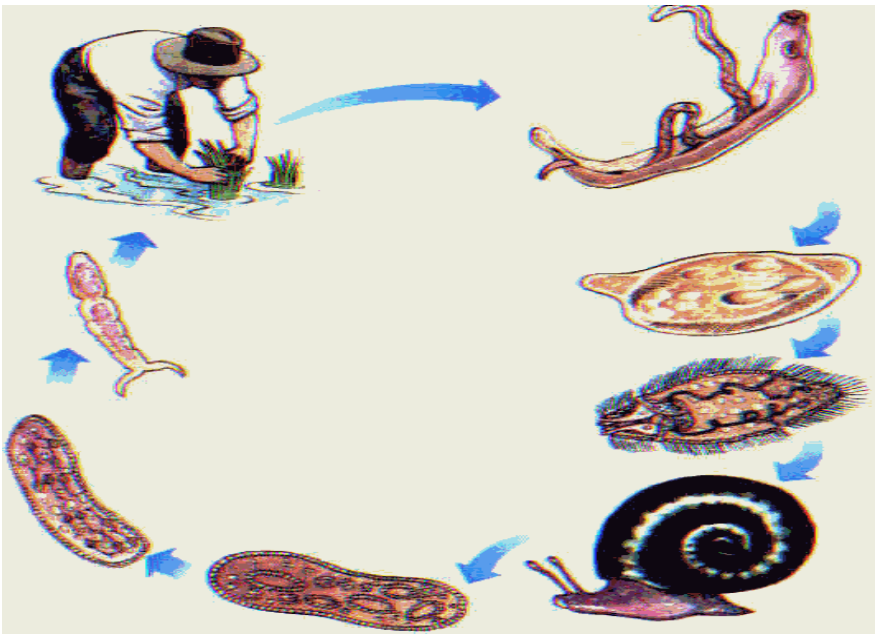
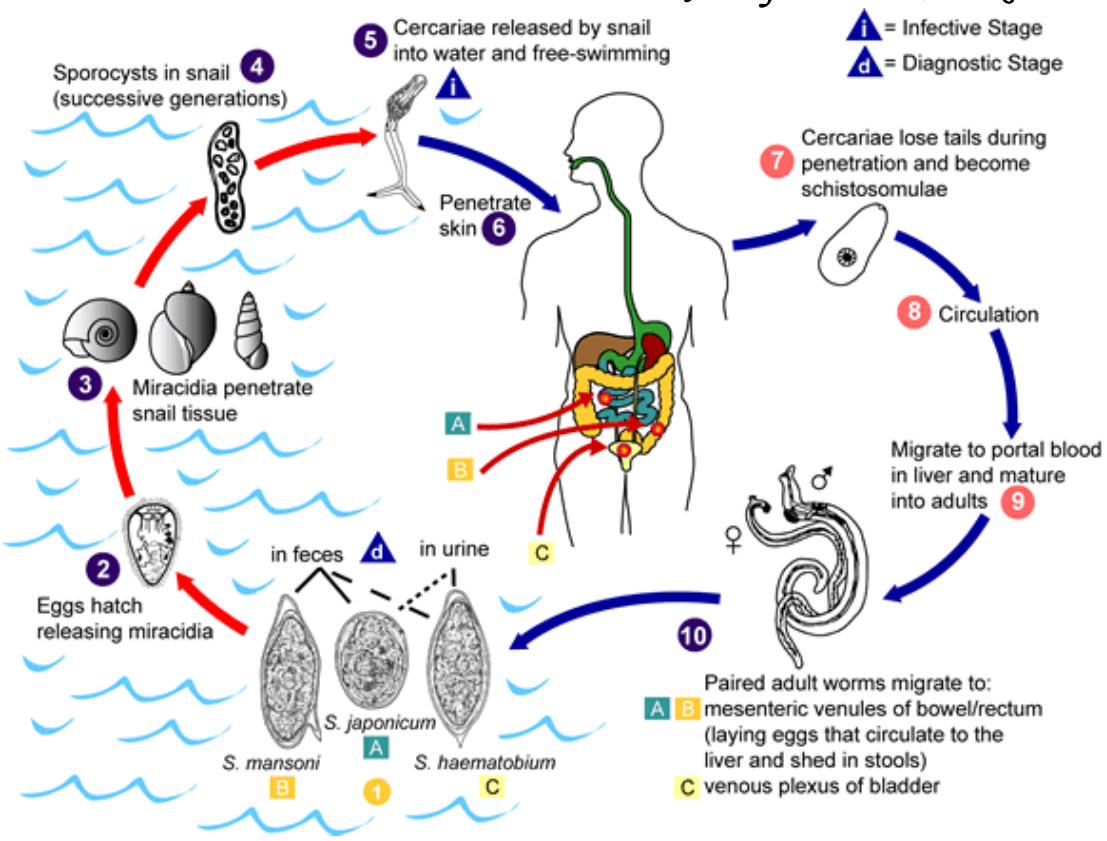


Figure: Stages of development of trematoda



Life cycle of schistosomes

Table: types of trematoda

Trematode	Mode of Transmission	Main Sites Affected	Intermediate Host (s)	Diagnostic Features of Eggs	Endemic Area(s)	Treatment
Schistosoma mansoni	Penetrate skin	Veins of colon	Snail	Large lateral spine	Africa, Latin America (Caribbean)	Praziquantel
Schistosoma japonicum	Penetrate skin	Veins of small intestine, liver	Snail	Small lateral spine	Asia	Praziquantel
Schistosoma haematobium	Penetrate skin	Veins of urinary bladder	Snail	Large terminal spine	Africa, Middle East	Praziquantel
Clonorchis sinensis	Ingested with raw fish	Liver	Snail and fish	Operculated	Asia	Praziquantel
Paragonimus westermani	Ingested with raw crab	Lung	Snail and crab	Operculated	Asia, India	Praziquantel

2.Fasciola hepatica

Adult

The adult *Fasciola hepatica* worm is flattened, leaf like shape, equipped with shoulders, somewhat oblong. Adult *Fasciola hepatica* measuring 3cm by 1cm in size, grayish in color. There are two suckers, oral sucker and ventral sucker, they located in cephalic zone. The intestine is branched, there are many branched testis, vitellaria situated in body laterals and the posterior end. The uterus is short and coiled filled with grayish eggs.

Life cycle

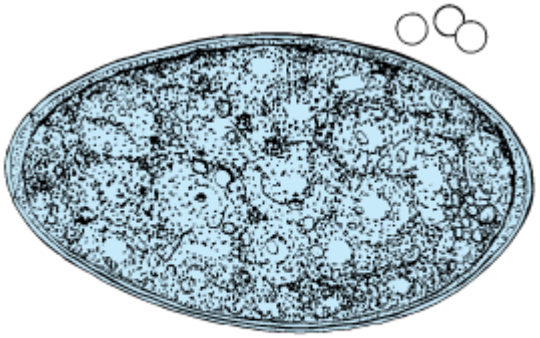
Fasciola hepatica, the sheep liver fluke, causes disease primarily in sheep and other domestic animals in Latin America, Africa, Europe, and China. Humans are infected by eating watercress (or other aquatic plants) contaminated by larvae (metacercariae) that excyst in the duodenum, penetrate the gut wall, and reach the liver, where they mature into adults. Hermaphroditic adults in the bile ducts produce eggs (unembryonated operculated ovum) , which are excreted in the feces. The eggs hatch in fresh water, and miracidia enter the snails. Miracidia develop into cercariae, which then encyst on aquatic vegetation. Sheep and humans eat the plants, thus completing the life cycle.

Clinical symptoms

Fascioliasis, an infection with a liver fluke *(Fasciola hepatica)*, **Symptoms** are due primarily to the presence of the adult worm in the biliary tract. it is marked by stomach and bowel pain, fever, a liver disease (jaundice), hives, and diarrhea. In early infection, right-upper-quadrant pain, and hepatomegaly can occur, but most infections are asymptomatic. Months or years later, obstructive jaundice can occur. Halzoun is a painful pharyngitis caused by the presence of adult flukes on the posterior pharyngeal wall. The adult flukes are acquired by eating raw sheep liver. Or by swallowing forms of the fluke found on water plants, as raw watercress.

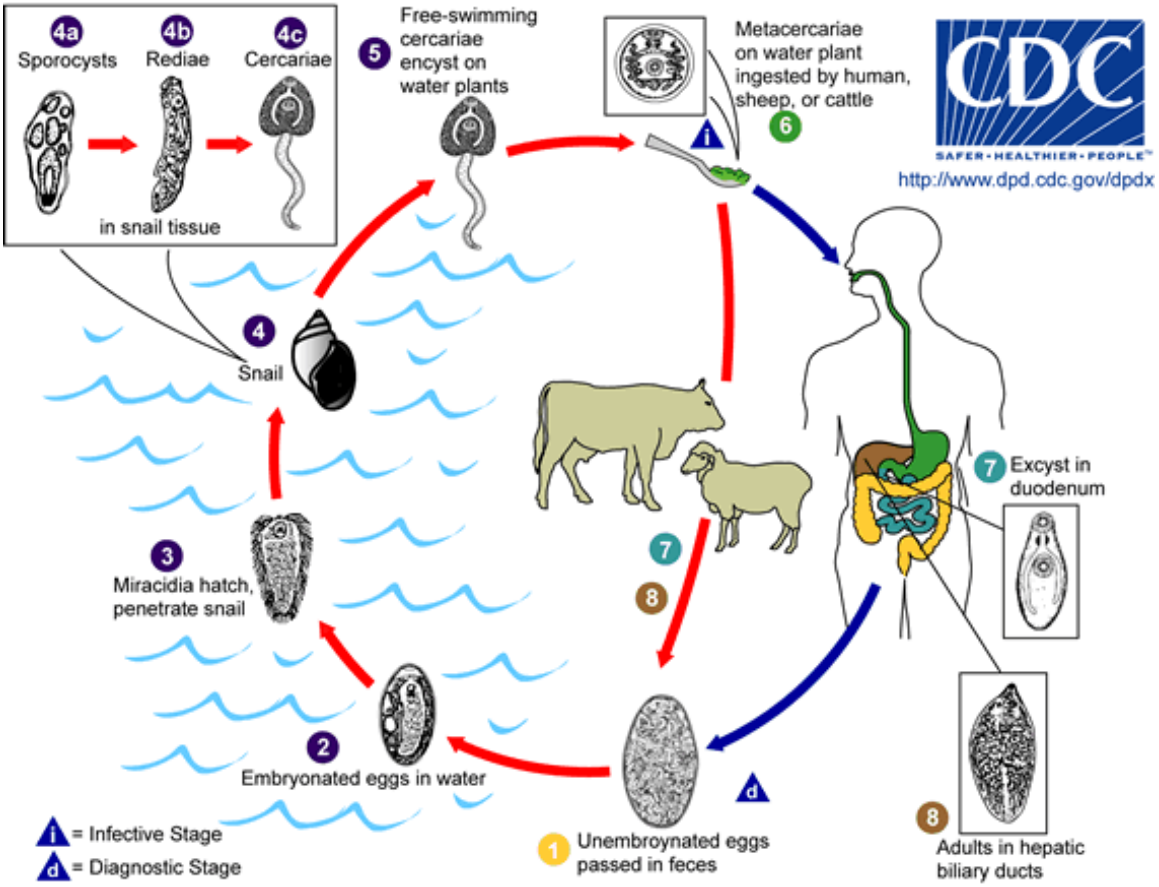
Diagnosis is made by identification of eggs in the feces. There is no serologic test. Praziquantel and bithionol are effective drugs. Adult flukes in the pharynx and larynx can be removed surgically. Prevention involves not eating wild aquatic vegetables or raw sheep liver.





Fasciola hepatica or *Fasciolopsis buski*.
Unembryonated operculated ovum.

Fasciola hepatica



***Fasciola hepatica* life cycle**

3-Fasciolopsis buski

It 's causes **Fasciolopsis** Humans are infected by eating aquatic vegetation that carries the cysts. After excysting in the small intestine, the parasites attach to the mucosa and differentiate into adults. Eggs are passed in the feces; on reaching fresh water, they differentiate into miracidia. The ciliated miracidia penetrate snails and, after several stages, develop into cercariae that encyst on aquatic vegetation. The cycle is completed when plants carrying the cysts are eaten.

Pathologic findings are due to damage of the intestinal mucosa by the adult fluke. Most infections are asymptomatic, but ulceration, abscess formation, and hemorrhage can occur. Diagnosis is based on finding typical eggs in the feces. Praziquantel is the treatment of choice. Prevention consists of proper disposal of human sewage.

Nematodes (roundworms)

The nematodes are elongated, non segmented worms that are tapered at both ends. Unlike other helminths, nematodes have a complete digestive system, including a mouth, an intestine that spans most of the body length, and an anus. The body is protected by a tough, non cellular cuticle. Most nematodes have separate, anatomically distinctive sexes. The mode of transmission varies widely, depending on the species and includes direct skin penetration by infectious larvae, ingestion of contaminated soil, eating undercooked pork, and insect bites. The parasites can invade almost any part of the body: liver, kidneys, intestines, subcutaneous tissue, or eyes. Generally, nematodes are categorized by whether they infect the intestine or other tissues. Alternatively, they can be divided into those for which the eggs are infectious and those for which the larvae are infectious.

1.Ascaris lumbricoides (Giant (roundworms)
2.Enterobes (pinworm)
3. Trichuris trichiura (Whip worm)
4.Anchylostoma (Hook worm)

1.Ascaris lumbricoides (Giant roundworms)

A more serious disease of worldwide occurrence is **ascariasis, caused by Ascaris lumbricoides**. The disease transmitted by ingesting the soil containing egg. Larva grow in the intestine, causes intestine obstruction, may pass to the blood and through the lung. Transmitted by ingestion of soil containing the organism's eggs. Humans are the sole host.

Adults

Adult *A. lumbricoides* worm usually assume a creamy – white color with a tint of pink . Fine striations are visible on the cuticle . Ascaris adult worms are the largest known intestinal nematodes. The average adult male is small only seldom reaching 30 cm in length. The male is characteristically slender and possesses a prominent incurved tail. The adult female measures 22 to 35 cm in length and resembles a pencil lead in thickness.

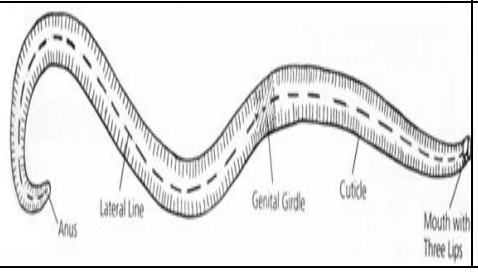
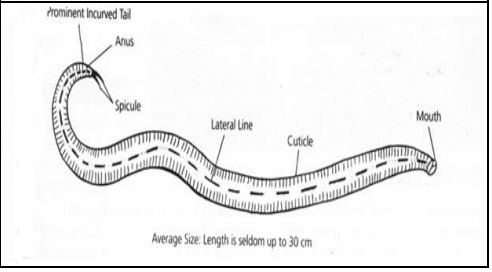
Table *A. lumbricoides* adults : Typical characteristics		
Characteristic	**Adult female**	**Adult male**
length	22 to 35 cm	Up to 30 cm
Color	Creamy – white pink tint	Creamy – white pink tint
Other features	Pencil – lead thickness	Prominent incurved tail
figure		

Table *A. lumbricoides* fertilized egg: Typical characteristics	
Size	40 to 75 µm by 30 to 50 µm
Shape	Rounder than nonfertilized version
Embryo	Undeveloped unicellular embryo
Shell	Thick , chitin
Other features	My be corticated or decorticated

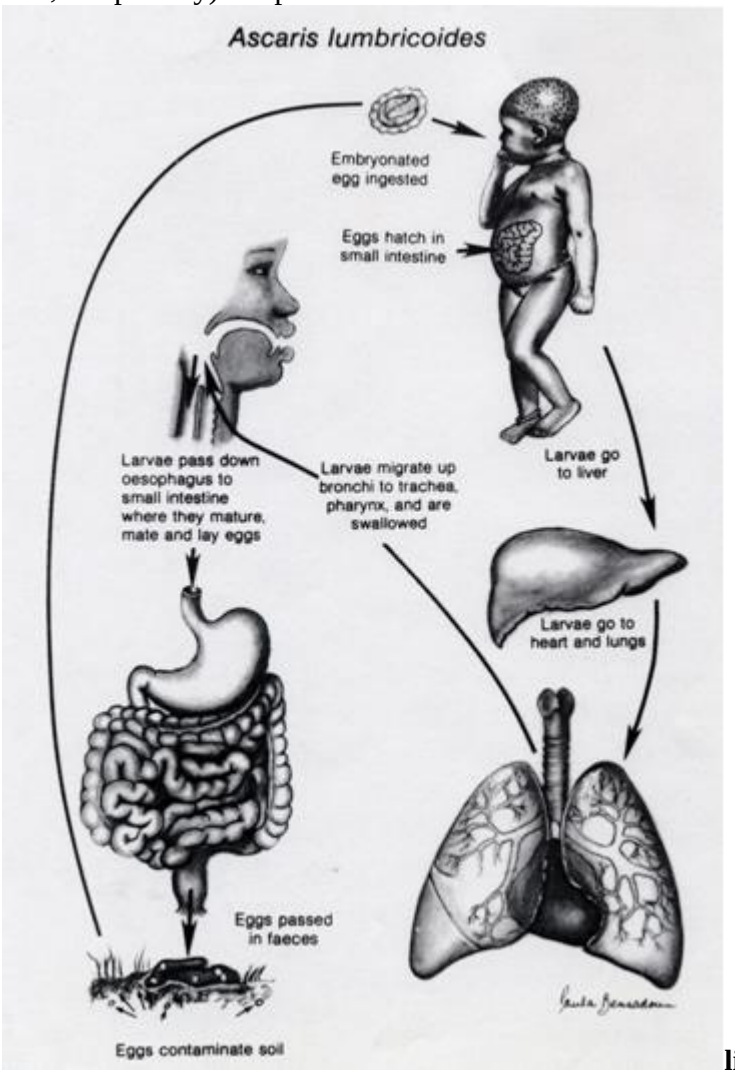
Table: *A. lumbricoides* nonfertilized egg: Typical characteristics	
Shape, Size	Varies,85 to 95 µm by 38 to 45 µm size variations possible
Embryo	Unembryonated ; amorphous mass of protoplasm
Shell	Thin
Other features	Usually corticated

Life cycle

The life cycle of *A. lumbricoides* is relatively complex compared with the parasites presented thus far. Infection begins following the ingestion of infected eggs that contain viable larvae. Once inside the small intestine, the larvae emerge from the eggs. The larvae then complete a liver lung migration by first entering the blood via penetration through the

intestinal wall. the first "stop" on this journey is the liver . From there, the larvae continue the trip via the blood stream to the second "stop" the lung. Once inside the lung, the larvae burrow their way through the capillaries into the alveoli. Migration into the bronchioles then follows. From here, the larvae are transferred through coughing into the pharynx, where they are then swallowed and returned to the intestine.

Maturation of the larvae occurs, resulting in adult worms, which take up residence in the small intestine. The adults multiply and a number of the resulting undeveloped eggs (up to 250,000 per day) are passed in the feces.



life cycle of *A. lumbricoides*

Clincal symptoms

Ascariasis / Roundworm Infection :

Patients who develop symtomatic ascariasis may be infected with as few as a single worm . such a worm may produce tissue damage as it migrates through the host. a secondary bacterial infection may also occur following worm perforation out of the intestine.

Patients infected with many worms may exhibit vague abdominal pain, vomiting, fever, and distention. Mature worms may entangle themselves into a mass that may ultimately

obstruct the intestine, appendix, liver, or bile duct. Such intestinal complications may result in death. In addition, discomfort from adult worms exiting the body through the anus, mouth, or nose may occur. Heavily infected children who do not practice good eating habits may develop protein malnutrition.
Diagnosis: egg in stool, (ovum have heavy protective tuberculated shall). **Treated** with mebendazole.

2. Enterobiasis (pinworm)

The most common nematode infection is **Enterobies (pinworm disease)** is caused by *Enterbius vermicularis*, which causes anal itching with white worms visible in stool or perianal region but otherwise does little damage. The disease transmitted by ingesting the egg. Diagnosis: egg in stool (unembryonated ovum, flattening on side, thin shell. Deposited on perianal skin). Treated with mebendazole.

3. Trichuris trichiura (Whip worm)

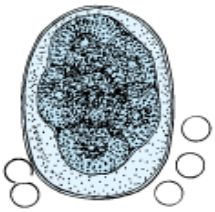
This disease is caused by **Trichuris trichiura.** The infection is usually asymptomatic; however, abdominal pain, diarrhea, and rectal prolapsed can occur. The disease transmitted by ingesting the egg. Diagnosis egg in stool, (unembryonated double plug ovum). Treated with mebendazole.

4. Anchylostoma (Hook worm)

This disease is caused by **Anchylostomaduodenale.** The worm attaches to the intestinal mucosa, causing anorexia, ulcer-like symptoms, and chronic intestinal blood loss, leading to anemia. This disease is transmitted through directed skin penetration by larvae found in soil. Diagnosis egg in stool (thin shall, 4-8 cell stage).



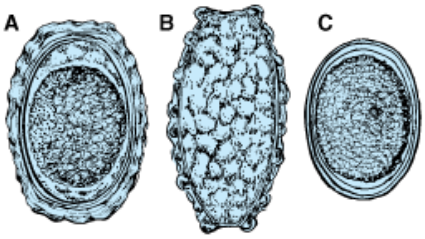
Trichuris trichiura. Unembryonated double-plug ovum.



Ancylostoma duodenale or *Necator americanus.* Note shape, thin shell, 4- to 8-cell stage.



Enterobius vermicularis. Embryonated ovum. Note flattening on one side, thin shell. Deposited on perianal skin.



Ascaris lumbricoides. **A**: Fertilized unembryonated ovum; **B**: unfertilized ovum; **C**: fertilized decorticated ovum. Note heavy protective tuberculated shell in **A**.

Figure eggs of Nematoda

Table of Nematoda types

Organism	Insect Vector	Stage That Infects Humans	Stage(s) in Humans Most Associated with Disease	Important Stage(s) Outside of Humans
Enterobius	None	Eggs	Female worm migrates out anus and lays eggs on perianal skin, causing itching	None
Trichuris	None	Eggs	Worms in colon may cause rectal prolapse	Eggs survive in environment
Ascaris	None	Eggs	Larvae migrate to lung, causing pneumonia	Eggs survive in environment
Ancylostoma and *Necator*	None	Filariform larvae enter skin	Worms in colon cause blood loss (anemia)	Egg → rhabditiform larvae → filariform larvae

Chapter six

Viruses

Introduction to the Viruses

Viruse are extremely small infective agents. A complete particle, or virion, has much simpler structure than a cell. It essentially consist of a block of genetic material (either DNA or RNA but not both) surrounded by proteinaceous coat that protects it from the environmental damage and aid in its transmission from host to host, the protein coat of virus is called the capsid, designed to protect the genome, the capsid and nucleic acid called Nucleocapsid.
The payload is the viral genome and may also include enzymes required for the initial steps in viral replication process that is obligatory intracellular. The pathogenicity of a virus depends on a great variety of structural and functional characteristics. Therefore, even within a closely related group of viruses, different species may produce significantly distinct clinical pathologies. Some viruses are **enveloped &other unenveloped (Naked viruses**).An important structural feature used in defining a viral family is the presence or absence of a lipid-containing membrane surrounding the nucleocapsid. This membrane is referred to as the envelope. A virus that is not enveloped is referred to as a naked virus. In enveloped viruses, the nucleocapsid is flexible and coiled within the envelope, resulting in most such viruses appearing to be roughly spherical. The envelope is derived from host cell membranes. However, the cellular membrane proteins are replaced by virus-specific proteins, conferring virus-specific antigenicity upon the particle.

Note:
Viruses containing lipid envelopes are sensitive to damage by harsh environments and, therefore, tend to be transmitted by the respiratory, parenteral, and sexual routes. Nonenveloped viruses are more stable to hostile environmental conditions and often transmitted by the fecal-oral route.

Virion: is a complete virus particle combining these structural elements.
Prions: This infectious protein is designated the prion protein without nucleic acid.
Viriod: This infectious nucleic acid without protein.

Viruses are divided into related groups, or families, and, sometimes into subfamilies based on: 1) type and structure of the viral nucleic acid, 2) the strategy used in its replication, 3) type of symmetry of the virus capsid, and 4) presence or absence of a lipid envelope. Within a virus family, differences in additional specific properties, such as host range, serologic reactions, amino acid sequences of viral proteins, degree of nucleic acid homology, among others, form the basis for division into genera (singular = genus) and species.

Genome
The type of nucleic acid found in the virus particle is perhaps the most fundamental and straightforward of viral properties. It may be RNA or DNA, either of which may be single-stranded (ss) or double-stranded (ds). The most common forms of viral genomes found in nature are ssRNA and dsDNA. However, both dsRNA and ssDNA genomes are found in viruses of medical significance. Single-stranded viral RNA genomes are further subdivided into those of positive polarity (that is, of messenger RNA sense, which can therefore be used as a template for protein synthesis), and those of negative polarity or are

antisense (that is, complementary to messenger RNA sense, which cannot therefore be used directly as a template for protein synthesis). Viruses containing these two types of RNA genomes are commonly referred to as positive-strand and negative-strand RNA viruses, respectively.

Type of symmetry of the virus capsid, capsids normally have one of three shapes
1.icosahedral (as in the poliovirus).
2-helical (as in the tobacco mosaic virus)
3.complex (as in the bacteriophages , or phages).

Viral Replication: the One-Step Growth Curve
A. Attachment of a virus to the host cell
B. **Eclipse period**
This is the eclipse period, and it represents the time elapsed from initial entry and disassembly of the parental virus to the assembly of the first progeny virion. This period falls within a range of 1 to 20 hours.
C. Exponential growth
The number of progeny virus produced within the infected cell increases exponentially for a period of time.

Steps in the Replication Cycles of Viruses
The individual steps in the virus replication cycle are presented below in sequence,
1. Adsorption (attachment to the host cell)
2. Penetration
3. Uncoating of the viral genome
4. Gene expression and replication
A. Mechanisms of DNA virus genome replication
B. Mechanisms of RNA virus genome replication
5. Assembly and release of progeny viruses

Effects of viral infection on the host cell
The response of a host cell to infection by a virus ranges from:
1) Little or no detectable effect.
2) Alteration of the antigenic specificity of the cell surface due to presence of virus glycoproteins.
3) Latent infections that, in some cases, cause cell transformation.
4) Ultimately, to cell death due to expression of viral genes that shut off essential host cell functions.

Table: the following tables shows the type of viruses and diseases & viruses vaccines

Family	Viruses	Type of NA	Diseases
Pox viruses	Variola	DNA	Smallpox
herpes viruses	Herpes simplex type1&2	DNA	Cold, genital sores, encephalitis
	Varicella-zoster	DNA	Chickenpox, shingles
	Cytomegalovirus	DNA	Cytomegalic inclusion disease of neonates, pneumonia in immunocompromised patients
	Epstein- Barr (EB)	DNA	Infectious mononucleosis (cancer)
Adeno viruses	Adeno viruses	DNA	Sore throat, conjunctivitis, haemorrhgic cystitis
Papova viruses	Papilloma	DNA	Warts, cervical cancer
Hepadna viruses	Hepatitis A, BC,D	DNA	Hepatitis B, liver cancer
Reo viruses	Rota viruses	RNA	Causes diarrhea in infant
Picprna viruses	rhino viruses	RNA	Common cold
Retero viruses(HIV)	Human immuno-deficiency viruses	RNA	AIDS
Rhabdo viruses	rabies viruses	RNA	Causes human rabies
paramyxo viruses	Mumps	RNA	Causes mumps in children &encephalitis
	Measles	RNA	Causes measles in children (rash, maculopapules)
orthomyxovirus	Influenza A,B	RNA	Influenza
Togo viruses	Rubella	RNA	Causes measls in children (rash)

Vaccines

The availability of vaccines has resulted in the global eradication of smallpox and the virtual elimination of poliomyelitis, tetanus, and diphtheria. Protection of individuals from disease by vaccination can take two forms: passive and active immunization.

Passive immunization is achieved by injecting a recipient with preformed immunoglobulins directed against an already present infection, whereas

active immunization involves injection of modified or purified pathogens or their products. Both provide protective immune responses. Active and passive immunization differ in significant ways, and the situations under which one or the other or a combination (active-passive immunization) is preferred depends on the infecting microorganism, age of the patient, anticipated imminent contact with a pathogen, or time elapsed since contact.

Passive Immunization

Passive immunization is achieved by injecting a recipient with preformed immunoglobulins obtained from human (or, occasionally, equine) serum. Passive immunization provides immediate protection to individuals who have been exposed to an infectious organism and who lack active immunity to that pathogen. Because passive immunization does not activate the immune system, it generates no memory response. Passive immunity is not permanent, but dissipates after a few weeks to months as the immunoglobulins are cleared from the recipient's serum.

A. Types of immunoglobulins used to give passive immunity (IgG)

This type of immunization is effective when given immediately before or after exposure to an infectious disease, such as hepatitis A.

Hyperimmune human immunoglobulin: This type of immunoglobulin contains high concentrations of antibodies directed against a specific pathogen or toxin (for example, varicella-zoster immunoglobulin or diphtheria antitoxin).

B. Adverse effects

There are risks associated with the injection of preformed antibody. For example, the recipient can mount an adverse response to the antigenic determinants of the foreign antibody, potentially leading to systemic anaphylaxis. [Note: This is particularly true when the immunoglobulins are obtained from a nonhuman source, such as a horse.]

Active Immunization

Active immunization is achieved by injection of viable or nonviable pathogens, or purified pathogen product, prompting the immune system to respond as if the body were being attacked by an intact infectious microorganism. Whereas passive immunization provides immediate protection, active immunization may require several days to months to become effective. Active immunization leads to prolonged immunity and is generally preferred over the short-term immunity provided by passive immunization with preformed immunoglobulins.

Simultaneous administration of active and passive immunizations may be required after exposure to certain infections, such as hepatitis B.

A. Formulations for active immunization

Vaccines are made with 1) live, attenuated microorganisms; 2) killed microorganisms; 3) microbial extracts; 4) vaccine conjugates; or 5) inactivated toxins (toxoids). Both bacterial and viral pathogens are targeted by these diverse means.

A: Live pathogens: When live pathogens are used, they are attenuated (weakened) to preclude clinical consequences of infection. Attenuated microbes reproduce in the recipient, typically leading to a more robust and long-lasting immune response than can be obtained through vaccination with killed organisms. However, with live, attenuated vaccines there is a possibility that the attenuated vaccine strain will revert to an active pathogen after administration to the patient. For example, vaccine-associated poliomyelitis occurs following administration of approximately one of every 2.4 million doses of live polio vaccine. Also, live, attenuated vaccines should not be given to immunocompromised individuals because there is the potential for a disseminated infection.

B: Killed microorganisms: Killed vaccines have the advantage over attenuated microorganisms in that they pose no risk of vaccine-associated infection. As noted above, killed organisms often provide a weak or short-lived immune response. Some vaccines, such as polio and typhoid vaccines, are available both in live and killed versions.

C: **Microbial extracts**: Instead of using whole organisms, vaccines can be composed of antigen molecules (often those located on the surface of the microorganism) extracted from the pathogen or prepared by recombinant DNA techniques. The efficacy of these vaccines varies. In some instances, the vaccine antigen is present on all strains of the organism, and the vaccine thus protects against infection by all strains. With other pathogens, such as pneumococcus, protective antibody is produced against only a specific capsular polysaccharide one among more than eighty distinct types. Immunity to one polysaccharide type does not confer immunity to any other type. For this reason, the pneumococcal vaccine is composed of 23 different polysaccharides, comprising the antigens produced by the most common types of disease-causing pneumococci. Some pathogens, such as influenza virus, frequently change their antigenic determinants. Therefore, influenza virus vaccines must also change regularly to counter the different antigens of influenza A and B virus strains in circulation.

D: Vaccine conjugates: Vaccines can produce humoral immunity through B cell proliferation leading to antibody production, which may or may not involve helper T cells. For example, pneumococcal polysaccharide and the polysaccharide of Haemophilus influenzae type b induce B-cell type-specific protective antibody without involvement of helper T cells. These T cell independent responses are characterized by low antibody titers, particularly in children younger than eighteen months. Thus, conventional H. influenzae polysaccharide vaccine does not provide protection for children three to eighteen months. Consequently, this organism has, in the past, produced severe infections in this age group. However, by covalently conjugating the Haemophilus polysaccharide to a protein antigen, such as diphtheria toxoid protein, Haemophilus vaccines produce a robust T cell dependent antibody response even in three-month-old infants. A conjugate vaccine for Streptococcus pneumoniae, and one for Neisseria meningitidis, are also currently available.

E: Toxoids: These are derivatives of bacterial exotoxins produced by chemically altering the natural toxin, or by engineering bacteria to produce harmless variants of the toxin. Vaccines containing toxoid are used when the pathogenicity of the organism is a result of the secreted toxin. Depending on the specific vaccine, administration is generally via intramuscular or subcutaneous routes. shows the formulation of some of the vaccines currently licensed in the United States. Details of the various vaccines are presented in the chapters in which the target microorganisms are discussed. In the case of rhinovirus infections the leading cause of the common cold least 100 types of the virus are known. It is not practical to develop a vaccine that confers protection to this large number of antigenic types.

Types of immune response to vaccines

Vaccines containing killed pathogens (such as hepatitis A or polio vaccine) or antigenic components of pathogens (such as hepatitis B vaccine) do not enter host cells; therefore, they give rise to a primary B cell mediated humoral response. These antibodies are ineffective in attacking intracellular organisms. By contrast, attenuated live vaccines (usually viruses) do penetrate cells. This results in the production of intracellular antigens that are displayed on the surface of the infected cell, prompting a cytotoxic T cell response, which is effective in eliminating intracellular pathogens.

Effect of age on efficacy of immunization

- **Passive immunity from mother:** Newborns receive serum IgG antibodies from their mothers, which gives them temporary protection against those diseases to which the mother was immune. In addition, maternal milk also contains secretory antibodies that provide some protection against intestinal and respiratory tract infections.
- **Active immunization**: The infant's antibody-producing capacity develops slowly during the first year of life. Although the immune system is not fully developed, it is desirable to begin immunization at two months of age because diseases are common in this age group, and can be particularly severe (for example, whooping cough, H. influenzae meningitis). As with infants, the elderly have a reduced antibody response to vaccines.

Adverse reactions to active vaccination

Adverse consequences of vaccinations range from mild to severe and even life-threatening. Symptoms vary among individuals and with the nature of the vaccination. Among the most common and mildest consequences of immunization are tenderness and swelling at the site of injection, and a mild fever.

Bacterial Vaccines

Vaccines against commonly encountered bacterial are described below.

Less common bacterial pathogens

Anthrax (Bacillus anthracis): Anthrax vaccine consists of a noninfectious sterile filtrate from the culture of an attenuated strain of B. anthracis that contains no dead bacteria. The filtrate is adsorbed to an adjuvant, aluminum hydroxide. [Note: Adjuvants are substances that when injected with an antigen, serve to enhance the immunogencity of that antigen.] The incidence of all forms of naturally occurring anthrax is low, particularly the inhalation form of the disease. Thus, there is no opportunity to conduct field trials of the vaccine against inhalation anthrax, the form most likely to be used in a biologic attack. Safety and efficacy of the vaccine are supported by studies in nonhuman primates where efficacy was close to 100%. Vaccine is recommended for goat hair and woolen mill workers, veterinarians, laboratory workers, and livestock handlers who are at risk as a result of occupational exposure.

Cholera (Vibrio cholerae): The vaccine contains killed bacteria and is given to travelers.

Typhoid fever (Salmonella typhi): The most commonly used vaccine contains an attenuated recombinant strain of S. typhi. It is given to individuals living in or traveling to high-risk areas, and to members of the military.

Plague (Yersinia pestis): The vaccine contains killed bacteria, and is given to high-risk individuals.

Viral Vaccines

Immunity to viral infection requires an immune response to antigens located on the surface of the viral particles, or on virus-infected cells. For enveloped viruses, these antigens are often surface glycoproteins. The main limitation of viral vaccines occurs with viruses that show a genetically unstable antigenicity (that is, they exhibit antigenic determinants that continuously vary, such as with influenza viruses or the human immunodeficiency virus). Common viral pathogens for which there are vaccines include the following:

A. Hepatitis A

Formalin-inactivated whole virus vaccine produces antibody levels in adults similar to those observed following natural infection, and approximately fifteen times those achieved by passive injection of immunoglobulin. Projections indicate that immunity from hepatitis A virus will probably last for approximately ten years after two doses of vaccine. Currently hepatitis A virus vaccine is not recommended for children younger than two years because residual anti-HAV passively acquired from the mother may interfere with vaccine immunogenicity.

B. Hepatitis B

The current vaccine contains recombinant hepatitis surface antigen. Efficacy is 95 to 99 % in healthy infants, children, and young adults. Its use is indicated for healthcare workers in contact with blood, and persons residing in an area with a high rate of endemic disease. Immunoglobulins obtained from hyperimmunized humans can provide passive immunity after accidental exposure (a needle stick, for the neonate of an infected mother). Active and passive treatments can be administered into different sites at the same time.

C. Varicella-zoster

This vaccine contains live, attenuated, temperature-sensitive varicella-zoster virus. Its efficacy in preventing chickenpox is approximately 85 to 100 %t in children, and this immunity is persistent.

D. Polio

Vaccination is the only effective method of preventing poliomyelitis. Both the inactivated polio vaccine and the live, attenuated, orally administered polio vaccine have established efficacy in preventing poliovirus infection and paralytic poliomyelitis.

Inactivated poliovirus (Salk) vaccine: Because the inactivated vaccine cannot cause poliomyelitis, it is safe for use in immunocompromised persons and their contacts. The

disadvantages of this inactivated vaccine are: 1) administration is by injection only; and 2) it provides less gastrointestinal immunity, resulting in the possibility of asymptomatic infection of the gastrointestinal tract with wild poliovirus, which could be transmitted to other persons.

Attenuated live poliovirus (Sabin) vaccine: Advantages of this vaccine include: 1) it can be administered orally; 2) it provides life-long protection from poliovirus for more than 95 % of recipients after the primary three-dose series; and 3) it provides early intestinal immunity. The main disadvantage of attenuated live virus vaccine is a small risk of infection, estimated to be 1 per 2.4 million doses.

E. Influenza

The traditional flu shot vaccine contains formalin-inactivated virus. A live, attenuated influenza vaccine is administered intranasal. The vaccine provides peak protection about two weeks after its administration. Vaccine efficacy of seventy to ninety percent is generally achieved in young adults. The vaccine is recommended for adults older than 65, high-risk persons six months or older, and those who might transmit the virus to persons at high risk. **Antigenic drift** requires that individuals be vaccinated against influenza annually prior to the winter flu season.

F. Measles, mumps, and rubella (MMR)

This combination vaccine contains live, attenuated virus, and should be administered to young children prior to entering school. Measles vaccine should also be administered to individuals traveling in endemic areas.

DNA Vaccines

DNA vaccines represent a new approach to vaccination. The proposed mechanism for these vaccines can be summarized as follows: The gene for the antigen of interest is cloned into a bacterial plasmid that is engineered to increase the expression of the inserted gene in mammalian cells. After being injected, the plasmid enters a host cell where it remains in the nucleus as an episome (that is, it is not integrated into the cell's DNA). Using the host cell's protein synthesis machinery, the plasmid DNA in the episome directs the synthesis of the protein it encodes. This antigenic microbial protein may leave the cells and interact with T helper and B cells, or it may be cleaved into fragments and presented as MHC I antigen complex on the cell surface, resulting in activation of killer T cells.

Table shows of viruses vaccines

Use	Vaccine	Type	Cell Substrate
Common	Hepatitis A	Killed	Human diploid fibroblasts (MRC-5)
	Hepatitis B	Subunit (HBsAg)	Yeast (recombinant DNA)
	influenza A and B	Killed	Embryonated chicken eggs
	Measles	Live	Chicken embryo fibroblasts
	Mumps	Live	Embryonated chicken eggs and chicken embryo fibroblasts
	Poliovirus (IPV)	Killed	Monkey kidney cells (Vero)
	Poliovirus (OPV)	Live	Monkey kidney cells
	Rabies	Killed	Human diploid fibroblasts (MRC-5) or rhesus fetal lung diploid cells or chicken fibroblasts
	Rubella	Live	Human diploid fibroblasts (WI38)
	Varicella-zoster	Live	Human diploid fibroblasts (MRC-5)
Special situations	Adenovirus[1]	Live	Human diploid fibroblasts (WI-38)
	Japanese encephalitis[2]	Killed	Mouse brain
	Smallpox	Live	Calf lymph
	Yellow fever[2]	Live	Embryonated chicken eggs

Viruses Medical Families

Family of Enveloped DNA Viruses

Human Herpesvirus Types 1and 2**,** Varicella-Zoster Virus**,** Human Herpesvirus Types 6 and 7**,** Human Herpesvirus Type 8**,** Epstein-Barr Virus**&**Human cytomegalovirus (HCMV)

All herpesviruses can undergo an alternative infection cycle, entering a quiescent state (latency) from which they subsequently can be reactivated. The cell type in which this occurs is usually not the same cell type in which productive, cytocidal infection occurs. Because the mechanism of latency, the cells in which it is established, the frequency of reactivation, and the nature of the recurrent disease are characteristic for each of the herpesviruses. Because these enzymes are virus-specific, they provide excellent targets for antiherpes agents (such as acyclovir), that are relatively nontoxic for the cell.

VIRUS	VIRUS SUBFAMILY	CLINICAL MANIFESTATIONS OF PRIMARY INFECTION	CLINICAL MANIFESTATIONS OF RECURRENT INFECTION	SITE OF INITIAL INFECTION	SITE OF LATENCY
Herpes simplex-1	α	Keratoconjunctivitis, gingivostomatitis, pharyngitis, tonsilitis	Herpes labialis ("cold sores")	Mucoepithelial	Trigeminal sensory ganglia
Herpes simplex-2	α	Genital herpes; perinatal disseminated disease	Genital herpes	Mucoepithelial	Lumbar or sacral sensory ganglia
Varicella-zoster virus	α	Varicella ("chickenpox")	Herpes-Zoster ("shingles")	Mucoepithelial	Dorsal root ganglia
Cytomegalo-virus	β	Congenital infection (*in utero*); mono-nucleosis-like syndrome	Asymptomatic shedding of virus	Monocytes, lymphocytes, and epithelial cells	Monocytes, lymphocytes
Epstein-Barr virus	γ	Infectious mono-nucleosis; Burkitt lymphoma	Asymptomatic shedding of virus	Mucosal epithelium, B lymphocytes	B lymphocytes

Herpes simplex virus, types 1 and 2

HSV-1 and HSV-2 are the only human herpesviruses that have a significant degree of nucleotide homology (about fifty percent). They therefore share many common features in replication, disease production, and latency.

A. Epidemiology and pathogenesis

Transmission of both HSV types is by direct contact with virus-containing secretions or with lesions on mucosal or cutaneous surfaces. Primary or recurrent infections in the oropharyngeal region, caused primarily by HSV-1, are accompanied by virus release into saliva; therefore kissing or saliva-contaminated fingers are major modes of transmission. In genital tract infections, caused primarily by HSV-2, virus is present in genital tract secretions. Consequently, sexual intercourse and infections of newborns during passage through the birth canal are major modes of transmission. Both HSV-1 and HSV-2 multiply in epithelial cells of the mucosal surface onto which they have been inoculated, resulting in production of vesicles or shallow ulcers containing infectious virus. In immunocompetent individuals, epithelial infection remains localized because cytotoxic T lymphocytes recognize the HSV-specific antigens on the surface of infected cells and kill these cells before progeny virus has been produced. A lifelong latent infection is usually

established in the regional ganglia as a result of entry of infectious virions into sensory neurons that terminate at the site of the infection.

Clinical significance
A useful generality is that HSV-1 is most commonly found in lesions of the upper body, and HSV-2 is more commonly the cause of genital tract lesions. Either can, however, infect and cause similar lesions at the opposite site.

Primary infections of the upper body: Many primary HSV infections are subclinical, but the most common symptomatic infections of the upper body are gingivostomatitis in young children and pharyngitis or tonsillitis in adults. The lesions typically consist of vesicles and shallow ulcers, which are often accompanied by systemic symptoms such as fever, malaise, and myalgia. Another clinically important site of infection is the eye, in which keratoconjunctivitis can lead to corneal scarring and eventual blindness. If HSV infection spreads to the central nervous system (CNS) it can cause encephalitis, which, if untreated, has a mortality rate estimated to be seventy percent. Survivors are usually left with neurologic deficits.

Primary infections of the genital tract: Primary genital tract lesions are similar to those of the oropharynx; however, based on the frequency of antibody in the population, the majority of these infections are asymptomatic.When symptomatic (genital herpes), local symptoms include painful vesiculoulcerative lesions on the vulva, cervix, and vagina, or penis. Systemic symptoms of fever, malaise, and myalgia may also be more severe than those that accompany primary oral cavity infections. In pregnant women with a primary genital HSV infection, the risk of infecting the newborn during birth is estimated to be thirty to forty percent (neonatal herpes). Because such infants have no protective maternal antibody, a disseminated infection, often involving the CNS, results. There is a high mortality rate if untreated, and survivors are likely to have permanent neurologic sequelae. A newborn is also at risk of acquiring infection from an infected mother by transfer on contaminated fingers or in saliva. However, infection in utero appears to occur only rarely.

Latency: In latently infected cells of the ganglia HSV-1 in trigeminal ganglia and HSV-2 in sacral or lumbar ganglia from one to thousands of copies of the viral genome are present as nonintegrated, circular molecules of DNA in the nuclei Expression of HSV genes is shut off in latently infected cells.

Reactivation: Several factors, such as hormonal changes, fever, and physical damage to the neurons, are known to induce reactivation and replication of the latent virus. The newly synthesized virions are transported down the axon to the nerve endings from which the virus is released, infecting the adjoining epithelial cells. Characteristic lesions are thus produced in the same general area as the primary lesions. [Note: Virus replication occurs in only a fraction of the latently infected neurons, and these nerve cells eventually die.] The presence of circulating antibody does not prevent this recurrence, but does limit the spread of virus to surrounding tissue. Sensory nerve symptoms, such as pain and tingling, often precede and accompany the appearance of lesions. In general, the severity of any systemic symptoms is considerably less than that of a primary infection, and many

recurrences, in fact, are characterized by shedding of infectious virus in the absence of visible lesions.

HSV-1: The frequency of oropharyngeal symptomatic recurrences is variable, ranging from none to several a years. The lesions occur as clusters of vesicles at the border of the lips (herpes labialis or cold sores, fever blisters) and heal without scarring in eight to ten days.

HSV-2: Reactivation of HSV-2 genital infections can occur with considerably greater frequency (for example, monthly) and is often asymptomatic, but still results in viral shedding. Consequently, sexual partners or newborn infants may be at increased risk of becoming infected resulting from lack of precautions against transmission. The risk of transmission to the newborn is much less than in a primary infection because considerably less virus is shed and there is maternal anti-HSV antibody in the baby. This antibody also lessens the severity of the disease if infection does occur. In the United States, HSV-1 infection of the eye is the second most common cause of corneal blindness (after trauma), HSV infections of the CNS account for up to twenty percent of encephalitis viral infections.

D. Treatment: The guanine analog, acycloguanosine (acyclovir),

Human Cytomegalovirus

Human cytomegalovirus (HCMV) is differs from HSV and VZV in several ways. Its replication cycle is significantly longer, and infected cells typically are greatly enlarged and multinucleated (hence, cytomegalo). There is only one recognized human species of HCMV, but there are many distinct strains that can be distinguished by antigenic differences as well as by restriction fragment analysis of their genomes. HCMV is the most common cause of intrauterine infections and congenital abnormalities in the United States. It also represents a serious threat to immunodeficient or immunosuppressed patients.

Epidemiology and pathogenesis

Initial infection with HCMV commonly occurs during childhood. Depending on geographic location and socioeconomic group, 35 to 90 percent of the population have antibody against the virus by adulthood.

Transmission: Infection in children is usually asymptomatic; these children continue to shed virus for months in virtually all body fluids, including tears, urine, and saliva. Transmission is by intimate contact with these fluids, although saliva may be the most common source. In adults, the virus can also be transmitted by: 1) sexual means because it is present in semen and vaginal secretions; 2) organ transplants; and 3) blood transfusions. Similarly, virus is present in breast milk and, thus, neonates can be infected by this route. HCMV can also cross the placenta and infect a fetus in utero. Initial replication of the virus in epithelial cells of the respiratory and gastrointestinal (GI) tracts is followed by viremia and infection of all organs of the body. In symptomatic cases, kidney tubule epithelium, liver, and CNS, in addition to the respiratory and GI tracts, are most commonly affected.

Latency and reactivation: A distinctive feature of HCMV latency is the phenomenon of repeated episodes of asymptomatic virus shedding over prolonged periods. Latency is probably established in monocytes and macrophages, but other cell types, such as those of the kidney, are also involved.

Clinical significance

In healthy individuals, primary HCMV infection is usually in apparent. Whereas most infections occur in childhood, primary infection as an adult may result in a mononucleosis syndrome clinically identical to that caused by Epstein-Barr virus (EBV). It is estimated that about eight percent of infectious mononucleosis (IM) cases are caused by HCMV.
Persistent fever, muscle pain, and lymphadenopathy are characteristic IM symptoms, as are elevated levels of abnormal lymphocytes and liver enzymes. Two specific situations have greater clinical significance, namely, congenital infections and infection of immunocompromised patients.

Congenital infections: HCMV is the most common intrauterine viral infection. However, there is a great disparity in incidence of fetal infection and severity of outcome, depending on whether the mother is experiencing a primary or recurrent infection. In women experiencing their first HCMV infection during pregnancy (who, therefore, have not yet produced antibodies against HCMV), 35 to 50 % of fetuses will be infected, and 10% of these will be symptomatic. The severity of the symptoms is most pronounced when infection occurs during the first trimester. Referred to as cytomegalic inclusion disease, results caused by the infection range from fetal death to various degrees of damage to liver, spleen, blood-forming organs, and components of the nervous system. The latter is a common cause of hearing loss and mental retardation. Even in infants who are asymptomatic at birth, hearing deficits and ocular damage (for example, chorioretinitis) may appear later and continue to progress during the first few years.

Family of Orthomyxoviridae & Paraorthomyxoviridae

Orthomyxoviruses are spherical, enveloped viruses containing a segmented.Viruses in this family infect humans, and animal. Orthomyxoviruses are divided into 3 types: influenza A, B, and C. Only types A and B are of medical importance.
Structure: Influenza virions are spherical, enveloped. Two types of spikes project from the surface: one is composed of hemagglutinin (H protein) and the second of neuraminidase (N protein). The RNA is composed of eight distinct segments of RNA.

Pathology and clinical significance

In humans, influenza is spread by respiratory droplets and is an infection solely of the respiratory. There is rarely viremia or spread to other organ systems. Destruction of respiratory epithelial cells is attributed to the host immune response, specifically cytotoxic T cells. Typically, influenza has an acute onset characterized by chills, followed by high fever, muscle aches, and extreme drowsiness. The disease runs its course in four to five days, after which there is a gradual recovery. The most serious problems, such as development of pneumonia, occur in the very young, the elderly, and people with chronic cardiac or pulmonary disease or those who are immunodeficient.

The immunology of influenza viruses

When individuals are infected with influenza virus, antibodies are made against the various viral proteins. However, it is the antibodies made against the H protein that are neutralizing and the best index of protection. The antigenic properties of the influenza virus proteins are also important because they serve as the basis for the classification of influenza viruses.

The classification into subtypes depends on antigens associated with the outer viral proteins, H and N, among human influenza viruses, only three H (H1, H2, and H3) and two N (N1 and N2) subtypes are found. Human influenza viruses are therefore designated, for example, as subtype H1N1, H2N2, and H3N2.

Antigenic variability of influenza viruses: In contrast to viruses such as polio or measles virus that have maintained antigenic stability since they were first isolated, influenza viruses have shown marked variation over the years in antigenic properties, specifically H and N proteins. Two distinct phenomena account for this observation: antigenic drift and antigenic shift.

Antigenic drift: This refers to minor antigenic changes in H and N proteins that occur each year. Antigenic drift does not involve a change in the viral subtype. This phenomenon can be easily explained by random mutations in viral RNA and single or a small number of amino acid substitutions in H and N proteins.

Antigenic shift: This phenomenon involves a much more dramatic change in the antigenic properties of the H and/or N proteins, and a change in subtype, for example, from H1N1 to H3N2. Antigenic shift occurs only infrequently, perhaps every ten or twenty years. For example, the appearance of a new, extremely virulent H1N1 virus, due presumably to antigenic shift, and H1N1 virus was replaced by subtype H2N2; in 1968, H2N2 was replaced by H3N2. Since 1977, multiple subtypes of influenza A have been circulating around the world.

Note: H5N1 (an avian flu virus strain) was first isolated in 1997 from a human. The virus affects individuals who live closely with domestic birds such as chickens.

Consequences of antigenic variation: When antigenic shift occurs and a new subtype of virus appears that has not been in circulation for many years, the immune systems of a large proportion of the population have never encountered that virus; these individuals are, therefore, immunologically unprotected. Thus, the conditions are set for influenza epidemic or even pandemic. Antigenic shift also means that the vaccine that was in use before the antigenic shift will not be effective in protecting against the new subtype of virus; therefore, it becomes necessary to develop a new vaccine as quickly as possible, incorporating the new virus subtype.

The molecular basis of antigenic variation: The dramatic changes associated with antigenic shift result from **reassortment** of viral RNA segments, a process observed with all RNA viruses having a segmented genome. Reassortment results when a cell is infected with two genetically distinct influenza viruses; the genomic RNAs of both parental viruses

are replicated, and progeny viruses are assembled that contain genomic RNA segments from one of the parental viruses, and other genomic segments from the second parent (Figure). In this way, new viruses can be generated that differ from both parents.

Diagnosis: Specific test is the quantitation of HI (hemagglutination inhibition) antibodies.
Treatment: Amantadine, rimantadine, Zanamivir and oseltamivir.

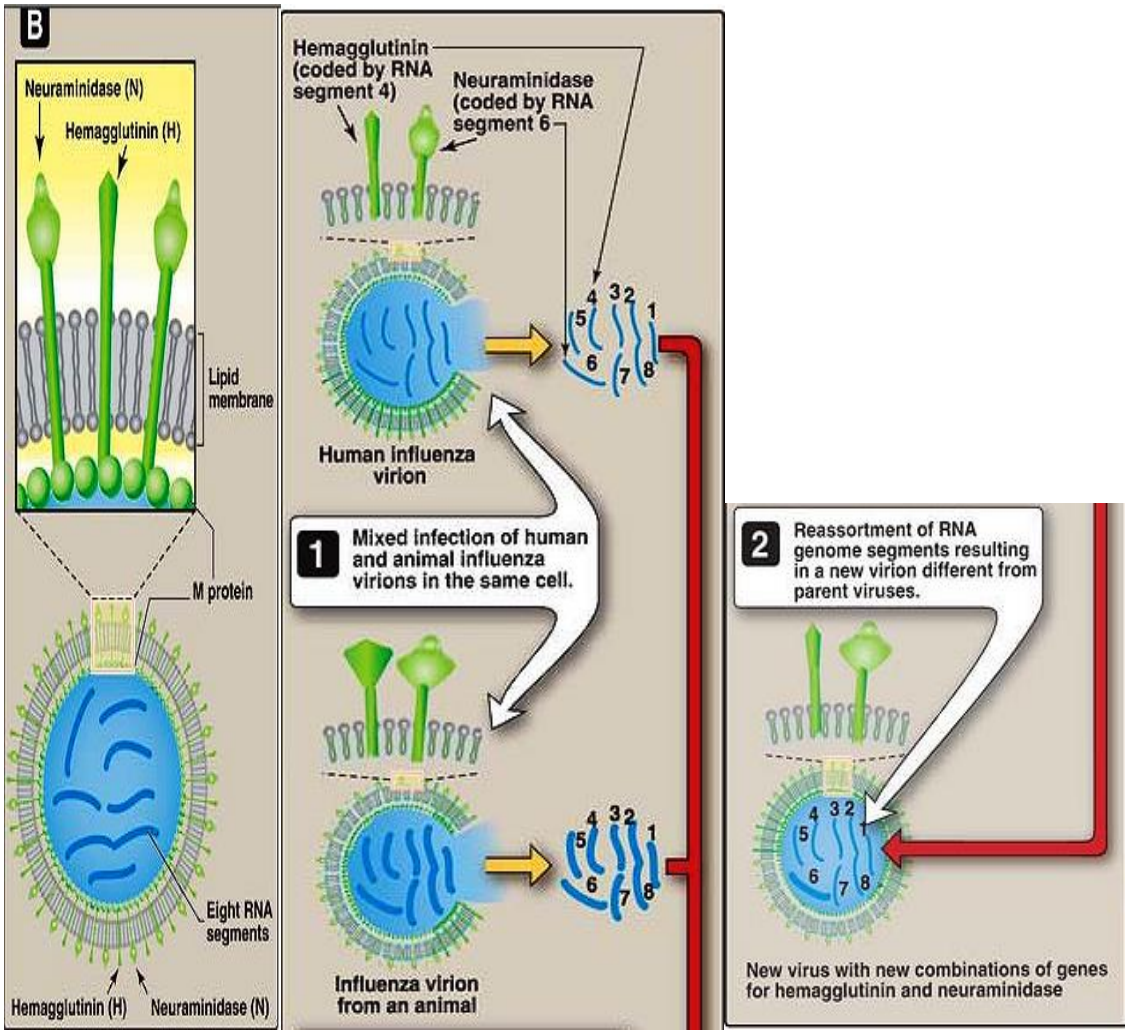


Figure: the immune response not effective against influnzae viures

Paramyxoviridae

The genera include; **parainfluenza** viruses (which cause upper respiratory tract infections), the **mumps** virus, the **measles** virus, and **respiratory syncytial virus** (a major respiratory tract pathogen). Paramyxoviruses are spherical, enveloped particles that contain a nonsegmented, and have envelope. The first, the HN protein (H, and N), is involved in the binding of the virus to a cell; measles virus lacks the neuraminidase activity. The second, the F protein (fusion), functions to fuse viral and cellular membranes, thus facilitating virus entry into the cytoplasm where viral replication occurs.

Mumps virus:

Mumps used to be one of the commonly acquired childhood infections. Adults who escape the disease in childhood could also be infected. In the prevaccine period, mumps was the most common cause of viral encephalitis. Complete recovery may be. The virus is spread by respiratory droplets. Although about one third of infections are subclinical, the classic clinical presentation and diagnosis center on infection and swelling of the salivary glands, primarily the parotid glands. However, infection is widespread in the body and may involve not only the salivary glands but also the pancreas, CNS, and testes. Orchitis (inflammation of the testis) caused by mumps virus may cause sterility.

Measles virus

Measles virus is transmitted by sneeze- or cough-produced respiratory droplets. The virus is extremely infectious, and almost all infected individuals develop a clinical illness. Measles virus replicates initially in the respiratory epithelium and then in various lymphoid organs. Measles begins with a prodromal period of fever, upper respiratory tract symptoms, and conjunctivitis. 2 to 3 days later, specific diagnostic signs develop; first, Koplik spots (small white spots on bright red mucous membranes of the mouth and throat and then a generalized macular rash, beginning at the head and traveling slowly to the lower extremities. Soon after the rash appears, the patient is no longer infectious. The major morbidity and mortality caused by measles are associated with complications of infection, especially those affecting the lower respiratory tract and the CNS. This is an autoimmune disease associated with an immune response to myelin basic protein.

Rubella virus {German measles}.

Respiratory secretions of an infected person are the primary vehicles for rubella virus transmission. Rubella causes a mild clinical syndrome that is characterized by a generalized maculopapular rash and occipital lymphadenopathy. In most cases, these symptoms may be hardly noticeable, and the infection remains subclinical. The clinical significance of rubella lies not in the primary infection described above, but rather in the fact that when a woman is infected during pregnancy, there can be severe damage to the developing fetus, especially in the first trimester (congenital rubella). This damage can include congenital heart disease, cataracts, hepatitis, or abnormalities related to the CNS, such as mental retardation, motor dysfunction, and deafness. Fetal damage resulting from rubella infection is preventable by use of the live attenuated rubella vaccine that is included with the routine childhood vaccinations. This vaccine, which has few complications, is effective in preventing congenital rubella because it reduces the reservoir of the virus in the childhood populations, and also ensures that women reaching childbearing age are immune to rubella infection.

Prevention: Measles is usually a disease of childhood, and is followed by life-long immunity. A live, attenuated measles vaccine, thus, two doses of the vaccine, in the form

of the **measles-mumps-rubella (MMR) vaccine** are now recommended, the first at twelve to eighteen months, the second at four to twelve years.

Rhabdoviridae (Rabies virus)

Rabies virus is enveloped, bullet-shaped viruses, to infect mammals.

Epidemiology

A wide variety of wildlife, such as raccoons, skunks, squirrels, foxes, and bats, provide a reservoir for the rabies virus. In third-world countries, domestic dogs and cats also constitute an important reservoir for rabies. Humans are usually infected by the bite of an animal, but in some cases, infection is via an aerosol (for example, of droppings from infected bats).

Pathology

Following inoculation, the virus may replicate locally, but then travels via the axoplasm of peripheral neurons to the brain, where it replicates primarily in the gray matter (Figure). From the brain, the rabies virus can travel along autonomic nerves, leading to infection of the lungs, kidney, adrenal medulla, and salivary glands. [Note: Contamination of saliva potentially leads to further transmission of the disease; for example, through a bite from an infected animal.] The incubation period is extremely variable, depending on the host's resistance, amount of virus transferred, and distance of the site of initial infection from the central nervous system (CNS). Incubation generally lasts one to eight weeks, but may range up to several months or, in unusual cases, as long as several years following exposure. Clinical illness may begin with an abnormal sensation at the site of the bite, then progress to afatal encephalitis, with neuronal degeneration of the brain and spinal cord. Symptoms include hallucinations, seizures, weakness, mental dysfunction, paralysis, coma, and finally death. Many, but not all, patients show the classic rabid sign of hydrophobia (in this case, hydrophobia refers to an infected individual's painful inability to swallow liquids, leading to avoidance). Once symptoms begin, death is inevitable.

Laboratory identification

Clinicallycharacteristic eosinophilic cytoplasmic inclusions (Negri bodies) may be identified in the brain, demonstration of the viral nucleic acid by RT-PCR.

Treatment and prevention

There is no effective treatment. However, a killed rabies virus vaccine is available for prophylaxis. Prevention of initial exposure is, however, clearly the most important echanism for controlling human rabies.

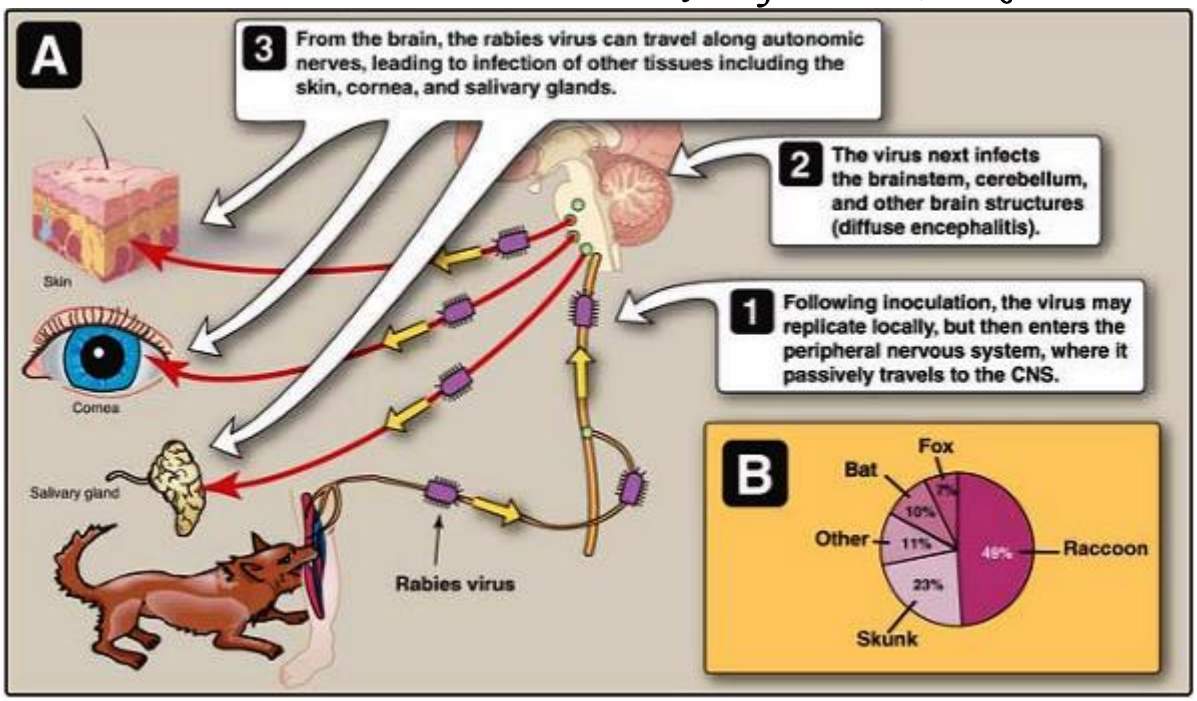


Figure Pathology of rabies virus.

Poliomyelitis virus

Clinical significance of poliovirus infection

Poliomyelitis is an acute illness in which the poliovirus selectively destroys the lower motor neurons of the spinal cord and brainstem, resulting in flaccid, asymmetric weakness or paralysis. The few cases of polio that occur (less than ten per year) are all caused by the reversion to virulence of the virus in the live-attenuated Sabin polio vaccine.

Transmission and pathogenesis: Poliovirus infections may follow one of several courses: 1) asymptomatic infection, which occurs in 90 to 95 percent of cases and causes no disease and no sequelae; 2) abortive infection; 3) nonparalytic infection; or 4) paralytic Poliomyelitis. The classic presentation of paralytic Poliomyelitis is flaccid paralysis, most often affecting the lower limbs. This is a result of viral replication in, and destruction of, the lower motor neurons in the anterior horn of the spinal cord. Respiratory paralysis may also occur, following infection of the brain stem. Poliomyelitis should be considered in any unimmunized person with the combination of fever, headache, neck and back pain, asymmetric flaccid paralysis without sensory loss, and pleocytosis (an increase in the number of lymphocytes in the spinal fluid).

Family of Hepatitis Viruses

Hepatitis B Viruses (HBV)

HBV is a leading cause inflammation of the liver, chronic hepatitis, cirrhosis, and hepatocellular carcinoma, accounting for one million deaths annually.

HBV is infects humans. Because of highly infectious virus in the blood of both symptomatic and asymptomatic patients, chronically infected individuals poses a serious threat to all healthcare workers, and immunization of individuals is generally required. A highly effective vaccine is available and included among routine childhood immunizations

- Viral proteins: The four proteins encoded by viral DNA are: 1) the capsid protein [hepatitis B capsid antigen (HBcAg)]; 2) envelope protein [a glycoprotein referred to as hepatitis B surface antigen (HBsAg)]; 3) multifunctional reverse transcriptase/DNA polymerase, which is complexed with the DNA genome within the capsid; and 4) a nonstructural regulatory protein designated the X protein.

Transmission

Infectious HBV is present in all body fluids of an infected individual. Therefore, blood, semen, saliva, and mother's milk, serve as sources of infection. The titer of infectious virus in the blood of an acutely infected patient can be as high as 10^8 virus particles per ml, but generally is lower in other body fluids. The majority of the population becomes infected at or shortly after birth from a chronically infected mother or from infected siblings.

Pathogenesis

Fully differentiated hepatocytes are the primary cell type infected by HBV. The primary cause of hepatic cell destruction appears to be the cell-mediated immune response. The cells involved are HLA -restricted cytotoxic T cells, which react specifically with the fragments of nucleocapsid proteins (HBcAg and HBeAg), expressed on the surface of infected hepatocytes. This response also contributes to control of the infection by eliminating virus-producing cells. Enhanced natural killer cell activity, as well as production of interferon, also contributes to limiting the extent of infection. Humoral anti-HBsAg antibody, which is the neutralizing antibody (Ab), does not appear until well into the convalescence period, when it may aid in clearing any remaining circulating free virus. Of greater importance, however, is that this antibody provides protection against reinfection. However, it is this same humoral antibody that is considered the source of extrahepatic damage seen in ten to twenty percent of patients, through the formation and deposition of HBsAg/anti-HBs Ab immune complexes and the consequent activation of complement.

Clinical significance:

HBV is of medical and public health importance, not only as the cause of acute liver disease but also as the cause of chronic, persistent infections that can result in the eventual death of infected individuals from cirrhosis and liver cancer. Chronically infected people serve as the reservoir of transmissible virus in the population. In most individuals, the primary infection is asymptomatic, and resolves as a result of an effective cell-mediated immune response.

Acute disease: Phases in acute HBV infections: Following infection, HBV has a long but variable incubation period of between 45 and 120 days. Following this period, a pre-icteric (pre-jaundice) phase occurs, lasting several days to a week. This is characterized by mild fever, malaise, anorexia, myalgia, and nausea. The acute, icteric phase then follows and lasts for one to two months. During this phase, dark urine, due to bilirubinuria, and jaundice (a yellowish coloration of mucous membranes, conjunctivae, and skin) are evident. There usually is an enlarged and tender liver as well. In eighty to ninety percent of adults, a convalescent period of several more months is followed by complete recovery.

Monitoring the course of acute HBV infection: Whereas liver-specific enzymes are important clinical determinants of all of the viral hepatitides, HBV infection is unusual in that the quantities of virions and virion components in the blood are so great that the time course of their appearance and clearance, along with that of the antibodies directed against them, serve as convenient markers of the stage of the disease and the likely future course

- Appearance of viral antigens: During the incubation period, HBsAg and HBeAg are the first indicators of HBV infection to appear in the blood. Their presence indicates an active infection, but does not distinguish between acute and chronic infections. Next, viral DNA, viral DNA polymerase, and complete virions become detectable. These continue to increase during the acute disease phase, when a patient's blood has the highest titer of infectious virus.

- Appearance of antiviral antibodies: Antibodies to the HBcAg rise concurrently with liver enzymes in the serum, whereas anti-HBe antibodies and still later, anti-HBs antibodies do not appear until the beginning of convalescence generally after the respective antigens have disappeared from the blood. In those patients in whom the infection resolves completely, anti-HBc and anti-HBs antibodies remain present for life, providing immunity to reinfection. Continued presence of HBsAg beyond six months and absence of anti-HBs indicates that the infection has become chronic

Fulminant hepatitis:

- In one to two percent of acute symptomatic cases, much more extensive necrosis of the liver occurs during the first eight weeks of the acute illness. This is accompanied by high fever, abdominal pain, and eventual renal dysfunction, coma, and seizures. Termed fulminant hepatitis, this condition is fatal in roughly eight percent of cases. Whereas it is not clear why the acute disease takes this course, a more highly virulent strain of HBV, coinfection with HDV or another hepatitis virus (for example, HCV), and/or perhaps an uncontrolled immune response by the patient, are thought to play a role.

Clinical significance: chronic disease

- In about two thirds of individuals, the primary infection is asymptomatic, even though such patients may later develop symptomatic chronic liver disease, indicating persistence of the virus. Following resolution of the acute disease (or asymptomatic

infection), about two to ten percent of adults and over twenty five percent of young children remain chronically infected. The high rate of progression to chronic liver disease seen in infants born to HBV-infected mothers is thought to be related to the less competent immune status of newborns. Adults with immune deficiencies also have a considerably higher probability of developing chronic infection than individuals with normal immune systems.

- Types of chronic carriers: The asymptomatic carriers of HBsAg are the most common type of persistently infected individuals. They usually have anti-HBe antibodies and little or no infectious virus in their blood. Later progression of liver damage or recurrence of acute episodes of hepatitis is rare in such patients. Those carriers with minimal chronic hepatitis (formerly,chronic persistent hepatitis) are asymptomatic most of the time, but have a higher risk of reactivation of disease, and a small fraction do progress to cirrhosis. Severe chronic hepatitis (formerly, chronic active hepatitis) results in more frequent exacerbations of acute symptoms, including progressive liver damage, potentially leading to cirrhosis and/or hepatocellular carcinoma (see below), chronic fatigue, anorexia, malaise, and anxiety. These symptoms are accompanied by active virus replication and the corresponding presence of HBeAg in the blood. Serum levels of liver enzymes and bilirubin are increased to varying degrees, reflecting the extent of necrosis. The risk of developing cirrhosis is highest in those carriers with more frequent recurrences of acute disease and those in whom HBeAg is not cleared from the blood, indicating continuing virus replication. Overall life expectancy is significantly shorter in those individuals with cirrhosis.
- Development of hepatocellular carcinoma (HCC, hepatoma): HCC is fairly uncommon in the United States, whereas it is ten to a hundred times more frequent in areas of high HBV endemicity. In all populations, males experience a higher rate of chronic HBV infections, a higher rate of progression to cirrhosis, and ultimately a higher rate of HCC, for which the male-to-female ratio is six to one. HCC typically appears many years after the primary HBV infection, and the tumor itself is rather slow growing and only occasionally metastasizes. Clinically, a patient with HCC exhibits weight loss, right upper quadrant pain, fever, and intestinal bleeding. Although there is no doubt that chronic HBV infection greatly increases the risk of HCC, the mechanisms relating HBV and HCC are not understood. However, by causing continuing liver necrosis accompanied by continuing regeneration of the damaged tissue, chronic HBV infection provides the opportunity for chromosome rearrangements and mutations. The presence of environmental carcinogens could further contribute to the disease process.

Laboratory identification

The diagnosis of hepatitis is made on clinical grounds, coupled with biochemical tests that evaluate liver damage. Elevations of aminotransferases, bilirubin, and prothrombin time, all contribute to the initial evaluation of hepatitis. ELISA.

Treatment: lamivudine.

Prevention: The availability of a highly effective vaccine has led to a several-pronged approach: 1) protection of those adults who are at risk because of lifestyle or occupation; 2) protection of newborns from infection by transmission from HBV-positive mothers (important because of the high rate of resulting chronic infections, and 3) protection of siblings and other children from infection by chronically infected family members.

Passive immunization {Hepatitis B immunoglobulin (HBIG)} Infants born to mothers who are HBV-positive are given HBIG plus hepatitis B vaccine at birth, followed by additional doses of vaccine at one and six months.

Hepatitis C viruses

Hepatitis C virus (HCV) was discovered in 1988 in the course of searching for the cause of non-A, non-B, transfusion-associated hepatitis. At that time, HCV accounted for ninety percent of the cases of non-A, non-B hepatitis. The hepatitis C viruses are heterogeneous and can be divided into six types on the basis of their nucleotide sequences.

Transmission and pathogenesis: Although HCV was initially identified as a major cause of posttransfusion hepatitis, intravenous drug users and patients on hemodialysis are also at high risk for infection with HCV. Tattoos are also a leading cause of HCV infection. In addition, there is evidence for sexual transmission of HCV, as well as for transmission from mother to infant. In the infected individual, viral replication occurs in the hepatocyte and probably also in mononuclear cells (lymphocytes and macrophages). Destruction of liver cells may result both from a direct effect of viral replication and from the host immune response.

Clinical significance: The majority of infections with HCV are subclinical, but about 25 percent of infected individuals present with acute hepatitis, including jaundice. More important, a significant proportion of infections progress to a chronic hepatitis and cirrhosis. Finally, some of these individuals go on to develop hepatocellular carcinoma many years after the primary infection.

Treatment by interferon+ ribavirin. Chronic hepatitis resulting in severe liver damage may be an indication for a liver transplant.

Hepatitis E virus (HEV)

The peak incidence is in young adults, and the disease is especially severe in pregnant women, in whom death can result from HEV infection. Viral RNA can be detected in the feces of infected individuals by RT-PCR, and nearly all serologically confirmed epidemics of HEV can be attributed to fecally contaminated water. The signs and symptoms are similar to those seen with other forms of acute viral hepatitis. No antiviral treatment nor vaccine is currently available.

Hepatitis D Virus (Delta Agent)

Hepatitis D virus (HDV) is found in nature only as a coinfection with HBV. It is significant because its presence results in more severe acute disease, with a greater risk of fulminant hepatitis and, in chronically infected patients, a greater risk of cirrhosis and liver cancer.

Transmission and pathogenesis

Because HDV exists only in association with HBV, it can be transmitted by the same routes. However, it does not appear to be transmitted sexually as frequently as HBV or HIV. Pathologically, liver damage is essentially the same as in other viral hepatitides, but the presence of HDV usually results in more extensive and severe damage.

Clinical significance

Disease HDV can occur in one of three variations. First, simultaneous primary coinfection with both HBV and HDV can cause an acute disease that is similar to that caused by HBV alone, except that, depending on the relative concentrations of the two agents, two successive episodes of acute hepatitis may occur. The risk of fatal fulminant hepatitis caused by the presence of HDV is also considerably higher than with HBV alone. The likelihood of progression to the second variation of HDV disease (chronic coinfection with HBV) is greatly increased as well. In this case, cirrhosis and HCC or deaths due to liver failure also develop more frequently than with HBV infection alone. The third variation primary HDV infection of a chronically HBV-infected individual leads to an episode of severe acute hepatitis after a short incubation period and develops into chronic HDV infection in more than seventy percent of the cases. Again in this situation, the risk of acute hepatitis becoming fulminant is greatly increased, and the persistent infection is often of the severe chronic type.

Hepatitis A virus

Hepatitis A virus (HAV). Although at one time HAV was also known as Enterovirus 72, HAV, of which there is only one serotype, causes viral hepatitis. As with the enteroviruses, transmission is by the fecal-oral route, and the virus is shed in the feces. For example, a common mode of transmission of the virus is through eating uncooked shellfish harvested from sewage-contaminated water. The main site of replication is the hepatocyte. liver function is significantly impaired, and the development of persistent infection and chronic hepatitis is uncommon. **Prevention** depends on taking measures to avoid fecal contamination of food and water. Vaccines prepared from whole virus inactivated.

Family of Rotaviruses

Epidemiology

Rotaviruses are divided into seven serogroups (A through G) of which group A is the most important cause of outbreaks of disease in humans. Transmission of rotaviruses is via the fecal oral route. There is a marked seasonal incidence associated with rotavirus infections, with the peak months in the United States being January through March. Because infectious particles are relatively stable, they can survive for extended periods on various surfaces. Rotavirus infections account for about fifty percent of cases of severe diarrhea in infants and young children (up to two years of age)

Clinical significance

Following ingestion, rotaviruses infect the epithelial cells of the small intestine, primarily the jejunum. [Note: Rotaviruses are able to reach the small intestine because they are resistant to the acid pH of the stomach.] The incubation period is usually 48 hours or less. Infection can be subclinical or may result in symptoms ranging from mild diarrhea and vomiting to severe, nonbloody, watery diarrhea with dehydration and loss of electrolytes. Although rotavirus infections are probably equally widespread around the world, the outcomes of infection vary significantly in different regions of the world. Despite the fact that more than ninety percent of children in the United States may have antibodies to rotaviruses by the age of three or four, mortality is low because patients who are severely ill are generally hospitalized, with fluid and electrolyte losses rapidly corrected.

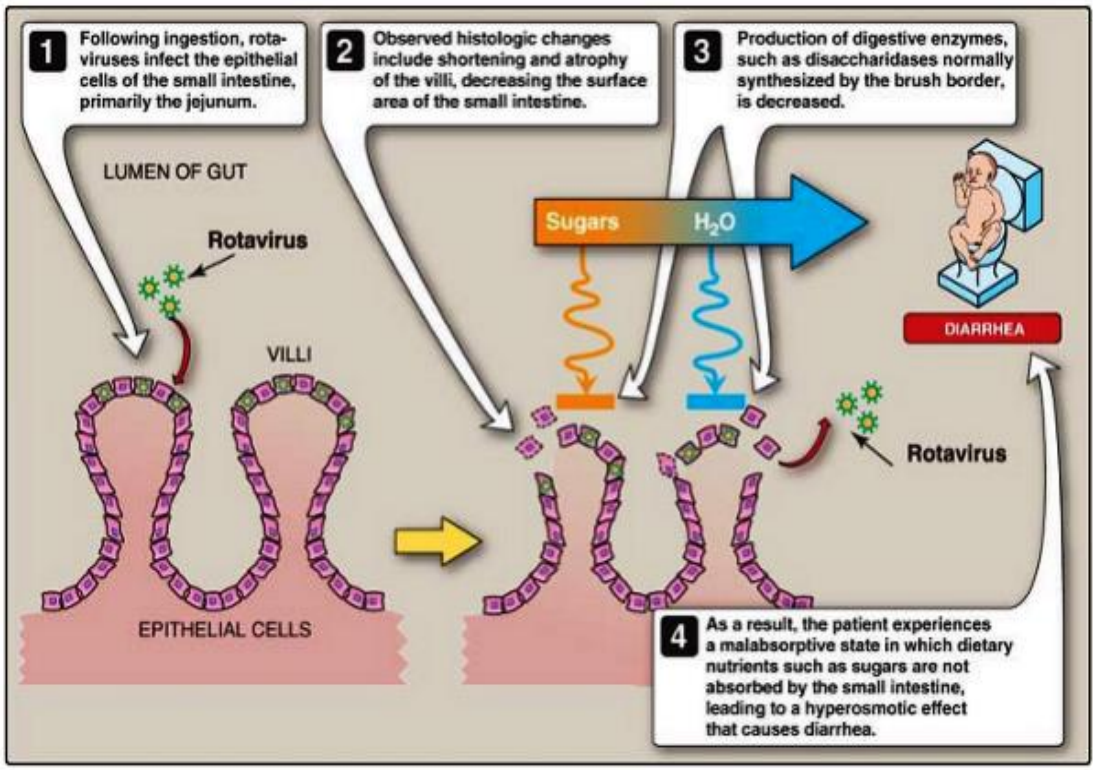


Figure: mechanism of Rotaviruses dirrhoea

Family of Human Immunodeficiency Virus (HIV)

Acquired immune deficiency syndrome (AIDS) was first reported in the United States in 1981. The virus infects helper T cells, lymphocytes, monocytes, and dendritic cells, which contain this protein in their cell membranes which preferentially binds to a CD4 molecule

Transmission of HIV

Transmission of HIV generally occurs by one of four routes; there has been no firm evidence for transmission by saliva, urine, nonsexual contact in which blood is not exchanged, or by an insect bite.

- **Sexual contact**: HIV, present in both semen and vaginal secretions, is transmitted primarily as cell-associated virus in the course of either homosexual or heterosexual contact. Disruption of mucosal surfaces by sexually transmitted diseases, particularly those such as syphilis and chancroid that result in genital ulcerations, may greatly facilitate HIV-1 infection.
- **Transfusions:** HIV has been transmitted by transfusion with whole blood, plasma, clotting factors, or cellular fractions of blood.
- **Contaminated needles**: Transmission can occur by inoculation with HIV-contaminated needles either accidentally, or through use of shared needles or syringes by drug users.

- **Perinatal transmission**: An HIV-infected woman has a fifteen to forty percent chance of transmitting the infection to her newborn, either transplacentally during passage of the baby through the birth canal, or via breast-feeding.

Pathogenesis and clinical significance of HIV infection

The pathology of HIV disease results from either tissue destruction by the virus itself or the host's response to virus-infected cells. In addition, HIV can induce an immunodeficient state that leads to opportunistic diseases that are rare in the absence of HIV infection. The progression from HIV infection to AIDS develops in fifty percent of HIV-infected individuals in an average of ten years, and, if untreated, it is uniformly fatal generally within two years of diagnosis. However, there is a significant fraction (about ten percent) of HIV-infected individuals who have not developed AIDS after twenty years. Development from HIV infection to end-stage AIDS progresses through several phases

- Initial infection: After the acquisition of HIV, the initially infected cells are generally macrophages within the genital tract. From this initial localized infection, HIV disseminates via the blood, and virus may then localize in dendritic cells throughout the lymphoid tissue. From the surface of follicular dendritic cells, HIV can then infect CD4+ lymphocytes moving through the germinal centers of lymph nodes. This process creates a reservoir of chronically HIV-infected cells within the lymphatic tissue of the body.
- Acute phase viremia: Several weeks after the initial infection with HIV, one third to two thirds of individuals experience an acute disease syndrome (also referred to as the primary infection) similar to infectious mononucleosis. During this period, there is a high level of virus replication occurring in CD4+ cells. Large amounts of virus and capsid protein (CA antigen) are present in the blood, and circulating antibody appears in one to ten weeks after the initial infection (seroconversion). A constant level of virus and virus-infected cells is maintained by a combination of replacement of the CD4+ cells killed by HIV infection with cells newly produced in lymphoid organs and the subsequent infection of these new cells with progeny virus. Lymph nodes also become infected during this time; they later serve as the sites of virus persistence during the asymptomatic period.
- Latent period: The acute phase viremia is eventually reduced significantly with the appearance of a HIV-specific cytotoxic T-lymphocyte response, followed by a humoral antibody response. A clinically asymptomatic or latent period lasting from months to many years follows the acute infection. During this latent period, the majority (ninety percent) of HIV proviruses are transcriptionally silent, so that only ten percent of the cells containing integrated HIV DNA also contain viral mRNA or viral proteins. There are transient peaks of viremia that are often correlated with stimulation of the immune system by infection with other pathogens or by immunization. Although there is continuous loss of those CD4+ cells in which HIV is replicating, active replacement through stem cell multiplication compensates for this loss, and the CD4+ count declines only slowly over a period of years. In addition, the host immune response is still sufficiently effective to maintain a relatively stable, low level of virus production. It has been estimated that 10^{11} virions and 10^{9} CD4 T cells are produced each day. Virus isolated during this period is also less cytopathic for CD4+ cells and replicates more slowly than does that isolated later during symptomatic AIDS. Despite

the nearly normal levels of CD4+ cells, however, impairment of T-cell responses to specific antigens is evident. The infection remains relatively clinically asymptomatic as long as the immune system is functional.

- Clinical complications of HIV infection during the latent period: During this period whose length is variable, but lasts on average about ten years there are multiple, nonspecific conditions, such as persistent, generalized lymphadenopathy (swollen lymph nodes), diarrhea, chronic fevers, night sweats, and weight loss. The more common opportunistic infections such as herpes zoster and candidiasis may occur repeatedly during this period, as well as when patients progress to AIDS. The CD4+ cell count remains normal or gradually declines, but is greater than 200/µl.
- Progression to AIDS: The progression from asymptomatic infection to AIDS is not sudden, but in fact occurs as a continuum of clinical states. A number of virologic and immunologic changes occur that affect the rate of this progression. For example, coinfection with a number of the herpesviruses, such as human herpesvirus type 6, can transactivate transcription from the silent HIV provirus, increasing HIV replication. Any stimulation of an immune response causing activation of resting T cells also activates HIV replication. Not only does this increase the number of infected CD4+ cells, but it also increases the opportunity to create generations of virus mutants. Eventually, a more highly cytocidal, more rapidly multiplying variant appears. In addition, these variants are often highly syncytium-inducing, promoting fusion between infected and previously uninfected cells. T-cell precursors in the lymphoid organs are also infected and killed, so the capacity to generate new CD4+ cells is gradually lost. The capacity to contain the infection is further compromised by the appearance of HIV mutants with altered antigenic specificity, which are not recognized by the existing humoral antibody or cytotoxic T lymphocytes. The eventual result of these accumulating, interacting factors is an increasingly rapid decline in CD4+ count, accompanied by loss of immune capacity.
- End-stage AIDS: Nearly all systems of the body can be affected as a result of HIV infection, either by HIV itself or by opportunistic organisms. The weakening immune system leads to many complications including malignancies.
  - Spread of HIV to additional body sites: Cell types other than CD4+ lymphocytes can be infected by HIV. Infection of these cells produces some of the additional manifestations of end- stage disease. Chief among these are infected cells of the monocyte-macrophage lineage, which are not killed as rapidly as CD4+ T cells and can transport the virus into other organs. For example, macrophages are the HIV-infected cells present in brains of patients with AIDS encephalopathy, which typically evolves over a period of one year, with gradual deterioration resulting in severe dementia. This appears to be unrelated to CD4+ depletion, but rather to an expanded host range of variant HIV. The basis for damage to neuronal cells is, however, not known. Similarly, the wasting syndrome seen in late stages of AIDS is probably related to HIV-infected macrophages induced to produce various cytokines, especially tumor necrosis factor. Virus has also been found in Langerhans cells in the skin, dendritic cells in lymph nodes, and monocytes in bone marrow, but their significance in the disease process is not clear. The eye is another site affected by HIV infection itself, which produces focal areas of ischemia in the retina. HIV infection of blood cell progenitors in the bone marrow leads to the anemia seen in most AIDS patients.

- Opportunistic infections in AIDS: Multiple recurrent bouts of infections with fungi, bacteria, and viruses occur as the CD4+ cell count declines. For example, the nervous system can be the site of opportunistic infections with Toxoplasma, Cryptococcus, JC virus, and Mycobacteria. The eye cannot only be infected with HIV, but also with opportunistic agents, the most prominent of which is cytomegalovirus (CMV) a cause of retinal destruction. The lungs are also primarily affected by opportunistic infections, P. jirovecci pneumonia being one of the most common. Mycobacterial infections are also a common problem in the lung; for example, currently thirty percent of AIDS patients die from tuberculosis. Serious gastrointestinal tract illnesses are due to opportunistic pathogens, but these may be in concert with HIV infection. CMV colitis is a common problem, but HIV is often present as well. Protozoal parasitic diseases, as well as infections with gram-negative enteric bacteria are other sources of gastrointestinal disorders. The immune deficiency also provides the opportunity for latent herpesvirus infections to recur repeatedly or become chronic and spread extensively. Mucocutaneous candidiasis (for example, oral, esophageal, or vaginal) is an ongoing problem in AIDS patients as well. In fact, vaginal candidiasis is the most frequent reason HIV-infected females seek medical attention. Malignancies associated with AIDS: A number of malignancies commonly arise in HIV-infected patients. The most characteristic neoplasm present in AIDS patients is Kaposi sarcoma (KS), which involves skin, mucous membranes, and deep viscera. Various lymphomas, including those of the CNS, are also common. These are probably the result of the immune compromise and not HIV itself. KS has been associated with human herpesvirus, type 8 (HHV-8, Figure 28.15). In AIDS patients, body cavity lymphomas are also usually associated with HHV-8 infection, whereas many other lymphomas are EBV-associated

Laboratory identification

Demonstration of virus or virus components: Amplification of viral RNA or DNA proviruses by the PCR technique is the most sensitive method for early detection of virus in blood or tissue specimens. Recent adaptations of the technique to obtain quantitative estimates of viral load (measured, for example, as the amount of viral RNA per milliliter of blood plasma) now permit evaluation of the stage of the disease, effectiveness of a drug regimen, and prognosis. For purposes of initial screening of the blood supply, ELISA testing for the CA antigen in serum can detect infection in individuals who are infectious but undetectable by screening for anti-HIV antibodies.

Chapter seven

Hospital acquired infection, nosocomial infection

Hospital acquired infection, nosocomial infection

Definition

A hospital—acquired infection, also called a nosocomial infection, is an infection that first appears between 48 hours and four days after a patient is admitted to a hospital or other health care facility

Description

Hospital-acquired infections can be caused by bacteria, viruses, fungi, or parasites. Depending on the causal agents involved, an infection may start in any part of the body. A localized e infection is limited to a specific part e of the body and has local symptoms. a generalized infection is one that enters the blood stream and causes systemic symptoms such as fever, chills, low blood pressure, or mental confusion. This can lead to sepsis, a serious, rapidly progressive multi—organ infection, that can result in death.

Hospital-acquired infections may develop from the performance of surgical procedures; from the insertion of catheters (tubes) into the urinary tract, nose, mouth, or blood vessels; or from material from the nose or mouth that is aspirated (inhaled) into the lungs. The most common types of hospital-acquired infections are urinary tract infections (UTIs), ventilator associated pneumonia, and surgical wound infections

Causes

All hospitalized patients are at risk of acquiring an in infection from their treatment or surgery. Some patients are at greater risk than others, especially young children the elderly, and persons with compromised immune system. he risk factors for hospital-acquired infections in children include parenteral, the use of antibiotics for more than 10 days, use of invasive devices, poor postoperative status, and immune system dysfunction.

Other risk factors that increase the opportunity for hospitalized adults and children to acquire infections are:

- a prolonged hospital stays to severity of underlying illness; use compromised nutritional or immune status use of indwelling catheters

failure of health care workers to wash their hands between patients or before procedures

prevalence of antibiotic-resistant bacteria fromthe overuse of antibiotics

Any type of invasive procedure can expose a patient to the possibility of infection. Some common procedures that increase the risk of hospital-acquired infections include:

- urinary bladder catheterization
- respiratory procedures such as intubation or mechanical ventilation
- surgery and the dressing or drainage of surgical wounds
- gastric drainage tubes into the stomach through the nose or mouth
- intravenous (IV) procedures for delivery of medication, transfusion , or nutrition

Urinary tract infection (UTI)

Urinary tract infection (UTI) is the most common type of hospital-acquired infection and has been shown to occur a after urinary catheterization.

Catheterization is the placement of a catheter through the urethra into the urinary bladder to a empty urine from the bladder; or to deliver medication relieve pressure or measures. Urine in the bladder; or for other medical reasons Normally a healthy urinary bladder is sterile, with no harmful bacteria or other microorganisms present. Although bacteria may be in or around the urethra, they normally cannot enter the bladder. A catheter, however, can pick up bacteria from the urethra and give them an easy route into the bladder, causing infection Bacteria from the intestinal tract are the most common type to cause UTIs. Patients with poorly functioning immune systems or who are taking antibiotics are also at increased risk for UTI caused by a fungus called Caindida. The prolonged use of antibiotics, which may reduce the effectiveness of the patient‘s own immune system, has been shown to create favorable conditions for the growth of this fungal organism

Pneumonia

Pneumonia is the second most common type of hospital-acquired infection. Bacteria and other microorganisms are easily introduced into the throat by treatment procedures performed to treat respiratory illnesses. patients, with-COPD , for example, are especially susceptible to infection because of frequent and prolonged antibiotic therapy and long-term mechanical ventilation used in their treatment. The infecting microorganisms can come from contaminated equipment or the hands of health care workers as procedures are conducted such as respiratory intubation, suctioning of material from the throat and mouth, and mechanical ventilation Hospital-acquired infections. Invasive surgical procedures increase a patient's risk of getting an infection by giving} bacteria a route into normally sterile areas of the body. An infection can be acquired_from contaminated surgical equipment or from the hands of health care workers following surgery, the surgical wound can become-infected from contaminated dressings or the hands of health care workers who change the dressing. Other wounds can also become easily infected, such as those caused by trauma, burns, or pressure sores that result from prolonged bed rest or wheel chair

Use. Many hospitalized patients need continuous medications, transfusions, or nutrients delivered into their bloodstream. An IV catheter is placed in a vein and the medications, blood components, or liquid nutritionals are infused into the vein bacteria from the surroundings, contaminated equipment, or health care workers‘hands can enter the body at the site of catheter insertion. A local infection may develop in the skin around the catheter. The bacteria can also enter the blood through the vein and cause a generalized infection. The longer a catheter is in place, the greater the risk of infection

Symptoms

Fever is often the first sign of infection. Other symptoms and signs o infection are rapid breathing mental confusion, low blood pressure, reduced urine output, and as high white blood cell count Patients with a UTI may have pain when urinating and blood in the urine

Symptoms of pneumonia may include difficulty breathing and inability to cough. A localized infection begins with swelling, redness, and tenderness on the skin or around a surgical wound or other open wound, which can progress rapidly to the destruction of deeper layers of muscle tissue, and eventually sepsis

Diagnosis

An infection is suspected any time a hospitalized patient develops a fever that cannot be

patients, especially the elderly, may not develop a feverin these `patients, the first signs of infection may be rapid breathing or mental confusion

Diagnosis of a hospital-acquired infection is determined by:

- evaluation of symptoms and signs of infection
- examination of wounds and catheter entry sites for redness, swelling, or the presence of pus or an abscess
- a complete physical examination and review of underlying illness laboratory tests, including CBC; urinalysis, looking for white cells or evidence of blood in the urinary tract; cultures of the infected area, blood, sputum or other body fluids or tissue to rind the causative organism
- chest x ray may be done when pneumonia is suspected to look for the presence of white blood cells and other inflammatory substances in lung tissue review of all procedures performed that might have led to infection

Treatment

Cultures of blood, urine, sputum, other body fluids, or tissue are especial y important in order to identify the bacteria, fungi virus, or other microorganism causing the inflection. Once the organism has been identified, it will be tested again for sensitivity to a range of antibiotics so that the patients can be treated quickly and effectively with an appropriate medicine to which the causative organism will respond.While waiting for these test results, treatment may begin with common broad—spectrum antibiotics such as penicillin, cephalosporins, tetracyclines , or erythromycin More and more often, some types of bacteria are becoming resistant to these standard antibiotic treatments, especially when patients with chronic illnesses are frequently given antibiotic therapy for long periods of time. When this happens, a different, more powerful, and more specific antibiotic must be used to which the specific organism has been shown to respond. Two strong antibiotics that have been effective against resistant bacteria are vancomycin and imipenem, although some

bacteria are developing resistance to these antibiotics as well. The prolonged use of antibiotics is also known to reduce the effectiveness of the patient's own immune system, sometimes becoming a factor in the development infection.

Fungal infections are treated with antifungal medications. Examples of these are amphotericin B, nystatin, ketoconazole itraconazole, and fluconazole.

Viruses do not respond to antibiotics. A number of antiviral drugs have been developed that slow the growth or reproduction of viruses, such as acyclovir, ganciclovir, foscarnet, and amantadine

Prevention

Hospitals take a variety of steps to prevent nosocomial infections, including:

- Identify high-risk procedures and other possible sources of infection.
- Strict adherence to hand—washing rules by health care worker and visitors to avoid passing infection microorganisms to or between hospitalized patients
- Strict attention to aseptic (sterile) technique in the performance of procedures, including use of sterile gowns, gloves, masks, and barriers.
- Sterilization of all reusable equipment such as ventilators, humidifiers, and any devices that come in contact with the respiratory tract.
- Frequent changing of dressings for wounds and use of antibacterial ointments under dressings
- Remove nasogastric (nose to stomach) and endotracheal (mouth to stomach) tubes as soon as possible.
- Prevent contact between respiratory secretions and health care providers by using barriers and masks as needed.
- Limitations on the use and duration of high-risk procedure such urinary catheterization .
- Isolation of patients with known infections.
- Sterilization of medical instruments and equipment to prevent contamination.
- Reductions in the general use of antibiotics to encourage better immune response in patients and reduce the cultivation of resistant bacteria.

Appendix:

MCQs IN MICROBIOLOGY

Appendix: MCQs IN MICROBIOLOGY

1. According to Pasteur statements which one of the following is true?
a. Living organisms discriminate between stereoisomers b. Fermentation is a aerobic process
c. Living organisms doesn't discriminate between stereoisomers d. Both a and b
2. Fluroscent substance used in fluorescent microscopy are
a. Quinine sulphate b. Auramine c. All of these d. None of these
3. Trepanema pallidum was discovered by
a. Schaudinn and Hoffman b. Louis Pasteur c. Burgey d. Laennec
e. None of these
4. Rh factor of the blood was discovered by scientist
a. Louis Pasteur b. Landsteiner and Weiner c. Janskey d. Moss
e. None of these
5. Neisseria gonorrhoeae was first described by
a. Neisser in 1879 b. Pasteur in 1878 c. Robert Koch d. None of these
6. Reverse isolation would be appropriatefor
a. a patient with tuberculosis b. a patient who has had minor surgery
c. a patient with glaucoma d. a patient with leukemia
7. The symptome "general feeling of illnessand discomfort " is called
a. Cystitis b. Malaise c. Anaphylactic shock d. Arthritis
8. T. pallidum was discovered by
a. Robert Koch b. Schaudinn and Hoffman c. Louis Pasteur
d. Edward Jenner
9. The first antibody to contact invading microorganisms was
a. IgG b. IgM c. IgA d. IgD
10. Pseudomonas aeruginosa was first named
a. Schroeter and Gessard b. Robert Koch c. Louis Pasteur d. Edward
11. Staphylococcus aureus was isolated by
a. Rosenbach b. Louis Pasteur c. Passet d. Sir Alexander Ogston Jenner
12. B.anthracis was isolated by
a. Louis Pasteur b. Robert Koch c. Antonyvon Leewenhok
d. None of these
13. Pick out the vector using in human Genome project
a. Phagemid vector b. Yeast artificial chromosomes c. Cosmid vectors
d. Yeast episomal plasmids
14. Salt and sugar preserve foods because they
a. Make them acid b. Produce a hypotonic environment
c. Deplete nutrients d. Produce a hypertonic environment
15. In a fluorescent microscope the objective lens is made of
a. Glass b. Quartz c. Polythene d. None of these
16. Streptococcus pneumoniae was isolated by
a. Robert Koch b. Edward Jenner c. Antony von Leewenhock
d. Louis Pasteur

17. Which one of the following fungi is the most serious threat in a bone marrow transplant unit?
a. Candida albicans b. Aspergillus c. Blastomyces d. Cryptococus
18. Direct microscopic count can be done withthe aid of
a. Neuberg chamber b. Anaerobic chamber c. Mineral oil
d. Olive oil
19. The image obtained in a compound microscope is
a. Real b. Virtual c. Real inverted d. Virtual inverted
20. Enzymes responsible for alcoholic fermentation
a. Ketolase b. Zymase c. Peroxidase d. Oxidase
21. Which type of spores are produced sexually?
a. Conidia b. Sporangiospores c. Ascospores d. None of these
22. Bacterial transformation was discoveredby
a. Ederberg and Tatum b. Beadle and Tatum c. Griffith d. None of these
23. Father of microbiology is
a. Louis Pasteur b. Lister c. A.V. Leeuwenhock d. Robert Koch
24. The antiseptic method was first demonstrated by
a. Lwanowski b. Lord Lister c. Edward Jenner d. Beijerinck
25. Small pox vaccine was first discovered by
a. Robert Koch b. Louis Pasteur c. Lister d. Edward Jenner
26. The term mutation was coined by
a. Pasteur b. Darwin c. Hugo devries d. Lamark
27. Compound microscope was discovered by
a. Antony von b. Pasteur c. Johnsen & Hans d. None of these
28. Father of Medical Microbiology is
a. Pasteur b. Jenner c. Koch d. A.L.Hock
29. Disease that affects many people at different countries is termed as
a. Sporadic b. Pandemic c. Epidemic d. Endemic
30. Prophylaxis of cholera is
a. Protected water supply b. Environmental sanitation c. Immunization with killed vaccines d. All of these
31. In electron microscope, what material is used as an objective lense?
a. Magnetic coils b. Superfine glass c. Aluminium foils d. Electrons
32. The main feature of prokaryotic organismis
a. Absence of locomotion b. Absence of nuclear envelope c. Absence of nuclear material d.Absence of protein synthesis
33. Vibrio Cholerae was discovered by
a. Koch b. Metchnikoff c. John Snow d. Virchow
34. Antiseptic methods were first introduced by
a. Lord Lister b. Iwanowski c. Beijernick d. Edward Jenner
35. Kuru disease in Humans is caused by
a. Bacteria b. Viroides c. Prions d. Mycoplasma
36. A mutation that produces termination codon is
a. Mis-sense mutation b. Neutral mutation c. Non-sense mutation
d. Reverse mutation

37. During conjunction the genetic material will be transferred through
a. Cell wall b. Medium c. Pili d. Capsule
38. Antiseptic surgery was discovered by
a. Joseph Lister b. Ernest Abbe c. Pasteur d. Beijerink
39. Tuberculosis is a
a. Water borne disease b. Air borne disease c. Food borne disease d. Atthropod borne disease
40. Phagocytic phenomenon was discovered by
a. Louis Pasteur b. Alexander Fleming c. Metchnikof d. Robert Koch
41. Mycobacterium lepree was discovered by
a. Robert Koch b. Hansen c. Edward Jenner d. Louis Pasteur
42. Hybridoma technique was first discoveredby.
a. Kohler and Milstein b. Robert Koch c. 'D' Herelle d. Land Steiner
43. The minimum number of bacteria required to produce clinical evidence of death in a susceptible animal understandard condition is called
a. LD50 b. ID c. MLD d. All of these
44. In Electron Microscope source of electrons is from
a. Mercury lamp b. Tungsten metal c. both a and b d. None of these
45. Griffith (1928) reported the phenomenon of transformation first in
a. H. influenzae b. Bacillus species c. Pneumococci d. E.coli
46. The resolution power of the compound microscope is
a. 0.2 micron b. 0.2 millimeter c. 0.2 Angstrom units d. 0.2 centimeter
47. The capacity of a given strain of microbial species to produce disease is known as
a. Pathogen b. Virulence c. Infection d. None of these
48. Monoclonal antibodies are associated with the name of
a. Burnet b. Medwar c. Milstein kohler d. Owen
49. Lederberg and Tatum (1946) described the phenomena of
a. Conjunction b. Transformation c. Mutation d. Plasmids
50. Hanging drop method for motility study was first introduced by
a. Robert Koch b. Louis Pasteur c. Jenner d. Leeuwenhock
51. Electron microscope gives magnification upto
a. 100 X b. 2000 X c. 50,000 X d. 2,00,000 X
52. Term vaccine was coined by
a. Robert Koch b. Pasteur c. Needham d. None of these
53. The inventor of Microscope is
a. Galileo b. Antony von c. Pasteur d. Koch
54. First Pasteur conducted fermentationexperiments in
a. Milk b. Food material c. Fruit juices d. Both a and c
55. Modern concepts of chemotherapy was proposed by
a. Paul Ehrlich b. Joseph Lister c. Elie Metchnikoff d. None of these
56. The role of phagocytosis was discovered by
a. Paul Ehrlich b. Joseph lister c. Elie Metchikoff d. Pasteur
57. L – forms are discovered by
a. Klein Berger b. Louis Pasteur c. Robert Koch d. Antony von Leeuwenhock

58. The causative organism of rocky mountainspotted fever was first described by
a. Howard Ricketts b. da Rocha-lima c. Both a and b d. Robert Koch
59. The term bacteriophage was coined by
a. De'Herelle b. F.W. Twort c. Beijernick d. Jwanosky
60. Viral infection of bacteria was discovered by
a. De'Herelle b. F.W. Twort c. Beijernick d. Jwanoksy
61. Eye cannot resolve any image less than
a. 1ìm b. 2ìm c. 7ìm d. 5ìm
62. Compound Microscope was discovered by
a. A.V. Lewenhoek b. Pasteur c. Janssen and Hans d. None of these
63. Electron Microscope was discovered by
a. Prof. Fritz b. Janssen and Hans c. Knoll and Ruska d. None of these
64. Magnification range of light microscope is
a. 1000x – 5000x b. 1000x – 2000x c. 500x – 1000x d. None of these
65. Condensation of light in light Microscope is by
a. Objective b. Condensor c. Ocular d. All of these
66. Light gathering capacity of Microscope is called
a. Numerical aperture b. Angular aperture c. Both a and b d. None of these
67. If 10x and 40x objectives are used (air is the medium), the numerical aperture is
a. 1.5 b. 2.0 c. 1.0 d. 1.8
68. The ability of Microscope to distinguish two objects into two separate objects, is called.
a. Resolving power b. Wave length c. N.A. d. None of these
69. Limit of resolution of compound microscope is
a. 0.018 Ao b. 0.1 mm c. 5 ìm d. 1 mm
70. Source of light in fluorescence microscopy is from
a. Mercury lamp b. Sunlight c. Both a and b d. None of these
71. Mycobacterium tuberculosis was first discovered by
a. Robert Koch b. Edward Jenner c. Louis Pasteur d. None of these
72. Which is the following enzyme acts as a spreading factor?
a. Hyaluronidase b. Coagulase c. Catalase d. DNase
73. The ability of a pathogen to spread in thshost tissues after establishing the infection is known as
a. Adhesion b. Invasiveness c. Toxigenicity d. None of these
74. The most important virulence factors are
a. Adhesions b. Invasivenessc. Toxigenicity d. Enzymese. All of the above
75. The lethal dose required to kill 50% of the lab animals tested under standard called
a. ID b. LD50 c. ID50 d. MLD
76. The transfer of genetic material duringtransformation is proved basing on Griffith's experiment by
a. Avery Macleod & Mc.Carthy b. Lederberg & Taulum c. Zinder & Lederberg d. Watson & Crick

77. Phagocytic theory was proposed by
a. Louis Pasteur b. Elie Metchnikoff c. Behring d. Widal
78. Anaphylaxia was first observed by
a. Parter & Richet b. Coombs c. Gell d. None of these
79. Primary mediators in anaphylaxis
a. Histamine b. Seratonin c. Heparin d. All of these
77. The virulence of a pathogen is usually measured by
a. LD b. MLD c. ID d. All of the above
78. Enhancement of virulence is known as
a. Exaltation b. Attenuation c. Both a and b d. None of these
79. Reduction of virulence is known as
a. Exaltation b. Attenuation c. Both a and b d. None of these
80. If a person can be infected by direct contact with infected tissue of another person, it is termed as
a. Indirect contact transmission b. Attachment c. Direct contact transmission
d. None of these
81. If the vectors transmit the infection mechanically they are called
a. Biological vectors b. Mechanical vectors c. Biological reservoir
d. Both a and c
82. Hybridoma technique was developed by
a. Kochler & Milston b. Niel's Jerne c. Both a and b d. None of these
83. Disease that effects many people at different countries is termed as
a. Sporadic b. Pandemic c. Epidemic d. Endemic

ANSWERS
1. a 2. c 3. a 4. b 5. b 6. a7. b 8. b 9. c 10. a 11. b 12. b13. b 14. d 15. c 16. d 17. b 18. a19. b 20. b 21. c 22. a 23. c 24. b25. d 26. c 27. c 28. c 29. b 30. b31. a 32. b 33. b 34. a 35. c 36. c37. c 38. a 39. b 40. c 41. b 42. a43. c 44. b 45. c 46. a 47. b 48. a49. a 50. d 51. d 52. b 53. b 54. c55. a 56. c 57. a 58. c 59. a 60. b61. d 62. c 63. c 64. b 65. b 66. a67. c 68. a 69. b 70. a 71. a 72. a73. b 74. e 75. b 76. a 77. d 78. a79. b 80. c 81. b 82. c 83. a

1. Cold like symptoms are caused by whichbacteria
a. Pseudomonas b. E.coli c. Haemophilus influenza
d. Haemophilus streptococcus
2. In Streptococcus fecalis, the conjugationtakes place at
a. Pili b. Cell membrane c. Cell wall d. Flagella
3. The infected mad dogs may contain
a. Nergi bodies b. Niagri bodies c. Negri bodies d. Neisser bodies
4. What disease the Nesser will produce?
a. Mumps b. Rubella c. Polio d. Measles
5. Rancidity in spoiled foods is due to
a. Lipolytic organisms b. Proteolytic organisms c. Toxigenic microbes
d. Saccharolytic microbes
6. The Baterium that is most commonly used in genetic engineering is
a. Escherichia b. Klebsiella c. Proteius d. Serratia
7. The functions of plasmid are
a. DNA replication b. Protein synthesis c. Cell wall synthesis d. None of the above
8. Mycoplasmas are bacterial cells that
a. Fail to reproduce on artificial meida b. Have a rigid cell wall c. Are resistant to penicillin d. Stain well with Gram's stain
9. The etiologic agent of botulism is a
a. Neurotoxin b. Endotoxin c. Enterotoxin d. All of the above
10. The bacterial cells are at their metabolic peak during
a. Lag phase b. Log c. Stationary d. Decline
11. Protein particles which can infect are called
a. Virons b. Prions c. Nucleoida d. None of these
12. Rickesia are stained with
a. Giesna and Castaneda stain b. Macchiavello and Gimnez stains c. Both a and b
13. Endotoxin produced by gram negative bacteria is present in
a. Peptidoglycan b. Lippolysacharide c. Theichoic acid d. Inner membrane
14. Main causative organism of chiken pox is
a. Fox virus b. Mumps virus c. Measles virus d. None of these
15. The mode of reproduction which occursin mycoplasma is
a. Budding b. Bursting c. Binary fission d. Binary fusion
16. Which one of the following is about Herpes viruses?
a. Icosahedral, with envelope, ds DNA b. Polyhedral with envelope, ds DNA
c. RNA, helical with enveloped. ds DNA, brick shape
17. Which one of the following producetypical fried egg appearance colonies onsolid media?
a. Mycobacteria b. Mycoplasts c. Mycoplasms d. Bacteroides
18. An organism that is osmophilic and hasa specific requirements for sodium chloride resembles
a. Halophile b. Basophile c. Barophile d. Xerophile

19. A population of cells derived from a single cell is called
a. Monclonal cells b. Clones c. Protoplasts d. Sub culture
20. Which of the following characters are related to viruses?
a. No growth on inanimate culture media b. Not sensitive to antibiotics
c. No energy producing enzymes d. Insensitive to interferon
21. Dengue fever is caused by –
a. Bacteria b. Virus c. Fungi d. Rickettsia
22. Which of the following is most similar toRickettsia and Chlamydia?
a. Bdellovibrio b. Clostridium c. Mycobacterium d. Mycoldaima
23. How would you distinguish pseudomonasspecies from E-coli?
a. Gram staining b. Morphology c. Glucose fermentation Vs Respiration
d. All of the above
24. The dengue fever virus is –
a. Arbo virus b. Echo virus c. Entero virus d. Orthomyxo virus
25. All of the following are DNA viruses except –
a. Parvo virus b. Paramyxo virus c. Herpes virus d. Pix virus
26. The viruses that live as parasites on bacteria are
a. Fungi b. Commensels c. Bacteriophages d. None of these
27. The anthrax disease is most frequently infected from
a. Cattle b. Sheeps c. Rats d. Both a and b
28. The colonies produced by Pseudomonas on Mac Conkey's medium are
a. Purple colored b. Pink colored c. Pale colored d. Green colored
29. Staining material of gram positive bacterium is
a. Fast green b. Haematoxylon c. Crystal violet d. Safranin
30. Neil mooseri reaction is related to
a. Rickettsiae b. Chlamydiae c. Spirochaetes d. Clostridium periringens
31. Daisy head colony is associated with
a. M.tuberculosis b. C.diphtheriaec. Cl. tetani d. None of these
32. Diagnosis of carrier of salmonella typhimay be shown by
a. Fecal culture b. Bile culture c. Urine culture d. All of these
33. Cholera red reaction is identified by
a. Sulphuric acid b. Nitric acid c. Hydrochloric acid
d. Carbolic acid
34. Bacteria that are responsible forfermentation of dairy milk are
a. Azetobacter b. Rhizobium c. Lactobacillus d. Hay bacillus
35. The fungal disease that affect the internalorgans and spread through the body are called
a. Mycoses b. Systemic mycoses c. Mycotoxicosis d. Superficial mycoses
36. The staining technique used to stain themetachromatic granules of Corynebacterium
a. Giemsa stain b. Alberts stain c. Acid fast staining d. Both a and b
37. The orderly increase in all componentsof protoplasm of a cell is called
a. Reproduction b. Cell division c. Growth d. All of the above
38. The causative organism of cholera, i.e.,Vibrio show the movement called

a. Gliding movement b. Darting movement c. Pseudopoidal movement d. None of these

39. Erythrocytes will get its ATP energy only by

a. Glycolysis b. Kreb's cycle c. Electron Transport d. HMP shunt

40. Virus will contain

a. Cell membrane b. Cell wall c. DNA d. DNA or RNA

41. The bacterial pili mainly contain

a. Carbohydrates b. Lipids c. Proteins d. Minerals

42. The wonder drug of Second World War is produced by

a. Algae b. Fungi c. Bacteria d. Plants

43. Toxins or enzymes which are not produced by streptococcus pyrogens

a. Hyaluronides b. Phosphate c. Hemolysin d. Streptokinase

44. Streptococci which are destroyed at 60°Cfor 30 minutes

a. Preptostreptococci b. Strepto viridians c. Strepto hemolyticus d. All of these

45. Somatic cell of the adult body are haploidin many except

a. Vertebrates b. Invertebrates c. Fungi d. Vascular plants

46. Congential diseases are

a.Diseases present at birth **b**.Deficiency disease **c**.Occur during life **d**. Spread from one individual to another

47. The enzyme needed in biological systems for joining two molecules is called

a. Lyases b. Diastases c. Polymerases d. Hydrolase

48. Identify the obligate anaerobes

a. Salmonella b. Vibrio cholera c. Cl. tetani d. Sarcinae

49. All prokaryotes are surrounded by a cell wall except

a. Mycoplasms b. Sperochetes c. Actinomycetes d. Methanogena

50. Enzyme hydrolyzing bacterial cell wall

a. Lysozome b. Reductase c. Protease d. Lysozyme

51. All of the following are energy source of bacteria except

a. Oxidation of inorganic compounds b. Oxidation of organic compounds
c. Absorption of heat d. Utilisation of visible light

52. The nucleus controls protein synthesis in the cytoplasm by sending

a. Chromatin b. A DNA template c. m RNA molecule
d. A pecialized protein

53. The site of energy production in a cell

a. Micro body b. Chromosome c. Ribosome d. Mitochondria

54. All of the following are acid fast structures except

a. Clostridium b. Bacterium spores c. Exoskeleton d. None of these

55. Which of the following are acid fast structures?

a. Mycobacteria b. Bacterial spores c. Nocardia d. All of these

56. Maximum application of animal cell culture technology today is in the production of

a. Insulin b. Interferons c. Vaccines d. Edible proteins

57. Bacterial ribosomes are composed of

a. Protein and DNA b. Protein and mRNA c. Protein and rRNA
d. Protein and tRNA

58. Which of the following are the characteristics of bacterial spore?

a. Highly refractile b. usually dehydrated c. Sensitive to formaldehyde d. All of these

59. Bioleaching is done by

a. Protozoa . Bacteria c. Algae d. All of the above

60. Inclusion bodies diagnostic of rabies are called

a. Elementary bodies b. Pascheur bodies c. Negri bodies d. Guarnieri bodies

61. Which of the following genera is mostlikely to contain organisms capable of surviving high temperature?

a. Vibrio b. Pseudomonas c. Torula d. Coxiella

62. What is the function of bacterial capsule?

a. Production of organism from phagocytosis b. Helps in adherence of bacteria to surface inits environment c. Both a and b d. None of these

63. The apparatus used to maintain a continuous culture

a. Chemostat b. Autostat c. Thermostat d. Both a and c

64. One flagelium at one end of the organ is called –

a. Monotrichate b. Amphitrichate c. Iophotrichate d. Peritrichate

65. Diphtheria is caused by

a. Corynebacterium b. Staphylococcus c. Streptococcus d. None of these

66. Koplic spots observed in the mucousmembrane is characteristic feature of the disease

a. Rubella b. Measles c. Mumps d. Influenza

67. A bacterium containing prophage is called as

a. Lytic b. Lysoge c. Lytogen d. None of these

68. The most infectious food borne disease is

a. Tetanus b. Dysentery c. Gas gangrene d. Botulism

69. An example for common air borne epidemic disease

a. Influenza b. Typhoid c. Encephalitis d. Malaria

70. Vrial genome can become integrated into the bacterial genomes are known as

a. Prophage b. Temperatephage c. Bacteriophage d. Metaphage

71. Techoic acid is –

a. Found in the walls of Gram positive bacteria b. Provide receptors for phages c. Make up outer wall of Gram negativebacteria d. Influence the permeability of the membrane

72. Virion means

a. Infectious virus particles b. Non-infectious particles c. Incomplete particles d. Defective virus particles

73. Virulence of the microorganisms can be reduced by

a. Attenuation b. A virulence c. Inactivation d. Freezing

74. The test used for detection of typhoid fever

a. WIDAL test b. ELISA c. Rosewaller test d. Westernblotting

75. Bacteriophage capable of only lytic growth is called

a. Temperate b. Avirulent c. Virulent d. None of these

76. Diphtheria bacillus is otherwise known as

a. Fried-Landers bacillus b. Kleb's hofflers bacillus c. Frchs bacillus d. Koch's bacillus

77. Acridine dyes are more effective against
a. Gram positive b. Gram negative c. Ricke Hsia d. Mycoplasma
78. In bacteria pigment bearing structures are
a. Chloroplast b. Protoplast c. Sphaeroplast
d. Chromatophores
79. The procedure of differential staining of bacteria was developed by
a. A.H. Gram b. H.C. Gram c. N.C. Gram d. H.A. Gram
80. Intermediate group of pathogen between bacteria and viruses which are intracellular parasites are called
a. Mucoplasmas b. Rickettsias c. Prions d. Virusoides
81. Bacillus is an example of
a. Gram positive bacteria b. Gram negative bacteria c. Virus d. Viroid
82. Amoebic dysentery in humans is causedby
a. Plasmodium b. Paramecium c. Yeast d. Entamoeba histolytica
83. Viral genome that can become integratedinto bacterial genome is called
a. Prophage b. Temperate phage c. Bacteriophage d. Metaphage
84. Cytochromes are
a. Oxygen acceptors b. ATP acceptors c. Electron acceptors
d. Protein acceptors
85. The cells having F plasmid in the chromosomes were termed as
a. Hfr b. F– c. Hbr d. C+
86. Recombination process occurring through the mediation of phages is
a. Conjunction b. Transduction c. Transformation d. Transfection
87. Mordant used in grams staining is
a. Crystal violet b. Iodine c. Saffranin d. All of these
88. Parasitic form must contain
a. Capsule b. Cell-wall c. Endospores d. Flagella
89. Gram staining is an example for
a. Simple staining b. Differential staining c. Negative staining
d. None of these
90. Following Cocci are non-motile except
a. Staphylococcus b. Meningococcus c. Gonococcus
d. Rhodococcus agilis
91. Aspergillus fumigatus can infect
a. Birds b. Animals c. Man d. All of them
92. Enterotoxin responsible for food poisoning is secreted by
a. Enterococci b. Entamoeba histolytica c. Enterobacteriaceae
d. Straphylococci
93. Autolysis is done by
a. Mitochondria b. Lysosomes c. Golgi bodies d. Peroxisomes
94. A facultative anaerobic is
a. Only grow anaerobically b. Only grow in the presence of O2 c. Ordinarily an anaerobe but can grow withO2 d. Ordinarily an aerobe but can grow inabsence of O2
95. The percentage of O2 required bymoderate anaerobe is
a. 0% b. < 0.5% c. 2 – 8% d. 5 – 10%
96. Interferon is formed by

a. Lymphocytes b. Lymphoblasts c. Fibroblasts d. All of these

97. Pigment bearing structure of bacteria are

a. Mesosomes b. Plasmids c. Mitochondria d. Chromophores

98. Spirochete is

a. Gonococci b. Strphylococci c. Treponema pallidum d. Streptococci

99. Histones are found in

a. Prokaryotes b. Eukaryotes c. Viruses d. None of these

100. Cell wall of gram negative bacteria is

a. Thick b. Lipids are present c. Teichoic acids are absent d. None of these

101. Cytoplasmic streaming is present in

a. Prokaryotes b. Animals c. Eukaryotes d. Both a and b

102. The motile bacteria is

a. S. typhi b. K. pneumoniae c. B. anthracis d. Shigella

103. The stain used to demonstrate fungus

a. Albert b. Nigerosin c. Lactophenol cotton blue d. None of these

104. Exotoxina are

a. Heat labile b. Heat stable c. Part of cell wall d. Polymerized complexes

105. The viruses that attack bacteria are

a. Bacterial viruses b. Bacterial pathogens c. Bacteriophages d. Various

106. The size of virus particle may range

a. 0.02–0.2 ìm b. 0.5–10 ìm c. 0.015–0.2 ìm d. 0.1–100 ìm

107. The bacterial cell multiplication is usually by

a. Mitosis b. Meiosis c. Conjugation d. Binary-fission

108. Rod shaped bacteria are known as

a. Cocci b. Comma forms c. Bacilli d. Plemorphic froms

109. All the groups of bacteria have cell wall

a. Mycobacteria b. Mycoplasmas c. Clostridia d. Rickettsia

110. The bacteria, which is motile at 22oC butnon-motile at 37oC is

a. Tranformation b. Transduction c. Conjugation d. Cell fusion

111. Teichoic acids and Teichuronic acids are found in

a. Gram positive bacteria b. Gram negative bacteria c. Fungi d. None of these

112. Meosomes are

a. Kind of ribosomes b. Formed during cell lysis
c. A part of cell wall d. Principal sites of respiratory enzymes

113. The characteristic shape of the bacteria is maintained because of

a. Capsule b. Cell wall c. Cell membrane d. Slime layer

114. Bacterial capsule is chemically composed of

a. Polypeptide b. Polynucleotides c. Polysaccharides d. Polypeptides or polysaccharides

115. The cell wall deficient form of bacteria is

a. Mycoplasma b. ‘L’ form c. Protoplast d. Spheroplast

116. Which of the following vaccine containsattenuated form of bacteria?

a. BCG b. TAB c. Polio d. Cholera

117. The differences between Gram positive and Gram negative bacteria is shown to reside in the

a. Cell wall b. Nucleus c. Cell membrane d. Mesosomes

118. Premunition is particularly seen in –

a. Ascaris b. Giardia c. Plasmodium d. None of these

119. The virulence determining antigens of microorganisms may be

a. Proteins and polysaccharides b. Carbohydrate – protein complexes
c. Polysaccharide – Phospholipid – Proteincomplexes d. All of these

120. The largest protozoa is –

a. Balantidium coli b. Entamoeba coli c. Trichomonus vaginalis
d. Toxoplasma gondii

121. Bacterial locomotion is accomplished by

a. Fimbria b. Flagella c. Cytoskeleton d. Both a and b

122. Fimbriae are demonstrated by

a. Culture b. Gram stain c. Biochemical reactions d. Haemaggulation test

123. The motile bacteria is

a. Salmonella typhi b. Klebsiella pneumoniae c. Bacillus anthracis
d. Shigella flexneri

124. Following cocci are non-motile except

a. Staphylococcus b. Meningococcu c. Gonococcus
d. Rhodococcus agilis

125. A polysaccharide capsule is present on cryptococci which –

a. Inhibits phagocytosisb. Is an aid to diagnose c. Cross reacts with rheumatoid factor d. All of these

126. Fungi differs with bacteria in that it –

a. Contain no peptidoglycan b. Are prokaryotic c. Susceptible to griseofulvin d. Have nuclear membranes e. All of these

127. Bacteria multiply by

a. Spore formation b. Simple binary fission c. Conjugation
d. Gametes

128. Bacterial spores are

a. Weakly acid fast b. Strongly acid fast c. Alcohol fast d. Non acid fast

129. Endospores can be stained with

a. Safranine b. Crystal violet c. Methylene blue d. Malachite green

130. The following bacteria produce pigment, except

a. Pseudomonas pyocyaneus b. Serratia marcescens c. D. pneumoniae
d. Staphylococcus aureus

131. The order of stains in Gram-stainingprocedure is

a. Crystal violet, Iodine solution, Alcohol,Saffranine **b.** Iodine solution, Crystal Violet, Saffranine,Alcohol **c.** Alcohol, Crystal Violet, Iodine solution,Saffranine **d.** All of these

132. The percentage of alcohol used in Gram staining Is

a. 75% b. 90%c. 60% d. 25%

133. Gram positive bacteria appear as

a. Pink b. Violet c. both a & b d. None of these

134. Gram negative bacteria appear as
a. Pink b. Violet c. both a & b d. None of these
135. The action of alcohol during Gram staining is
a. Allows the color b. It adds color c. Decolorises the cells d. None of these
136. Lipid contents is more in
a. Gram negative bacteria b. Gram positive bacteria c. Same in both
d. None of these
137. Cell-wall is
a. Thick in Gram positive than Gram negative b. Thick in Gram negative than Gram positive
c. Equal in both d. In Gram negative cell-wall is absent
138. The Lipid content present in Gram positivebacterial cell-wall is
a. 1-10 % b. 1-5 % c. 2-8 % d. None of these
139. Rickettsiae stained by this technique responds as
a. Gram positive b. Gram negative c. Between positive and negative
d. None of these
140. Fungal disease in human is caused by –
a. Inhalation of conidia b. Invasion of mucous membrane
c. Contamination of wounds with conidia or myceliat fragments d. All of these
e. None of these
141.The main difference in true bacteria and mycoplasma is that it does not possess –
a. Flagella b. Cell wall c. ATP synthesis d. A capsule
142.Wet mount slide preparations are used in microbiology as they allow to see
a. Size and shape of individual organisms b. Characteristic arrangement or grouping of cells c. Motility of the organism d. All of thesee. None of these
143. Ziehl – Neelson stain is a _____
a. Simple stain b. Counter stain c. Differential stain d. None of them
144. Cholera occurs in _____ form
a. Endemic b. Epidemic c. Sporadic d. alle. None of these
145. Staphylococcus aureus are characterized by
a. Formation of acid in sucrose, dextrose b. Liquification of gelatin due to production ofgelatinase
c. Strains are catalase positive d. All of abovee. None of these
146. Diagnosis of bacterial disease can be made by
a. Finding bacteria in pathological fluids or blood b. Isolation of bacteria by culture from exudates or blood. c. Both a and b d. None of these
147. Tinea capitis is
a. Ring worm of the foot b.Ring worm of scalp c.Ring worm of non-hairy skin of body d. Both a and c
148. Those fungi which do not have a sexual stage are classified as
a. Phycomycetes b. Ascomycetes c. Basidiomycetes d. Fungi imperfect in
149. Most molds are capable of growing inthe temperature range between
a. 0o – 25oC b. 0o – 35oC c. 10o – 25oC d.10o – 35oC
150. Lab diagnosis of Leishmaniasis is done by
a. CFT b. Peripheral smear c. Blood culture d. All of these
151. Screening test for AIDS is

a. Western blot test b. ELISA test c. Both a and b d. VDRL test

152. The Largest virus is

a. Parvo virus b. Pox virus c. Rhabdo virus d. None of these

153. The smallest virus is

a. Parvo virus b. Rhabdo virus c. Pox virus d. Adeno virus

154. The extra cellular infections virus particleis called

a. Capsid b. Nucleocapsid c. Virion d. None of these

155. AIDS virus is

a. RNA virus b. DNA virus c. Retro virus d. Entero virus

156. If only one stain is used for staining aspecimen

a. Simple staining b. Negative staining c. Differential staining d. None of these

157. Other than the sample (specimen) the remaining portion is stained then it is called

a. Simple staining b. Negative staining c. Differential staining d. None of these

158. If more than one stain is used, such staining is called

a. Simple staining b. Negative staining c. Differential staining d. None of these

159. Special feature of Retro viruses

a. Reverse transcriptase b. RNA directed DNA polymerases c. Both a & b d. Boils

160. HIV is belonging to

a. Retro Viridae b. Rhabdo Viridae c. Toga Viridae d. Paramyxo Viridae

161. During staining for Electron Microscopy, the method which improves contrast ofspecimen is

a. Positive staining b. Negative staining c. Shadow staining d. None of these

162. Coagulase test is used for

a. Salmonella b. Staphylococcus c. Bordetella d. Pneumococcus

163. Archaeo bacteria are known as

a. Halophiles b. Red extreme halophiles c. Osmophiles d. Extreme thermophiles

164. Gasgangarene bacillus is

a. Facultative anaerobe b. Obligate anaerobe c. Facultative aerobe d. Obligate aerobe

165. Mc Fadyean's reaction is used to detect

a. Bacillus anthracis b. Brucella c. Corynaebacterium d. None of these

166. Anthrax is a

a. Vector borne b. Zoonotic infection c. Wound bone d. Soil borne

167. Of the following, this is a capsulated organism

a. Bacillus anthracis b. Escherichia-coli c. Corynebacterium d. Brucella

168. "Prozone phenomenon" is encounteredin

a. A typical mycobacteria b. Brucella c. Streptococcus d. Bordetella pertusis

169. Influenza virus is identified by using

a. Haemaggulutinin inhibition test b. Tissue culture method c.Embryonated eggs d. Plaque formation

170. Streptolysin 'S' is

a. Oxygen unstable b. Thermostable c. Oxygen stable d. None of these

171. Streptolysin O is inactivated by

a. CO2 b. Nitrogen c. Oxygen d. Serum

172.haemolytic streptococci are also knownas

a. Str. Pyogenes b. Virulence group c. Viridans group d. None of these

173. Streptococcus pyogenes classification is based on

a. Protein M b. Protein T c. Protein R d. Polysaccharide C

174. Parasitic form must contain

a. Capsules b. Cell-wall c. Endospores d. Flagella

175. Streptococcus forms causes which type ofinfections?

a. Fever b. Zoonotic c. Pyogenic d. None of these

176. Transformation was observed mainly in

a. Bacteriophages b. Temperate phages c. Lemda–phage d. All of these

177. Capsulated forms of bacteria are

a. Virulent b. A virulent c. Useful d. Symbiotic

178. The bacterial cells participating inconjugation are

a. Conjugants b. Fertile cells c. Exconjugants d. None of these

179. Phagocytes are

a. Monocytes b. Macrophages c. Basophils d. All of these

180. The microorganism engulfed by phagocyte resides in a vacuole is known as

a. Phagosome b. Lysosome c. both a and b d. None of these

181. Sh.dysenteriae is also known as

a. Sh.shiga b. Sh.schmitzi c. Both a and b d. Sh.para dysenteriae

182. Acid fast bacteria are

a. Neisseria b. Staphylococci c. Mycobacteria d. All of the above

183 Mycobacteria are stained with

a. Gram's staining b. Simple staining c. Both a and b d. Ziehl – Neelsen's staining

184. Niacin test is positive in case of

a. Corynebacterium b. M. tuberculosis c. M. bovis d. M. avium

185. Lepromin test

a. Is negative in tubercular leprosy b. Positive in lepromatous type
c. Indicated delayed hypersensitivity test d. Indicates infection

186. Presence of viable bacteria in the bloodstream is called

a. Viraemia b. Septicaemia c. Bacteraemia d. Bactericidal

187. Presence of viruses in the blood streamis known as

a. Viraemia b. Bacteraemia c. Septicaemia d. Pyemia

188. Prophylaxis of cholera is

a. Protected water supply b. Environmental sanitation c. Immunisation with killed vaccines d. All of these

189. □-haemolytic bacteria is

a. Streptococcus pyogenes b. Str. Pneumoniae c. Str. Viridians d. Str. faecalis

190. The natural reservoir of infection forcholera is

a. Flies b. Horse c. Man d. None of these

191. Cholera vaccine gives protection for

a. 1 – 3 months b. 3 – 6 months c. 6 – 9 months d. 9-12 months

192. Vibrio cholera differs from vibrio eltor by

a. It shares some Inaba, Ogawa subtypes with eltor b. Resistant to polymuxin c. Eltor is non-motile d. Causes less subclinical infections as comparedto eltor

ANSWERS

1. c 2. c 3. c 4. d 5. a 6. a 7. d 8. c 9. a 10. b 11. b 12. C 13. b 14. d 15. c 16. a 17. c 18. A19. b 20. d 21. b 22. c 23. c 24. A 25. b 26. c 27. d 28. c 29. c 30. A 31. b 32. d 33. a 34. c 35. b 36. B37. c 38. b 39. a 40. d 41. c 42. B43. b 44. d 45. c 46. a 47. c 48. C 49. a 50. d 51. c 52. c 53. d 54. A 55. d 56. c 57. c 58. d 59. b 60. C61. c 62. c 63. a 64. a 65. a 66. C67. b 68. d 69. a 70. b 71. a 72. C73. a 74. a 75. a 76. b 77. a 78. D79. b 80. b 81. a 82. d 83. a 84. C85. a 86. b 87. b 88. b 89. d 90. A91. b 92. d 93. b 94. d 95. c 96. d97. d 98. c 99. b 100. c 101. c 102. A103. c 104. a 105. c 106. c 107. d 108. C109. b 110. d 111. a 112. d 113. b 114. D115. b 116. a117. a 118. c 119. d 120.a 121. d 122. d 123. a 124. d 125. a 126. E 127. b 128. a 129. d 130. c 131. a 132. B133. b 134. a 135. c 136. a 137. a 138. B139. b 140. d 141. b 142. d 143. c 144. A145. d 146. c 147. c 148. d 149. b 150. D151. b 152. b 153. b 154. C 155. c 156. A157. b 158. c 159. c 160. a161. b 162. B 163. b 164. b 165. a 166. b 167. a 168. B 169. a 170. c 171. c 172. c 173. a 174. A175. d 176. b 177. c 178. a 179. d 180. A181. c 182. c 183. d 184. b 185. c 186. C187. a 188. d 189. a 190. c 191. b 192. d

1. The medium used in membrane filter technique was
a. EMB agar b. EMR-Vp medium c. Lactose broth d. Endo agar
2. Lysol is a
a. Sterilent b. Disinfectant c. Antiseptic d. Antifungal agent
3. Which of the following is a neutral stain?
a. Picric acid b. Gmiemsa c. Neutral red d. Malachite green
4. Peptone water medium is an example for
a. Synthetic medium b. Semisynthetic medium c. Differential medium
d. None of these
5. The method in which the cells are frozen dehydrated is called
a. Pasteurization b. Dessication c. Disinfection d. Lypophilization
6. The technique used to avoid all microorganisms is accomplished by
a. Sterlization b. Disinfection c. Surgical sterilization d. Disinfection Sterilization
7. Thermal death time is
a. Time required to kill all cells at a giventemperature b. Temperature that kills all cells in a given time
c. Time and temperature needed to kill all cells d. All of the above
8. A culture medium the exact compositionof which is not known was called as
a. Simple b. Complex c. Defined d. Natural
9. Elek's gel diffusion test is used for thedetection of
a. Tetani toxin b. Cholera toxin c. Diophtheria toxin
d. Toxoid
10. Temperature required for pasteurization is
a. Above 150oC b. Below 100oC c. 110oC d. None of these
11. Separation of a single bacterial colony is call
a. Isolation b. Separation c. Pure culturing d. All of these
12. Which of the following is ionizing radiation?
a. U.V. rays b. IR c. -xrays d. None of these
13. Which of the following induces dimidiation of thymine?
a. X-rays b. U.V. rays c. ã-rays d. None of these
14. When food material are preserved at a temperature just above freezing temperature, the process is called.
a. Freezing b. Pasteurisation c. Chilling d. Frosting
15. Which of the following method of sterilization has no effect on spores?
a. Drying b. Hot air oven c. Autoclave d. None of these
16. Treponema pallidum can be best indentified using
a. Fluorescence microscope b. Bright field microscope c. Dark field microscope d. Flourescence microscope
17. Autoclaving is carried at
a. Dry heat b. Atmospheric pressure c. 120oC d. All of these
18. Temperature in pasteurization is
a. 62.8oC b. 35.7oC c. 68.2oC d. 60.8oC
19. The bacterial culture prepared by pure culture method is
a. Inoculum b. Suspension c. Dilution d. None of these

20. Algae are rich in
a. Carbohydrates b. Proteins c. Vitamins d. All of these
21. L-Lysine is produced from
a. Corynebacterium glutamicum b. Clostridium botulinum c. Mycobacterium sps d. pseudomonas
22. The orderly increase in the quantity of allof the cellular components is known as
a. Reproduction b. Growth c. Binary fission d. None of these
23. Theobacillus thio oxidans grow at pH
a. 7.0 b. 1.0 c. 6.0 d. 9.5
24. Slow freezing requires the conditions
a. 0oC to 15oC for 15 min. b. – 6 oC to – 10oC for 10 min. c. – 15oC to 3 to 72 hrs. d. None of these

25. Discontinuous heating is called
a. Pasteurization b. Sterilization c. Fermentation d. Tindalisation
26. Isolation is
a. Purification of culture b. Introduction of inoculums c. Separation of a single colony d. To grow microorganisms on surfaces
27. The condition required for autoclave
a. 121oC temp.and 15 lbs. pressure for 20 min. b. 120oC temp.and 20 lbs. pressure for 30 min
c. 150oC temp.for 1 hr. d. 130oC temp for 2 hr.
28. Lysozyme is effective against
a. Gram negative bacteria b. Gram positive bacteria c. Protozoa
d. Helminthes
29. Blood agar medium is
a. Enrichment medium b. Enriched medium c. Selective medium
d. Differential medium
30. Infrared radiation is a method of sterilization By
a. Dry heat b. Moist heat c. Chemical method d. Mechanical method
31. Lyophilization means
a. Sterilization b. Freeze-drying c. Burning to ashes
d. Exposure to formation
32. Temperature used for hot air oven is
a. 100oC for 1 hour b. 120oC for 1 hour c. 160oC for 1 hour
d. 60oC for 1 hour
33. Phenol co-efficient indicates
a. Efficiency of a disinfectant b. Dilution of a disinfectant c. Purity of a disinfectant d. Quantity of a disinfectant
34. This is an agar plate method and iscommonly used for estimation of the number of bacteria in milk.
a. Standard Plate Count (SPC) b. Spread plate c. Lawn culture
d. Roll tube method
35. Agar is obtained form
a. Brown algae b. Red algae c. Green algae d. Blue-green algae

36. A gram positive organism which produces swarming on culture medium is
a. Salmonella b. Clostridium c. Staphylococci d. Proteus
37. Enhancement of virulence in bacteria is known as
a. Pathogenicity b. Attenuation c. Exaltation d. Toxigenicity
38. For effective sterilization in an autoclave the temperature obtained is
a. 50oC b. 100oC c. 120oC d. 180oC
39. Spores are killed by
a. 70% alcohol b. Glutaraldehyde c. Autoclaving d. Both b and c
40. Glassware are sterilized by
a. Autoclaving b. Hot air over c. Incineration d. None of these
41. Tyndallisation was proposed by
a. Tyndall b. Pasteur c. Koch d. Jenner
42. Viruses can be cultivated in
a. Lab media b. Broth c. Living cells d. None of these
43. By pasteurization
a. All the microorganisms can be removed b. Only pathogenic forms can be removed c. Only non-pathogenic forms can be removed d. All of these are correct
44. The temperature required for pasteurizationis
a. Above 100oC b. Below 100oC c. 100oC d. None of these
45. In the medium other than nutrients, if any substance is used in excess, that mediumis
a. Enriched medium b. Special medium c. Enrichment medium d. None of these
46. Example for indicator medium is
a. Nutrient Agar b. Nutrient broth c. Wilson and Blair d. Czapeck-dox medium
47. Example of Anaerobic medium is
a. Robertson cooked-meat medium b. Nutrient agar c. Nutrient broth d. Mac-Conkey's agar
48. The differentiate lactose and non-lactose fermentors, the medium used is
a. Wilson & lair b. Blood Agar c. Tetra thionate broth d. Mac-Conkey's Agar
49. Best method for getting pure culture is
a. Streak-plate b. Agar slant c. Both a & b d. None of these
50. To transfer cultures from one place to another, the device used is
a. Slant b. Needle c. Inoculation loop d. Autoclave
51. The bacterial culture prepared by pureculture is
a. Inoculum b. Suspension c. Dilution d. None of these
52. Separation of a single colony is
a. Pure-culturing b. Isolation c. Separation d. Both a and b
53. Growth period of the culture is
a. Inoculation b. Incubation c. Incineration d. Isolation
54. At the temperature 160oC for one hour, complete sterilization occurs in
a. Autoclave b. Hot air oven c. Laminar flow d. Incubator
55. In autoclave, the principle involved is
a. Dry heat b. Moist heat c. Steam under pressur d. Both b and c

56. The spores of the bacteria which canwithstand the moist heat effect also
a. Bacillus subtilis b. Coxiella burnetti c. Bacillus stearothermophilus d. Pseudomonas

57. Factors on which disinfectivity of a disinfectant depends
a. Concentration of the substance b. Time of action c. pH of the medium and temperature suitable for the chemical d. All of the above

58. Aldehydes, which are most powerful disinfectants
a. Formaldehyde b. Acetaldehyde c. Glutamal aldehyde d. Both a and c

59. Accridine dyes are more effective against
a. Gram positive b. Gram negative c. Mycoplasmas d. Rickttsiae

60. The sterilizing agent is
a. Ethelene oxide b. Oxygen c. Nitrogen d. Carbon tetrachloride

61. Salts of heavy metals used as disinfectants are
a. Thiomersal b. Phenyl mercury nitrate c. Mercurochrome d. All of these

62. Cultures are prepared by penetrating the inoculation loop with suspension into the medium, they are
a. Stock cultures b. Stabcultures c. Sub-cultures d. None of these

63. The principle involved in the streak plate method is
a. Separation b. Streaking c. Isolation d. Dilution

64. Culture media for fungi are
a. Potato dextrose agar (PDA) b. Sabouraud's agar c. Czapekdox agar d. All of the above

65. Spores of actinomycetes are verysensitive, killed at room temperature of
a. 52oC for 30 min. b. 65oC for 30 min. c. 70oC for 30 min. d. 43oC for 30 min.

66. The term that is used for the bacteria which can withstand pasteurization but does not grow at higher temperatures
a. Thermophiles b. Extreme thermophiles c. Thermoduric d. Facultative thermophiles

67. A common laboratory method of cultivating anaerobic micro-organisms is
a. Gas pack system b. Brewer jar system c. Pyrogallic acid over the cotton d. None of these

68. Alkaliphiles grow at pH value between
a. 1 to 6 b. 6 to 9 c. 1 to 11 d. 7 to 12

69. The micro-organisms grow at high salinity are
a. Osmophiles b. Halophiles c. Both a and b d. None of these

70. Non-lactose fermenting colonies seen on Mac Conkey's medium are
a. Salmonella typhi b. Escherichia coli c. Klebsiella pneumonia d. Shigella shigae

71. Wilson and Blair medium is used forisolation of
a. Staphylococci b. Salmonella typhosa c. Vibrio cholera d. Shigella shigae

72. Laboratory diagnosis of enteric fever is based on

a. Blood culture b. Urine and stool culture c. Widal test d. All of the above

73. Shigella was first isolated by

a. Shiga b. Schmitz c. Sonnei d. Robert Koch

74. Which of the following are gas producing Salmonella?

a. S.typhi b. S.enteritidis c. S.cholerasuis d. S.typhimurium

75. Kauffmann white scheme is used to detect

a. Salmonella spp. b. Shigella spp. c. E.coli d. None of these

76. On Mac Conkey's medium Esch. Coli forms

a. Colorless colonies b. Greenish pigmentation c. Pink coloured colonies
d. Medusa head appearance

77. C.diphtheriae requires

a. LJ medium b. Mac Conkey's medium c. Potassium tellurite medium d. PDA medium

78. Culture medium for Mycobacterium tuberculosis

a. L J medium b. Mac Conkey's medium c. Wilson blair medium d. None of these

79. Lepra bacillus is best cultured on

a. Armadillo's brain b. Foot pad of mice c. Liver of guinea pig d. Any of the above

80. Culture medium for clostridia spp.

a. 76 Lower stein Jensen's medium b. Mac Conkey's medium c. Robertson's cooked meat medium d. None of these

81. Clsotridium welchii is positive for

a. Elek's gel precipitation test b. Nagler's test c. Weil felix test
d. Bacitracin test

82. Nagler's reaction detects

a. Coagulase b. Hyaluronidase c. Lecithinase d. None of these

83. Incubation period of Cl. welchii is

a. 8-12 hours b. 7-10 hours c. 5-7 hours d. 2-4 hours

84. The average incubation period of tetanus is

a. 2-3 days b. 7-10 days c. 14-21 days d. 3-4 weeks

85. Salt agar is used for

a. Streptococcus b. Staphylococcus c. Vibrio d. Shigella

86. Culture medium of Leishmania is

a. Sabousand's medium b. NNN medium c. Wilson Blair medium
d. Czapek – dox medium

87. A simple asexual spore which develops by budding is known as

a. Chlamydospore b. Blastospore c. Arthospore d. Conidia

88. Culture medium used for fungus is

a. Sabouraud's medium b. Nutrient agar c. Nutrient broth
d. Minimal agar medium

89. For sterilization of fermentation equipment the method followed is

a. Radiation b. Chemicals c. Heating d. All of these

90. Listed below are substances which areassayed by organisms mentioned in A to E. Match them correctly

1. Crystal Violet I.P. A. Pasteurella pestis

2. Ampicillin I.P.
3. Plaque Vaccine I.P.
4. Rifampicin

B. Bacillus cerus
C. Micrococcus luteus
D. Lactobacillus aureus
E. Lactobacillus aureus
F. Bacillus subtillus

91. Match the following terms with their respective formulations A to E:

1. Lysol
2. Black fluids
3. White fluids
4. Iodophores

A. Higher boiling fractions of the tar acids
B. Prepared from refined tar acids
C. Solution of cresol with soap
D. Basic molecules has varying numbers of amino groups
E. Iodine combined with complex organic chemicals

92. Match the following tests with their respective applications A to E:

1. Schick test
2. Mantoux test
3. Sterility test
4. Potency test

A. Tuberculosis
B. Detection of extraneous microorganisms
C. Diphtheria toxin
D. Detection of infection caused by Rickettsia prowazeki
E. Usefulness of immunological products

93. Match the following equipments with their respective methods of sterilization A to E:

1. Glass syringes
2. Disposable
3. Respiratory parts
4. Dialysis machine

A. Autoclave
B. Chemical instrument
C. Dry heat
D. g-Radiation
E. Chicken pox in children

94. The items listed from A to D can be identified by the tests given below :

1. Coomb's test
2. Coagulase test

A. Candida albicans
B. Virulent staphylococcus aureus
C. Mycobacterium tuberculosis
D. Non-agglutinating antibodies

95. D.pneumoniae can be cultivated in

a. Glucose broth b. Serum broth c. Agar and blood agar d. Chocolate agar e. All of these

96. D.pneumoniae can be identified by

a. Microscopic exam b. Culture of sputum/blood c. Animal inoculation d. All of these e. None of these

97. The diagnosis of tuberculosis is carried out by

a. Emulator b. Antiformin method c. Petroff's method d. Concentration method e. All of these

98. The size of the virus can be determinedby

a. Micrography b. Ultra-centrifugation at high speed c. Ultra-filteration d. All of these

99. Differential staining of bacteria spore is related to

a. Albert's staining b. Lugol' s staining c. Moller's staining d. Indian ink preparation

100. Electron microscope studies does not helpin identifying the section of bacterialspore

a. Core b. Spore cortex c. Capsule d. All of these

101. Wilson and Blair bismuth sulphite medium is used for the growth

a. Salmonella typhi b. Shigella dysenteriae c. Vibrio cholerae d. E. coli

102. Which Rickettsia can be grown on bloodagar media?

a. Lactobacilli b. Streptobacillus c. Bacillus anthrax d. Vibrio cholerae

ANSWERS

1. b 2. b 3. c 4. b 5. d 6. A 7. b 8. a 9. c 10. b 11. a 12. C 13. b 14. c 15. a 16. b 17. c 18. A 19. a 20. d 21. a 22. b 23. b 24. C 25. d 26. c 27. c 28. b 29. b 30. D 31. b 32. c 33. a 34. a 35. b 36. D 37. c 38. c 39. d 40. b 41. a 42. C 43. b 44. b 45. a 46. c 47. a 48. D 49. c 50. b 51. a 52. b 53. b 54. B 55. d 56. c 57. d 58. d 59. a 60. A 61. d 62. b 63. d 64. d 65. b 66. C 67. c 68. d 69. c 70. a 71. b 72. D 73. c 74. b 75. a 76. c 77. c 78. A 79. b 80. c 81. b 82. c 83. a 84. B 85. b 86. b 87. b 88. b 89. D 90. 1.d, 2.c, 3.a, 4.e 91. 1.c, 2.a, 3.b, 4.e 92. 1.c, 2.a, 3.b, 4.e 93. 1.c, 2.d, 3.e, 4.b 94. 1.d, 2.a 95. e 96. E 97. e 98. d 99. c 100. c 101. a 102. A

1. Ergot disease is caused by
a. Puccinia b. Rhizopus c. Claveceps d. Penicillium
2. Mycotoxins are produced by
a. Bacteria b. Fungi c. Algae d. Protozoans
3. The following disease are caused by Mycoplasma except
a.Pneumonia in human beings b.Little leaf of Brinjal c. Dwarf disease of Mulbery d.Citrus canker
4. A disease that can be transmitted by an infectious agent from one individual toanother was called
a. Epidemic b. Pandemic c. Communicable d. Comma
5. The following organisms lack definite cell wall
a. Mycoplasma b. L-forms c. Both a and b d. Bacteria
6. Nagler reaction detects
a. Corynebacterium diphtheria b. Clostridium tetani c. Clostridium perfringens d.Clostridium botulinum
7. The Bacteria move in response to magnetic field is
a. Spirochets b. Treponema c. Aquaspirillum Magnetotacticum d. None of these
8. Virulent factor in pneumococcus is
a. Cell wall b. Capsule c. Mesosomes d. Emdotoxins
9. Which of these is a trace element for bacteria?
a. Mg+2 b. Na+ c. Ca+2 d. Mn+2
10. Most bacteria require vitamins as
a. Growth Factors b. Sources of energy c. Sources of carbon d. Sources of electron donars
11. The primary mode of transmission of poliomyelitis virus:
a. Flies b. Milk c. Person to person d. Food and water
12. Genetic constitution of the cell is
a. Phenotype b. Genotype c. Cryptotype d. Histotype
13. The primary mode of transmission of poliomyelitis is
a. Oral route b. Blood c. Milk d. Person to person
14. Cerebral malaria is caused by
a. Plasmodium vivox b. P.ovale c. P.falsiparum d. P.malaria
15. Size, shape and mode of arrangements is typical of certain microorganisms. Match them correctly:

1. Streptococci	A. Comma and S shaped form
2. Sarcina	B. Gram positive arranged in
3. Bacillus Anthracis	C. Multiples of eight
4. Vibrios and Spirilla	D. Large bacilli, rectangular and gram positive
	E. Gram negative cocci
	F. Rod shaped-acid fast

16. Match the following microorganisms with their respective characteristic A to E :
1. Bacteria
A. Much similar, contains one type of nucleic acid, do not reproduce by binary fission
2. Rickettsia B. Parasites on bacteria, highly specific to one type of

3. Viruses C. Living organism, unicellular, motile, microscopic and show reproduction

4. Bacteriophages D. Grows in atmospheric oxygen, visible without microscope, produces, disease

E. Tiny microorganism, enable to grow outside living cells, retained by bacteria proof filters.

ANSWERS

1. c **2**. b **3**. d **4**. c **5**. c **6**. C **7**. c**8**. d**9**. b**10**. a**11**. d **12**. B **13**. c **14**. c **15.**24. 1.b, 2.b, 3.d, 4.a **16**. 1.c,2 .e, 3.a, 4.b

1. Multiple antibiotic resistances is mediated by

a. Episome b. Plasmid c. Colplasmid d. Both b and c

2. "Antagonism " is seen in

a. Lag phase b. Plasmids c. Log phase d. None of these

3. the first phase of a growth curve is

a. Log phase b. Lag phase c. stationary phase d. Both a and b

4. In gram positive and gram negative bacteria the electron transport contains

a. Naphthquinone b. Plastoquinone c. Ubiquinone d. Both a and b

5. Mycotoxins are formed during the end of

a. Lag phase b. Log phase c. Death phase d. Stationary phase

6. Cells are active and synthesizing new protoplasm. This stage of growth is called

a. Lag phase b. Stationary phase c. Log phase d. All of these

7. Tubercular bacilli grow best in

a. Absence of O2 b. Presence of CO2 c. Presence of O2 d. None of these

9. Rapid bacterial growth phase is known as

a. Log b. Lag c. Lack d. None of these

10. Clostridium welchii spore formation can be induced only on specified media such as

a. Wilson-Blair medium b. Macconkey medium c. Ellner medium d. Thayee-Martion medium

11. Mycotoxins are formed during the end of

a. Lag phase b. Log phase c. Death phase d. Stationary phase

12. Bacteria which need oxygen for growth are called

a. Thermophilic bacteria b. Microaerophilic bacteria c. Facultative anaerobic bacteria d.Mycobacteria

13. pH required for the growth of bacteria is

a. 6.8 – 7.2 b. 5.6 – 8.2 c. 3.0 – 6.0 d. 8.0 – 14.0

14. Drug resistance in bacteria is mainly determined by factor:

a. F b. R c. Col d. Lysogenic factor

15. The ion that is required in trace amounts for the growth of bacteria is

a. Calcium b. Magnesium c. Cobalt d. Sodium

16. The most important vitamin for the growth of bacteria is

a. B-complex b. Vitamin A c. Vitamin D d. Vitamin C

17. The principle in microbiological assays is

a. At certain range the concentration of growth factor will bear a linear relationship to the amount of nutrients added
b. Concentration of growth factor has a linear relationship with the growth of the organism
c. Both a and b d. None of the above

18. If the source of energy for bacteria is from chemical compounds they are said to be

a. Phototrophs b. Autotrophs c. Chemotrophs d. Chemolithotroph

19. In the synthesis of cell components the major element required is

a. Nitrogen b. Sulphur c. Carbon d. Oxygen

20. For the formation of cell-components theelements required are

a. Nitrogen b. Oxygen c. Sulphur d. All of these

21. For the synthesis of amino acids cysteine,cystine and methionine the elementrequired is

a. Sulphur b. Oxygen c. Nitrogen d. None of these

22. Sulphur can be utilized by bacteria in the form of

a. Organic compounds b. Inorganic compounds c. Elemental compounds
d. All of the above

23. Phosphorous is an essential componentof

a. Nucleotides b. Nucleic acids c. Phospholipids and Heichoic acids d. All the above

24. Trace elements are

a. Zn+2, Cu+2, Mn+2 b. MO6+, Ni2+, B3+ and CO2+ c. Both a and b
d. None of these

25. Most bacteria do not require the ion

a. Mg2+ b. Ca2+ c. Na+ d. Fe+2

26. Vitamin function as

a. Co-enzymes b. Co-melecules c. Building blocks of cell d. None of these

27. The vitamin required for Lactobacillus species is

a. Riboflavin b. Niacin c. Pyridoxine d. Folic acid

28. Vitamin K is necessary for the species

a. Lactobacillus spp. b. Bacillus anthracis c. Bacteroides melaninogenicus
d. All of these

29. The bacteria which are able to grow at0°C but which grow at 20°C to 30°C, are known as

a. Psychrophiles b. Facultative psychrophiles c. Average psychrophiles
d. Mesophiles

30. Radical shifts can be prevented by adding

a. Acids b. Alkali c. Buffer d. None of these

31. The orderly increase in the quantity of all the cellular components is known as

a. Reproduction b. Growth c. Binary fission d. None of these

32. The most common mode of cell division in bacteria is

a. Binary fission b. Transverse binary fission c. longitudinal binary fission
d. None of these

33. How much time a bacterium take for the complete duplication?

a. 30 min. b. 10 min. c. 20 min. d. 25 min.

34. The generation time is

a. The time required for the cell to divide b. The total division of the cell during its life time c. The total no. of cells formed d. None of these

35. In bacteria, the increase in population is in the manner

a. Geometric progression b. Multiplication c. Doubling d. None of these

36. Physiologically the cells are active and are synthesizing new protoplasm in which stage of the growth in bacteria

a. Log phase b. Lag phase c. Stationary phase d. None of these

37. The most active stage in the sigmoid curve of bacteria in which maximum growth is attained

a. Lag phase b. Stationary phase c. Decline phase d. Log phase

38. Log-phase is also known as

a. Death phase b. Exponential phase c. Lag-phase d. None

39. The no. of generations per hour in a bacterium is

a. Growth rate b. Generation time c. Sigmoid curve d. None of these

40. In the sigmoid curve (or) growth curve of bacteria how many stages are there

a. 3 b. 4 c. 2 d. 5

41. The reproduction rate is equal to death rate in which stage

a. Decline phase b. Stationary phase c. Lag phase d. Log phase

42. Minimum growth temperature is

a. The growth of organisms at lowest temperature b. The lowest temperature at which the microorganisms grow c. The maximum temperature at which the growth is stable d. None of these

43. Optimum growth temperature is greater that 45oC is

a. Mesophiles b. Thermophiles c. Psychrophiles d. None of these

44. The organisms which can grow both in presence and absence of oxygen

a. Aerobes b. Anaerobes c. Faculative anaerobes d. Strict aerobes

45. The organisms which can grow best in the presence of a low concentration of oxygen

a. Aerophilic b. Microaerophilic c. Aerobic d. Anaerobic

46. The compound that is added to the medium to absorb oxygen for the creation of anaerobic conditions

a. Sodium Thioglycollate b. Nitrous acid c. Citrate d. None of these

47. The utilization of light energy to drive the synthesis of ATP is called as

a. Photolysis b. Photophosphorylation c. Photosynthesis d. Respiration

48. During cyclic phosphorylation NADP is formed or not.

a. No NADP formation b. No NADP utilization c. NADP is converted into NADPH d. All are correct

49. Which of the following organisms requires tryptophan for growth?

a. H.influenza b. Vibrio c. Gonococci d. S.typhi

50. Match the following growth characteristics with their respective temperature ranges A to E :

1. Psychrotrophs	A. Grows between 55 to 65oC
2. Mesophils	B. May survive above 60oC
3. Thermophils	C. Grow well between 25 to 45oC

4. Vegetable bacteria D. Grow below 25oCE. Multiply slowly at 0-4oC

51. Match the following microorganisms with respective sources A to E:

1. Achrommobacter . A. Bread spp
2. Aspergillus flavus B. Water supply
3. Oscillatiria C. Meat scytonema
4. Clostridium D. Salad nigereticans
E. Milk and cheese products

52. Match the following microorganisms with their respective appearance of colonies on bismuth Sulphite agar from A to E:

1. Salmonella typhi A. Brown
2. Salmonella B. No growth choleraesuis
3. Shigella flexneri C. Green
4. Escherichia coli D. Yellow
E. Black

53. The suitable temperature to transport viral culture is –

a. 30oC b. 5oC c. 25oC d. 45oC e. None of these

54. Growth curve does not include following phases of bacteria –

a. Decline phase b. Stationary phase c. Lag phase
d. Synchronous growth

55. Bacteria are more sensitive to antibiotics at which phase of growth curve?

a. Decline phase b. Stationary phase c. Lag phase d. Log phase

ANSWERS

1. b 2. d 3. b 4. a 5. a 6. A 7. b 8. d 9. a 10. c 11. a 12. B 13. a 14. d 15. c 16. a 17. b 18. C 19. c 20. d 21. d 22. a 23. d 24. D 25. c 26. c 27. b 28. a 29. c 30. C 31. b 32. c 33. c 34. c 35. a 36. C 37. d 38. c 39. b 40. b 41. d 42. b
43. a 44. a 45. b 46. b 47. c 48. A 49. d50. 1.b, 2.c, 3.d, 4.a 51.
1.e,2.a,3.b,4.c52.1.e,2.c,3.a,4.b 53. b 54. d 55. D

1. A peculiar cytochrome is observed inbacteria and it can react with molecularoxygen, what is it?
a. Cyt b b. Cyt c c. Cyt d d. Cyt o
2. The genetic material in HIV is
a. ds DNA b. ss DNA c. s RNA d. None of these
3. Which one of the following mutagens actonly on replicating DNA?
a. Ethidium bromide b. Nitrosogeranidine c. Acridine orange d. None of above
4. Poly A tail is frequently found in
a. Histone in RNA b. Bacterial RNA c. eukaryotic RNA d. TRNA
5. Which of the following is an example of RNA virus?
a. SV 40 b. T4 phage c. Tobacco mosaic virus d. Adeno virus
6. Genomic DNA is extracted, broken into fragments of reasonable size by a restriction endonuclease and then inserted into a cloning vector to generate_____ chimeric vectors. The cloned fragments are called
a. Clones b. Genomic library c. mRNA d. None of these
7. Transgenic animals are produced when GH gene fused with
a. MT gene b. GH c. GRF d. FIX
8. In which medium the hydridoma cells grow selectively?
a. Polyethylene glycol b. Hypoxanthine aminopterin thyminine
c. Hypoxathing-guaning phosphoribosyl transferase d. Both b and c
9. The enzymes which are commonly used in genetic engineering are
a. Exonuclease and ligase b. Restriction endonuclease and polymerase
c. Ligase and polymerase d. Restriction endonuclease and ligase
10. A successful hybridoma was produced by fusing
a. Plasma cells and plasmids b. Plasma cells and myeloma cells
c. Myeloma cells and plasmids
d. Plasma cells and bacterial cells
11. **The technique involved in comparing theDNA components of two samples is known as**
a. Monoclonal antibody techniques b. Genetic finger printing c. Recombinant DNA technology d. Polymerase chain reaction
12. Plasmids are ideal vectors for gene cloning as
a. They can be multiplied by culturing b. They can be multiplied in the laboratory using enzymes c. They can replicate freely outside the bacterial cell
d. They are self replicating within the bacterial cell
13. Humans normally have 46 chromosomes in skin cells. How many autosomes would be expected in a kidney cell?
a. 46 b. 23 c. 47 d. 44
14. Pasteur effect is due to
a. Change from aerobic to anaerobic b. Providing oxygen to anaerobically respiring structures c. Rapid utilization of ATP d. Nonsynthesis of ATP
15. A mechanism that can cause a gene to move from one linkage group to another is
a. Trans location b. Inversion c. Crossing over d. Duplication
16. The smallest unit of genetic material that can undergo mutation is called
a. Gene b. Cistron c. Replicon d. Muton

17. The two chromatids of metaphase chrosome represent
a. Replicated chromosomes to be separated at anaphase b. Homologous chromosomes of a diploid set c. Non-homologous chromosomes joined at the centromere d. Maternal and paternal chromosomes joined at the centromere
18. Malate dehydrogenase enzyme is a
a. Transferase b. Hydrolase c. Isomerase d. Oxido reductase
19. In E.Coli att site is in between
a. Gal and biogenes b. Bio and niacin genes c. Gal and B genes d. None of these
20. The best vector for gene cloning
a. Relaxed control plasmid b. Stringent control plasmid c. Both a and b d. None of these
21. A gene that takes part in the synthesis of polypeptide is
a. Structural gene b. Regulator gene c. Operator gene d. Promoter gene
22. DNA replicates during
a. G1 – phase b. S – phase c. G2 – phase d. M – phase
23. A human cell containing 22 autosome and a 'Y' chromosome is probably a
a. Male somatic cell b. Zygote c. Female somatic cell d. Sperm cell
24. Crossing-over most commonly occurs during
a. Prophase I b. Prophase II c. Anaphase I d. Telophase II
25. DNA-replication is by the mechanism of
a. Conservative b. Semiconservative c. Dispersive d. None of the above
26. Production of RNA from DNA is called
a. Translation b. RNA splicing c. Transcription d. Transposition
27. Nucleic acids contain
a. Alanine b. Adenine c. Lysine d. Arginine
28. What are the structural units of nucleic acids?
a. N-bases b. Nucleosides c. Nucleotides d. Histones
29. The most important function of a gene is to synthesize
a. Enzymes b. Hormones c. RNA d. DNA
30. One of the genes present exclusively on the X-chromosome in humans is concerned with
a. Baldness b. Red-green colour baldness c. Facial hair/moustache in males d. Night blindness
31. Peptide linkages are formed in between
a. Nucleotides b. Amino acids c. Glucose molecules d. Sucrose
32. The nucleic acid of polio viruses is
a. DNA b. RNA – (+) type c. t-RNA d. m-RNA
33. Rabies virus is
a. Nake RNA virus b. Naked DNA virus c. Enveloped RNA virus d. Enveloped DNA virus
34. Example for DNA virus:
a. Polio virus b. Adeno virus c. Echo virus d. Poty virus
35. In genetic engineering breaks in DNA are formed by enzymes known as
a. Restriction enzymes b. Ligases c. Nucleases d. Hydralases
36. DNA transfer from one bacterium to another through phages is termed as

a. Transduction b. Induction c. Transfection d. Infection

37. Microorganisms usually make acetyl CO-A by oxidizing

a. Acetic acid b. Pyruvic acid c. □-ketoglutaric acid d. Fumaric acid

38. The method of DNA replication proposed by Watson and Crick is

a. Semi conservative b. Conservative c. Dispersive d. Rolling loop

39. The distance between each turn in the helical strand of DNA is

a. 20 Ao b. 34 Ao c. 28 Ao d. 42 Ao

40. Self-replicating, small circular DNA molecules present in bacterial cell are known

a. Plasmids b. Cosmids c. Plasmomeros d. plastides

41. Western blotting is the technique used in the determination of

a. RNA b. DNA c. Proteins d. All of these

42. m RNA synthesis from DNA is termed

a. Transcription b. Transformation c. Translation d. Replication

43. Western blotting is a technique used in the determination of

a. DNA b. RNA c. Protein d. Polysaccharides

44. Building blocks of Nucleic acids are

a. Amino acids b. Nucleosides c. Nucleotides d. Nucleo proteins

45. DNA finger printing is based on

a. Repetitive sequences b. Unique sequences c. Amplified sequences d. Non-coding sequences

46. The enzyme required for DNA from RNA template:

a. RNA polymerase b. Reverse transcriptase c. DNA polymerase d. Terminal transferase

47. Double standard RNA is seen in

a. Reo virus b. Rhabdo virus c. Parvo virus d. Retro virus

48. Example for DNA viruses:

a. Adeno virus b. Bacteriophage T1, T2, T3, T4, T5, T6 c. Papova virus d. Herpes virus and cauliflower moisaic e. All of the above

49. The following are the RNA viruses, except

a. Reo viruses b. Retro viruses c. Bacteriophage □□C d. Tmv and Bacteriophages Ms2, F2 e. Dahila mosaic virus and Bacteriophages □× 174, M12, M13

50. The two strands of DNA are joined no covalently by

a. Ionic bonds b. Covalent bonds c. Hydrogen bonds between bases d. Polar charges

51. The bases Adenine and Thymine are paired with

a. Double hydrogen bonds b. Single hydrogen bonds c. Triple hydrogen bonds d. Both b and c

52. The no. of hydrogen bonds existing between Guanine and Cytosine are

a. 5 b. 2 c. 3 d. None of these

53. The length of each coil in DNA strand is

a. 15 Ao b. 34 Ao c. 30 Ao d. 5 Ao

54. Nucleic acids are highly charged polymers due to

a. There is phosphodiester bond between 5'- hydroxyl of one ribose and 3'–hydroxyl of next ribose

b. They have positive and negative ends c. Nucleotides are charged structures

d. Nitrogenous bases are highly ionizedcompounds

55. The best studied example for specialized transduction is

a. P1 phage b. P22 phage c. ë-phage d. Both a and c

56. The diagrammatic representation of the total no. of genes in DNA is

a. Genome b. Gene map c. Gene-structure d. Chromatin

57. During specialized transduction

a. Large a mound of DNA is transferred b. A few no. of genes are transferred
c. Whole DNA is transferred d. None of these

58. The cell donating DNA during transformation is

a. Endogenate b. Exogenate c. Mesozygote d. Merosite

59. Genetic information transfer DNA to RNA is called –

a. Transcriptase b. Transduction c. Transformation d. Recombination

60. The gene transfer occurs by –

a. Transformation b. Transduction c. Conjugation d. Cell fusion

ANSWERS

1. d 2. a 3. c 4. c 5. c 6. B7. a 8. b 9. a 10. b 11. b 12. d 13. d 14. b 15. a 16. d 17. a 18. d 19. a 20. a 21. a 22. b 23. b 24. a 25. b 26. c 27. b 28. c 29. a 30. b 31. b 32. b 33. c 34. b 35. b 36. a37. a 38. a 39. b 40. a 41. b 42. a43. a 44. c 45. b 46. b 47. a 48. e49. e 50. c 51. a 52. c 53. b 54. A55. c 56. b 57. b 58. b 59. a 60. a

1. Which of the following is called serum Hepatitis?
a. HCV b. HAV c. HBV d. HIV
2. Which of the following was a non-neural vaccine for rabies?
a. HEPV b. Card vaccine c. BPL d. Simple
3. Which type of antibodies will associate in blood cell coagulation?
a. IgE b. IgA c. IgM d. IgG
4. In a antigen haptens are
a. Immunogenic b. Non-immunogenic c. Antigenic d. None of these
5. The antibody that is first formed after infection is
a. IgG b. IgM c. IgD d. IgE
6. Antibodies in our body are produced by
a. B-lymphocytes b. T-lymphocytes c. Monocytes d. RBC's
7. The points at which crossing over has taken place between homologus chromosomes are called
a. Chiasmata b. Synaptonemal complex c. Centromeres d. Protein axes
8. How much of globulin is present in human serum?
a. 8% b. 12% c. 16% d. 4%
9. The substance which acts as antimetabolites are called
a. Activators b. Substrates c. Inhibitor d. Cofactor
10. Enzymes are chemically
a. Lipids b. Proteins c. Carbohydrates d. None of these
11. Monoclonal antibodies are produced by
a. Hybridoma technology b. Biotechnology c. Fermentation Technology
d. None of these
12. First line of body defence is
a. Antibody molecules b. Unbroken skin c. Antigen molecules d. Phagocytic cells
13. What is the strength of the bond between antigen and antibody?
a. Affinity b. Avidity c. Covalent d. None of these
14. Syphillis is caused by
a. Staphylococcus aureus b. Yersinia psdtis c. Treponema pallidum
d. Streptococcus syphilitis
15. Nergibodies produced by rabies virus show characteristic _____ inner granues
a. Basophilic b. Eosinophilic c. Neutrophilic d. Acidophilic
16. The widely used yeast for the production of single cell protein is
a. Saccharomyces cerevisiae b. Rhizopus c. Candida utilis d. All of the above
17. Analysis of protein antigen is by
a. Southern blot b. Northern blot c. Western blot d. None of these
18. Which of the following can provide naturally acquired passive immunity for the new born.
a. IgA b. IgG c. IgE d. IgM
19. AIDS disease is caused by a virus which belongs to
a. Retro virus group b. Rhabdo virus group c. Hepatitis virus group
d. Adeno virus group
20. Complement based agglutination reaction is known as
a. Haem agglutination b. Coplement fixation c. Conglutination d. Schultz Dale Phenomenon

21. Reverse transcriptase is an enzyme involved in the synthesis of
a. DNA b. Soluble RNA c. m-RNA from DNA d. Nucleotides
22. The cellular immune response is mediated by
a. B cells b. T cell c. BT cells d. Endothelial cells
23. The major immunoglobulin present in the human serum is
a. IgG b. IgA c. IgE d. IgG
24. Reagenic type antibody is
a. IgG b. IgA c. IgM d. IgE
25. Blood group antigens are
a. Species specific b. Isospecific c. Autospecific d. Organ specific
26. The reaction of soluble antigen with antibody is known by
a. Precipitation b. Flocculation c. Agglutination d. Complement fixation
27. Interferon is composed of
a. Lipids b. Lipoprotein c. Glycoprotein d. Nucleic acid
28. Agglutination reaction is strongest with the immunoglobulin:
a. IgM b. IgG c. IgA d. IgD
29. The use of monoclonal antibodies is
a. Immunotherapy b. Gene therapy c. Blood transfusion d. Organ transfusion
30. Hybridoma technique is used for
a. Monoclonal antibodies b. Polyclonal antibodies c. Both a and b d. None of these
31. Test used for AIDS is
a. Widal test b. ELISA c. Aggluatination d. CFT
32. Antibody having high valency is
a. IgG b. IgA c. IgD d. IgM
33. Intensity of attraction between antigen and antibody molecule is known as
a. Affiniy b. Avidity c. Reaction d. None of these
34. Active immunity is induced by
a. Infection b. Placental transfer of antibodies c. Injection of antibodies
d. Injection of gamma- globulins
35. Pasteur developed the vaccines for
a. Anthrax b. Rabies c. Chicken cholera d. All of the above
36. Delayed type of hypersensitivity is seen in
a. Penicillin allergy b. Contact dermatitis c. Arthus reaction
d. Anaphylaxis
37. The following are used for the preservation of virus, except
a. Freezing (–20°C–70°C) b. Lyophilization c. Ether d. Formaldehyde
38. Antibody formation depends on
a. Age of the person b. Amount of antigen c. Well being of the person d. All of the above
39. Local immunity is important in
a. Influenza b. Allergy c. Polio d. All of these
40. Role of magnesium in vaccine is

a. Adjuvant b. Stabilizer c. Conditioner d. All of these

41. Immunity is lifelong following

a. Diphtheria b. Tetanus c. Measles d. Yellow fever

42. To prepare vaccine for small pox, the material used by Edward Jenner is

a. Small pox material b. Chicken pox material c. Cow-pox material d. Measles material

43. During recombination, the strain that donates genetic material frequently with high rate:

a. Hfr-Strain b. F+-Strain c. F-Strain d. both a and c

44. The character acquired by the cell due to recombination is

a. Inheritable b. Syppressed c. Dominating d. Heritable

45. T-cells are produced from

a. Bone marrow b. Thymus c. Spleen d. None of these

46. Antibodies are produced from

a. T-cells b. â-cells c. NK cells d. Eosinophils

47. Incomplete antigens are called

a. Immunogens b. Epitomes c. Haptens d. Paratope

48. To be antigen, the chemical molecule (protein) needs

a. High molecular weight b. Chemical complexity c. Both a and b d. None of these

49. The parts which filter lymph are

a. Lymph nodes b. Spleen c. Thymus d. Bone marrow

50. The primary cells involved in immune response are

a. NK-cells b. K-cells c. Lymphocytes d. None of these

51. **Plasma cells are the end cells of**

a. T-cells b. B-cells c. Killer cells d. Nk-cells

52. Basophils have receptors for antibodies

a. IgG b. IgAc. IgM d. IgE

53. Because of denaturation, antigensbecome functionless, these are called:

a. Cross-reactive antigens b. Epitopes c. Hidden epitopes d. Forssman antigens

54. Capacity of antigen to breakdown into small fragments each with a single epitopic region is known as

a. Solubility b. Froeignness c. Denaturation d. None of these

55. Antigenic specificity is due to

a. Chemical complexity b. Solubility c. Steric configuration d. All of these

56. Antibodies are

a. Proteins b. Glycoproteins c. Phospholipids d. None of these

57. General purpose antibody is

a. IgA b. IgG c. IgM d. IgD

58. Antibody present in colostrums is

a. IgG b. IgA c. IgM d. IgE

59. Which antibody is called millionaire molecule?

a. IgA b. IgM c. IgG d. IgD

60. IgE is discovered by
a. Ishizaka b. Porter c. Richet d. None of these
61. Antigen-antibody reactions are
a. Reversible b. Irreversible c. Specific d. Both a and b
62. Serological reactions are useful for
a. Detection of antigens b. Detection of antibodies c. Both a and b
d. None of these
63. For the separation of antigens the methodused is
a. Immuno electrophoresis b. Flocculation c. Agglutination
d. None of these
64. Counter immune electrophoresis is useful for detection of
a. One antigen/antibody b. Two antigens/antibody c. More than two
d. None of these
65. When a particular antigen is mixed with antibody in the presence of an electrolyte at suitable temperature and pH theparticles are clumped, this is called:
a. Precipitation b. Agglutination c. Electrophoresis d. CIE
66. Toxins and viruses can be detected by
a. Precipitation b. Agglutination c. Neutralisation
d. None of these
67. Which is most antigenic?
a. Exotoxins b. Endotoxins c. Viruses d. All of these
68. Shick test is used for the detection of
a. Diphtheria b. T.B. c. Cholera d. Typhoid
69. Secondary function of complements are
a. Haemolysis b. Phagocytosis c. Both a and b d. None of these
70. Very effective, less time consuming and at a time so many samples can be detected by
a. ELISA b. CFT c. Neutralization d. Agglutination
71. â-cells are involved in
a. Humoral immunity b. Cell-mediated immunity c. Active immunity
d. Passive immunity
72. Innate immunity is
a. Specific b. Non-specific c. Active d. Passive
73. Innate immunity is developed by
a. Mechanical barriers b. Chemical barriers c. Both a and b
d. None of these
74. Acquired immunity is
a. Natural b. Artificial c. Active & Passive d. All of these
75. Acquired immunity can be developed by
a. Natural means b. Artificial means c. Both a and b d. None of these
76. Immediate type hypersensitivity reactions are
a. Type-I b. Type-II c. Type-III d. All a, b and c
77. Immediate type of hypersensitivity reactions are mediated by
a. T-cells b. □-cells c. Mast cells d. Macrophages
78. Example for cell-mediated immunity are

a. Tuberculin type b. Contact dermatitis c. Granulomatous d. All of these

79. Mountax reaction is used for detection of

a. T.B. b. Diphtheria c. Cholera d. None of these

80. All the antibodies produced from a â-cellare having

a. Similar specificity b. Different specificities c. Similar size d. None of these

81. Hybridoma formation in hybridoma technique is from

a. Spleen cell–Myeloma cell b.Spleen cell–Spleen cell c.Myeloma cell–Myeloma cell d.None of these

82. Anthrax vaccine is prepared by

a. Attenuated bacilli b. Killing the bacilli c. Live bacilli d. None of these

83. Attenuated, oral poliomyelitis vaccine is

a. BCG b. Measles vaccine c. Sabin vaccine d. TAB vaccine

84. Killed, polio vaccine is

a. Sabin vaccine b. Salk c. BCG d. TAB

85. Measles vaccine is given to children at the age of

a. 1 year b. 7 months c. between 9 months and 10 years d. None of these

86. Pertussis vaccine is

a. Heat killed b. Formalin killed c. Attenuated d. live

87. DPT is

a. Triple vaccine b. Double vaccine c. Tetanus toxoid d. All of these

88. DPT, is used as vaccine for

a. Diphtheria b. Pertussis vaccine c. Tetanus toxoid d. All of these

89. DPT is given to children at the age of 16-24 months, as the dose is

a. 0.5 ml at intervals of 4 weeks b. A booster dose of 0.5 ml c. Both a and b d. None of these

90. If more than one kind of immunizingagent is included in the vaccine, it is

a. Cellular vaccine b. Recombinant vaccine c. Mixed vaccine d. Toxoid vaccine

91. Vaccines are prepared from killed microbes, they are

a. Inactivated (killed) vaccine b. Attenuated vaccines c. Autogenous vaccine d.None of these

92. Vaccines used against viral infections are

a. Measles and Mumps vaccine b. Cholera vaccine c. Typhoid vaccine d. Anti-rickettsial vaccine

93. If the microbes used in the vaccine are obtained from patient, they are

a. Anti viral vaccines b. Anti bacterial vaccines c. Autogenous vaccines d. None of these

94. Vaccines prepared from toxins andchemicals are

a. Cellular vaccines b. Sub-cellular vaccines c. Attenuated vaccines d. Heterologous vaccines

95. Example for live vaccine is

a. Rubella & BCG b. Polio & TAB c. Diphtheria & Tetanus d. Hepatitis A & Rabies

96. DPT is given for the prevention of

a. Diphtheria, Tetanus b.Diphtheria, Pertusis c.Diphtheria, Tetanus &pertussis d.None of these

97. The live vaccines are available against the following viruses, except:

a. Influenza b. Measles c. Rabies d. Polio

98. HIV can be transmitted through

a. Blood b. Semen c. Vaginal fluid d. All of these

99. Match the following terms with their respective definitions A to E used in virology:

1. Haemagglutination — A. A phenomenon of acquiring resistance to infection by a infection by a second virus
2. Virus titre — B. A virus does not cause cytopathogenic changes in tissue culture
3. Virus interference — C. Determination of the number of infective units in the virus suspension
4. Interferon — D. A substance by which viruses can attack themselves to RBC

E. Substance used to destroy virus

100. Match the following vaccines with their respective contents A to E:

1. Typhoid vaccine — A. Killed rickettsia
2. Typhus vaccine — B. Killed bacteria
3. Measles vaccine — C. Attenuated viruses
4. Smallpox — D. Killed viruses

E. Attenuated bacteria

101. Match the following immunglobulins with their respective occurrences A to E:

1. IgM — A. In the seromucous secretions
2. IgG — B. After the primary antigenic stimulus
3. IgA — C. Synthesized during secondary response
4. IgE — D. Plasma

E. Serum

102. Match the following viral vaccines with their source materials A to E:

1. Influenza — A. Fluid from cultures of human diploid cells
2. Rabies — B. Dermal scraping from infected animals
3. Smallpox — C. Allantoic fluid from fertile hen's eggs
4. Yellow fever — D. Fluid from cultures of rabbit kidney

E. Aqueous homogenate of chick embryo

103. Animals are naturally immune to infection caused by

a. V. Cholera b. S.typhosa c. Both a and b d. None of these

104. The immunity acquired by inoculation of living organism of attenuated virulenceis

a. Artificial active immunity b.Passive immunity c.Natural active immunity d.Local immunity

105. Organisms can be attenuated for inoculation by

a. Growing it at a temperature higher than optimum b. By passage through animals of different species which are less susceptible to it c. By continous cultivation in presence of antagonistic substance d. Any one of the above e. None of these

106. Passive immunity lasts for the period of about

a. 10 days b. 2 – 3 months c. 10 years d. None of the above

107. The markers helpful in detecting anti immunity are

a. Hyper gamma globulinaemia b. Circulating antibodies c. Response to cortisone d. Lymphoid hyperplasia e. All of these

108. Following substance may act as anantigen

a. Egg albumin b. RBC and serum c. Vegetable protein d. Snake venom e. All of these

109. H antigen are present in

a. Motile organ b. Non-motile organ c. Both a & b d. None of these

110. Antitoxin is used for _____ immunization.

a. Active b. Passive c. Both a and b d. None of these

111. The agglutinin test is used for _____

a. Identification of isolated bacteria b. Typing of bacterial species c. Study of antigenic structure of bacteria d. All of these e. None of these

112. In blood transfusion it is essential that

a. Blood of hologous group should always besame b. Direct matching between patient's serum and donor's corpuscles be performed c. Both a & b d. None of these

113. To be anaphylactic, the sensitizingsubstance should be

a. Protein in nature b. Should have a large molecular weight c. Soluble in tissue fluids d. All of the above e. None of these

114. The basics of pathology in asthama,allergic rhinitis, urticaria are

a. Local vasodilation b. Increased capillary secretion c. Excess eosinophils in tissue secretion andblood d. All of these

115. Which test is used for detecting susceptibility of an individual to diphtheria toxin?

a. Schick tests b. Dick test c. V-P test d. Precipitin test

116. Syndromes associated with Human Tlymphotropic virus type I(HTLV-I) are

a. Adult T-cell lymphoma b. Hairy cell leukemia c. Adult T cell leukemia d. All of these

117. Plague and Tularemia vaccine can beprepared from

a. Chemical fraction of the causative bacteria b. Heat killed suspension of virulent bacteria

c. Formalin inactivated suspension of virulent bacteria d. Avirulent live bacteria e. All of these

118. AIDS patients suffer from pneumonia due to

a. Pneumocystisis carinii b. Cryptospodidium c. S.pneumoniae d. Toxoplasma

119. Statements applicable to human lice:

a. Cause pruritic skin lesions. b. Are wingless c. Transmit epidemic typhus, relapsing fever and Trench fever d. Pediculus humanus and phthirus pubis are two species e. All of these

120. Natural killer cells

a. Belongs to B-cell lineage b. Belongs to T-cell lineage c. Display cytotoxic effect on tumour cell d. Require previous antigen exposure for activation

121. Immunoglobulin is associated with anaphylactic delayed hypersensitivity reaction

a. IgE b. IgA c. IgD d. IgM e. IgG

122. The most abundant antibody found in serum is

a. IgA – 1 b. IgG – 1 c. IgG – 2 d. IgG – 3 e. IgG – 4

123. Patients suffering from AIDS havefollowing immune abnormalities

a. Decreased CD4 + T cells b. Increased CD8 + T cells c. Hypergammaglobulinemia d. CD4 +/CD8 + ratio greater than 21 e. Both b & d

124. Immunoglobulin which cannot activatecomplement

a. IgM b. IgE c. IgA d. IgG

125. Hydatid disease is identified by

a. Schick test b. Dick test c. Casoni test d. Freis test

126. Prausnitz kustner reaction is generated by

a. IgA b. IgE c. IgG d. IgD

127. Immunoglobin which are found in asthma at elevated level:

a. IgA b. IgE c. IgM d. IgD

128. What is the similarity between IgM &IgG?

a. A compliment fixation b. Placental transport c. Heat stability at 56oC d. Sedimentation coefficient

129. What is the technique for quantitativeestimation of immunoglobulin?

a. Single diffusion in one dimension b. Single diffusion in two dimension c. Double diffusion in one dimension d. Double diffusion in two dimension

130. Cell mediated immunity can be identified by

a. Sheep bred blood corpuscles roasette formation b. Microphase inhibiting factor c. Skin test for delayed hyper sensitivity d. All of these

131. Out of the following which are the examples of autoimmune disease?

a. Acquired Haemolytic anaemia b.Rheumatoid arthritis c. Hashiomoto disease d. All of these

132. Which of the following is a true statementregarding Purified Protine Derivative (PPD) used in tuberculin test?

a. Prepared from tubercle bacilli b.It is inferior to old tuberculin c.Consists of filtrate of glycerol broth d. None of these

133. Which of the following are inactive viralvaccines?

a. Influenzae b. Rabies c. Russian spring summer encephalitis d. All of these

134. Antigenic variation is most extensive in

a. Influenza virus b. Small pox virus c. Measles virus

d. Herpes virus

135. Which is the correct statement related to hepatitis B virus?

a. Paramyxo virus b. Orthomyxo virus c. Reo viruses d. Retro viruses

ANSWERS

1. c 2. a 3. c 4. b 5. b 6. A7. a 8. a 9. c 10. b 11. a 12. B13. b 14. c 15. a 16. c 17. c 18. B19. a 20. a 21. a 22. a 23. a 24. D25. b 26. a 27. b 28. a 29. a 30. A31. b 32. d 33. a 34. a 35. d 36. B37. c 38. d 39. d 40. b 41. c 42. C43. a 44. d 45. b 46. b 47. c 48. C49. a 50. c 51. b 52. d 53. c 54. A55. c 56. b 57. b 58. b 59. b 60. A61. d 62. c 63. a 64. a 65. b 66. C67. a 68. a 69. c 70. a 71. a 72. B73. c 74. d 75. c 76. d 77. b 78. D79. a 80. a 81. a 82. a 83. c 84. A85. c 86. b 87. a 88. d 89. c 90. C91. a 92. a 93. c 94. b 95. a 96. C97. c 98. d 99. 1.d, 2.c, 3.b, 4.a 100. 1.b, 2.a, 3.d, 4.c101. 1.b, 2.c, 3.a, 4.e 102. 1.c, 2.a, 3.b, 4.e103. c 104. a 105. d 106. a 107. e 108. C109. a 110. b 111. d 112. c 113. b 114. E115. a 116. b 117. e 118. d 119. e 120. C121. a 122. a 123. e 124. b 125. c 126. B127. b 128. a 129. b 130. d 131. d 132. A133. d 134. a 135. c

1. Food poisoning is caused by

a. Clostridum tetani b. Clostridum Welchi c. Diptheria d. Clostridium botulinum

2. Koplic's spots will develop in

a. HIV b. Measles c. Mumps d. Rubella

3. Viral DNA is resistant to DNA of the host cell because it contains

a. 5'-HMC b. 5'-HMA c. 5'-CHM d. 5'MHC

4. Which of the following is an example of live vaccine?

a. pertussis b. mumps c. cholera d. rabies

5. Triple toxoid vaccine gives protection against

a. Diphtheria, tetanus and rabies b. Tetanus, whooping cough, Tuberculosis c. Whooping cough, tetanus and Diphtheria d. Whooping cough, cancer and T.B.

6. Higher dose of chloramphenicol affectsthe eukaryotic cells because

a. They have 30 S ribosomes b. They have mitochondria c. They have 70 S ribosomes d. None of the above

7. AIDS is caused by

a. Retrovirus b. Prion c. Rhabdovirus d. Retroprison

8. Penicillin is a

a. Primary metabolite b. Secondary metabolite c. Tertiary metabolite d. None of the above

9. The rejection of an organ transplant suchas a kidney transplant, is an example of_____ Hypersensitivity.

a. Immediate b. Delayed c. Allergy d. None of these

10. Listeriosis was _____ disease.

a. Food borne b. Water borne c. Milk borne d. Air borne

11. Pus-forming forms are called as

a. Pyoderm b. Pyogenic c. Pyrogen d. None of the above

12. In Elisa technique, the antibodies arelabeled by

a. Acridine orange b. Alkaline phosphate c. Neutral red
d. Bromothymol blue

13. _____ is a genetic disease charachterizedby a total or partial inability to synthesizeglobulins.

a. Apitososis b. Agamma globulinemia c. Gammaglobulinemma
d. Sickle-cell anemia.

14. A study involving analysis of risk for genetic defects in a family is

a. Genetic Engineering b. Genetic counseling c. Genetic drift
d. Genetic equilibrium

15. Viral antigens are likely

a. Proteins b. Glyco proteins c. Lipo proteins d. Both a and b

16. The suitable assay method for antibiotics is

a.Enzymatic assay b.Turbidometric assay c.End point determination assay
d.Metabolic assay

17. ELISA test is used for the identification of

a. Janudice b. AIDS c. Cancer d. Diabetis

18. Incubation period for infective Hepatitis disease

a. 45 – 80 days b. 15 – 35 days c. 35 – 50 days d. 5 – 15 days

19. All of the following are bacteriostatic chemotherapeutic agents except

a. Bacitracin b. Chloramphenicol c. Novobiocin
d. Tetracycline

20. Kinetosomes are observed in

a. Algae b. Fungi c. Protozoa d. Viruses

21. Beta-lactum ring is present in

a. Erythromycin b. Penicillin c. Tetracyclins
d. Chromphenical

22. Antibiotic produced from Streptomyces orientalis is

a. Streptomycin b. Penicillin c. Vancomycin d. Both a and b

23. The drug of choice for dermal, oral andvaginal candidiasis is

a. Griseofulvin b. Amphoterein B c. Gentian violet
d. Nystatin

24. Botulism means

a. Food adultration b. Food poisioning by streptococcus bacteria
c. Chemical contamination of food d. Food processing

25. Chloramphenicol is obtained from

a. Streptomyces griseus b. Streptomyces venezuelae c. Streptomyces pyrogenes d. None of these

26. Streptomycin is obtained from

a. Streptococcus species b. Streptomyces griseus c. Straphylococcus aureus
d. None of these

27. The treatment required for small bodies of water is

a. Disinfection b. Filtration c. Purification d. All of these

28. Surface ropiness is caused by

a. Alkaligenes viscolactis b. Streptococcus c. both a and b d. None of these

29. Septicaemia is

a. Bacteria in blood b. Toxin in blood c. Pus in blood d. Multiplication of bacteria and toxins in blood

30. In AIDS, Kaposis sarcoma may respond to

a. Interleukin – 2 infusion b. Azathioprine c. Alpha interferon d. None of these

31. Ciprofloxacin acts by inhibiting

a. Cellwall synthesis b. RNA synthesis c. Folate synthesis d. DNA gyrase

32. Lyme disease is caused by

a. Bacteria b. Fungi c. Spirochaete d. Virus

33. Toxic shock syndrome is caused by

a. Staph. albus b. Staph. Aureus c. Strep. viridana d. None of these

34. Black water fever is caused by

a. P. vivax b. P. falciparum c. P. ovale d. None of these

35. Mantoux test detects

a. M. tuberculosis b. Cynaobacteria c. Clostridia d. Both a and b

36. The antibiotic acting on cell wall is

a. Bactracin b . Penicillin c. Cyclosporine d. All of these

37. Aflatoxin is produced by

a. Aspergillus sps b. Penicillium sps c. Alternaria sps d. None of these

38. Penicillin is discovered by

a. Fleming b. Pasteur c. Koch d. None of these

39. Antibiotics used in combination may demonstrate

a. Synergism b. Antaginism c. both d. None of these

40. The drug of choice in anaphylactic shockis

a. Histamine b. Corticosteroid c. Epinephrine d. None of these

41. Drugs of choice for treatment of Mycoplasma infections:

a. Tetracyclines b. Erythromycin c. a and b d. Penicillins

42. A number of viruses are known to infect mycoplasmas, called

a. Bacteriophages b. Mycoplasma phages c. Virions d. Tiny strains

43. The following are true about Rickettsiae.

a. Unicellular organisms b. Prokaryotic intracellular parasites c. Presence of 70 S ribosomes d. It causes hemolysis in human beings e. Gram negative plemorphic rods

44. The causative agent of scrub typhus:

a. R.Quintana b. R.rickettsii c. R.orinetalis d. R.prowazekii

45. Lymphogranuloma venerum (LGV) is asexually transmitted disease is caused by
a. Copthalmia b. C.trachomatis c. C.pneumonias d. C.psittasi
46. Intradermal test employed for diagnosis of LGV is
a. Frei test b. Mantoux test c. Schick test d. Dick test
47. Which algae is pathogenic to human?
a. Cephaloeuros b. Ulothrix c. Macrocystis d. Prototheca
48. Erythromycin is obtained from
a. S.griseus b. S.rimosus c. S.scabies d. S.erythraeus
49. Common cold is caused by
a. Adeno virus b. Corono virus c. Hepatitis virus d. Pox virus
50. The causative agent of conjunctivitis:
a. Adeno virus b. Corono virus c. Paramyxo virus d. None of these
51. Antibiotics used for treatment of cholera are
a. Tetracyclines b. Penicillins c. Streptomycines d. None of these
52. Salmonella typhi is causative organism of
a. Undulent fever b. Remittent fever c. Dengue fever d. Enteric fever
53. Which of the following Salmonella paratyphi is the commonest in India?
a. A b. B c. C d. None of these
54. In enteric fever, the organ lodging maximum number of the organism is
a. Liver b. Gall bladder c. Small intestine d. Large intestine
55. True about Enteric fever is
a. Bacteraemia in first week b.Carrier in 90% c.All serotypes cause the disease d.Rosy spots on 18th day
56. Gastroenteritis is caused by
a. Shigella b. V.cholerae c. V.cholera Parahaenolyticus
d. S.typhi
57. E.coli produces the following toxins:
a. Enterotoxins b. Endotoxins c. Verocytotoxins d. Hemolysins
58. The following infections caused by Esch.Coli, except
a. Urinary tract infections b.Septic infections of wounds c.Diarrhoea d.Dysentery e.Meningitis
59. Diphtheria is caused by
a. Corynebacterium diphtheria b. C. Bovis c. C. Jeikeium d. C. equi
60. Causative organism of diphtheria was first demonstrated by
a. Robert Koch b. Lois Pasteur c. Klebs and Loeffler d. Volhard and Fahr
61. Coryne bacterium is
a. Gram positive b. Resistant to Penicillin c. Gram negative d. Resistant to Chloramphenicol
62. C. diphtheriae consists of
a. Startch granules b. Polymeta phosphate granules c. Lipid granules d. None of these
63. The incubation period of diphtheriae is
a. Upto 2 weeks b. Upto 1 week c. 2–4 weeks d. None of these
64. Diphtheria virulence test is

a. Ascoli's thermoprecipitation test b. Eleck's gel precipitation test c. C.R.P test d. M.R.T. test

65. Diptheria toxoid is prepared by using

a. Aldehyde b. Formali c. Phenols d. None of these

66. Diphtheria is an example of

a. Bacteraemia b. Pyaemia c. Septicemia d. Toxaemia

67. Main symptom of tuberculosis is

a. Tubercle formation b. Liquid formation c. Both a and b d. None of these

68. BCG vaccine is for the prevention of

a. Brucellosis b. Diphtheria c. Botulism d. Tuberculosis

69. Dose of BCG vaccine is

a. 0.2–0.5 ml b. 0.1 ml c. 0.05 ml d. 0.2 to 0.3 ml

70. Negative Mantoux test is important in

a. Pulmonary Koch's syndrome b. Sarcoidosis c. Carcinoma bronchus d. Lymphoma

71. Bacilli Calmette Guerin (BCG) contains the avirulent strains of

a. Human tubercle bacilli b. Avian tubercle bacilli c. Bovine tubercle bacilli d. A typical mycobacteria

72. Drugs used against tuberculosis (TB) are

a. Refampicin, Isoniazid b. Pyrazinamide, Streptomycin c. Both a and b d. None of these

73. The greatest number of tubercle bacilli is present in

a. Large sized tuberculomas b. Miliary tuberculosis c. Tuberculous lymphadenitis d. Tuberculous cavity of the lung

74. Histoid Hansen is a veriety of

a. Tuberculoid Leprosy b. Borderline tuberculoid c. Borderline lepramatous d. Lepronmetous leprosy

75. Streptococcus pyogens produces all of the following lesions, except

a. Impetigo contagiosa b. Erysipeals c. Boil d. Paronchia

76. Causative agent of Scarlet fever:

a. Staphylococcus aureus b. Streptococcus viridans c. Stre. Pyogens d. None of these

77. Rheumatic fever is most commonly caused by

a. Str. viridans b. Str. Pyogenes c. Stph. Aures d. None of these

78. Penicillin is the drug of choice for

a. Scarlet fever b. Whooping cough c. Brucellosis d. Cholera

79. In human being str. pneumoniae causes

a. Septicaemia b. Paronychia c. Pneumomnia d. None of these

80. Virulence factor for Stre. pneumoniae:

a. Capsular polysaccharide b. Specific soluble substance c. Vi-antigen d. Forsmann antigen

81. Conjunctivitis in a new born is caused by

a. Streptococcus b. Pneumococcus c. Meningococci d. None of these

82. Influenza is belonging to

a. Orthomyxoviridae b. Retroviridae c. Both a and b d. None of these

83. Influenza virus contains

a. Eight segments of RNA b. Two strands of RNA c. Single RNA d. None of these

84. 'Reye's syndrome' is caused by

a. St.pneumoniae b. St.pyogenes c. Influenza d. None of these

85. Geraman measles is also known as

a. Rubella / 2-day measles b. Rubella / 3day measles c. Rubella / 4-day measles d. Rubella / 1-day measles

86. The commonest cause of rubella in new bornes

a. Congential rubella b. Post natal rubella c. Expanded rubella syndrome (ERS) d. Both a and c

87. Mumps virus is belonging go

a. Retroviriae b. Paramyxoviriae c. Orthomyxo viridae d. None of these

88. Measles is characterized by

a. Negribodies b. Babes-Ernest granules c. Koplik's spots d. Fever

89. Brucella causes

a. Pertusis b. Plague c. Brucellosis d. None of these

90. Mediterranian fever is caused by

a. M. tuberculosis b. S. typhi c. C.neoformans d. Brucella

91. Which of the following test is specific for Brucellosis?

a. Frei b. Weil c. Castaneda strip d. Rose water

92. Malignant pustule is caused by

a. Anthrax b. Tetanus c. Diphtheria d. None of these

93. The commonest form of anthrax in man is

a. Alimentary b. Cutaneous c. Pulmonary d. Hepatic

94. The animals most frequently infected with anthrax are

a. Sheep b. Cattle c. Goats d. All of these

95. Virus causing Rabies is

a. Orthromyxo virus b. Paramyxo virus c. Rhbdo virus d. Toga viruses

96. Rhabdo viruses are belonging to the family:

a. Rhabdo viridae b. Toga viridae c. Paramyxo viridae d. None of these

97. Rabies Virus isolated from natural humanor animal infection is termed as

a. Street virus b. Fixed virus c. Both a and b d. None of these

98. Rabies virus can multiply in

a. The central nervous system only b. The peripheral nerves c. Muscle tissues d. All the above

99. Neurological complications following rabies vaccines is common with

a. Chick embryo vaccine b. HDCS vaccine c. Semple vaccine d. BPL vaccine

100. Which anti rabic vaccine has been recommended by WHO as the most effective?

a. Duck embryo vaccine b. HDCS vaccine c. Sheep brain vaccine d. BPL vaccine

101. The causative agent of tetanus is

a. Clostridium botulinum b. Cl. Tetani c. Cl. Welchii d. Cl. perfringens

102. The mode of spread of tetanus neurotoxin from blood to brain is

a. Via lymphaties b. Arterial blood c. Cranial nerves d. None of these

103. Tetanus is caused by spread of

a. Exotoxin in sympathetic system b. Exotoxin in para sympathetic system
c. Endotoxin in sympathetic system d. Endotoxin in parasympathetic system

104. The first symptom of tetanus is

a. Lock jaw b. Trismus c. Anorexia d. Dyspagia

105. Of which clostridia, the neurotoxin is most powerful?

a. Cl. tetani b. Cl. Welchii c. Cl. botulism d. Cl. septicum

106. Toxin produced by C. botulism is

a. Botulin b. Tetanospasmin c. Tetanolysin d. Cholaragen

107. "Toxic shock syndrome" is caused by the toxin of

a. Staphylococcus aureus b. Streptococcus pyoge c. Vibrio cholera
d. Candida

108. Causative agent of syphilis

a. T. pallidum b. T. pertenue c. T. carateum d. T. endemicum

109. Spirochaelis are sensitive to

a. Penicillin b. Chloramphenicols c. Erythromycin d. Tetracyclins

110. Specific test for syphilis is

a. VDRL test b. ELISA c. FTA d. None of these

111. VDRL test is a

a. Agglutination test b. Slide flocculation test c. Precipitation test d. None of these

112. The following characters are true about Neisseria gonorrhoeae except

a. Gram-negative, aerobic bacteria b. Non-motile diplococcic c. Oxidase positive organisms d. Air borne infection

113. Gonorrhoea is

a. Air borne disease b. Water borne disease c. Sexually transmitted venereal disease d. Both a and c

114. Bartholin cyst is caused by

a. Candida b. Streptococcus c. Staphylococcus d. Gonococcus

115. Neisseria gonorrhoeae causes

a. Urethritis b. Conjuctivitis c. Arthritis d. All of the above

116. Virulence in gonococcus is due to

a. Pili b. Cell membrane c. Its cellular location d. Cyclic enzymes

117. Japanese encephalitis is caused by

a. Toga Viruses b. Arbo Viruses c. Para myxo Viruses d. Ortho myxo Viruses

118. In India, Japanese b encephalitis was first isolated from the mosquitoes of the

a. Culex tritaeriorhynchus b. Culex annulirostris c. Culex vishnui d. None of these

119. Dengue virus is transmitted from man to man by the

a. Sand fly b. Ticks c. Aedes aegypti d. Culex

120. Yellow fever is caused by

a. Bunya virus b. Calci virus c. Arbo virus d. None of these

121. Vector for leishmaniasis is

a. Tick b. Mite c. Sand fly d. Tsetse fly

122. Splenomegaly is an important manifestation of

a. Kala-agar b. Typhoid c. Malaria d. All of these

123. Which of the following is most severly affected in Kala-azar?

a. Liver b. Spleen c. Adrenal gland d. Bone marrow

124. In India, malaria most often spreads by

a. Anophels cucifacies b. Anopheles fluvatis c. Anopheles stephensi d. Anopheles minimus

125. Man is intermediate host for

a. Guinea Worm b. Filaria c. Malaria d. Kala-azar

126. Which of the following preferably infects reticulocytes?

a. P. ovale b. P.vivax c. P.falciparum d. P.malaria

127. In which type of material parasite in the exoerythrocytic stage absent?

a. P.ovale b. P.vivax c. P.falciparum d. P. malariae

128. In falciparum malaria, all of the following stages are seen except

a. Ring stage b. Schizont c. Gametocyte d. None of these

129. Sporozite vaccine in malaria has

a. Induces antibodies b. Prevents only asexual forms with reproduction c. No effects on clinical illness d. None of the above

130. Growing trophozoites and schizonts are not seen in the peripheral blood in malaria due to

a. P. falciparum b. P.vivax c. P.ovale d. P. malaria

131. Thin blood smear for malaria is used to identify

a. Plasmodium b. Gametocytes c. Type of parasite d. Schizont

132. The radical teatment of malaria is to half

a. Gametocyte b. Exo-erythrocytic phase c. Erythrocytic phase d. All of these

133. Symptoms of acute aflatoxicosis

a. Osteogenic sarcoma b. Lymphatic leukemia c. Malaise & Anorexia d. Both a and b

134. Most important Penicillium toxins are

a. Citrinin b. Patulin c. Penicillic acid d. All of the above

135. Penicillic acid is produced by

a. A. ochraceus b. P. puberulum c. Both a and b d. None of the above

136. Fungi producting mycelium are called

a. Moulds b. Filamentous fungi c. Both a and b d. Yeasts

137. Candidiasis is caused by

a. Candida albicans b. Aspergillus spp. c. E. floccosum d. M. audouinii

138. Candida albicans is capable to form

a. Single cells b. Pseudomonas c. Multicellular forms d. None of these

139. Aspergillus fumigatus can infect

a. A. niger b. A. fumigatus c. A. flavus d. A. oryzae

140. A.fumigates can produce

a. Endotoxins b. Exotoxins c. Enterotoxins d. None of these

141. The drug of choice for dermal, oral and vaginal candidiasis is

a. Griseofulvin b. Amphotericin B c. Gentian violet d. Nystatin

142. The following Penicillium species are pathogenic except

a. P. commune b. P. bicolor c. P. glaucum d. P.notatum

143. Tinea versicolor is caused by

a. Candida albicans b. Malassezia furfur c. Aspergillus niger d. None of these

144. Causative agent of Tinea nigra

a. Malassezia furfur b. Exophiala werenekii c. Candida albicans d. Aspergillus flavus

145. Causative agent of African histoplasmosis

a. Histoplasma capsulatum b. Histoplasma duboissi c. Aspergillus niger d. Aspergillus flavus

146. Sun ray fungus is

a. Actinomyces irraeli b. Chromoblastomycosis c. Streptomyces griseus d. Cryptococcosis

147. Which agent on addition to a colony inhibits its growth and on removal the colony regrows is?

a. Bacteriostatic b. Bactericidal c. Antibiotic d. Antiseptic

148. Griseofluvin is obtained from

a. Penicillium notatum b. Streptomyces griseus c. Penicillium griseofluvin d. None of these

149.B -lactum ring is present in

a. Erythromycin b. Penicillin c. Tetracyclins d. Chloramphenicol

150. All of the following drugs act on cell membrane, except

a. Novobiocin b. Nystatin c. Chloromycetin d. Colicins

151. **Cycloserine related to the amino acid in structure**

a. Serine b. Aspergine c. Alanine d. None of these

152. In Tuberculosis therapy mainly used antibiotic is

a. Penicillin b. Streptomycin c. Chloramphenol d. Cycloserine

153. The antibacterial action of penicillin is due to its effect on

a. Cell membrane permeability b. Cell wall synthesis c. DNA synthesis d. Protein synthesis

154. The antibiotic produced from Bacillus subtilis is

a. Vancomycin b. Bactiracin c. Both a and b d. None of these

155. bacitracin sensitivity test is done for

a. Pneunocci b. Group 'A' Streptococci c. Gonococci d. Staphylococci

156. The effect of antibiotics on micro organisms is mainly due to

a. Inhibition of cell-wall synthesis b. Damage to the cytoplasmic membrane c. Inhibition of nucleic acid and protein synthesis d. All of the above

157. The antibiotic acting on cell wall is

a. Penicillin b. Bacitracin c. Cyclosporin d. All of the above

158. Erythromycin belongs to chemical class of antibiotics

a. â-lactose b. Tetracyclines c. Macrolides d. Aminoglycosides

159. Bacterial resistance to antibiotics is transmitted by

a. Transduction b. Transformation c. Mutation d. Plasmids

160. Erythromycin inhibits protein synthesis by

a. Attaching to 30 S ribosome unit b. Attaching to 50 S unit or ribosome c. By the attachment to t-RNA d. By the attachment to m-RNA

161. The function of (THFA) Tetrahydrofolic acid coenzyme include

a. Amino acid synthesis b. Thymidine synthesis c. Protein synthesis d. Both a and b

162. Resistant to drugs in tuberculosis develops by

a. Transduction b. Transformation c. Conjugation d. Mutation

163. Which of the following is penicillinase resisting acid labile penicillin?

a. Amoxycillin b. Cloxacillin c. Carbenicillin d. Methicillin

164. Which of the following does not inhibit cell wall synthesis?

a. Penicillin b. Carbenicillin c. Amikacin d. Vancomycin

165. The anti tumor antibiotics act by inhibiting

a. Cell wall synthesis b. RNA synthesis c. Cell membrane synthesis d. The DNA structure & function

166. Drug resistance to sulphonamides is due to

a. Production of PABA b. Folic acid synthetase c. Drug alteration d. Low affinity for drug synthesis by bacteria

167. Amoxycillin is combined with clavulanic acid to inhibit

a. DNA gyrace b. Cell synthesis c. Protein synthesis d. Beta-lactamase enzymes

168. Drug of choice for methicillin resistant staph. Aureus is

a. Ampicillin b. Erythromycin c. Neomycin d. Vancomycin

169. Nalidixic acid activity is due to

a. The inhibition of DNA synthesis b. Inhibition of protein synthesis c. The inhibition of cell wall synthesis d. Both b and c

170. The best test for the susceptibility of a microorganism to antibiotics and other chemotherapeutic agents:

a. Tube-dilution test b. Paper-disc test c. Both a and b d. None of these

171. The smallest amount of chemotherapeutic agents required to inhibit the growth of the organism in Vitro is known as

a. MIC (minimum inhibitory concentration) b. Thermal death point (TDP)
c. Death rate d. None of these

172. Clear-zones formation around antibiotic disc is due to

a. Growth of the bacterium surrounding of the disc b. Lysis of the bacterial cells surrounding the disc c. The destruction of paper disc (antibiotic) d. None of these

173. Bacitracin is obtained from

a. B. subtilis b. B. anthracis c. B. cereus d. B. anthracoid

174. Vancomycin is obtained from

a. Streptococcus species b. Aspergillus niger c. Streptomyces orientalis d. Bacillus anthracis

175. â-lactum antibiotics are

a. Penicillin b. Cephalosporin c. Both a & b d. None of these

176. Following are the test organisms used for the I.P microbiological assay of antibiotics match them correctly:

1. Rifampicin	A. Escherichia Coli
2. Tetracyclin	B. Klebsiella pneumonia
3. Streptomycin	C. Micrococcus luteus
4. Chloramphenol	D. Bacillus subtilis

E. Bacillus cereus

177.The drugs mentioned below are produced by the species mentioned from Ato E. Match them correctly:

1. Rifampicin
2. Nystatin
3. Amphotericin B
4. Candicidin

A. Streptomyces griseus
B. Bacillus polymyxa
C. Streptomyces mediterranei
D. Streptomyces nodosus
E. Streptomyces noursei

178. Match the correct method of sterilization listed A to E for the following drugs :

1. Tetracyclin injection
2. Insulin injection
3. Quinine injection
4. Morphine injection

A. Sterilised by dry heat
B. Sterilised by heating with a bacteria
C. Sterilised by aseptic method
D. Prepared by aseptic method
E. Sterilised by heating in an autoclave

179. Match the following rickettsial disease with their respective organisms:

1. Epidemic typhus
2. Scrub typhus
3. Trench typhus
4. Murine typhus

A. Rickettsia rickettsi
B. Rickettsia prowazeki
C. Rickettsia typhus
D. Rickettsia Quintana
E. Rickettsia typhus

180. Match the following antimicrobial with their respective side effects A to E:

1. Acridines
2. Benzalkonium preservative in eye drops
3. Parahydroxy
4. Formalin

A. Showed adverse effects on proteins
B. exhibit synergism and chloride unsuitable for
C. Haemolytic benzoates
D. Very toxic
E. Toxic to leucocytes and retard granulation process

181. Match the following antibiotics with their respective modes of administration A to E:

1. Penicillin V
2. Benzathine
3. Methicillin sodium
4. Ampicillin

A. Intramuscular suspension
B. Oral penicillin
C. Both as oral and injection
D. Locally applied
E. Intramuscular injection

182. Match the following antibiotics with respective strains A to E used for their production :

1. Tetracyclin
2. Chloramphenicol
3. Erythromycin
4. Cycloserine

A. Streptomyces erythreus
B. Streptomyces garyphalous
C. Streptomyces niveus
D. Streptomyces viridifaciens
E. Streptomyces venezuelae

183. Match the following strains with their respective produced antibiotics A to E:

1. Streptomyces
2. Streptomyces
3. Streptomyces
4. Streptomyces

A. Oxytetracycline griseus.
B. Neomycin sulphate aureofaciens
C. Viomycin rimosus
D. Chlortetracycline griseus var.
E. Streptomycin purpurea

184. Match the following antibiotics with their respective disease A to E to be cured :

1. Streptomycin
2. Cycloserine
3. Novobiocin
4. Griseofulvin

A. Staphylococcus infections
B. Tuberculosis
C. Fungal tuberculosis
D. Pulmonary tuberculosis
E. Anti-spirochaetes

185. Match the following antibiotics with their respective side effects A to E:

1. Novobiocin
2. Neomycin
3. Cycloserine
4. Chloramphenol

A. Damages 8th cramial nerve
B. Damages CNS
C. Damages haemopoietic system
D. Skin rashes
E. Kidney problems

186. Match the following antibiotics with their modes of action A to E:

1. Tetracyclines
2. Erythromycin
3. Novobiocin
4. Griseofulvin

A. Form an irreversible complex with sterols
B. Chelation of light divalent salts
C. Blocks protein synthesis
D. Interferes with the conjugation of bilirubin
E. Influences mitosis

187. Match the following dosage forms with their respective antibiotics A to E:

1. Tablets
2. Intravenous injection
3. Capsules
4. Intramuscular

A. Vancomycin Hcl
B. Colistin
C. Polymixin B sulphate
D. Gentamycin injection
E. Paromomycin sulphate

188. Match the following side effects with their respective antibiotics A to E:

1. Nephrotoxic
2. Rashes
3. Hypersensitivity
4. Gastric irritation

A. Triacetyloleandomycin
B. Polymixin B sulphate
C. Cephaloridine
D. Gentamycin
E. Sodium fusidate

189. Match the following antibiotics with their respective activity spectra A to E:

1. Bacitracin
2. Gentamycin
3. Sodium fusidate
4. Framycetin

A. Gram negative
B. Mainly staphylococci
C. Mainly Ps. Aeruginosa
D. Gram positive

190. Match the following enzymes with their activities A to E:

1. Hyaluronidase
2. Collagenase
3. Lecithinase
4. Leucocidins

A. Inactivate leucocytes and aid bacterial invasion
B. Reversibly catalyzes the breakdown of a major component
C. Disintegrates a constituent of muscle, cartilage and bone
D. Haemolysis of erythrocytes and the necrosis of other cells
E. Clots plasma and surrounds the bacteria

191. Match the following aggresins with their respective modes of action from A to E:

1. Hyaluronidase
2. Haemolysis
3. Streptokinase
4. Lecithinase

A. Destroys RBC's and other tissues
B. Breaks down connective tissues, increases permeability of tissue space
C. Causes lysis of RBC's and other tissues
D. Digest the fibrin of blood

E. Dissolves collagen

192. Match the following terms with their respective effects A to E:

1. Brucella melitensis A. Causes trachoma, conjunctivitis and nongonococcal gamets
2. Flavobacterium B. Causes influenza like fever species
3. Chlamydia C. Causes Malta fever in goats trachomatis
4. Leptospira D. Contaminates pharmaceutical icterohaemorrhagiae products

E. Weil's disease (jaundice)

193. Virus causing mumps is also responsible for

a. Measles b. Hepatitis A c. Rabies d. Variola

194. Epidemic pleurodynia and myocarditis of new born infants are both caused by

a. Group B cox sack virus b. Reovirus c. Polyomavirus d. Cytomegalovirus

195. Human papillomavirus causes following tumors:

a. Hepatic carcinoma b. Cervical cancer c. Condyloma acuminatum d. Plantar wart

196. Viral infection is caused due to

a. Acute self limited illness b. No apparent symptoms c. Chronic infection with persistent viral shedding d. All of these

197. Viruses which do not carry enzymes for DNA synthesis as a part of their virion are

a. Hepatitis B virus b. Poxyviruses c. Heepes simplex virus d. Retroviruses e. All of these

198. Following virus is known to establish latent infections:

a. Adeno virus b. Varicella-zoster virus c. Cytomegalovirus d. Hepes simplex virus e. All of these

199. Viruses which have teratogenic property are

a. Herpes simplex virus b. Cytomegalovirus c. Rubella virus d. All of these

200. Kawasaki syndrome is

a. Most prevalent in Japan and Hawaii b. Patients show rickettsia like bacteria in skin biopsies c. Strain involved may be propionibacterium d. All of these

201. Mode of action of quinolone antibiotics on growing bacteria was thought to be

a. Inhibition of â lactamase b. Prevention of the cross linking of glycine c. Inhibition of DNA gyrase d. Inhibition of reverse transcriptase

202. The role that human play in the plague life cycle is

a. Secondary reservoir b. Primary transmission vector c. Primary host d. Accidental intruder in rat flea cycle e. None of these

203. Patient with presence of penile chancre should be advised by physician –

a. To take rest at home b. To swab the chancre and culture on Thayer- Martin agar c. To Gram stain the chancre fluid d. To repeat VDRL test in 10 hours e. Perform dark field microscopy for treponemes

204. Which organism is responsible for causing fever to a man dealing with goats?

a. Trepanema Pallidum b.M.tuberculosis c.Clostridium novyl d.Brucella melitensis e.None of these

205. Diphtheria toxins are produced from the strains of C.diphtheriae, which are

a. Encapsulated b. Sucrose fermenters c. Of the mitis and strain
d. Glucose fermentors e. Lysogenic for □□prophase

206. Skin of the healthy person has normal microbial flora which includes
a. Enterobacteriaceae b. Aerobic diphtheria bacilli c. Anaerobic diphtheriae bacilli d. Nonhemolytic staphylococci e. All of these
207. Which of the following organisms can infect humans if improperly cooked meat is used?
a. Trichinella spiralis b. Taenia saginata c. Taenia solium d. Diphyllobothrium latum e. Both a and c
208. The parasite related to ancylostoma duodenale is
a. Wuchereria bancrofti b. Necatur americanes c. Loa loa
d. Trichinella spiralis
209. Which of the following amoeba does not live in large intestine ?
a. Entamoeba coli b. Entamoeda histolytica c. Endolimax nana
d. Entamoeba gingivalis
210. Which of the following is not related to congenital syphilis?
a. Aneurysm b. Saddle nose c. Still birth d. Hutchiso's teeth
211. Streptococcus pyogens produce infection –
a. Streptococcal sore throat b. Acute glomerulo nephritis c. Rheumatic fever
d. None of these
212. Salmonella which can cause prolong septicaemia.
a. Salmonella anetum b. Salmonella cholerasuis c. Salmonella typhimurium
d. Salmonella entritidis
213. E.coli produce which type of toxins?
a. Exotoxins b. Endotoxins c. Leucocidin d. Both a and b
214. Main causative organism of gas gangreneis
a. B.anthrax b. Clostridium tetani c. Cl.deficile d. Cl.perfringens
215. Causative organism of whooping cough is
a. Bordetella pertussis b. Bordetella parapertussis c. Bordetella bronchi septica
d. None of these
216. Pfeiffer phenomenon is related to
a. Vibrio cholerae b. B.anthrax c. Rickettsial pox d. All of these
217. Diagnostic test for the identification of primary syphilis:
a. VDRL test b. Treponema pallidum immobilization test c. Kahn's test
d. Dark ground microscopic examination
218. Sporadic summer diarrhea may be caused by
a. E.coli b. Enterobacter c. Hafnia d. Serratia
219. Biological false reaction in VDRL is related to
a. Lepra bacilli b. Corynebacterium diphtheria c. Cl.welchi d. None of these

ANSWERS
1. d 2. b 3. a 4. c 5. c 6. B 7. a 8. b 9. a 10. a 11. b 12. B 13. b 14. d 15. d 16. c 17. b 18. D 19. a 20. c 21. b 22. c 23. d 24. C 25. b 26. a 27. d 28. d 29. d 30. C 31. d 32. c 33. b 34. b 35. a 36. D 37. a 38. a 39. c 40. c 41. c 42. B 43. d 44. c 45. b 46. a 47. d 48. D 49. b 50. a

51. a 52. d 53. a 54. B 55. a 56. c 57. b 58. e 59. a 60. C 61. a 62. b 63. c 64. b 65. b 66. D 67. a 68. D 69. b 70. a 71. c 72. C 73. d 74. d 75. d 76. c 77. b 78. A 79. c 80. a 81. a 82. a 83. b 84. C 85. b 86. d 87. b 88. b 89. c 90. D 91. c 92. a 93. b 94. d 95. c 96. A 97. a 98. d 99. c 100. b 101. b 102. C 103. a 104. b 105. c 106. a 107. a 108. A 109. b 110. a 111. b 112. d 113. c 114. D 115. d 116. a 117. b 118. C 119. c 120. C 121. c 122. d 123. b 124. a 125. c 126. B 127. c 128. b 129. a 130. a 131. c 132. C 133. d 134. d 135. c 136. a 137. a 138. B 139. d 140. a 141. c 142. d 143. b 144. B 145. b 146. a 147. a 148. c 149. b 150. D 151. c 152. d 153. b 154. b 155. b 156. D 157. d 158. c 159. d 160. b 161. d 162. D 163. d 164. c 165. d 166. b 167. d 168. D 169. a 170. c 171. a 172. b 173. a 174. C 175. c 176. 1.d, 2.e, 3.a, 4.a 177. 1.c, 2.e, 3.d, 4.b 178. 1.d, 2.c, 3.e, 4.b 179. 1.b, 2.c, 3.d, 4.e 180.1.e,2.c,3.b,4.a 181. 1.b, 2.a, 3.e, 4.c 182. 1.d, 2.e, 3.a, 4.b 183. 1.e, 2.d, 3.a, 4.c 184.1.b,2.d,3.a,4.c 185. 1.d, 2.e, 3.b, 4.c 186. 1.b, 2.c, 3.d, 4.e 187. 1.b, 2.a, 3.e, 4.c 188.1.b,2.c,3.a,4.e 189. 1.e, 2.a, 3.d, 4.b 90. 1.b, 2.c, 3.d, 4.a 191. 1.b, 2.a, 3.d, 4.c 192.1.c,2.b,3.a,4.e 193. a 194. a 195. a 196. d 197. e 198. E 199. d 200. d 201. c 202. d 203. e 204. D 205. e 206. e 207. e 208. b 209. d 210. A 211. a 212. b 213. d 214. d 215. a 216. A 217. d 218. a 219. a

1. The best medium for the production of Penicillin is

a. Nutrient agar b. Corn steep liquor c. Sulfite waste liquor d. Whey

2. Industrially important Antibiotic producing organisms shall be isolated by

a.Disk plate method b.Direct plate method c.Serial dilution method d.Crowded plate method

3. Industrial alchohol will be produced by using starter culture

a. Top yeast b. Middle yeast c. Bottom yeast d. Feeder yeast

4. Pyruvate decarboxylase acetaldehyde + CO2=This reaction is specially observed in

a. Lactic acid fermentors b. Ethanol fermentors c. Algae d. Plants

5. The pyruvate, dehydrogenase →□multienzyme complex does not occur in

a. Aerobic bacteria b. Microphilic bacteria c.Facultative anaerobic bacteria d.Strictly anaerobic bacteria

6. A major ingradient of penicillin production media is

a. Corn meal b. Corn steep liquor c. Cane steep liquor d. None of these

7. the outstanding example of traditional microbial fermentation product is

a. Vinegar b. Penicillin c. Citric acid d. Tetracyclin

8. Which of the following involves the formation of nitrate from ammonia

a. Ammonification b. Denitrification c. Nitrification d. Nitrogen fixation

9. First genetically engineered and biotechnologically produced vaccine was against

a. AIDS b. Small pox c. Herpes simplex d. Hepatitis B.

10. one of the standard cloning vector widely used in gene cloning is

a. Ti pasmid b. EMBL 3 c. pBR 322 d. EMBL 4

11. In alchoholic fermentation, CO2 is evolved during

a.Decarboxylatin of pyruvic acid **b**.Formation of acetaldehyde

c.Oxidation of acetaldehyde **d**.Both a and b

12. In the industrial production of streptomycin, the secondary metabolite or byproducts is

a. Vitamin – B12 b. Vitamin – C c. Vitamin – B6 d. Ethanol

13. Tobacco and tea leaves are fermented to give flavour and taste. This type of fermentation is known as

a. Alcohol fermentation b. Curing c. Degradation d. Lactic acid fermentation

14. Vinegar fermentation involves

a. Yeasts only b.Yeasts with lactic bacteria c.Yeasts with acetic acid bacteria d.Yeasts with butric acid bacteria

15. Carcinoma refers to

a. Malignant tumours of the connective tissue b. Malignant tumors of the skin or mucous membrane c. Malignant tumours of the colon d. Malignant tumors of the connective tissue

16. By-product of acetone-butanol fermentation include

a. Riboflavin b. Penicillin c. Isopropanol d. All of these

17. Transgenic animals are for improvement of the quality of

a. Milk b. Meat c. Eggs d. All of the above

18. Thermo resistant bacteria are important in the preservation of foods by

a. Freezing b. Canning c. Chemicals d. Irradiation

19. The fungus used in the industrial production of citric acid:

a. Rhizopus Oryzac b. Fusarium moniliformae c. Rhizopus nigricans d. Aspergillus nigricans

20. Penicilin is commercially produced by

a. P.notatum b. P.chrysogenum c. P.citrinum d. P.roquefortii

21. The most commonly used microorganism in alchohol fermentation is

a. A spergilus niger b. Bacillus subtilis c. Sacharomyces cerevisiae d. Escherichia coli

22. Vitamin B12 can be estimated and determined by using organism

a. Lactobacillus sps b. Lactobacillus Leichmanni c. Bacillus subtilis d. E.Coli

23. Batch fermentation is also called

a. Closed system b. Open system c. Fed-Batch system d. Sub-merger system

24. To differentiate lactose and non-lactosefermentors the medium used is

a. Mac Conkey's medium b. Stuart's medium c. Sugar medium d. Citrate medium

25. The micro-organism useful for fermentation are

a. Bacteria b. Yeast c. Fungi d. None of these

26. Industrial microbiology, mainly depends on the phenomenon

a. Pasteurisation b. Fermentation c. Vaccination d. Both b and c

27. Streptokinase is also termed as

a. Fibrionolysin b. Catalase c. Coagulase d. Hyaluronidase

28. Streptokinase is produced by

a. Staphylococcus aureus b. Streptococcus pneumoniae
c. Str. Faecalis d. Str. pyogenes

29. Large vessel containing all the parts and condition necessary for the growth of desired microorganisms is called
a. Bio reactor b. Auto reactor c. Impeller d. None of these
30. Basic principle in industrial microbiology is
a. Suitable growth conditions b. Fermentation c. Providing aseptic conditions d. All of these
31. For thorough mixing of medium of medium and inoculum the part of fermentor useful is
a. Shaft b. Headspace c. Impeller d. Sparger
32. Infermentor the top portion left without broth is called
a. Shaft b. Head space c. Impeller d. Sparger
33. Over heating of fermentator during fermentation is controlled by
a. Cooling jacket b. Steam c. Cool air d. None of these
34. Antifoam agent is
a. Silicon compounds b. Corn oil c. Soyabean oil d. All of these
35. The capacity of laboratory fermentors is
a. 12–15 liters b. 2000 gallons c. 500 liters d. 10000 gallons
36. For the production of ethanol the raw material used is
a. Molasses b. Cellulose c. Sulphite waste liquor d. None of these
37. Different methods of strain improvement are
a. Protoplast fusion b. Recombinant DNA technique c. Genetic recombination d. All of these
38. Protoplasts can be prepared from
a. Gram positive bacteria b. Gram negative bacteria c. Both a & b d. None of these
39. Upto the production of desirable production in the fermentor is called
a. Upstream process b. Downstream process c. Surface fermentation d. None of these
40. The purification and recovery of the production after fermentation is called
a. Upstream process b. Downstream process c. Surface fermentation d. None of these
41. If the microorganisms are allowed to nutrient medium is called
a. Submerged fermentation b. Surface fermentation c. Dual fermentation d. All of these
42. Submerged fermentations are
a. Batch fermentation b. Continuous fermentation c. Both a and b d. None of these
43. Batch fermentation is also called
a. Closed system b. Open system c. Fed-batch system d. None of these
44. If more than one microorganism is used to obtain the required product, that type of fermentation is called
a. Batch b. Continuous c. Dual d. Fed-batch
45. L. lysine is produced from

a.Coryne bacterium glutamicum b.Corynebacterium sps. c.Mycobacterium sp d.None of these

46. Methods used to get immobilized enzymes:

a. Adsorption b. Encapsulation c. Covalent bonding d. All of these

47. Raw-material used for the production of alcohol is

a. Molasses b. Starch c. Sulphite waste water d. All of these

48. Microorganisms used for alcohol production

a. Saccharomyces sereviceae b. Bacillus subtilis c. Penicillium chrysogenum d. None of these

49. For streptomycin production the microorganisms required are

a. Streptomyces griseus b. Streptomyces niger c. Saccharomyces cereviceae d. All of these

50. The by-product during streptomycin production is

a. Vitamin A b. Proline c. Vitamin B12 d. None of these

51. For acetic acid production the methods followed are

a. Orleans process b. Rapid process c. Submerged process d. All of these

52. For amylase production the micro organism required is

a. B. subtilis b. S. cereviceae c. A. nigar d. None of these

53. Pectinase is industrially produced from

a. S.cereviceae b. Trichoderma Koningi c. A. nigar d. None of these

54. Cellulose are produced from

a. S.cereviceae b. Trichoderma Koningi c. A. nigar d. None of these

55. Industrial Production of Vitamin-B12 is from

a. Propionibacterium sps. b. Pseudomonas sps. c. Both a and b d. None of these

56. Clostridium acetobutylicum is used for the production of

a. Acetone - Butanol b. Ethanol c. Vitamin-B12 d. None of these

57. In the production of ethanol industrially the yeast used is

a. K.pneumoniae b. Kluyreromyces fragilis c. S. cerevisiae d. Both b and c

58. Citric acid is used as

a. Flavouring agent in food b. As an antioxidant c. As preservative d. All of the above

59. Citric acid is produced in aerobic conditions by the fungi

a. Aspergillus b. Penicillin c. Mucor d. All of these

60. The raw material for citric acid production is

a. Corn b. Molasses c. Starch d. None of these

61. Aspergillus niger is used generally for the production of

a. Ethanol b. Penicillin c. Citric acid d. Lactic acid

62. In the citric acid production, the pH to be maintained in the fernmenter is

a. 7.0 b. 5.0 to 6.0 c. 8.0 to 9.0 d. 1.0 to 6.0

63. The required temperature for the production of citric acid is

a. 10oC – 80oC b.30oC – 50oC c. 20oC – 50oC d.25oC – 30oC

64. The penicillin produced in large scale submerged fermentations are

a. Penicillin-A b. Penicillin-D c. Penicillin-G d. None of these

65. The strain of fungi used for the large scale production of penicillin is

a. Penicillium chrysogenum b. P-notatum c. Streptomyces Aurecus d. Saccharomyces sps

66. amino penicillic acid is prepared from penicillin sps by

a. Acylase b. Punicillin acylase c. Penicillinone d. None of these

67. The pH, to be maintained for the production of penicillin is

a. 7.5 b. 6.5 c. 8.0 d. 5.0

ANSWERS

1. b 2. d 3. c 4. b 5. b 6. B 7. a 8. c 9. b 10. c 11. d 12. A 13. b 14. c 15. d 16. a 17. d 18. b19. d 20. b 21. a 22. b 23. a 24. a25. b 26. b 27. a 28. d 29. a 30. b31. c 32. b 33. a 34. d 35. a 36. c37. d 38. b 39. b 40. b 41. b 42. c
43. a 44. c 45. a 46. d 47. d 48. a49. a 50. c 51. d 52. a 53. c 54. b55. c 56. b 57. d 58. d 59. d 60. a61. c 62. b 63. d 64. c 65. a 66. b67. b

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