Afr. J. Infect. Diseases
www.africanethnomedicines.net

Review Paper

ISSN: 2006-0165©2008

INSIGHT INTO TROPICAL HUMAN INFECTIOUS DISEASES: AN UPDATE

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Abstract

Knowledge on infectious diseases encompasses a vast and constantly changing arena, and consistent research work is imperative to understand and combat the new problems resulting from emerging infectious diseases. Public health workers and epidemiologists aim at lowering morbidity and mortality due to diseases by preventing infections. For the rapidly expanding majority of the world's population, who live in the largely tropical areas of Africa, Asia and Latin America, the greatest threats to health remain tropical infectious diseases. Emerging infectious diseases are new, emerging or drug-resistant infections, whose incidence in humans may increase in the near future. With rapidly increasing international travel, the globalization and industrialization of food supply and exploding populations, infectious diseases pose unprecedented threats around the globe. Thus the imperative need is to promptly recognize, isolate and appropriately manage tropical infectious diseases. This review paper has attempted to provide the much needed insight into the different aspects of the major tropical infectious diseases affecting humans throughout the world.

Introduction

Infectious diseases

The impact of infectious diseases on humans includes acute or chronic illness of individuals, widespread effects on infected populations and effects on nutrition and development (Yekutiel, 1980). Infectious diseases affect individuals and populations in epidemic or endemic patterns of occurrences. An infectious disease results when the pathogen invades a host, starts multiplying and interferes with the normal life functions of the host. Normally the immune system of the host responds to the pathogen and destroys it. But sometimes, the pathogen circumvents the immune system, causing serious disease or death.

The distribution and incidence of tropical infectious diseases is related to geography, evolution, climate and human factors (Patz et al., 1995). In olden days, infections were the main means of human "population control" worldwide, often killing enormous number of people in epidemic outbreaks, as was the case with diseases such as bubonic plague and typhoid fever. Even in the present age, infections tend to cause more deaths during war and famine than do actual injuries and starvation. Fortunately, many infectious diseases can now be treated by means of antibiotics and other drugs and by a variety of preventative methods. Almost all infections contracted by humans pass from other humans or animals. Some infections originate from outside the body like rabies, hepatitis, dysentery etc. Others are endogenous (caused by factors within the organism) infections which occur when the host's resistance is lowered, either by malnutrition, illness or immune depression. (Lederberg et al., 1992).

One of the main purposes of epidemiology is the study of how the infectious agent is maintained in nature so that adequate measures can be taken to control its infection. The pattern of transmission of particular diseases can

be suspected on the basis of age-specific incidence, geographic and seasonal patterns, and other demographic characteristics. Diseases limited to a certain geographic area and season suggest the presence of a vector in the life-cycle that determines transmission in that particular region (Morse, 1995). Horizontal transmission refers to spread of infection from individual to individual in a given population. In contrast, vertical transmission refers to spread of infectious agents from parent to offspring. Globally, as assessed in terms of disability-adjusted life years (DALYs), which measures morbidity and mortality, infectious diseases in 1990 accounted for 36.4% of total DALYs (Murray and Lopez, 1996). For the major infectious diseases of the tropics, improvements in sanitation, living conditions and general public health would be critical in helping to control the impact of the diverse infectious agents that currently contribute to human morbidity and mortality.

What is a parasite?

A parasite is an organism that depends on another organism, known as a host, for food and shelter. A parasite usually gains all the benefits of this relationship. In contrast, the host may suffer from various diseases, and discomforts as a result of the parasitic infection. The life cycle of a typical parasite commonly includes several developmental stages during which it may undergo two or more changes in body structure as it lives and moves through the environment and one or more hosts.

The various infectious diseases may be caused by infections due to bacteria, viruses, chlamydia, rickettsia, trematodes, cestodes, nematodes and arthropods. The brief outline about each of these infectious agents is discussed below:

INFECTIONS CAUSED BY BACTERIA

Enteric Escherichia coli Infections (Diarrhea)

Diarrhoeal diseases in developed and developing countries are significant causes of morbidity and mortality and are caused by a variety of microbial pathogens. It is estimated that 3.3 to 6 million children die annually from diarrhoeal illnesses the world over (Guerrant et al., 1990). Enterovirulent *Escherichia coli* constitute one of the most common causes of diarrhoeal illness in tropical regions and developing countries particularly where sanitation facilities are limited. It causes diarrhoea, hemorrhagic colitis, thrombocytic purpura and sometimes death. Fever, severe abdominal cramps and malaise are common. Replacement of lost fluid and electrolytes is the cornerstone of therapy. This is best accomplished with the oral rehydration formulation recommended by the WHO (Hirschhorn and Greenough, 1991; Blake et al., 1993). Besides anti diarrheal drugs and proper sanitation and hygiene are the control measures advised to diminish risk of fecal-oral transmission.

Typhoid fever

Typhoid fever is an acute systemic illness in humans caused by infection with bacteria *Salmonella typhi.* It is characterized by prolonged fever and bacterial invasion of and multiplication within the phagocytic cells of the liver, spleen, lymph nodes and Peyer's patches. Paratyphoid fever is a pathologically and clinically similar but generally milder illness that is caused by the serotypes of *Salmonella*. Enteric fevers refer to either typhoid or paratyphoid fever. The stool and urine of carriers and those with or recovering from acute infections are the sources of the organism and is acquired by ingestion of contaminated food or water (Rowland, 1961). Epidemics occur mainly from endemic areas, where sanitation conditions are poor. Usual clinical manifestations are fever, abdominal pain, myalgia, headache, diarrhoea etc. Clinical course of antibacterial reduces complications. Widal's test is the standard serological test for typhoid fever. Control measures include adequate nutrition, fluid and electrolyte balance, sanitation measures, good nursing care and vaccination. The standard *S. typhi* parenteral vaccine offers partial protection to people from endemic and non-endemic areas.

Nontyphoidal Salmonellosis

In humans this infection is associated with food products and is the most frequent etiologic agent of food-borne disease outbreaks. It causes diarrhoea among travellers and among young children of developing countries. In 1994, an estimated 224,000 cases of *S. intrepidities* gastroenteritis developed among persons in the U.S. who ate a nationally distributed ice-cream product (Hennessy et al., 1996). Infection with nontyphoidal *Salmonella* most often results in nausea, vomiting, diarrhoea, fever. HIV-infected persons have an estimated 20 to 100 fold increased risk of salmonellosis compared with the general population (Levine et al., 1991). Main therapy is replacement of fluid and electrolyte losses.

Shigellosis

Shigellosis, a disease associated with poverty, poor hygiene and crowded living conditions remains a common cause of diarrhoea, dysentery, and death in poor countries and is a contributor to childhood malnutrition (Cohen et al., 1991). Unlike *E. coli* and *Salmonella* strains, *Shigella* does not produce flagella and is non-motile. Transmission is from person to person by means of contact spread via the focal-oral route. Symptoms are malaise, fever, diarrhoea, dysentery, anorexia. It affects nutrition in children. Antimicrobial therapy is the cornerstone of treatment for shigellosis. Oral rehydration is also useful. Personal hygiene sanitation practices are to be followed. Prevention is by immunization.

Campylobacter infections

Campylobacters are bacteria that usually produce an acute gastrointestinal illness. In developing countries of tropical areas, this disease is an important cause of morbidity and mortality in young children (Murray et al., 1995). It causes bacteremia, diarrhea, meningitis, endocarditis and abcesses in immunocompromised patients (Bokkenheuser, 1970; Nachamkin, 1995). The Gold standard for detection of Campylobacter infection is culturing the organism from stool, blood or other site of infection. Antimicrobial treatment of infection, safe disposal of sewage and purification of water supplies are fundamental to the control of this infectious disease.

Cholera

It is a diarrhoeal illness caused by *Vibrio cholerae* which in its severe form, *cholera gravis*, can rapidly and fatally dehydrate adults as well as children. Diarrhoeal illnesses constitute one of the leading infectious causes of death and disability worldwide. It is estimated that 3.3 to 6 million children die annually from diarrhoeal illnesses, the vast majority in Africa, Asia and Latin America (Bern et al., 1992). Since cholera is spread by contaminated water and food, its transmission is facilitated wherever populations are not served by treated water supplies and sanitation. The most common route is by food vehicle caused by raw or undercooked seafood. During outbreaks or seasonal epidemics, cholera spreads via multiple modes of transmission. Transmission is via food or water vehicles and humans are the only known hosts of cholera. Symptoms are nausea, diarrhoea, vomiting and dehydration. Diagnosis is by stool culture, biochemical tests, serological tests and PCR techniques. There are three pillars to the therapy of patients with cholera: aggressive rehydration therapy, (fluid therapy, by replacement of water and electrolyte deficits, intravenous rehydration, oral rehydration) administration of antibiotics, treatment of complications and safe water and food. There are currently three licensed cholera vaccines available in various countries around the world (Levine and Kaper, 1995; Esrey, 1996). *Vibrio parahemolyticus* is the most commonly isolated non-cholera *Vibrio*, associated with gastroenteritis outbreaks in Japan in 1950. In addition to *V. cholerae* and *V. parahaemolyticus* nine other *Vibrio* species infecting humans have also been recognized causing diarrhoea and wound infections. All cases are linked to consumption of raw or undercooked seafoods causing gastroenteritis, abdominal pain, nausea and vomiting. Volume replacement and antimicrobial therapy is the most important element of therapy. *Clostridium difficile* has recently emerged as the most important pathogen causing antibiotic associated diarrhoea and colitis. It is acquired nosocomially in the context of concurrent antimicrobial therapy. *C. perfringens* has been the cause of both sporadic and epidemic cases of necrotizing enteritis. *C .botulinum* is the cause of food-borne botulism (Bleck, 1995).

Helicobacter pylori infections

The gram-negative bacteria *Helicobacter pylori* is highly adapted for persistent colonization of the human stomach. Approximately one-half of the global human population is colonized by this organism. *H. pylori* colonization of the stomach is associated with an increased risk of gastric adenocarcinoma, peptic ulcer disease and gastric lymphoma (Marshall and Warren, 1984; Cover, 2006., Blaser and Atherton, 2004). It is associated with inflammation of the gastro-duodenal mucosa, peptic ulceration and gastric cancer. It is identified by simple microbiological or serological techniques or endoscopy.

Meningococcal infections

This infection causing meningitis continues to pose an important public health threat in the tropics because

mortality is high and serious neurological and other sequelae are frequent. Although focal outbreaks and epidemics occur in more developed areas, the incidence of epidemic meningococcal meningitis and meningococcemia exhibits high geographic variability (Reido et al., 1983). *Neisseria meningitides* is exclusive to humans. The human nasopharynx is the natural reservoir for meningococci; transmission is facilitated by airborne droplets or close personal contact.

Haemophilus Infections

Haemophilus influenzae is among the most common causes of bacterial respiratory infection and meningitis in infants through out the world. It is part of the normal human nasopharyngeal flora and spreads by airborne or direct contact routes from person to person. *H. aegypticus* is responsible for epidemic acute purulent conjunctivitis. Antibiotics are used for treatment (WHO, 1998).

Pneumonia

It is now the leading infectious cause of mortality and morbidity among children in developing countries (Garenne et al., 1992). Most fatal pneumonia in humans is caused by *Haemophilus influenzae* or *Streptococcus pneumoniae*. It is also a leading bacterial pathogen in the cause of meningitis and sinusitis throughout the world including tropical countries (Musher, 1991). Penicillin is the drug of choice but resistance to this drug has been reported. Hence combination therapies are adopted. Pneumococcal infections are diagnosed by culture from clinical samples such as CSF, blood, pleural fluid, or purulent sputum and PCR diagnosis.

Streptococcal and Staphylococcal Infections

Group a streptococcus (GAS) is a global pathogen that causes a wide variety of clinical infections and post infectious sequelae (Stevens, 1996). *Staphylo pyogenes* causes multiple clinical syndromes. The common ones are pharyngitis, sinusitis, otitis media, pneumonia, erysipelas, scarlet fever, impetigo, cellulites, etc. Fever is the common sign. Common *Staphlococcus aureus* infections are pyomyositis, impetigo, abcesses, carbuncles, cellulites. The normal reservoir is human skin and antibiotic treatment is imperative.

Pertussis or whooping cough

It continues to be a major cause of morbidity and mortality in children especially in areas, with limited access to immunization and support facilities. It is estimated that in 1990, 60 million cases of this illness were responsible for as many as 340,000 deaths per year world wide (Muller et al., 1986). It causes respiratory and systemic infection in humans. The disease results from a localized infection of the respiratory tract that has its primary manifestation as paroxysmal cough. Early treatment is the key to successful intervention. Pertussis vaccines are but effective in preventing the disease or reducing severity of infection with the causative agent, *Bordetella pertussis*.

Diptheria

It *i*s caused by infection with *Corynebacterium diptheriae*. Prior to the introduction of effective childhood vaccination programs, diphtheria was a major cause of childhood mortality. Symptoms are sore throat, anorexia, malaise, fever, pharyngitis, and respiratory obstruction. The mainstay of treatment is diphtheria antitoxin. Vaccination of infants with diphtheria toxoid in combination with tetanus toxoid and whole-cell pertussis vaccines (DTP) in many countries has resulted in dramatic reductions in diphtheria incidence globally (Efstratiou and Maple, 1994).

Tuberculosis (TB)

It is a chronic necrotizing granulomatous disease caused by the acid-fast organism *Mycobacterium tuberculosis*. It is spread by inhalation of aerosolized infectious droplet nuclei from patients with active pulmonary TB and the lung are the main portal of entry. The most common manifestation of TB in humans is pulmonary disease, but nearly all organ systems can be involved. TB occurs worldwide but the preponderance of mortality and morbidity occurs in developing countries. Miliary and meningeal TB in children and TB occurring in human

immunodeficiency virus (HIV) - coinfected persons are important causes of death in areas of high prevalence. TB is a global health problem. The World Health Organization (WHO) currently estimates that one third of the world's population has been infected with *M .tuberculosis*. With more than 8 million cases and 2 million deaths annually TB is a major cause of morbidity and mortality worldwide (Corbett et al., 2003). The approach to TB control advocated by the WHO is DOTS (directly observed therapy, short course), which focuses on the treatment of sputum smear-positive pulmonary TB with standardized short course chemotherapy under proper case management conditions (WHO, 2002a). Primary tuberculosis is usually acquired by the inhalation of droplet nuclei containing viable tubercle bacilli which are phagocytosed by alveolar macrophages to establish a primary nidus of infection. Persons who do not develop effective CMI responses following primary TB infection may develop progressive primary tuberculosis. Paediatric TB occurs in children from repetitive close contact with an infectious typically sputum smear positive adult. Persons who are mildly immunocompromised may present with typical reactivation TB with cough, sputum production, malaise, weight loss and a radiographic picture of apical fibrocavitary disease. The coexistence of cancer and TB has been recognized. Extrapulmonary TB manifests as tuberculosis pleurisy, lymphadenitis, bone and joint TB, military TB, meningitis, renal and genital TB, pericarditis, abdominal TB etc. (Martin and Blaser, 2006).

Drug and Drug resistance in TB

Soon after introduction of effective drugs for TB treatment, failures due to drug resistance were noted when patients were treated with single agents. Drug resistance develops from random chromosomal mutations which occur spontaneously in wild type strains of *M. tuberculosis* at low rates. The term multi-drug resistant (MDR) refers to isolates resistant to at least isoniazid and rifampicin (Huong et al., 2006). Modern TB treatment is based on the use of potent short course, multiple drug regimens which sterilize the sputum rapidly. Primary preventive measures include BCG (Bacille Calmette-Guerin is a living attenuated strain of *M. bovis*) vaccination and interruption of transmission, case finding and treatment. BCG is a component of the United Nations Expanded Programme on Immunizations (UNEPI) and is the world's most widely used vaccine. It is usually given as a single intradermal injection shortly after birth (Fine,1995).The mycobacterium (*Mycobacterium tuberculosis)* which causes TB are spread in air-borne droplets and the nature of the disease is such that a person may be infected for many weeks before the symptoms become apparent. And when they do and treatment is sought, it may not always be effective. If left untreated, one person with active TB will infect 10-15 people in a year's time. Many of the strains of mycobacteria that cause TB are now resistant to the three most commonly used anti-TB drugs; rifampicin, streptomycin and isoniazid. This means the individuals affected continue to suffer and also continue to pass on the drug resistant strains of mycobacteria to other people. The sooner such strains are detected, the quicker the individuals can be properly treated and fewer people become infected (WHO, 2004a, 2005; Mitchison, 2004).

DNA fingerprinting technique uses the distinctive DNA pattern or 'fingerprint' of each strain of the tuberculosis mycobacteria and has benefits in the control of multi-drug resistant TB. PCR-based dot blot hybridization uses DNA extracted from the pathogen to be identified and then amplifies it using the polymerase chain reaction. The amplified DNA is directly"dotted" onto a nylon membrane. Next, radioactively labelled DNA probes, specific to the pathogen's DNA, are added. The membrane is then exposed to X- ray film. Spots on the X - ray film will appear only when the pathogen has first been bound to the nylon membrane and the radioactively labelled probe has then been bound to the pathogen.

Approximately 778,000 patients with multidrug resistant (MDR-TB) would be treated in accordance with WHO recommendations over the next decade (WHO, 2006c). From a public health perspective, DOTS (Directly Observed Therapy, Short Course) the internationally recommended standardized management strategy for TB, alone may suffice in some settings (De Riemer et al., 2005). Both malaria and TB are becoming more resistant to the drugs that are currently available for treatment and drug resistant strains are posing a global threat. The International Atomic Energy Agency (IAEA) is responding by sponsoring a program to build technical competency in molecular and radioisotope-based techniques.

Leprosy

Leprosy is a chronic infection caused by *Mycobacterium leprae* affecting the cooler body areas: skin, upper respiratory passages, anterior segments of eyes, superficial segments of peripheral nerves etc. The traditional pervasive social stigma attached to leprosy may be traced to the disfigurement, deformity, mutilations, and blindness related to damage to these tissues. The term Hansen's disease is a frequent synonym for leprosy. Leprosy and its transmission do not require a tropical environment which is the reason that it has afflicted people in every part of the

world. Today the higher prevalence in the tropics is best attributable to socioeconomic factors. In 1995 the World Health Organization (WHO) estimated that there were 1.83 million people in the world with active leprosy (Noordeen, 1996). Direct skin to skin contact or formites may transmit leprosy but the naso-respiratory route is believed to be the most common (De Wit et al., 1993). The type of antibacterial multidrug therapeutic (MDT) regimen employed is usually based on the WHO simplified classification of disease, that is Paucibacillary Regimen (PB) or Multibacillary Regimen (MB) and utilizes the WHO recommended combination of drugs (WHO, 1982a).

Anthrax

Bacillus anthracis is the causative agent. The four clinical forms of human anthrax-cutaneous, oral-oropharyngeal, gastrointestinal and inhalational are determined by the portal entry of the etiologic bacterium. The preceding pain, ring of vesicles, black eschar, absence of pain and pus and presence of oedema surrounding the cutaneous lesion are diagnostically suggestive. Enzyme immunoassays are useful for diagnosis and antibiotics and supportive care is necessary (Christie, 1974).

Brucellosis

This is an occupational disease in abattoir workers, butchers, veterinarians and farmers who become infected through the skin and conjunctiva. *Brucella* species that infect humans are all considered to be biovars of *B. melitensis* (causing Malta fever). Outbreaks have been reported from common sources often from contaminated edible products (Corbel, 1997). Because it is a zoonotic disease, the best control measure is prevention with pasteurization of dairy products and safety and quarantine measures by veterinarians and farmers.

Plague

It is a severe febrile illness caused by the bacillus *Yersinia pestis* and causes catastrophic epidemics (Perry and Fetherston, 1997; WER, 2004). It is an incidental zoonotic infection of humans and other mammals. Humans acquire infection by bite of rodent fleas (*Xenopsylla cheopis* and *X. braziliensis*). The principal clinical forms of plague are bubonic, septicemic and pneumonic. The latter two are secondary complications of the former. Although antibiotics can successfully treat plague, the fatality rate is high when treatment is delayed > 24 h after the onset of symptoms (Smego et al., 1999). Epidemics can be prevented using standard public health measures. Plague is one of the three quarantinable diseases (plague, cholera, yellow fever) subject to the WHO International Health Regulations (1969), Geneva, WHO 1983. The global means to control plague are both ancient (taboos, quarantine, and isolation) and modern (vaccinations, mathematical modelling and molecular epidemiology) (Blaser, 2006).

Tetanus

In the tropical and developing countries tetanus and particularly neonatal tetanus continues to be a significant cause of morbidity and mortality. In 1975 it was estimated that about 1 million cases of the disease occurred annually in the world (Edsall, 1975). *Clostridium tetani* is the bacillus responsible for this disease and transmission is by spores and bacteria proliferate when the redox potential of the bacillus is low. Tetanus toxin causes all the clinical manifestations of tetanus. It has four clinical subtypes namely; generalized, localized, cephalic and neonatal paralysis of muscles of the face, jaw and neck resulting in complications by affecting the neuromuscular junction and CNS. Blood, serum and CSF studies are the routine test procedures. Active immunization with tetanus toxoid is one of the most effective preventive measures in medicine.

III. SPIROCHAETAL INFECTIONS

Treponemal infection

This disease of humans has a worldwide distribution and it includes endemic treponematoses which include yaws (*T. palladium*) and endemic syphilis. It is transmissible by direct contact.

Relapsing fever and other Borrelia infections

Relapsing fever is an arthropod -borne zoonosis and occurs in two major forms: epidemic and endemic. The epidemic form is transmitted by lice and endemic relapsing fever by ticks. In this century alone, at least a

million deaths are attributable to relapsing fever (Bryceson et al., 1970). Lyme disease, another tick-borne zoonosis caused by a *Borrelia* sp., which occurs in temperate to sub arctic regions. The agents of relapsing fever and Lyme disease are spirochaetes which is a separate phylum of bacteria (Holt, 1978).The vector of epidemic relapsing fever is the body louse, *Pediculus humanus corporis*, which feeds on humans. Common predisposing factors for epidemic are famine, war and the movements and congregations of refugees affecting millions of people. Infection begins with contact with a tick or louse bearing spirochaetes. The clinical hallmarks are fever, rigors, hyperpyrexia headache, arthralgia, myalgia etc. Smear detection, immunoassays and PCR helps in detection and antibiotics are useful for treatment. Insecticide spraying would reduce tick population.

Leptospirosis

It is a zoonotic disease caused by pathogenic spirochaetes of the genus *Leptospira*. Human infection can occur with infected animals or through direct contact with infected animals or through indirect contact with contaminated urine of infected animals (Faine, 1982). Epidemiologic studies indicate that infection is commonly associated with certain occupations such as farming, veterinary practices etc. Mainly it causes febrile illness but at times leads to jaundice called Weil's syndrome. The most commonly used immunologic method is the microscopic agglutination test (MAT) for detecting specific antileptospiral antibodies (Carter and Ryan, 1975). Antibiotics are used for treatment. Strategies to prevent and control this disease focus on reducing direct contact with infected animals and contaminated water or soil (MMWR, 1997). Prevention of indirect exposure is to wear rubber boots and protective clothing. The major problem with vaccination of humans is that immunity is serovar -specific and it is very difficult to select all the potentially important serovars in the vaccines (Torten et al., 1973).

IV. CHLAMYDIAL INFECTIONS

Trachoma

Trachoma is a chronic conjunctivitis which progresses to scarring of the conjunctiva, painful inturned eyelashes and corneal scarring with blindness. Blinding trachoma is still highly endemic in many developing countries, where it affects 600 million people and at least 6 million are blind from it (Thylefors et al., 1995). Blinding trachoma in developing countries is commonly associated with infections by *Chlamydia trachomatis* serovars A, B, or C. Transmission is by direct or indirect contact with infected materials and by flies. The intracellular bacterium *Chlamydia trachomatis* remains the most prevalent cause of sexually transmitted bacterial infections in humans with an estimated 90 million new infections occurring globally every year (WHO, 2001). In women, this infection can cause the onset of pelvic inflammatory disease and tubal factor infertility (Peripert, 2003). It can be cured by antibiotic therapy. Emphasis should be on personal and environmental hygiene.

V. RICKETTSIAL AND EHRLICHIAL INFECTIONS

Rickettsia species of the spotted fever group (SFG) cause human diseases in tropical countries. The primary vectors are ticks and mites. Fever, headache and myalgias are the common features of SFG rickettsial infections. The major target for all SFG rickettsiae is the endothelial cell. Antibiotics are preferred. Avoidance of tick bite is the only preventive method available.

Typhus group Rickettsioses

Murine typhus is among the most prevalent febrile illness occurring in the tropics. Louse borne typhus is not only the most devastating infections in world history but a continous cause of morbidity and mortality among isolated impoverished populations in tropical locations. Like spotted fever group, rickettsiae typhus group rickettsiae are small, obligate, intracellular, gram-negative bacteria that have an arthropod host as part of their ecological niche. Typhus group rickettsiae that are pathogenic for humans have an insect vector (louse or flea) and are transmitted mainly in the insect's faeces (Winkler, 1990). Immunodiagnostic techniques and DNA tests help to confirm the disease as the symptoms are difficult to recognize from other infections. Control relies on reducing the vector density and vaccines to some typhoid fevers are effective.

Scrub typhus

It is a chigger (larval mite) borne zoonosis which is of greatest public health importance in tropical Asia.

The etiological agent *Orientia tsutsugamushi* (formerly Rickettsia) is maintained by highly efficient transovarial transmission in trombiculid mites and is transmitted to humans by the chigger during feeding (Traub and Wisseman, 1974). Cough, tacypnea and infiltrates on chest radiography are among the most frequent presentations of scrub typhus. Laboratory diagnosis can be made by ELISA, PCR assays and other serological techniques. Scrub typhus responds to antibiotics.

Q fever

Q fever is caused by *Coxiella burnetti* and is found worldwide. It is a zoonotic disease. Symptoms are fever, severe headache, retroorbital pain, cough and aseptic meningitis.

VI. FUNGAL INFECTIONS

Dermatophytes are a closely related homogenous class of keratinophilic filamentous fungi that are associated with the stratum corneum of the skin, as well as hair and nails of the living host. These fungi are classified in the genera, Epidermophyton, Keratinomyces, Microsporum and Trichopyton. Clinical manifestations are inflammation and lesions. The fungus colonizes the stratum corneum and then grows in a radial manner without penetrating viable tissue. *Tinea capitis* is a worldwide disease of children in which dermatophytes cause scaling of the stratum corneum with or without inflammation and invasion of scalp hair follicles which subsequently results in alopecia**.** *Tinea corporis* is colonization of the glabrous skin by dermatophytes. *Tinea cruris* is an infection involving the groin, perineum and perianal regions that is prevalent in people living in tropical regions where high humidity contributes to skin maceration. *Tinea pedis* is an infection of feet caused by dermatophytes. *Tinea unguium* occurs when a dermatophyte invades the nail unit and causes toe nail infections (Weitzman and Summerbell, 1995). Topical antifungal drugs are the most commonly recommended treatment for most dermatophytes. Prevention measures are good hygiene measures as these are easily transported from one person to another especially if fomites are shared. Para coccidioidomycosis is a systemic mycotic infection caused by the dimorphic fungus *Paracoccidioides brasiliensis.* Candidiasis is caused by *Candida albicans* and causes septicaemia. Cutaneous candidiasis causes lesions of the skin and treatment is commonly by antifungal therapy.

VII. DISEASES CAUSED BY VIRUS

Despite the availability of an ffective preventive measure (live measles vaccine), measles continues to cause more deaths than any other single infectious agent. The WHO estimates that in 1997 there were over 36 million measles cases worldwide and nearly a million Measles-related deaths, more deaths than the other vaccine - preventable diseases combined. Globally it accounts for over 10% of deaths among children fewer than 5 years of age and is especially lethal in infants under 1 year of age. Children may die from acute measles infection or from its sequelae, including encephalitis, pneumonia, diarrhoea, and malnutrition. The disease thrives in cities especially in deprived urban areas where crowding, poor sanitation, and low measles vaccination coverage ensure the ongoing circulation of the virus (measles is caused by an RNA virus of the genus *Morbilli* virus of the Paramyxoviridae family). It is transmitted from person to person via respiratory droplets or airborne spray to mucous membranes in the upper respiratory tract or the conjunctiva (Morley, 1974). Humans are only reservoir for the virus. It can be diagnosed by Koolik's spots on buccal mucosa in over 80% of cases shortly before rashes appear and it is pathognomonic of measles infection. Measles virus isolation remains the gold standard for the laboratory confirmation of a suspected measles case and serological tests are also done in laboratories. Other than supportive care, including fluids (such as oral rehydration therapy) antipyretics, nutritional therapy, there is no specific treatment for uncomplicated measles infection. However antibiotics may be indicated for treatment of secondary bacterial infections. Immunization with live measles vaccine has been demonstrated to be protective for over 20 years and immunity is thought to be for life. By 1990, the estimated overall global coverage for children by 2 years of age was approximately 80% (WHO, 1998).

Infections due to Herpes Simplex Virus, Varicella -Zoster Virus, Cytomegalovirus, Epstein - Barr virus

There are currently nine herpes viruses known to afflict humans and in eight cases the human is the only known host. The human herpes viruses are distributed throughout the world with no significant geographic variation. In many developing areas including the tropics children appear to acquire most herpes virus infections with the

exception of VZV (varicella), at an earlier age than in developed countries, presumably due to greater transmissibility and exposure from overcrowding and poor general hygiene (Tyring, 1997). Symptomatic primary infections with the alpha herpes viruses usually present as muco-cutaneous vesicular eruptions.

Table 1. WHO classification of AIDS-defining conditions

CD4 cell count< 200/ul
Candidiasis, pulmonary or oesophageal
Cervical cancer
Coccidioidomycosis
Cryptosporidiosis
Cytomegalovirus infection
Herpes esophagitis
HIV encephalopathy
Histoplasmosis
Isosporiasis
Kaposi's sarcoma
Lymphoma
Mycobacterial disease
Pneumocystis carinii infection
Pneumonia, bacterial
Progressive multifocal leukoencephalopathy (polyomavirus)
Salmonellosis

Cancer

Cervical cancer and precancer are caused by persistent infection with human papillomavirus (HPV) (Schiffman et al., 2005).

Smallpox

Smallpox although now eradicated poses a potential threat to all countries should strains of the virus be accidentally released or deliberately disseminated as an act of biological terrorism. Small- pox was once, by far, the most serious of all infectious diseases, killing 25% to 30% of unvaccinated persons who became infected. Under the aegis of the WHO, eradication was achieved during a 10-year program culminating in the occurrence of the last case in 1977 (Fenner et al., 1988). Smallpox was as infectious as chicken pox but less infectious than measles. Natural

infection occurs following implantation of the virus on the oropharyngeal or respiratory mucosa. Virions multiply in spleen, bone marrow and lymph nodes. Except for lesions in the skin, mucous membranes and lymphoid hyperplasia other organs are seldom involved. Secondary bacterial infection is not common and death when it occurs probably results from the toxaemia associated with circulating immune complexes and soluble variola antigens. Symptoms are high fever, malaise, headache, backache, maculopapular rashes which become vesicular and then pustular. Diagnosis of poxvirus infection is confirmed rapidly by electron microscopic identification and PCR. Supportive therapy, maintenance of fluids and nutrition and good nursing care are the best that can be offered to most patients. Small pox vaccine, an orthopox virus provides durable protection against infection.

The increase in the incidence of avian influenza worldwide in both poultry and human introduces the potential for a pandemic that could pose a significant threat to both human health and the global economy (Whitley and Monto, 2006).

Diseases caused by Influenza virus, respiratory synctial virus, parainfluenza virus, rhinovirus and respiratory adenoviruses

Acute respiratory infections (ARI) are prevalent worldwide and viral diarrhoea is the leading cause of death in developing countries (Kilbourne, 2006). The disparity between developing and developed countries with regard to ARI epidemiology is the case-fatality rate of lower respiratory infection (LRI) mainly pneumonia, brochiolitis and influenza in children under 5 years of age. In impoverished populations these common viral infections may occur simultaneously with measles, diarrhoea, and malnutrition resulting in complex interactions of pathologic conditions which carry the potential to become serious diseases (Richard et al., 2006). Respiratory viruses have worldwide distribution, efficient person to person transmission and an impact on all age groups and in general replication is restricted to the respiratory mucosa of humans. Respiratory synctial virus (RSV) is the most frequently detected followed by parainfluenza viruses, adenoviruses and influenza viruses. Influenza viruses occur throughout the world, causing highly contagious respiratory infections with high morbidity and excess mortality particularly in infants and the elderly (La Force et al., 1994). Symptoms are fever, chills, malaise, headache, myalgia, respiratory symptoms. Secondary bacterial infections especially pneumonia are the most common complications of influenza. Laboratory diagnosis is by virus isolation or immunological methods. Antiviral and antipyretics are used for treatment. Vaccines are available for preventing influenza. The clinical spectrum of illnesses caused by RSV ranges from mild URI to severe LRI including pneumonia, bronchiolitis, tracheobronchitis, fever, otitis media. The differential diagnosis of acute bronchiolitis includes asthma, pneumonia, congenital heart and lung diseases and cystic fibrosis. URI caused by RSV requires no specific treatment and antibiotics are needed only when bacterial otitis media or sinusitis is present. Seasonal influenza is a major cause of vaccine preventable disease mortality causing estimated 250,000-500,000 deaths annually worldwide and 30,000-50,000 deaths in the US resulting in significant economic impact. Despite the availability of an effective vaccine there continues to be significant morbidity and mortality as a result of seasonal influenza (Thompson et al., 2004).

Hepatitis

Viral hepatitis is an important cause of morbidity and mortality worldwide particularly in the tropics. Sporadic cases are frequent among visitors and outbreaks of the enterically transmitted viruses are occasionally reported when food or water becomes contaminated with human faeces.

Hepatitis A (HAV)

Hepatitis A is endemic in most tropical and subtropical regions of the world but is seldom diagnosed since most infections are acquired during childhood. (Gust and Ruff, 1993).The HAV belongs to the family Picornaviridae which includes the enteroviruses and the rhinoviruses. The virus is found in high titres in stools of infected persons and the principal mode of transmission is focal oral. Person to person contact with infected family members remains one of the most important mechanisms of HAV transmission. Some of the vehicles involved in outbreaks of HAV include raw or partially cooked shellfish, milk, salads etc. HAV is transmitted by the oral-faecal route replicating in the intestine, after which it reaches the liver through the portal circulation reenters the intestine and eliminated in faeces. During acute HAV infection, there is an initial viremia and faecal shedding of virus. Hepatitis A is very often a subclinical infection of the liver running an anicteric course, particularly in children. Fever, onset of dark urine (choluria), malaise, anorexia, nausea, jaundice, hepatomegaly etc are the clinical manifestations.

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manifestations. There is no specific therapy for HAV infection and management is essentially supportive (Shapiro and Margolis, 1993). Good hygiene practices and restriction of activities of ill persons who work in the food industry are important. Hepatitis A vaccine (inactivated) is advised for use in persons more than 2 years old to prevent HAV infection.

Hepatitis B (HBV)

It still remains a major health problem throughout the world. It may evolve into chronic hepatitis and is a common cause of cirrhosis and hepatocellular carcinoma (HCC). Approximately 300 million people are chronic carriers of the virus and about three fourths of these live in Asia (Gust and Ruff, 1993).Transmission of HBV from an infected mother to her infant during birth (perinatal or vertical transmission) results in the highest rate of persistent infection. The clinical course of acute or chronic hepatitis B infection is indistinguishable from other forms of viral hepatitis and the diagnosis relies on serologic markers. There is no specific treatment for acute viral hepatitis B. By the time jaundice and symptoms present in acute hepatitis B, viral replication is usually decreasing and may have actually ceased. IFN (Interferon) has been shown to be the most efficacious antiviral agent in the treatment of chronic HBV infection. IFNs are a chemically heterogenous family of proteins grouped according to structure, antiviral effects and immunomodulatory properties. Two vaccines produced by recombinant DNA technology are currently available.

Hepatitis C (HCV)

Populations exposed to blood products (i.e. haemophiliacs) are at increased risk of contracting HCV infection, although improved screening of blood with first-generation assays has dramatically reduced the incidence of transfusion associated hepatitis C. HCV infection is also very frequent among IV drug users and is acquired by sharing contaminated needles (van den Hoek et al., 1990). Perinatal transmission of HCV is uncommon, but PCR assay for HCV RNA determines the frequency more accurately. The clinical course of acute HCV infection is indistinguishable from other acute viral hepatitides. A major impediment to the development of an effective vaccine for hepatitis C infection is the extensive antigenic variation of the virus.

Hepatitis D (HDV)

Hepatitis D virus (HDV) infection is caused by a defective RNA virus, formerly called delta virus, and requires the presence of HBV for its replication. HDV infection is transmitted by the parenteral route through contact with blood or body secretions. Direct parenteral inoculation is the most efficient route, accounting for the epidemic dissemination of the HDV agent among IV drug users. HDV is dependent on HBV for its expression and pathogenicity. HDV can infect a person who is already a carrier of HBV (superinfection) or can be transmitted simultaneously with HBV (coinfection).The severity of liver disease seems to be directly associated with the level of HBV replication. Besides accelerating the course of HBV-related chronic liver disease, HDV, is highly associated with fulminant hepatitis, which is more commonly seen with super infection than with coinfection (Aragona et al., 1987). Also the mortality rate of patients with fulminant hepatitis is higher in patients with super infection. There is no effective vaccine against HDV. Diagnosis is by serological tests and liver biopsy.

Hepatitis E (HEV)

Hepatitis E is an enterically transmitted form of viral hepatitis that appears to be common in tropical and subtropical areas and epidemics have been reported in Asia, Africa, the Middle East, and Central America. It is a distinct agent which has been associated with fulminant hepatic failure among pregnant women, but has no known chronic sequelae. There is no effective treatment for HEV infection other than general supportive measures. The search for the presence of risk factors for viral hepatitis becomes an essential part of the evaluation of the patient with acute elevation of aminotransferases and jaundice. A history of recent or remote IV drug use, blood transfusions, haemodialysis or working in the healthcare field is suggestive of acute hepatitis B, C, or D (Wald, 1995). Fever, arthralgia, general malaise and nausea are common manifestations. Boiling water before use during epidemics of HEV has been shown to reduce disease transmission. There is ongoing research directed toward the development of an effective vaccine against HEV infection in humans.

New hepatitis viruses

Analysis of non-A, non-B hepatitis cases showed that a significant proportion were also non-A, -B, -C,-D, and –E, with the discovery of new additional agents. Clinically, HGV seems to have a tendency to cause chronic hepatitis in post-transfusion patients, although it is milder than HCV infection. Antibodies to the new GB viruses have been tested in different populations with recombinant antigens for use by ELISA (Zuckerman, 1995). Further research should be directed toward determining the epidemiologic, biologic, and clinical behaviour of these newly discovered viruses. It seems likely that in the near future additional viral agents will be implicated in the pathogenesis of acute and chronic viral hepatitis in humans. Contact with icteric persons in the recent past, ingestion of potentially contaminated food, and a history of travel to areas endemic for viral hepatitis should be carefully sought, and a positive history is indicative of viral A hepatitis. A history of recent or remote IV drug use, blood transfusions, haemodialysis, or working in the healthcare field is suggestive of acute hepatitis B, C, or D. The diagnosis of different types of viral hepatitis relies on the investigation of serologic markers. The following serologic tests should be ordered to establish the diagnosis of acute viral hepatitis: IgM anti-HAV, HBsAg, IgM anti-HBcAg, and anti-HCV (or HCV RNA detection methods in selected cases). A very small percentage of patients with viral hepatitis present with fulminant hepatic failure. Hospitalization with close monitoring is indicated and referral to a specialized center for liver transplantation is appropriate.

Viral Hemorrhagic fevers

Several viruses regularly cause a syndrome which is referred to as hemorrhagic fever (HF). They belong to four different families of RNA viruses, but all are lipid-enveloped and zoonotic in their maintenance strategies. Virtually all the arenaviruses are maintained by chronic infection of a single rodent host and spread to humans by aerosols of rodent excreta. The geography and knowledge of the incubation period are important data in suspecting an exported viral HF. The syndrome characteristically begins with fever, myalgia and malaise. It also involves vascular damage to the gastrointestinal and other systems. Involvement of the vascular system is usually manifested by vascular dysregulation (mild hypotension, postural hypotension, flushing, injected conjunctivae), vascular damage (nondependent oedema, organ dysfunction), and haemorrhage (MMWR, 1988). Haemorrhage is usually diffuse in microvascular beds throughout the body and occurs particularly in patients with thrombocytopenia or marked platelet dysfunction. Many patients have little or no bleeding, but some present with extensive cutaneous and mucosal haemorrhage. Severe cases will have shock, central nervous system involvement or extensive hemorrhagic phenomena. The clinical findings in the different HFs vary in a characteristic way for each virus but any individual patient is difficult to classify without virologic diagnosis. The different viruses also differ in their pathology and pathogenesis. Correct diagnosis in most of the diseases depends on demonstration of the infecting virus or one of its products in acute serum samples. ELISA is rapid and RT-PCR is more sensitive.

Arenavirus Infections

Arenaviruses are rodent-borne pathogens that are important causes of hemorrhagic fever (HF) in Africa and South America. In West Africa, the only identified HF is Lasssa fever. Arenaviruses cause chronic infection of their rodent host, sometimes with prolonged or lifelong viremia and vertical transmission (Monath, 1975). They spread from rodents to humans by aerosol route. Serologic assays, (CF), IFA test, ELISA, have been used to define the antigenic relationships of viruses within the Arenaviridae.

Bunyaviral fever

Two bunyavirus infections found in tropical Africa are associated with haemorrhage and are sometimes fatal; these are Rift Valley fever (RVF) and Crimean-Congo hemorrhagic fever (CCHF). Viruses of the family Bunyaviridae contain negative-sense, trisegmented, single-stranded RNA.

Rift Valley fever (RVF)

RVF is a mosquito-borne virus serologically related to the sandfly fever viruses. It infects primarily cattle, but on occasion causes large epidemics in people. It is maintained over dry seasons in the eggs of *Aedes* mosquitoes. The infected eggs remain dormant in the depressions of East Africa. After heavy rains, the eggs hatch and are believed to give rise to a new batch of infected mosquitoes which initiate mosquito-livestock mosquito transmission. RVF transmission also results from direct and aerosol exposure to blood and amniotic fluids of cattle and sheep as

an occupational disease in butchers, veterinarians and farmers. Person-to-person transmission is not recorded. RVF presents with sudden onset of fever, malaise, severe myalgia with lower backache, chills, headache, retro-orbital pain, photophobia and anorexia. RVF is associated with three complications: Encephalitis presents a week or more after the febrile phase as severe headache, meningismus, confusion and vertigo with cerebrospinal fluid (CSF) pleocytosis. Optic neuropathy presents 7 to 20 days after the primary fever (Meegan and Shope, 1981).

Crimean-Congo Hemorrhagic fever

CCHF is a tick-borne virus of tropical Africa and temperate Europe and Asia as far east as Western China. CCHF virus distribution coincides with that of its primary vector, *Hyalomma* ticks. These ticks in the immature stages feed on rodents and ground birds and in the adult stage on cattle and other large mammals. People are infected when in contact with cattle or when camping, hunting, farming or engaging in recreation in natural habitats of *Hyalomma* and other *ixodid* ticks (Hoogstraal, 1979). Symptoms are sudden onset of flu-like symptoms, including dizziness; neck pain and stiffness, myalgia, arthralgia, nausea and abdominal pain. Serological tests and ELISA are the diagnostic tests. Livestock serve as amplifying hosts and their immunization is the key to the prevention of amplification and transmission. Prevention and control of CCHF are achieved by control of *Hyalomma* ticks.

Hantavirus infections

Hantaviruses are rodent-borne agents with a broad geographic distribution in the Americas, Asia, and Europe. They cause inapparent, chronic infections of rodents and spread to humans through infectious urine, saliva or faeces. The human host suffers one of two major diseases: hemorrhagic fever with renal syndrome (HFRS) or hantavirus pulmonary syndrome (HPS) (Schmaljohn and Hjelle, 1997). The viruses are virtually noncytopathic and disease is believed to be largely immunopathologic. Prevention rests on rodent avoidance when feasible but candidate vaccines for HFRS are being tested. The number of hantaviruses that have been recognized over the past years has increased exponentially as polymerase chain reaction (PCR) has allowed rapid identification of genetic sequences without the need for laborious and uncertain virus isolation. Chronic infection of a specific rodent host and persistent shedding of virus in urine, faeces, and saliva is the key to the maintenance of hantaviruses in reservoir populations as well as infection of humans. Typically severe disease has a characteristic progression beginning with fever which gives way to a brief period of hypotension followed by renal failure and finally diuresis. It also causes pulmonary syndrome. The diagnosis of all known hantavirus diseases is best made by IgM capture ELISA. Other serological tests are Western Blot assay and indirect fluorescent antibody tests. Reduction of human-rodent contact is the key to prevention.

Phlebotomus (sandfly) fever

Many of the PF group viruses appear to be maintained in their insect vectors by vertical (transovarial) transmission. After an incubation period of 3 to 5 days, PF begins suddenly with fever, severe frontal headache, retro-orbital pain, photophobia, malaise, anorexia, nausea, vomiting and low back pain. Serological techniques can be used for the diagnosis of the disease. PF is a self-limiting non-fatal disease and recovery is complete.

Oropouche fever

It is a midge-borne viral disease which has emerged during the past 40 years as an increasing public health problem in tropical America. The number of persons affected has varied with each outbreak but the two largest recorded epidemics each involved about 100,000 people. Oropouche virus is maintained in two distinct cycles:

(1) an epidemic urban cycle involving the biting midge *Culicoides paraensis* and possibly the *Culex quinquefasciatus* mosquito and

(2) a silent maintenance cycle in which forest animals (principally sloths) are the vertebrate hosts, and a yet unidentified arthropod serves as the vector. Oropouche fever is characterized by the abrupt onset of fever, chills, severe headache, generalized myalgia, arthralgia, anorexia, weakness, dizziness and photophobia. *C. paraensis* is a daytime biter and because of its tiny size readily passes through window screens (Tesh, 1994). Spraying in and around houses with residual insecticides seems to be the best method of control of adult peridomestic populations of this midge vector. Cleaning up of rotting vegetation around houses also helps to eliminate *C. paraensis* breeding sites.

Filovirus infections

There are two recognized filoviruses, Marburg and Ebola viruses and they have distinct subtypes. The filoviruses are zoonotic agents but their natural cycle is unknown. They are highly pathogenic for primates including humans. Viruses are highly infectious by small-particle aerosols. Ebola HF begins after an incubation period of 5 to

10 days. The viruses attack endothelial cells, macrophages, and other cells to produce severe hemorrhagic fever with high mortality for which there is no specific treatment. The adaptation of virus to interhuman spread has been suggested as leading to the possibility of more efficient transmission or the virus becoming attenuated. The virologic properties of Ebola virus may favour chronic infection of a mammalian reservoir. Laboratory diagnosis is by ELISA and RT-PCR. No specific antiviral therapy exists. To control filovirus infections, attention must be given to improving sterilization of parenteral equipment in hospitals.

Yellow fever

Yellow fever is the prototype virus of the Flaviviridae a unique virus family of approximately 70 enveloped positive-sense, single-stranded RNA viruses, the majority of which are transmitted by arthropods (mosquitoes or ticks).The control of *Aedes aegypti* in key centres of yellow-fever transmission and, improved sanitation prevented its spread to parts of America. The virus is transmitted between wild non-human primates and diurnally active mosquitoes that breed in tree holes. Humans are infected when they are exposed during activities that bring them into contact with vectors that have acquired virus from monkeys, such as clearing forest (Monath, 1987).The density of infected vectors and human disease incidence are low, and case distribution tends to be focal and sporadic. Alternatively *A. aegypti*, a domestic mosquito that breeds in manmade containers, may transmit yellow fever virus between humans as the sole viremic hosts in the cycle (urban yellow fever). The presence of a zoonotic cycle stimulated efforts to control the disease through vaccination which has been partially successful. The primary transmission cycle involves monkeys and diurnally active tree hole-breeding mosquitoes. An additional possible maintenance mechanism of yellow fever virus involves transmission by ticks. Yellow fever occurs only in tropical South America and sub-Saharan Africa and does not occur in Asia. During the past decade (1986 to 1995), a total of 23,543 cases and 6421 deaths were officially reported to the World Health Organization (WER, 1996). The incidence of yellow fever is highest during months with peak rainfall, humidity and temperature corresponding to the activity of *haemogogus* mosquitoes, which breed in tree holes containing rainwater. Systematic studies utilizing GIS and virus field studies would help to elucidate yellow fever risk. The clinical disease varies from non-specific influenza–like illness to life-threatening hemorrhagic fever. The onset is sudden with fever, chills, malaise, headache, lumbosacral pain, myalgia, anorexia, nausea and dizziness. Leukopenia and neutropenia are typical. Diagnosis is by serological tests, particularly ELISA. Patients should benefit from supplemental oxygen, fluid and electrolyte management, circulatory support, blood replacement and heparin treatment. Surveillance for and elimination of breeding sites, treatment of potable water with temephos and the use of residual organophosphorus insecticides are the preventive methods to be adopted. Immunization is by far the best preventive strategy. Yellow fever 17D vaccine is a safe and effective live attenuated vaccine giving long-lasting protective immunity.

Dengue and Dengue Hemorrhagic fever

Dengue fever has emerged as a serious international public health threat with almost half of the world's population at risk for infection. More than 50 million cases of Dengue fever are estimated to occur each year (WHO, 2002b). At some point in the past, probably with the clearing of the forests and development of human settlements, dengue viruses moved out of the jungle and into rural environment where they were and still are transmitted to humans by peridomestic mosquitoes such as *Aedes albopictus*. The species first infested port cities and then moved inland as urbanization expanded. Because *Ae. aegypti* had evolved to become intimately associated with humans, preferring to feed on them and to share their dwellings, this species became a very efficient vector of dengue and yellow fever viruses. Therefore, when these viruses were introduced into port cities infested with *Ae. aegypti*, epidemics occurred. As a result of major demographic changes, rapid urbanization on a massive scale, global travel and environmental changes, the world- particularly the tropical world, faces enormous challenges from emerging infectious diseases (TDR, 2005). Since the original isolates, thousands of dengue viruses have been isolated from all parts of the tropics; all have fit into the four-serotype classification. There are four dengue virus serotypes: DEN-1, DEN-2, DEN-3 and DEN-4. They belong to the genus *Flavivirus*, family. Flaviviridae (of which yellow fever is the type species), which contains approximately 70 viruses. There are three major complexes within this family: tick-borne encephalitis, Japanese encephalitis and dengue. All flaviviruses have common group epitopes on the envelope protein that result in extensive cross-reactions in serologic tests. These make unequivocal serologic diagnosis of flaviviruses difficult; this is true of the four dengue viruses. Infection with one dengue serotype provides lifelong immunity to that virus, but there is no cross-protective immunity to the other serotypes. Thus, persons living in an endemic area can be infected with three, and probably four, dengue serotypes during their lifetime.

Humans are infected with dengue, viruses by the bite of an infective *Ae. aegypti* mosquito. *Ae. aegypti* is a small, black and white, highly domesticated urban mosquito that prefers to lay its eggs in artificial containers

commonly found in and around homes in the tropics, for example, flower vases, old automobile tires, buckets that collect rainwater, and trash in general. Containers used for water storage are especially important in producing large numbers of adult mosquitoes in close proximity to dwellings where people live and work. The adult mosquitoes prefer to rest indoors, are unobtrusive, and prefer to feed on humans during daylight hours. After a person is bitten by an infective *Ae. aegypti* mosquito, the virus undergoes an incubation period of 3 to 14 days (average, 4 to 7 days), after which the person may experience acute onset of fever accompanied by a variety of nonspecific signs and symptoms. With increased epidemic transmission, hyperendemicity (the cocirculation of multiple dengue virus serotypes) developed in Southeast Asian cities, and epidemic DHF, a newly described disease, emerged. Currently, dengue fever causes more illness and death than any other arboviral disease of humans. Each year, an estimated 50 to 100 million cases of dengue fever and several hundred thousand cases of DHF occur, depending on epidemic activity. DHF is a leading cause of hospitalization and death among children in many Southeast Asian countries. The emergence of epidemic dengue and DHF as a global public health problem in the past 15 years is closely associated with demographic and societal changes that have occurred over the past 50 years. A major factor has been the unprecedented population growth and, with that, unplanned and uncontrolled urbanization, especially in tropical developing countries.

The substandard housing and the deterioration in water, sewer, and waste management systems associated with unplanned urbanization have created ideal conditions for increased transmission of mosquito-borne diseases in tropical urban centres. A second major factor has been the lack of effective mosquito control in dengue-endemic areas. In addition, the geographic distribution and population densities of *Ae. aegypti* have increased, especially in urban areas of the tropics, because of increased numbers of mosquito larval habitats in the domestic environment. These habitats include non biodegradable plastics and used automobile tires, both of which have increased dramatically during this same period of time. Another major factor in the global emergence of dengue and DHF is increased air travel by humans which provides the ideal mechanism for the transport of dengue and other pathogens between population centres of the world. Many travellers become infected while visiting tropical areas, but become ill after returning home, resulting in a constant movement of dengue viruses in infected humans to all areas of the world and ensuring repeated introductions. In 1998, dengue viruses and *Ae. aegypti* mosquitoes have a worldwide distribution in the tropics with over 2.5 billion people living in dengue-endemic areas (WHO, 1986b; Gublern and Trent, 1994). Currently, dengue fever causes more illness and death than any other arboviral disease of humans. Dengue virus infection in humans causes a spectrum of illness, ranging from inapparent or mild febrile illness to severe and fatal hemorrhagic disease. Infection with all four serotypes causes a similar clinical presentation that may vary in frequency and severity. The incubation period varies from 3 to 14 days (average, 4 to 7 days). In dengue-endemic areas, dengue infections are often clinically non-specific, especially in children, with symptoms of a viral syndrome that has a variety of local names. Important risk factors influencing the proportion of patients who have severe disease during epidemic transmission include the strain and serotype of the infecting virus, the immune status of the individual, the age of the patient, and the genetic background of the human host (Gubler, 1988). Classic dengue fever is primarily a disease of older children and adults. It is characterized by sudden onset of fever and a variety of nonspecific signs and symptoms, including frontal headache, retro-orbital pain, body aches, nausea and vomiting, joint pains, weakness, and rash. (Hayes and Gubler, 1992).

Hemorrhagic manifestations in dengue fever patients are common and may range from mild to severe. Skin haemorrhages are the most common, including petechiae and purpura, as well as gum bleeding, epitaxis, menorrhagia, and gastrointestinal haemorrhage. Clinical laboratory findings associated with dengue fever include neutropenia followed by lymphocytosis, often marked by atypical lymphocytes. Thrombocytopenia is also common in dengue fever. DHF is primarily a disease of children under the age of 15 years, although it may also occur in adults. The differential diagnosis during the acute phase of illness should include measles, rubella, influenza, typhoid, leptospirosis, malaria, viral hemorrhagic fevers, and any other disease that may present in the acute phase as a non-specific viral syndrome. Blood tests will help detect thrombocytopenia and evidence of a vascular leak syndrome. It is convenient for both clinicians and epidemiologists to classify DHF into four grades of illness based on severity. Grade I DHF is mild; the only hemorrhagic manifestations are scattered petechiae or a positive tourniquet test. Grade II DHF is more severe, with one or more overt hemorrhagic manifestations. Grades III and IV represent more severe forms of disease (DSS). Grade III illness is characterized by mild shock with signs of circulatory failure; the patient may be lethargic or restless and have cold extremities, clammy skin, a rapid but weak pulse, narrowing of pulse pressure to 20mm Hg or less, or hypotension. Grade IV, the most severe form of DHF or DSS is characterized by profound shock with undetectable pulse and blood pressure. As with dengue fever, leucopenia is common; thrombocytopenia and hemoconcentration are constant findings in DHF and DSS. Hemoconcentration, indicating plasma leakage, is almost present in classic DHF, but is more severe in patients with shock. Hepatosplenomegaly is a common, but not a constant finding. According to the immune enhancement

hypothesis, patients experiencing a second infection with a heterologous dengue virus serotype have a significantly higher risk of developing DHF or SS. The other hypothesis assumes that dengue viruses, like all animal viruses, vary and change genetically as they move through human or mosquito populations, and that there are some virus strains that have greater epidemic potential. It appears that only certain strains of virus may be involved in producing severe disease by immune enhancement. Patient management is by early and effective fluid replacement of lost plasma with electrolyte solutions, plasma .With adequate fluid replacement, DSS is reversible. Prognoses depend on early recognition of shock, based on careful monitoring. A decrease in the platelet count, which usually precedes the rise in haematocrit, is of great diagnostic and prognostic value. A definitive diagnosis of dengue infection can only be made in the laboratory and depends on either isolating the virus or detecting specific antibodies in the patient's serum. Virus can be often isolated from acute phase blood samples taken in the first 5 days of illness (Gubler and Sather, 1990).

Viral RNA can be often detected by polymerase chain reaction (PCR) in serum or tissues or antigen by immunohistochemistry in tissues. Serological tests would help to detect antibodies. Prevention and control depends on controlling the mosquito vector, *Ae.aegypti*, in and around the home, where most transmission occurs. Space sprays with insecticides would kill adult mosquitoes and larval source reduction is by eliminating or cleaning water-holding containers that serve as larval habitats for *Ae. aegypti* in the domestic environment.

Other flavivirus infections

About 60 flaviviruses are known to circulate mostly in the tropics. Twenty-three of these cause encephalitis, hemorrhagic diseases or fevers. In addition to the four dengue serotypes and yellow fever the ones with potential clinical importance in the tropics are Japanese encephalitis (JE), West Nile fever (WN), Kyasanur forest disease (KFD) and Zika viruses. Most of these viruses cause fever with or without arthritis or rash. JE virus causes epidemic encephalitis in Asia and the Pacific islands. While most epidemics are north of the Tropic of Cancer, JE, is still a serious problem in Thailand, Vietnam, Cambodia, Myanmar, Laos, Malaysia, Singapore, the Philippines, SriLanka, and southern India. The virus is transmitted by *Culex* mosquitoes that breed primarily in rice paddies and maintain the transmission cycle in ardeid birds such as herons and other waterfowl (WHO, 1979). WN virus is closely related to JE virus and has similar clinical and epidemiological features, causing encephalitis and involving passerine and water birds as vertebrate reservoir hosts. WN virus infection is enzootic in most of Africa, extending into the temperate regions of Europe and the Middle East. Epidemics of WN encephalitis are not seen in the tropics. KFD virus is the cause of a hemorrhagic disease localized to six small forest foci in Karnataka state, India (Webb and Rao, 1961). About 400 to 500 cases have been reported each year since the disease was first recognized in 1957.The virus belongs to the tick-borne encephalitis serocomplex. The cycle in the forest is maintained in Haemaphysalis ticks sand small mammals and birds. KFD virus is readily isolated from monkeys that are often found sick or dead in the enzootic foci. KFD is clinically and virologically similar to Omsk hemorrhagic fever, a disease of the temperate regions of Omsk and Novosibirsk oblasts of Russia. KFD has an incubation period of 2 to 7 days, followed by acute onset of severe frontal headache, continuous high fever, myalgia, cramps, diarrhoea, and vomiting. Death occurs in about 8 % of patients, usually secondary to pulmonary edema.

Zika virus has been diagnosed in 12 naturally acquired cases that occurred in Nigeria, Senegal, Central African Republic, and Indonesia Antibody prevalence of 50% or more in many African populations indicates that Zika virus infection is probably much more common than recognized (Adekolu-John and Fagbami, 1983). Patients have a self-limited disease characterized by fever and rash. The virus is maintained in a cycle involving Aedes mosquitoes and forest primates. The tropical flaviviruses associated with encephalitis include JE, WN, SLE, Rocio, MVE, and Ilheus viruses. These are closely related mosquito-borne viruses with differing geographic distribution, but with much similarity in clinical presentation and pathogenesis and most infections are subclinical. For most of these agents, the clinical presentation is one of three syndromes: fever, fever with aseptic meningitis, or fever with encephalitis. WN virus characteristically causes a mild illness and most cases present with fever, associated with a maculopapular or roseolar rash of the upper body in about 50% of cases. JE, Rocio, and MVE virus infections on the other hand are rarely mild and usually present as frank encephalitis. Both WN and SLE viruses can cause hepatitis (instead of encephalitis) in the tropics. Analysis of JE and SLE virus strains show that tropical isolates differ genetically from the epidemic viruses of the temperate region, suggesting possibly that viral factors are responsible for the putative lower virulence. The incubation period of flavivirus encephalitis ranges between 4 and 21 days, but is usually 1 to 2 weeks. Fever, severe frontal headache, malaise, and gastrointestinal symptoms preceed the central nervous system (CNS) manifestations by 1 to 3 days. The fever maybe 40^{o} C or higher and conjunctival and pharangeal hyperaemia is common. A wide variety of CNS signs and symptoms ensue, including convulsions, pareses, paralyses, cranial nerve defects, ataxia, hyperactive deep tendon reflexes and sensory abnormalities. In

severely affected patients, coma is followed by death through respiratory or cardiac failure (Monath, 1988).

Diagnosis is by serological methods, PCR and virus isolation methods. There is no specific treatment for flavivirus infections. Supportive treatment of encephalitis cases includes intravenous fluid and electrolyte management, assisted respiration if needed, anticonvulsants, and nursing care. Physical and psychological rehabilitation is important in surviving patients, because sequelae are common and result in major disability. Control of flavivirus disease can be achieved by vaccination of humans. It must be recognized, however, that the natural cycles of the flaviviruses causing human encephalitis involve mosquitoes and wild or domestic animal hosts. Therefore, vaccination of humans will not interrupt the transmission cycles in nature. Effective vaccines are available to immunize pigs, the amplifying host of JE virus. Vaccines for JE virus are licensed for human use in the United States and in countries of Asia. The principal vectors of the encephalitis viruses are Culex mosquitoes. These are mainly active in the rainy season in the tropics, breeding in rice paddies, irrigated fields or in river floodplains .larvae are killed by periodic short-term draining of the irrigation water. Use of larvicides can diminish emergence of adult Culex mosquitoes. During epidemics, Culex control can be achieved by aerial spray of pesticides such as ultra-low-volume fenitrothion. Resistance of Culex to organophosphate compounds and carbamates has been detected in Japan and SriLanka. The wide-scale re-emergence of JE in recent years in regions such as southern India and SriLanka speaks to the need for increased mosquito surveillance and improved vector control.

Alphavirus Infections

The alphaviruses comprise a genus (Alphavirus) in the family Togaviridae of enveloped, single-stranded, plus-stranded RNA viruses that occur nearly worldwide. Alphaviruses are zoonotic pathogens that are maintained primarily in rodents, primates, and birds by mosquito vectors. Human disease occurs when people intrude on enzootic transmission habitats and are bitten by infected mosquitoes, or when alphaviruses emerge to cause epizootics and epidemics. Five alphaviruses commonly infect people in the tropics and have epidemic potential: Venezuelan equine encephalomyelitis (VEE), chikungunya (CHIK), Ross River, O'nyong-nyong (ONN), and Mayaro viruses (Tsai and Monath, 1997). Several other alphaviruses, including eastern and western equine encephalomyelitis and Sindbis viruses cause human disease primarily in temperate regions. Definitive diagnosis of alphavirus infections generally requires virus isolation or serologic confirmation. Rapid diagnosis can be achieved using ELISA and other serological tests.

Venezuelan equine encephalomyelitis (VEE)

VEE was first recognized in Venezuela in 1936, and has caused periodic equine epizootics and epidemics in many regions of tropical America. A major epidemic in northern Venezuela and Colombia in 1995 involved approximately 100,000 persons. VEE viruses occur in two distinct transmission cycles: enzootic and epidemic or epizootic. Enzootic cycles involving small mammalian hosts and mosquitoes in the subgenus *Culex* (Melanoconion) occur in tropical forest and swamp habitats in the New World ranging from Florida to Argentina (Weaver et al., 1996). Enzootic viruses (antigenic subtypes ID-F, II-VI) are avirulent for equines, but are pathogenic for humans and can cause fatal disease. In contrast, epidemic or epizootic VEE viruses (subtypes IAB, IC) are virulent for both equines and humans, but have no known interepizootic maintenance cycles. Epizootic transmission involves several species of mammalophilic mosquitoes, including *Aedes* and *Psorophora*, which transmit the virus among equines circulating high levels of viraemia. People also become infected by mosquitoes that previously engorged on viraemic equines. However, human-mosquito-human transmission is uncertain. Human VEE infection causes a disease spectrum ranging from inapparent infection to acute encephalitis. Symptoms include fever, lethargy, headache, chills, dizziness, bodyaches, nausea, vomiting. throat inflammation. Laboratory findings include leucopenia and lymphopenia. The absence of rash and joint pain can be used to rule out Mayaro and dengue fevers. Definitive diagnosis of VEE is by virus isolation and reverse transcription-polymerase chain reaction assays and ELISA. Treatment is supportive and symptomatic and anticonvulsive therapy. Control of VEE relies on interruption of transmission by mosquito vectors and vaccination of equine hosts. Vector control relies on aerial application of insecticides such as Malathion, optimally applied soon after floodwater species emerge from the aquatic immature stages, prior to dispersal, infection, and incubation required for transmission.

Ross River virus

The first outbreak of Ross River virus infection was described in 1928 where the disease is known as epidemic polyarthritis. Ross River virus is maintained in Australia primarily in a wild vertebrate –mosquito cycle,

with *Culex annulirostris* and *Aedes vigilax* serving as the principal vectors. Epidemiologic studies during outbreaks in South Pacific in 1970-80 implicated *Aedes polynesiensis* as the vector and suggested a human-mosquito-human cycle. The disease is characterized by headache, malaise, myalgia, joint pain and rashes. Diagnosis is by serological methods. Treatment is symptomatic. There is no vaccine currently available; the best prevention is to avoid mosquito bites when travelling in rural areas where the disease occurs.

Chikungunya virus

CHIK virus was first isolated during a 1952 epidemic in Tanzania. Chikungunya comes from Swahili, meaning "that which bends up" and refers to the characteristic posture assumed by patients typically suffering severe joint pains. CHIK has occurred sporadically in India and Southeast Asia for at least 200 years. The known geographic distribution of the virus includes most of sub-Saharan Africa, India, Southeast Asia, Indonesia and the Philippines (Bessaud et al., 2006). Transmitted by *Aedes* spp., mosquitoes, Chikungunya virus (CHIK) causes febrile illness with joint pain. It is characterized by fever, chills, headache, photophobia, backache, nausea, vomiting, arthralgia, rash. Sylvan transmission cycles involving forest-dwelling *Aedes* spp. and wild primates maintain endemicity throughout tropical Africa. In tropical Asia, urban *Aedes aegypti* and *Aedes albopictus* –human cycles are thought to maintain CHIK between epidemics (WER, 2006). Widespread domestic water storage could have facilitated vector breeding and human contact. Climatic effects, particularly elevated temperature, on virus development in vector mosquitoes also could have enhanced transmission efficiency. Drought affected populations may be at heightened risk for chikungunya fever, and underscore the need for safe water storage during drought relief operations. CHIK fever follows heavy rains. Vector precautions and control measure should be maintained after chikungunya fever outbreaks because as in Asia, large epidemics may recur as population immunity declines (Jupp and McIntosh, 1988). Chikungunya Integrated satellite-based drought monitoring and epidemiologic surveillance could identify areas at risk for chikungunya fever epidemics, and allow countries to institute timely prevention or control programs (Paul and Epstein, 2007).Treatment is symptomatic and includes antipyretic and anti-inflammatory drugs. It is generally a self-limited infection. Vaccines have been produced, but are not available commercially. Control measures consist of avoidance of mosquito bites and vector control.

O'nyong-nyong virus

The name is derived from the description by the Acholi tribe, meaning "joint breaker". The virus was first isolated in 1959 during an epidemic involving an estimated 2 million people in Uganda, Kenya, Tanzania, Mozambique, Malawi and Senegal. The transmission cycle of ONN virus involves *Anopheles funestus* and *Anopheles gambiae* mosquitoes; ONN virus is the only known alphavirus with Anopheles vectors. Infection is self-limited and is treated symptomatically (Tesh, 1982) Prevention and control are limited to reductions of Anopheles vector populations and vector avoidance. Because *Anopheles* mosquitoes usually bite at night, these include measures effective in preventing malaria infection, such as bednetting impregnated with permethrin and application of repellents when outdoor nighttime exposure is anticipated.

Rabies

Rabies is a viral disease producing almost uniformly fatal encephalitis in humans and other mammals. It is the most fatal of all viral diseases and it remains one of the most common causes of mortality in tropical countries. Exposure to potentially rabid animals has profound economic and medical implications; about 4 million people receive pre-exposure treatment (PET) annually to prevent rabies. The family *Rhabdoviridae* are negatively stranded RNA viruses consisting of two genera which infect animals (*Lyssavirus* and *Vesiculovirus*) and the plant rhabdovirus group. The type species of the subfamily *Lyssaviridae* is rabies virus (serotype 1), and that of the subfamily *Vesiculoviridae* is vesicular stomatitis virus. Rabies is enzootic, and sometimes epizootic, in many mammals, most commonly wild and domestic canids (e.g., dogs, foxes, coyotes), mustelids (e.g.skunks, badgers, martens), viverids (e.g., mongoose, civets, genets), precyonids (e.g., raccoons and their allies) and insectivorous and hematophagous bats. Rabies occurs throughout the world. The epidemiology of human rabies is that of animal rabies in the community. Rabid wild animals are usually responsible for human rabies in regions where dogs are vaccinated. In 1994, 34,110 cases of human rabies were reported to the World Health Organization (WHO, 1997). Paralytic rabies expresses the majority of its pathologic changes in the spinal cord, with severe inflammation and neuronal necrosis. The viral inoculum is a relationship of rapidity with the extent of exposure to the saliva of a rabid animal. Salivary contamination of a preexisting wound, mucous membranes, or the respiratory tract (with

aerosolized virus) may also spread virus. The initial symptoms of rabies resemble those of other systemic viral infections, including fever, headache, malaise and upper respiratory and gastrointestinal tract disorders and neurologic problems. Human rabies is typically seen in two forms: furious and paralytic (or dumb). Furious rabies is dominated by encephalitis and presents with hydrophobia, delirium, and agitation. The spinal cord and brain stem are more involved in the paralytic form. Hydrophobia is the symptom most identified with furious rabies. Virus can enter both motor and sensory nerves. The human response to rabies infection is insufficient to prevent disease. Viral replication at an immunologically active site (CNS) also limits host response. Rabies virus can produce immunosuppression. Serological tests and RT-PCR is the diagnostic procedure of choice in suspected rabies which can be done on CSF, saliva or tissues of patients. There is no established treatment for rabies once symptoms have begun; almost all patients succumb to the disease or its complications within a few weeks of onset. Wild animal vaccination can be an effective veterinary public health measure. After exposure, rabies prevention begins with good wound care, which reduces the risk of rabies by up to 90%. Anti-rabies vaccines are also available.

G. RETROVIRAL INFECTIONS

Human Immunodeficiency Virus and Acquired Immunodeficiency in the tropics

The Acquired Immunodeficiency Syndrome (AIDS) is the end stage clinical manifestation of a chronic infection with Human Immunodeficiency Virus (HIV). Recent results with new antiviral drugs have shown that HIV infection is treatable and curable. Human immunodeficiency virus (HIV) was the etiologic agent of AIDS and is widely prevalent in both developing countries and developed world (Gallo et al., 1984). In the first decade 10 million people became infected and since 1990, the pandemic has quadrupled in size, to 42 million by the end of 1997 (WHO, 1995a; Quinn, 1996). HIV/AIDS is not strictly a tropical infection: approximately 20 million persons have become infected worldwide, and no country on the planet has been entirely spared. However, 90% of all infected persons live in sub-Saharan Africa, South and Southeast Asia, or South America. The HIV epidemic is having devastating effects on infant health and survival in sub-Saharan Africa (Newell, 2004). By the year 2010 it is anticipated that over 93% of all new infections will be occurring in developing countries. Due to the overwhelming immunosuppression associated with HIV infections, it is also anticipated that many endemic bacterial and parasitic diseases in these regions will increase dramatically. Diseases such as tuberculosis, toxoplasmosis, cryptosporidiosis, isosporiasis and fungal infections, including cryptococcosis and *Pencillium marneffei*, will become more prevalent and result in increasing morbidity and mortality. By December 1997; 42.3 million people had been infected with HIV, including 38.5 million adults and 3.8 million children (UNAIDS Report, 1997). Of these, it is estimated that 11.7 million people have died of AIDS since the start of the global pandemic, although only 1.5 million cumulative cases of AIDS, in adults and children have been officially reported to the World Health Organization (WHO). Thus, 30.6 million people are estimated to be currently living with HIV infection, including 29.5 million adults and 1.1 million children. Short term projections predict that a minimum cumulative total of 50 to 60 million cases of HIV infection will occur by the year 2010. HIV infections are transmitted through heterosexual and homosexual transmission; parenteral or perinatal or blood -borne transmission through use of inadequately sterilized needles, syringes etc. In a recent review , more than 100 pathogens, including viruses, bacteria, fungi, protozoa, helminthes, and arthropods, were identified as having caused opportunistic diseases in HIV - infected persons. The co-occurrence of other sexually transmitted diseases increases both the risk of acquisition and the risk of transmission of HIV infection. Molecular epidemiology of HIV genotype reveals the global pandemic to be composed of multiple, genetically distinct virus subtypes. A number of genetic variants have evolved in equatorial Africa and at least five of these variants have seeded major clonal epidemics outside this region. The level of virus in the blood and viral genotype are potential biologic determinants of transmissibility. In most developing countries tuberculosis is the most important opportunistic infection observed among HIV- infected patients in the tropics because it occurs frequently, is transmissible to both HIV- infected and uninfected persons, is relatively easily treated, and can be prevented. It is estimated that the HIV epidemic will be responsible for 20% of the projected increase in global TB.
In addition to TB, bacterial pneumonia, particularly pneumonia caused by *Streptococcus pneumoniae* and *Haemophilus influenzae* occurred frequently. The underlying HIV- associated immunosuppression may also result in a more aggressive course of disease due to the failure of immunologic control of the secondary organism such as TB. Immune activation hastens the course of HIV infection by increasing viral expression and viral burden in the infected person. Thus the rapid course of HIV disease that has been observed in the developing world is due in part to the persistent immune activation associated with chronic parasitic infection as well as infection with other agents.

Table 1. WHO classification of AIDS-defining conditions

CD4 cell count< 200/ul
Candidiasis, pulmonary or oesophageal
Cervical cancer
Coccidioidomycosis
Cryptosporidiosis
Cytomegalovirus infection
Herpes esophagitis
HIV encephalopathy
Histoplasmosis
Isosporiasis
Kaposi's sarcoma
Lymphoma
Mycobacterial disease
Pneumocystis carinii infection
Pneumonia, bacterial
Progressive multifocal leukoencephalopathy (polyomavirus)
Salmonellosis

Chronic immune activation exists due to infection with numerous pathogens and this could theoretically accelerate the course of HIV disease. An additional interaction between HIV and tropical parasites is the role of malnutrition and its effects on the immune system. Several pathogens including CMV, herpes simplex virus, hepatitis B virus, human herpesvirus type 6, human T-cell lymphotropic virus type 1, pseudorabies virus, and microbes such as *Mycoplasma* have been shown to enhance HIV expression. Thus there exist complex interactions between tropical diseases and HIV infection (Table 1). HIV results in immune dysfunction which may increase the individual's susceptibility to new infection and reactivation of old infection with tropical diseases. In view of the complex intertwining of these factors, it becomes important to consider prevention in the public health approach to HIV disease: prevention of infection with tropical diseases, through the effective treatment of debilitating diseases and provision of adequate access to nutrition and ultimately the prevention of HIV infection itself, through vaccine development. Like all retroviruses, the life cycle of HIV is characterized by alternating stages in which the genetic information is carried by DNA or by RNA, the proviral and the viral stage respectively (Weniger et al., 1994). Although the HIV epidemic has reached every country on the globe, the epidemic has important geographic differences. Homosexuality caused the epidemic in America but heterosexuality drove the epidemic in Africa. The WHO categorized the former as pattern I countries and the latter as pattern II countries. Disease progression is a direct reflection of virus replication. When viral replication is high HIV infection progresses to clinical immunodeficiency. HIV antibody assays on blood serum or plasma are used to establish a diagnosis.

VIII. OTHER DISEASES

Neurologic Disease

Neurologic disorders occur in a large percentage of tropical infectious diseases, and they are the proximate causes of death and disability in many of these conditions. The major of them are meningitides, encephalitides neurocysticercosis, echinococcosis, cerebral paragonimiasis etc.

Anaemia

Anaemia is a major problem in tropical areas largely because of interactions between three factors peculiar to these geographic regions: (1) a high prevalence of infections and infestations capable of causing anaemia, especially those caused by malaria, helminthes, tuberculosis and human immunodeficiency virus (HIV); (2) the common presence of dietary deficiencies; and (3) a high frequency of inherited red blood cell (RBC) disorders which result in anemia which is caused by haemoglobin deficiency (WHO, 1968). The three etiological factors in anaemia in the tropics are (1) infections or infestations, (2) dietary deficiency, and (3) inherited disorders. With some agents the pathogenesis may be multifactor, as in human immunodeficiency virus (HIV) infection, ACD, and malaria. Laboratory diagnosis is by Hb estimation.

Ocular Disease

Many tropical infectious diseases have ocular manifestations. A number of such infections (i.e. trachoma, onchocerciasis, measles/ xerophthalmia, leprosy) are the cause of significant visual loss and blindness worldwide. Ocular involvement by tropical infectious diseases may manifest as conjunctivitis, keratitis, uveitis, retinitis, optic neuritis, ulcerative lesions of the eyelid.

Adenovirus

Worldwide adenovirus is probably the most common cause of viral ocular infection. Ocular involvement by adenovirus usually manifests as one of the three clinical syndromes: nonspecific follicular conjunctivitis, pharyngoconjunctival fever, or epidemic keratoconjunctivitis. Diagnosis is by clinical recognition, viral cultures, immunofluorescence and serological and molecular techniques. Topical steroids are used for treatment.

Enterovirus

The enterovirus includes enterovirus, poliovirus, coxsackie virus and echovirus. Ocular manifestations of enteroviral infections include acute hemorrhagic conjunctivitis and cranial dysfunctionin acute enteroviral neuropathy. Influenza, measles, rubella and mumps and Varicella Zoster virus also cause ocular manifestations.

The hepatobiliary tract is the target of a wide variety of tropical infections. Some diseases such as chronic hepatitis and hepatosplenic schistosomiasis are major causes of morbidity and mortality throughout the tropics. Acute hepatitis, chronic hepatitis and jaundice are the common biliary diseases.

IX. DISEASES CAUSED BY PROTOZOA

The major types of organisms that cause parasitic infections include species of protozoa, helminths or worms and arthropods. Protozoa are single-celled organisms that carry out most of the same physiological functions as more complex organisms. More than 45,000 species of protozoa are known, many of which are parasitic. As parasites of humans, this group of organisms has historically been the cause of more suffering and death than any other category of disease causing organisms. Intestinal protozoa occur throughout the world. They are especially common in areas where food and water sources are subject to contamination from animal and human waste. Typically, protozoa that infect their host through water or food do so while in an inactive state, called a cyst. A cyst consists of a protozoan encased in a protective outer membrane. The membrane protects the organism as it travels through the digestive tract of a previous host. Once inside a new host, the parasite develops into a mature form that feeds and reproduces.

Enteric amoebiasis

Amoebic dysentery is one of the most common parasitic diseases. It often afflicts travellers who visit tropical and subtropical regions. The condition is characterized by diarrhoea, vomiting, colitis and weakness. Diarrhoeal illnesses constitute one of the leading infectious causes of death and disability worldwide. It is estimated that 3.3 to 6 million children die annually from diarrhoeal illnesses, the vast majority in Africa, Asia, and Latin America (Bern et al., 1992). It is caused by a protozoan known as *Entamoeba histolytica*. The trophozoites are located in the large intestine of the human host where they divide by binary fission. Another protozoan that causes severe diarrhoea is *Giardia lamblia* and transmission is by oral ingestion of the cyst form of the parasite. Diagnostic measures are microscopic detection of the parasite in stool, liver, abscess pus or colonic biopsies, antigen detection, PCR, colonoscopy and serology. Prevention of amoebiasis requires interruption of faecal-oral spread of the infectious cyst stage of the parasite by improved hygiene, sanitation and water treatment (Moyenuddin et al., 1987).

Intestinal flagellate and ciliate infections

Giardia lamblia is responsible for the frequent occurrence of diarrhoea in many developing countries where sanitation measures are wanting. It is a flagellate protozoan inhabiting the small intestine of man and other mammals. *Balantidium coli* is the largest intestinal ciliate protozoan of man causing diarrhoea and inflammatory colitis. The three modes of transmission are water-borne, direct faecal-oral and food-borne. Infection follows ingestion of cysts (Flanagan, 1992).

Intestinal coccidial infections

Cryptosporidium, *Isospora*, *Cylclospora* and *Sarcocystis* are intestinal coccidians. Intestinal coccidian are characterized by the faecal excretion of oocysts, which are the product of a sexual cycle of reproduction in the epithelium of the small intestine. Their unique characteristics of chlorine resistance and dependence on cell-mediated immunity for resolution of infection may place them among the most problematic organisms of the next century. The clinical spectrum of cryptosporidial infection ranges from asymptomatic passage of oocysts to severe cholera-like gastroenteritis with biliary tract disease. Like *Cryptosporidium*, *Isospora* generally produces a self - limited diarrhoeal illness in healthy persons and prolonged, severe disease in immunocompromised patients. The disease caused by *Cyclospora* is often indistinguishable from that due to *Cryptosporidium.* The hall mark symptoms are watery diarrhoea, abdominal cramps, nausea, anorexia, and bloating. In patients with AIDS, Cyclosporiasis can be as severe as Cryptosporidiosis, with protracted or fulminant diarrhoea and weight loss. Cryptosporidiosis in immunocompetent patients is self-limited and symptomatic therapy should be directed at rehydration and nutrition. Oral rehydration should be used as needed to prevent volume depletion. Antimotility agents may be used. Unlike Cryptosporidiosis, Isosporiasis can be treated successfully in patients with or without AIDS. The only way to acquire any of the coccidia is to ingest infectious oocysts; the diseases can be completely prevented by elimination of oocysts from food and water and by avoidance of faecal material from infected people. Since oocysts are extremely resistant to chlorination, water treatment plants must rely on mechanical means to remove them: flocculation, sedimentation, and filtration (Le Cevallier et al., 1991).These methods should be adequate to prevent water-borne outbreaks. However the Nevada outbreak of 1994 proved that these techniques can fail even without obvious malfunction. Muscle and intestinal sarcocystosis caused by *Sarcocystis* is found throughout the world.

Malaria

The most deadly vector borne disease, malaria, kills over 1.2 million people annually, mostly African children under the age of five. Poorly designed irrigation and water systems, inadequate housing, poor waste disposal and water storage, deforestation and loss of biodiversity, may be contributing factors to the most common vector-borne diseases including malaria. Malaria is a global crisis and also a public health problem today in more than 90 countries, inhabited by a total of some 2400 million people, 40% of the world's health population. Worldwide prevalence of the disease is estimated to be in order of 300-500 million cases per year and more than 90% of all cases are in sub-Saharan Africa. It is estimated that around 100 million clinical cases result in at least 50,000 deaths may occur every year in tropical Africa alone, where changes in the epidemiological situation in the last few years have resulted in an increased frequency of the disease (Greenwood et al., 2005). In 2002, 400 million episodes of clinical malaria occurred in Sub-Saharan Africa (Snow et al., 2005). *Plasmodium falciparum* is the main cause of severe clinical malaria and death. The vast majority of deaths occur among young children in Africa, especially in

remote rural areas with poor access to health services. Other high risk groups are pregnant women, and non-immune travellers, refugees or labourers all entering endemic areas.

Four species of the genus *Plasmodium* infect humans when injected into the bloodstream by mosquitoes. *P. falciparum* is found throughout tropical Africa, Asia and Latin America, *P. vivax* is worldwide in tropical and some temperate zones, *P. ovale* occurs mainly in tropical West Africa and *P. malariae* is found worldwide with patchy distribution.

Transmission is by inoculation via the bite of infected blood-feeding female mosquitoes of the genus *Anopheles*, which transfer parasites from human to human. In humans, parasites multiply exponentially in the liver and then infect red blood cells. Mosquitoes ingest parasites with a blood meal, the parasites undergoing another reproductive phase inside the mosquito before being passed on to another human.

Typical cycles of fever, shaking chills and drenching sweats may then develop. The periodicity of these cycles depends on the species of parasite, coinciding with parasite multiplication and destruction of red blood cells (RBC), which also leads to anaemia. Falciparum malaria may not show this cyclic pattern and can be fatal if untreated or treated with insufficiently effective drugs. Death may be due to infected RBC blocking blood vessels supplying the brain (cerebral malaria), or damage to other vital organs. Epidemics are often associated with non-immune people moving to highly-endemic areas, where they quickly succumb to infection. Severe anaemia is often the attributable cause of death in areas with intense malaria transmission.

Prevention and control strategies include protection measures against the mosquito vector which can be personal (individual or household) protection measures e.g. repellents, bednets, or community/population protection measures e.g., use of insecticides or environmental management to control transmission. Measures which protect against disease include chemoprophylaxis. Prophylatic drugs are chloroquine, chloroquine + proguanil, or mefloquine, doxycycline, or sulphadoxine-pyrimethamine in areas where chloroquine-resistant parasites are found. In endemic areas, malaria control relies on diagnosis and prompt treatment of infected individuals based on clinical symptoms and microscopic diagnosis (White, 1996). Regular assessment of a country's malaria situation is necessary, allowing early detection, containment or prevention of epidemics.

While malaria remains under control in most developed and stable areas, the situation is deteriorating in all frontier areas of economic development, i.e. in areas where the exploitation of natural resources or illegal trade occurs, in jungle areas or areas burdened with problems of civil war and other conflicts, and where mass movements of refugees exist. Increasing drug resistance, decreasing efficacy of vector control efforts and weak health infrastructure also affects the control of this disease. In addition, the expected rise in global temperature might herald an increase in the geographical distribution of malaria transmission. The development of environmental friendly insecticides, biopesticides, biocontrol agents like larvivorous fishes and good antimalarial drugs, might make the control of malaria an obvious possibility.

One of the most alarming problems in the treatment and control of malaria is the spread of resistance to anti-malarial drugs in most tropical areas like the spread of chloroquine resistance of *P. falciparum*. In spite of drug resistance, malaria is a curable disease, not an inevitable burden. Although there is only a limited number of drugs, if these are used properly and targeted to those at greatest risk, malaria disease and deaths can be reduced, as has been shown in many countries.(Krogstad, 1996).

Genetic resistance to malarial infection, of all infections, been best characterized, largely because of polymorphisms of the red blood cell membrane and the red blood cell's contents can be easily recognized by relatively simple laboratory methods such as blood group typing, enzyme assays, and haemoglobin electrophoresis. An effective vaccine would constitute a powerful addition to malaria control tools. Intensive research efforts are ongoing to elucidate the protective immune responses, identification of antigenic targets and development of vaccine delivery systems associated with malaria. Vaccines, produced by recombinant technology or peptide synthesis, showed a low efficacy in protecting against clinical malaria. Different parasite-specific features and characteristics of the interaction of human immune system and the parasites, might explain the difficulties in developing effective immuno-therapeutic intervention strategies. The three main types of vaccine being developed are: 'Anti-sporozoite' or 'pre-erythrocytic' vaccines, designed to prevent infection. 'Anti-asexual blood stage' vaccines, designed to reduce severe and complicated manifestations of the disease.' Transmission-blocking vaccines', (TBV) designed to arrest the development of the parasite in the mosquito, thereby reducing or eliminating transmission of the disease. One class of vaccine is being developed specifically for this purpose--the malaria transmission-blocking vaccines (TBV) based upon antigens expressed on the surface of the sexual and mosquito mid-gut stages of malaria parasites. These antigens are the targets of antibodies induced by vaccination of the host and ingested with the parasites in a mosquito blood meal. The antibodies act by inhibiting the parasite's development within the mosquito itself and they thereby prevent the onward transmission of the parasites. TBVs would help reduce malaria incidence and malaria-related morbidity and mortality. Promising recombinant TBV candidate antigens for the two main human malaria

parasite species, *Plasmodium falciparum* and *Plasmodium vivax,* have been produced and tested in the laboratory; one has undergone early clinical trials (Stowers and Carter, 2001).

A rational approach to the problem of malaria is required in combination with intensive research efforts on different approaches to control malaria. Expansion of our understanding of the biology, epidemiology, pathogenesis and clinical manifestations of this complex, heterogeneous disease will be critical to the development of additional strategies for control. In 1998, an initiative of WHO, UNDP, UNICEF and World Bank was announced to reduce the malaria burden: Roll Back Malaria. The RBM partnership includes governments, development agencies, commercial organizations, civil society, research groups and the media**.**

Malaria and Tuberculosis present a major barrier to poverty eradication in Africa. It has been estimated that malaria alone costs Africa more than US$12 billion annually and slows economic growth on the continent by up to 1.3% each year. The economic impact is felt disproportionately by poor families which spend about 25% of their annual income on malaria prevention and control. TB, especially when associated with AIDS, accounts for US$ 11 billion in future lost income (WHO, 1989). It affects what should be the most productive age group in society, effectively impoverishing both households and the nation. Because malarial parasites are constantly evolving and acquiring more resistance to available drugs, routine surveillance for drug resistance is an essential activity for all malaria control programmes.

Trypanosomiasis

Members of the genus *Trypanosoma* are widespread as trypomastigotes in the blood of vertebrates. They are transmitted from one vertebrate to another through biological vectors. Transmission to the invertebrate vector takes place through blood feeding. Transmission to the vertebrate host takes place through one of the two routes: injection during feeding or faecal contamination of the site where the insect vector has been feeding (WHO, 1986a). Trypanosomiasis is transmitted by tabanid flies and tsetse flies (*Glossina* sp.) and transmission is mechanical. *T. brucei rhodesiense* and *T. brucei gambiensi* causes African sleeping sickness (in humans) and *T. brucei brucei, T. congolense and T. vivax* causes nagana (in cattle). *T. brucei gambiense* causes Gambian fever and *T. brucei rhodesiense* causes Rhodesian fever. T. *equiperdum*, *T. evansi* and *T. equinum* are transmitted by tabanid flies causing Acute Dourine, Sura and Malde Caderas in horses and deer. *T. cruzi* causes Chagas disease or American trypanosomiasis, a zoonotic disease transmitted by reduvid bugs. WHO estimates 50,000-70,000 deaths per year and 1.78 million disability-adjusted life-years lost (WHO, 2006a).Symptoms are malaise, fever, lesions, oedema of face and lower extremities as well as hepatosplenomegaly and generalized lymphadenopathy. Metacyclic (infective) trypomastigotes are inoculated through the skin when a tsetse fly takes a blood meal. The parasites develop into long slender trypomastigotes which multiply at the site of inoculation and later in the blood, lymphatic system and tissue fluid. The trypomastigotes are carried to the heart and various organs and in later stages of infection invade the CNS. Trypomastigotes are taken up by the tsetse fly when it sucks blood (male and female). The parasites develop in the midgut of the fly and multiply. From about 2-3 weeks the trypomastigotes move to the salivary glands transforming through epimastigotes into metacyclic trypomastigotes. The tsetse fly remains infective for life - about 3 months.

In Chagas disease the trypomastigotes circulate in human blood and are taken up by tsetse flies during feeding. The metacyclic trypomastigotes of *T. cruzi* develop in the rectum of triatomine bugs and are infectious to mammals. Epimastogotes are able to multiply and colonize the whole intestinal tract of the vector. (Kollien and Schaub, 1998). After multiplying into trypomastigotes in the midgut of the flies they reach the salivary glands where they are transformed into epimastigotes and after multiplication are transformed back to metacylclic trypomastigote which are infective to human host. Upon feeding the metacyclic forms from flies enter serum and lymph where they multiply by binary fission. Slender forms change into stumpy forms which enter tsetse for transmission again.

Diagnosis is based on clinical signs and symptoms, xenodiagnosis, blood tests, serological tests. Signs of infection are swollen lymph nodes, spleen and liver, fever, headache, muscular weakness, drowsiness and finally unconsciousness leading to coma. Control programs are based on tsetse control, reservoir host control, chemotherapy and chemoprophylaxis. Vector control can be achieved by clearing of trees and bushes, application of insecticide, source reduction, personal protection measures and control of land use i.e. farming and grazing practices and control of reservoir hosts.

Leishmaniasis

This is another parasitic disease of tropical Africa, Asia and America. The WHO estimates that 350 million people are at risk of leishmaniasis worldwide (Guan, 1991). *Leishmania* spp. are kinetoplastids that develop intracellularly as amastigotes in vertebrate and promastigotes in vectors. Transmission takes place by blood sucking

sandflies of genus *Phlebotomus*. A fly becomes infected by taking blood from an infected mammal and in the midgut the amastigotes transform into promastigotes and multiply there. When promastigotes are reintroduced into a susceptible host they are phagocytised by macrophages and multiply by binary fission. Multiplication takes place and some of the amastigotes infect circulating phagocytic cells or those in the superficial layers of the skin waiting to be picked up by another sandfly during feeding. Diagnosis is based on clinical signs and symptoms and serological tests (Desjeux, 1992). The antibody based tests are Fluorescent antibody test (IFA) and ELISA, microscopic and histologic and parasite DNA tests by PCR examination of amastigote in tissue. Clinically, visceral leishmaniasis shows fever and anaemia. Control efforts are directed principally at control of sand fly vectors, elimination of reservoir hosts, immunization, treatment and personal protection.

Giardiasis

Giardia sp. is a cosmopolitan intestinal parasite of humans and animals. Human infections result from accidental ingestion of cysts from contaminated water, food, poor sanitary practices etc. *Giardia duodenalis* is the common parasite of humans. It is pathogenic giving rise to serious diarrhoea and is called giardiasis. Epidemics are associated with drinking water. *Giardia* sp. reproduces by binary fission and the trophozoites live in lumen of intestine. Diagnosis depends on faecal smear examination to detect cysts or trophozoites. Control measures include public health and personal protection measures by sewage treatment, treatment of drinking water, chemotherapeutic treatment of infected persons. Metronidazole is the drug of choice and chemotherapy includes use of three drugs quinacrine, metronidazole and furazolidine (Hill, 1993).

X. SYSTEMIC COCCIDIA
Toxoplasmosis

The extra-intestinal or tissue coccidian parasites comprise a large group of organisms that are important to human and animal health. The life cycle of the tissue coccidian involve two hosts usually a carnivore and a herbivore, *Toxoplasma gondii* causes toxoplasmosis. The definitive host is the domestic cat and the percentage of prevalence in humans could be upto 90%, as measured by serology. Congenital transmission in humans takes place when a pregnant woman inhales or ingests infective oocysts. Chemotherapy and control measures are suggested to tackle the infection. Toxoplasmosis affects domestic animals too (Frenkel, 1990).
Sarcocystis* and *Neurospora affects ruminants and domestic animals.

HELMINTH INFECTIONS

Helminths are wormlike organisms including nematodes (roundworms), cestodes (tapeworms) and trematodes (flukes). Leeches are also helminths and are considered ectoparasitic, since they attach themselves to the outside skin of their hosts.

XI.TREMATODE INFECTIONS

Trematodes or flukes are another class of helminths that have parasitic species. Flukes usually have an oral sucker, sometimes ringed with hooks to attach themselves to the host's tissues. It is estimated that over 40 million people are infected with flukes, approximately 21 million with lung flukes, 20 million with liver flukes and unknown millions with intestinal flukes (WHO, 1995c).

Schistosomiasis

Schistosoma mansoni, *S. japonicum* and *S. haematobium* are the most important human blood flukes causing schistosomiasis in tropical countries. Humans are definitive hosts, snails are the intermediate hosts and there are reservoir hosts too. These flukes infect human hosts directly by burrowing into the skin of a person wading or swimming in infected water. The adults reside in the liver and mesenteric venules and the eggs are passed to outside with feces or urine. The eggs in fresh water hatches into miracidia which find a suitable snail, and enter its body. The miracidia in snail's body transform from sporocyst to cercaria which later in water penetrates skin of the vertebrate host to continue the life cycle. Diagnosis of infection is based on clinical signs and symptoms and serological tests. Diarrhoea, fever, dermatitis are the symptoms. Damage to liver and urinary system occurs. Control measures include chemotherapy, health education, control of intermediate hosts, sanitary measures and environmental modification (WHO, 1993).

Some digenetic trematodes are of medical importance. *Fasciola hepatica* the common liver fluke causes fascioliasis or liver rot. Definitive hosts are mainly ruminants and humans are occasionally infected.

Clonorchiasis

Clonorchis sinensis causes Clonorchiasis mainly in oriental countries. Humans are the definitive hosts. Adults live in the liver. It is estimated that there are 7 million infections and 290 million people are at risk. Heavy infections are characterized by chills, fever, liver enlargement and splenomegaly and hepatomegaly. Control measures involve mainly chemotherapy, change of food habits (as metacercariae from snail infect fish) and snail control. Praziquantel is the drug of choice for the treatment of humans (Rim, 1986).

Paragonimiasis

Paragonimus westermani is an important lung fluke which causes paragonimiasis. Adult flukes are found in lungs. Carnivores and humans are the hosts. Snail is the intermediate host and crayfish and crabs are the second intermediate hosts. Cercariae exist in these hosts. Humans are infected by eating paratenic hosts such as wild boars and pigs. Lung lesions are caused in this disease. Diagnosis is by sputum and feces examination for detecting eggs. Serodiagnostic tests like complement fixation and precipitation tests are also helpful. Control measures include changing food habits, adequate cooking, controlling snails, drug treatment and eliminating reservoir hosts. The current drug of choice is praziquantel (WHO, 1995c).

XII.CESTODE INFECTIONS
Taeniasis, Cysticercosis

Tapeworms are a class of worms characterized by their flat, segmented bodies. The segments hold both male and female reproductive organs, allowing self-fertilization. Segments that contain fertilized eggs break off or dissolve, passing the eggs out of the host. Adult tapeworms typically reside in the intestinal tract of vertebrates, attaching themselves to the stomach lining with hooks or suckers on their head.

Common tapeworms that attack humans are *Taenia saginata*, *Taenia solium*, and *Diphyllobothrium latum*. These parasites use intermediate hosts, such as cattle, swine, and fish respectively, before entering the human body. Parasites such as these infect an intermediate host organism while in early developmental form. But they do not grow to maturity until they have been transmitted to the final host. In the case of *Taenia* sp., for example, tapeworm eggs are passed into cattle or swine through infected soil. They develop into an intermediary stage that embeds in the muscle and connective tissue of the animal. Infected animals that are processed for meat but improperly cooked still harbour the parasite, which is passed on when consumed by humans. The tapeworms develop into adults that attach to the intestinal lining of the host. Since tapeworms lack mouth and digestive tract all nutrients are acquired through the external surface. They are intestinal parasites of a wide range of hosts. *Taenia saginata* is the beef tapeworm and *T. solium* is the pork tapeworm. More than 39 million infections are estimated throughout the world. Humans become infected by eating cysticerci in raw or partially cooked beef. Diagnostic techniques involve stool examination and serological tests (Geerts, 1995). Abdominal pain and appendicitis are the severe reactions. Meat inspection and adequate cooking of meat are the precautionary measures to be adopted.

Diphyllobothriasis

Diphyllobothrium latum causes diphylllobothrisis or broad fish tapeworm disease in humans. Humans are the principal final host but animal reservoirs exist. It is a cosmopolitan species typically present in subarctic and temperate zones. It is the largest tapeworm that infects humans. First intermediate hosts are copepods usually cyclops. The second intermediate hosts are planktivorous fish which eat the infected copepods (Von Bonsdorff and Bylund, 1982). It affects the intestine and causes anemia. Tapeworm anaemia called pernicious anaemia is the most significant host-parasite interaction.

XIII. NEMATODE INFECTIONS
Filariasis

Filariasis is a major public health problem throughout many regions of the tropics. The debilitating disease is caused by several species of filarial nematodes including *Wuchereria bancrofti* and *Brugia malayi*, the agents of

lymphatic filariasis. The disease is associated with episodes of acute and chronic inflammation causing irreversible changes in the skin leading to elephantiasis of the affected limbs. This causes gross disfigurement and sexual dysfunction and the affected individuals are often ostracized from their societies. In January 2001, the World Health Organization launched a program aimed at global elimination by 2020 of the lymphatic filarial parasites *Wuchereria bancrofti* and *Brugia malayi*. Globally, over 1 billion persons are at risk of lymphatic filariasis (WHO, 2005b). Filariod nematodes are found in the blood, lymphatics or extra-intestinal tissues of the body and are transmitted by bloodsucking arthropods such as mosquitoes, blackflies and biting midges. These nematodes have evolved a mutualistic symbiosis with intracellular bacteria of the genus *Wolbachia*, which are required for nematode embryogenesis and survival. *Wolbachia* holds promise as a novel chemotherapeutic target for the control of filarial infection and disease (Tayler, 2002). Future prospects for the development of an anti-Wolbachia treatment regimen suitable for integration into mass drug administration programs are also under study (Johnston and Taylor, 2007)
Treatment: The treatment of filariaisis consists of chemotherapy directed against the adult worms (macrofilaricidal) and against the microfilariae (microfilaricidal) combined with symptomatic treatment to relieve the damage caused by the body's immunological reaction to dead or dying worms. Diethylcarbamazine also known as Heterazan, is a piperazine derivative used mainly as a microfilaricide even though it does have macrofilaricidal properties. It is usually effective in treating Tropical Pulmonary Eosinophilia (TPE) and its mechanism of action is thought to involve sensitizing the microfilariae to phagocytosis. Ivermectin is an effective microfilaricide and its mechanism of action is thought to involve the activation of GABA (gamma-aminobutyric acid) pathways and chloride channel permeability. Lymphatic incompetence due to filarial infection causes lymph stasis and causes development of opportunistic microbial infections (Michael et al., 1996). Antiseptic hygiene minimises these complications. Surgery can remove and provide bypasses around the damaged lymphatics.
Prevention: Transmission of LF can be interrupted by annual mass treatment with drugs that target mf??, the stage of the parasite that circulates in the blood. Mass drug administration is done with DEC and DEC –medicated salt on a campaign basis (WHO, 2006b).

Loiasis and Mansonella infections

Loiasis infection with the filarial nematode *Loa loa*, is limited to, and highly endemic in western and Central Africa. Characteristic clinical features include Calabar swellings (transient localized angioedema) and subconjunctival migration of the adult parasite (eyeworm). Infective larvae are transmitted to the host by the bite of an infected female fly of the *Chrysops* species. These migrate to the subcutaneous tissues including subconjunctiva. The microfilariae are released into the bloodstream from where they are ingested by the vector in a blood meal to complete the cycle. The diurnal periodicity of microfilariae in the host coincides with the feeding pattern of the vector *Chrysops* sp., which are day biting flies and live in the canopy of the rain forest (Beaver, 1989).

Onchocerciasis

The genus *Onchocerca* represents filarioid nematodes whose adults are found in subcutaneous areas and in connective tissues. *Onchocerca volvulus* causes onchocerciasis or river blindness. This infection is common in Africa and is caused by the vector blackfly (*Simulium* sp.). An estimated 17 million people are infected with *O. volvulus* in Africa alone. Globally about 85 million persons are at risk of infection and about 1 to 2 million persons have been blinded or have severe visual impairment (Duke, 1990). Adults lie in the subcutaneous tissues and start producing microfilariae (mf). The mf circulate in the skin and are transmitted by black flies where they develop to L3 in the flight muscles which in turn invade the host directly by puncturing the wound made by the vector. Diagnosis is mainly by finding mf in skin snips and ELISA tests and by clinical signs and symptoms. The effects on the host are damage to skin, lymph nodes and eye resulting in blindness. Reducing vectors by source reduction, aerial spraying of insecticides are the measures to be undertaken. The Global 2000 River Blindness Program and Onchocerciasis Elimination Program, are credited for establishing sustainable national programs for mass community- based ivermectin distribution (WHO, 1995b)

Dracunculiasis

Guinea worm disease (Dracunculiasis), caused by the nematode *Dracunculus medinensis*, is a disabling disease of rural parts of India, Yemen and Africa. People become infected when they drink water containing tiny freshwater crustaceans called copepods or water fleas, which act as intermediate hosts and harbour infective larvae. When the ingested copepods are killed by the digestive juices in the stomach, the larvae are released and move to the

small intestine where they penetrate the intestinal wall and migrate to the connective tissues of the thorax. The gravid females migrate to body surface and worms emerge from the lower extremities. In infected persons, the mature female reaches the skin and forms a painful papule in the dermis. It becomes a blister and ruptures exposing the worm. Local erythematic and urticarial rashes are the symptoms. On contact with freshwater, the worm discharges the motile larvae into the water which are ingested by copepods. The seasonal emergence of the worm coincides with the harvest or planting season. Stagnant sources of drinking water are the sites of transmission of infection. The global dracunculiasis eradication campaign (GDEC) began in 1981 when the Centres for Disease Control and Prevention (CDC) proposed that eradication of dracunculiasis was the ideal indicator with which to measure the success of International Drinking Water and Sanitation Decade (1981-1990), since this disease is only transmitted via unsafe drinking water (Hopkins and Foege, 1981). In 1986 the global incidence of this disease was estimated to be 3.2 million cases with about 100 million people living in areas at risk of disease but now it has been eradicated from many countries (Hopkins et al., 1991).

Toxocariasis

Human toxocariasis is frequently caused by infective larvae of the canine ascarid nematode *Toxocara canis*, a ubiquitous parasite of dogs in temperate and warm climates. The life-cycle involves one phase with developmental arrest in intermediate hosts (including humans) as well as a complete phase within its definitive canine host that involves vertical transmission from the dog to its offspring. Human infection can be prevented by public health measures to prevent dog faeces containing the eggs from contaminating the environment (Gillespie, 1988).

Trichinellosis

One of the most infamous nematodes is *Trichinella spiralis*. At one stage of its life cycle, this nematode lives in the muscle tissue of animals, including swine. Eventually, these organisms make their way into the intestinal tissue of humans who happen to ingest infected, undercooked pork.

Acanthocephala in Humans

Humans have been reported with Acanthocephalan parasites like *Acanthocephalus bufonis*, *A. rauschi*, *Bolbosoma* sp., *Corynosoma stromosum*, *Moniliformis moniliformis*, *Macracanthorhynchus hirudinaceus*. Intermediate hosts are crustaceans, cockroaches and fish. The fish foods pose a risk for both anisakosis and acanthocephalosis. Treatment with anthelmintics would be of effect.

Angiostrongyliasis

Angiostrongylus cantonensis causes eosinophilic meningitis, meningitis or tropical eosinpphilia in humans mainly in Southeast Asia and Africa. The final site of adult worms is the pulmonary arteries and central nervous system.

Enterobiasis

Enterobius vermicularis causes enterobiasis or pinworm infection in humans. The females oviposit in the perianal region; eggs have infective larvae in them and are carried to the mouth of the person on the fingers or are inhaled with dust. Eggs also hatch within the body of the host giving rise to autoinfection. Most of the development takes place in the tissue of the large intestine. Enterobiosis is treated as a household infection by treating the entire household members and maintaining cleanliness and personal hygiene (Warren, 1974).

Trichuriasis

Trichuris trichiura or whip-worm is an intestinal parasite. Eggs passed in the faeces contain a zygote and are not infectious until embryonation which takes place in the soil. Following ingestion, the larva is released in the stomach and passes into the intestine and crypts of the caecum. Warm damp soil is the best medium for transmission once it is faecally contaminated. The systemic consequences are anaemia and impaired growth (Cooper and Bundy, 1987).

Ascariasis

Ascaris lumbricoides is another nematode living in the small intestine of man and other animals. Human ascariosis is cosmopolitan. The largest parasitic roundworm, common among humans living in tropical developing countries, is *Ascaris lumbricoides.* This roundworm can grow to a length of 35 centimeters (15 inches) within the small intestine of its host. Estimates of worldwide human ascarid infections run as high as 1 billion. Eggs are shed in the faeces. They develop in egg capsules and hatch in stomach and small intestine; larvae reach hepatic portal system, lymphatics and liver. Migrate to lungs after moulting in liver, are released out during coughing swallowed and again reach small intestine. Larvae in the lung cause fever, chills and paroxymal cough. Intestinal pain, peritonitis, loss of weight nausea and eosinophilia are the other complications. Sanitary measures and chemotherapy are the control measures. Success of geohelminth infection control depends on close liaison with the community, the setting of clear goals, a commitment to the improvement in environmental infrastructure and sanitation, and local health education, as well as regular targeted population-based chemotherapy over a sustained period. Previously anthelmintic pyrantel was in use but due to parasite resistance it was switched over to single-dose albendazole. Two major pharmaceutical companies from their leadership are donating ivermectin and albendazole to the WHO-sponsored filariasis program (WDR, 1993).

Hookworm infections

The prevalence of hookworm infection in human population ranges from 450 million to 900 million persons worldwide (Chan, 1994). Because hookworm infection occurs in the poorest parts of the world, it interacts with other factors to impose a significant drain on the health of the people. Inadequate diet and continual blood loss sap their strength and sense of well being. Other helminthic and protozoan infections are common in warm climates, so that any one person is almost certain to harbor at least one pathogenic parasite so polyparasitism makes for health problems that are difficult to sort out and alleviate.

Ancylostoma duodenale and Necator americanus has been reported from humans to cause hookworm disease, hookworm anaemia and ancylostomosis. The eggs are passed with the faeces in an early stage of embryonation and develop to the ensheathed, nonfeeding stage. Infection takes place either by mouth or by penetration of the unbroken skin. Worms penetrating the skin migrate through the blood stream to the lungs, are coughed up, swallowed and carried to the small intestine, where they mature. The third stage larva (L3) ingested with food or water usually remain in the gut and do not migrate through the body. Migration via the lungs does occur but worms may reach the intestines by other routes as well. Adults are blood suckers. Many of the hookworms of medical and veterinary importance are cosmopolitan in tropical and subtropical areas. The ways in which hookworms cause damage to their hosts is by skin penetration, migration through the lungs and pharyngeal regions to intestine, causing inflammatory reactions. Anaemia results from depletion of iron and haemoglobin damage and physiological damage by blood loss due to chronic infection. Control measures include mass chemotherapy, sanitary disposal of human waste, wearing shoes, administration of iron salts and ensuring adequate dietary protein (Schad and Anderson, 1985).

Strongyloidiasis

Humans occasionally become infected with spirurids such as *Gongylonema pulchrum, Gnathostoma spinigerum* and *Thelazia californensis*. *G. spinigerum* is obtained by humans by accidentally ingesting infected insects (Neva, 1986). *G. pulchrum* has a more complex life cycle involving two intermediate hosts, a copepod and a cold blooded vertebrate. *Thelazia sp* is transferred to human eyes from mouthparts of muscoid flies such as *Fannia* spp. There are a few hundred cases of spiurid infections in humans but their prevalence is not well documented; all are zoonoses with their normal hosts being ruminants, suids and carnivores. *Dipetalonema perstans* occurs in North Africa and Africa. Its vectors are *Culicoides* spp. *Mansonella* occurs in tropical areas and is transmitted by *Culicoides* and *Simulium* spp. A parasitic roundworm that affects dogs is *Dirofilaria immitus*, or heartworm. This worm infects the heart tissues and eventually weakens the cardiac (heart) muscles to the point of failure. If left untreated, heart-worm can kill a dog.

XIV. VECTOR-BORNE DISEASES

A vector-borne disease is one in which the pathogenic microorganism is transmitted from an infected individual to another individual by an arthropod or other agent, sometimes with other animals serving as

intermediary hosts. The transmission depends upon the involvement of three factors namely, the pathologic agent, a virus, protozoa, bacteria or helminth (worm); the vector, which are commonly arthropods such as ticks or mosquitoes and the human host. In addition, intermediary hosts such as domesticated and/or wild animals often serve as a reservoir for the pathogen until susceptible human populations are exposed. Nearly half of the world's

Table 2. Some disease agents transmitted by mosquitoes to humans

Disease agent and disease	Vectors
Virus	
Eastern equine encephalitis	*Coquilletidia perturbans*
Venezuelan encephalitis	*Cx. pipiens*, etc.
Western equine encephalitis	*Cx. tarsalis*
Dengue	*Ae. aegypti, Ae. albopictus*
Japanese encephalitis	*Cx. tritaeniorhynchus*
St. Louis encephalitis	*Cx.pipiens, Cx. nigripalpus*
Yellow fever	*Ae. aegypti, Ae. africanus*
LaCrosse encephalitis	*Ae. triseriatus*
Protozoan	
Malaria	*Anopheles* spp.
Malaria	*Culex* spp.
Filarioid Nematode	
Wuchereria bancrofti	*Culex, Mansonia*
Brugia malayi	*Culex, Mansonia*
Dirofilaria immitis	*Culex, Aedes* spp.

population is infected by vector-borne diseases, resulting in high morbidity and mortality. The distribution of the incidence of vector-borne diseases is grossly disproportionate, with the overwhelming impact in developing countries located in tropical and subtropical areas. Weather, mainly temperature and humidity, affects vector population dynamics and disease transmission. Climate change might affect the distribution of vector-borne diseases as seen recently by the emergence of newer diseases as a result of global warming. A comprehensive model should consider both the direct impacts (such as changes in temperature or rainfall) and indirect impacts (such as changes in hydrology or agriculture) of global warming on the agent, vector, intermediary host, and the human host. New strategies for prevention and control of vector-borne diseases are emphasizing "Integrated Vector Management" - as an approach that reinforces linkages between health and environment, optimizing benefits to both (Dye, 1994).

Malaria and other vector-borne diseases are a major public health problem in WHO's SouthEast Asian

Region. In the wake of increasing resistance to both drugs and pesticides, there is a need to establish integrated vector management strategies that are less reliant on chemical methods of disease control. These strategies should involve other sectors and local communities in managing the ecosystem to reduce health risks and increase the sustainability of programmes to control vector-borne diseases.

Table 3. Diseases common among impoverished persons compared to prosperous persons in the same countries

Disease	Exposure
Chaga's disease	Poor housing and reduviid bug
Onchocerciasis	Black fly exposure
Leishmaniasis	Occupational exposure
Malaria	Poor housing location and anopheline mosquito exposure
Filariasis and anopheline mosquitoes)	Fetid water pooling in poor neighbourhoods breeding (culicine
African sleeping sickness	Crowding in tsetse-infested areas
Tuberculosis and leprosy	Crowded living conditions
Schistosomiasis	Exposure to infected water for work or transport
Respiratory and gastrointestinal diseases	Poor accesss to water for hygiene and drinking
Hantavirus pulmonary syndrome	Rodent-infested rural households
Sexually transmitted diseases (STDs) including HIV infection	Poverty and illiteracy

Ecology of vector-borne diseases

Research focuses upon the impact of environmental changes (climate change and habitat modifications, such as irrigation and deforestation) on the risk of some of the world's major vector-borne diseases (chiefly malaria in Africa and West Niles virus surveillance in the UK). These studies explore the temporal and spatial patterns of vectors, pathogen exposure and disease patterns in human communities in order to better inform public health policy makers about disease risk. Environmental management tools are also being developed and tested to reduce disease transmission, each method tailored to specific ecological conditions (e.g. mosquito larval control, improvements in house design and sanitation). Behavioural, molecular and physiological studies of disease vectors are also being carried out in order to better understand the mechanisms that govern the distribution and abundance of insect vectors and which may provide new targets that could be exploited for disease monitoring or control. The modern trend is to control vectors is by the large-scale application of biopesticides, mainly *Bacillus spp*. which have been successful as ecofriendly agents. Research studies show that these mosquitocidal bacteria are best produced on a cost-effective basis using enviromental bio-organic wastes (WHO, 1982b; Poopathi et al., 2003a, 2003b).

Global warming

Global warming is causing explosive reemergence of various vector-borne diseases The changing ecological conditions of the countries of the world is causing fundamental changes in the earth's heat budget which in turn is accelerating the global hydrologic cycle: warming of water, melting of ice and increase in water vapour increasing droughts and higher humidity condenses into heavier downpours and conducive breeding grounds for mosquitoes leading to disease outbreaks (Patz et al., 1995).

A major focus of the vector group is the development of simple tools for the control of vector-borne diseases (biocides, house-screening and source reduction). The group has recently received major awards (National Institute of Health, USA; Environmental Health Project of the USAID; Medical Research Council) for research programmes aimed at controlling malaria in Africa. This support reflects the group's position as one of the world's leading players in the study of the ecology and control of vector-borne diseases (WHO, 2006d).

XV. DISEASES CAUSED BY ARTHROPODS

Arthropods are organisms characterized by exterior skeletons and segmented bodies. Examples include the crustaceans, insects, and arachnids. The arthropods are the most diverse and widely distributed animals on the planet. Many arthropod species serve as carriers of bacterial and viral diseases, as intermediate hosts for protozoan and helminth parasites, and as parasites themselves. Certain insect species are the carriers of some of humanity's most dreaded diseases, including malaria, typhus, and plague (Markell et al., 1992). As consumers of agricultural crops and parasites of our livestock, insects are also humankind's number-one competitor for resources.

Diseases transmitted by Dipterans

These are important to humans for a variety of reasons. House fly and face fly are pests and also serve as mechanical or biological vectors of infectious agents. Tsetse fly transmits the agent causing African sleeping sickness; mosquitoes transmit malaria, lymphatic filariasis and viruses. Biting midges transmit filarioid nematodes and viruses such as bluetongue virus; tabanids transmit tularemia. Since these flies are blood suckers they can be serious pests regardless of whether they are vectors of infectious agents. *Toxorhychitis* spp, *Anopheles* spp, *Culex*, *Aedes*, *Mansonia*, etc. are blood feeders. Mosquitoes transmit arboviruses (arthropod borne viruses) such as Cache Valley virus, Crimean Haemorrhagic fever virus, Japanese encephalitis virus, dengue virus, Kyasanur forest disease etc (Table 2). All mosquitoes require water for the development of larvae and pupae. Their breeding habitats differ. In the biological transmission of disease agents of all sorts, the female is infected at one feeding, lays eggs and then must take another blood meal for transmission to occur. Biological and behavioural characteristics of mosquitoes are important in relation to their ability to serve as vectors of disease agents (Clasher, 1994). Economics, rapid transport and the adaptability of potential vectors intersect both in the distribution of vectors and their likelihood of transmitting a pathogen to humans. The feeding preferences of mosquitoes are important in their abilities to serve as vectors as well as their being pests of humans and domesticated animals. Black flies transmit *Onchocerca volvulus*. Biting midges transmit viruses and nematodes. *Musca domestica,* the house fly, serves as mechanical vector of various microbial agents such as the pathogenic enteric gram-negative bacteria *Salmonella* spp., and the dysentery amoeba, *Entamoeba histolytica.* Control measures include source reduction, insecticide use and screening of buildings (Goddard, 1996). The screwworms *Cochliomyia hominivorax* and *Chrysoma bessiana* are obligatory parasites and invade broken skin of the host, wound and sores. *Auchmeromyia luteola,* the Congo floor maggot, is the only blood sucking larva (haematophagous *Calliphorid*) that is known to attack humans**.** Mosquitoes are the most notorious carriers of disease and parasites. Female mosquitoes rely on warm-blooded hosts to serve as a blood meal to nourish their eggs. During the process of penetrating a host's skin with their long, sucking mouth parts, saliva from the mosquito is transferred into the bite area. Any viral, protozoan, or helminth infections carried in the biting mosquito can be transferred directly into the blood stream of its host. Among these diseases are malaria, yellow fever, filariasis, elephantiasis, and heartworm.

Diseases transmitted by flies

Flies also harbor diseases that can be transmitted to humans and other mammals when they bite to obtain a blood meal for themselves. For example, black flies can carry *Onchocerca* spp (which causes river blindness), sandflies can carry *Leishmania* spp that cause kala-azar and tsetse flies can carry the trypanosomes that cause sleeping sickness. Livestock, such as horses and cattle can be infected with a variety of botflies and warbles that infest and feed on the skin, throat, nasal passages, and stomachs of their hosts.

Diseases transmitted by fleas and lice

These are two of the most common and irritating parasitic insects of humans and livestock. Lice commonly live among the hairs of their hosts, feeding on blood. Some species are carriers of typhus fever. Fleas usually infest birds and mammals, and can feed on humans when they are transferred from pets or livestock. Fleas are known to carry a variety of devastating diseases, including the plague. Another prominent class of arthropods that contains parasitic species is the arachnids. Included in this group are spiders, scorpions, ticks, and mites.

Crustacea as disease agents

Freeliving copepods are intermediate hosts for tapeworms (Diaptomus and Cyclops for *Diphyllibothrium* spp.) nematodes (Cyclops for *Dracunculus medinensis*) and acanthocephalans. There are three sucking lice on humans *Pediculus humanus*, the body louse *Pediculus capitis* and the head louse *Pthirus pubis*, the crab louse. The body louse is the vector of various disease agents. Typhus is a rickettsial disease caused by *Rickettsia prowazeki*. Wherever there is crowding, interruption of sanitary services or displacement of people (like poverty, war or natural disasters), typhus appears. Mortality from endemic typhus ranges from 10 to 100% in any one outbreak. Epidemic relapsing fever caused by *Borellia recurentis* causes death rates less than 5%. Trench fever is a shortlived acute disease with a low mortality and is caused by the bacterium *Bartonella quintana*. This disease accompanies war and other human tragedies and has been seen in many countries.

Bed bugs as disease agents

Cimicids are vectors of disease agents and are cosmopolitan in distribution. *Cimex lectularis* is the human bedbug and *C.hemipterus* is the tropical bedbug. Fleas are also vectors of disease agents. *Cimex felis* or *C. canis* dwell on dogs and cats and give rise to flea allergic dermatitis. The transmission of tapeworms *Hymenolepis nana* and *Dipylidium caninum* by fleas to *Pulex irritans* the human flea transmits the bacillus *Yerstinia pestis* which causes plague. *P. irritans* transmits murine typhus caused by *Rickettsia typhi* to humans especially children can become a serous illness. Plague is a flea borne zoonosis caused by the bacterium *Yersinia pestis* and the disease is referred to as Bubonic Plague and Black Death. Urban outbreaks take place primarily through rats. It causes epidemics and can be treated by antibiotics.

Diseases borne by Ticks

Ticks are important as they feed on human blood and transmit disease agents like viruses, rickettsia, bacteria and filarioid nematodes. Through hematophagy they cause a great deal of damage by removing blood from their hosts damaging the skin at the site of feeding and causing tick paralysis in humans. The lifecycle involves egg, lava, nymph and adult. *Dercamentor andersoni* is the rocky mountain wood tick or the spotted fever tick and is the vector for Colorado tick fever and Rocky mountain spotted fever. Lyme borlliosis causing rheumatoid arthritis is caused by spirochaete, *Borellia burgdorferi*, transmitted by ixodid tick, *Ixodes scapularis*. Human granulocuytic ehrlichiosis is caused by *Ehrlichia equi* like rickettsial agent vectored by deer tick, *I. scapularis* (Wharton, 1976). Tick paralysis is an acute ascending paralysis that results when one or more ticks attach to engorge and humans are the common hosts. *Dermacentor* spp. and *Ixodes* spp. are the common ones that cause infection.

Diseases borne by Mites

Mites are very small arachnids that infest both plants and animals. One common type of mite is the chigger, which lives in grasses. As larvae, they may grab onto passing animals and attach themselves to the skin, often leading to irritating rashes or bite wounds. Scabies are another mite that causes mange in some mammals by burrowing into the skin and producing severe scabs, lesions, and loss of hair. Fleas are common parasitic insects that are known to carry a variety of devastating diseases, including the plague. Ticks also live their adult lives among grasses and short shrubs. They are typically larger than mites. The adult female tick attaches itself to an animal host for a blood meal. Tick bites themselves can be painful and irritating. More importantly, ticks can carry a number of diseases that affect humans. The most common of these diseases include Rocky Mountain spotted fever, Colorado tick fever and Lyme disease (Burgdofer, 1977).

The parasitic mites are cosmopolitan, feed on hosts with their chelicerae and take blood and cause damage by feeding. There is no head and the capitulum bears the mouthparts. The developmental pattern is egg, to larva to

nymph and to adult. They cause rashes and host-dust allergies. *Sarcoptes scabiei* var *humani* is the scab mite attacking humans (Wharton, 1976). Scrub typhus is caused by *Rickettsia tsutsugamushi* and the vector is *Leptotrombidium akamushi.* It causes rashes, fever, CNS abnormalities and mortality has also been reported. Treatment is by use of broad spectrum antibiotics.

XVI. CONTROL OF PARASITES AND DISEASES

Determinants of parasitic disease in indigenous communities are complex and the contributing factors include poverty, lack of health knowledge, poor environmental infrastructure and housing, remoteness from health services etc. To be successful, parasite control programs must be consistent, coordinated and sustained and accompanied by local health education. Improvements in health infrastructure, standard surveillance techniques, reporting and targets would enable monitoring of progress. Parasitic diseases can mainly be controlled by use of drugs, vaccines and antibiotics (Mayberry, 1996).The reduction of parasite burden would be achieved through coordinated, programmed use of proven, safe drugs at a community level and programs should be monitored for the development of drug resistance. Two critical factors for the success of such a program are political will, and a will on the part of the communities themselves, together with local healthcare providers, government and indigenous health organizations (Benneson, 1985). Leadership in parasite elimination programs should come from both the local and medical communities through an alliance of local people and public health, infectious diseases and practitioners.

Many parasitic infections can be treated by a variety of medical procedures, such as the use of antibiotics. The best way of controlling infection, however, is prevention. Scientists have developed and continue to test a number of drugs that can be taken as a barrier to certain parasites (Yekutiel, 1980). Other measures of control include improving sanitary conditions of water and food sources, proper cooking techniques, education about personal hygiene, and control of intermediate and vector host organisms (Table 3). Poorly designed irrigation and water systems, inadequate housing, poor waste disposal and water storage, deforestation and loss of biodiversity, all may be contributing factors to the most common vector-borne diseases including malaria, dengue and leishmaniasis (Esrey et al., 1991).

Integrated Vector Management (IVM)

Integrated vector management (IVM) strategies are designed to achieve the greatest disease control benefit in the most cost-effective manner, minimizing negative impacts on ecosystems (e.g. depletion of biodiversity) and adverse side-effects on public health from the excessive use of chemicals in vector control. Rather than relying on a single method of vector control, IVM stresses the importance of first understanding vector ecology and local patterns of disease transmission, and then choosing the appropriate vector control tools, from the range of options available. These include environmental management strategies that can reduce or eliminate vector breeding grounds altogether through improved design or operation of water resources development projects as well as use of biological control (e.g. bacterial larvicides and larvivorous fish) that target and kill vector larvae without generating the ecological impacts of chemical use (Bang, 1985). At the same time, when other measures are ineffective or not cost-effective, IVM makes judicious use of chemical methods of vector control, such as indoor residual sprays, space spraying, and use of chemical larvicides and adulticides. These reduce disease transmission by shortening or interrupting the lifespan of vectors. IVM provides a framework for improved personal protection/preventive strategies that combine environmental management and chemical tools for new synergies; e.g. insecticide-treated nets (ITNs) (WHO, 2004b). Trials using insecticide-treated bednets in some malaria-endemic African countries have shown very substantial reductions in child and infant mortality. IVM also supports effective, accessible and affordable disease diagnosis and treatment within the framework of a multi-disease control approach. IVM requires a multi-sectoral approach to vector-borne disease control (Rajagopalan and Das, 1987). For instance Health Impact Assessments infrastructure development, e.g. water resource, irrigation and agriculture, can help IVM is not a panacea. However, in many settings, the use of IVM strategies has yielded sustainable reductions in disease and transmission rates. In addition, certain IVM field experiences have been documented as cost-effective in terms of disease control and potential generators of economic co-benefits in terms of development and growth - although more work needs to be done linking health and economic outcomes (Sachs and Malaney, 2002).

Malaria and TB are becoming more difficult and more expensive to cure because resistance to the most widely available drugs is increasing. Mosquitoes have become resistant to many kinds of insecticides, which make eradication programmes prohibitively expensive for many countries and, in some regions; the parasites have also developed drug resistance. Recent developments in the field of molecular genetics have led to the identification of

genetic mutations that result in drug resistance. Multiple drug resistance is the consequence of an accumulation of mutations in these genes. The mutations can be identified by molecular methods and used to predict drug resistance in a matter of days. Furthermore, these methods are far more sensitive and allow hundreds of samples to be analysed simultaneously. The two most important diagnostic techniques are PCR and DNA finger printing. PCR (polymerase chain reaction) and dot blot hybridization provides an effective means of surveillance for drug resistance but it also indicates where alternative drugs are likely to be more effective for treating the individual patient.

XVII. ROLE OF HUMAN GENOMICS

Human genomics is now more than ever poised to ask and answer challenging questions pertaining to the relationship between the inherent variability observed in populations and human diseases. Whether it is susceptibility or resistance, sensitivity to drugs or indeed differences in the host response to infection, it is evident that common genetic variants will emerge as a key component of a comprehensive understanding of many infectious diseases (Risch, 2000). Even diseases considered to be affected by genetic modification. Major improvements in the technological platforms for genotyping the most common variants in the genome – the single nucleotide polymorphism (SNP) together with bioinformatics tools for manipulating large sets of data on genetic information now enable researchers to look comprehensively on the entire genome.

XVIII. INTERNATIONAL COLLABORATION

The success of these global initiatives will depend, to a large extent, upon precise and rapid surveillance for drug resistance without which control efforts will be ineffective (LeDuc, 1996). Drug resistant TB has developed largely where TB control authorities have poorly implemented the DOTS (Directly Observed Therapy, Short-course) strategy. According to the WHO, there is no cure for some of the known multi-drug resistant strains. There is a real danger that without new intervention and control strategies conventional drugs will become useless. Multi-drug resistant TB and malaria are global problems and no country is immune (Sudre et al., 1992).

XIX. CONCLUSION

Emerging infectious diseases- Need for surveillance

Infectious diseases are the leading cause of death worldwide. Emerging infectious diseases are defined as new, reemerging or drug-resistant infections whose incidence in humans has increased within the past two decades or whose incidence threatens to increase in the near future (Lederberg, 1996). The causes of resurgence of infectious diseases include changes in human industrial practices, economic development, and changes in land use, increase in international travel and commerce and adaptation of the microbes including development of resistance to antimicrobial agents (Table 4). Surveillance of infectious diseases is one of the key components of basic public health systems, since it provides the information necessary to allow the recognition of changes in trends of diseases and the appearance of new or unusual events. Prompt dissemination of surveillance data to those responsible for disease prevention and control is critically important. There are a number of sources of surveillance data on infectious diseases where a national surveillance system exists. Routine passive surveillance strategy focus on strengthening surveillance and response capability, addressing research priorities, improving prevention and control strategies, and strengthening the public health infrastructure at the local, state, and federal levels. Internationally only three diseases plague, cholera and yellow fever are officially reportable to the WHO as required by the International Health Regulations. Global eradication programs such as those underway for poliomyelitis and guinea worm infection (dracunculiasis) have surveillance activities as a critical component of their strategy. In addition, the WHO has also developed a strategy for addressing emerging infections (WDR, 1993). A major stimulus to the implementation of GFLP (Global and other programs, such as those aimed at the elimination of leprosy and Chagas' disease, was the successful global elimination of smallpox in 1977 and major advances in programs to control polio, measles, dracunculiasis (guinea worm) and onchocerciasis (river blindness). The Ebola virus outbreak in Zaire in 1995, plague in India in 1994, and leptospirosis in Nicaragua in the fall of 1995 underscores the importance of surveillance, prompt epidemiologic investigation, and availability of adequate diagnostic laboratory capacity. These outbreaks also provide evidence of the adverse effects on commerce and industry that infectious disease outbreaks can cause. The microbiology and pathology laboratory plays a critical role in recognizing new, emerging, and re-emerging infectious diseases by establishing specific causes for the disease syndromes seen by clinicians, and by reporting new or unusual pathogens that they encounter. The laboratory may also serve as a key surveillance point for information gathering and dissemination as is the case for antimicrobial resistance data. The availability of high -

quality diagnostic reagents may be the single greatest limiting factor to effectively addressing new and emerging infectious diseases, especially in the case of viral diseases. The WHO network of collaborating centres for arboviruses and hemorrhagic fevers involve 36 laboratories in 27 countries which were selected for their technical expertise and special capabilities, and they often serve as national reference centres for viral diseases (WHR, 1996).

Table 4. International emerging infections

1. Ebola hemorrhagic fever
2. Dengue hemorrhagic fever
3. Chikungunya fever
4. Venezuelan equine encephalitis
5. Cholera
6. Meningococcal meningitis
7. Lassa fever
8. *Streptococcus iniae* infection
9. Variant Creutzfeldt-Jakob disease
10. *Escherichia coli* O 157:H7 hemorrhagic colitis
11. *Salmonella hartford*
12. *Salmonella stanley*
13. Human granulocytic ehrlichiosis
14. *Cyclospora caytanensis*
15. Fluoroquinolone-resistant *Neisseria gonorrhoeae*
16. Raccoon rabies
17. *Cyclospora* gastroenteritis
18. Malaria
19. Yellow fever
20. Multi drug resistant TB

These laboratories are called upon to assist in the identification of new or unusual viruses and serve as referral facilities to many national laboratories. One of the most important aspects of a global strategy to monitor emerging diseases especially those caused by viruses is ensuring that the proper level of biosafety containment is available to allow safe handling of pathogenic organisms. The network of collaborating laboratories helps in active monitoring

for new and emerging infectious diseases. A key element in surveillance activities is the ability to rapidly and reliably exchange information on disease incidence and distribution. Future challenges would certainly include more problems with drug-resistant infections, the threat of another influenza pandemic and the likelihood of increasing problems of dengue hemorrhagic fever and the risk of urban yellow fever and global HIV epidemic and opportunistic infections (Tenover and Hughes, 1996). The roles of hepatitis B and C viruses in chronic liver disease and hepatocellular carcinoma, human papillomavirus in cervical cancer and *Helicobacter pylori* infection in peptic ulcer disease and gastric cancer are now well established. Responding to these threats will require strengthened surveillance and the formation of multidisciplinary response teams at the local, state, national and international levels with expertise in epidemiology, laboratory science, and disease control (MMWR, 1995).

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