Communicable Disease Control

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*This material is intended for educational use only by practicing health care workers or students and faculty in a health care field.*
Preface

This lecture note was written because there is currently no uniformity in the syllabus and, for this course additionally, available textbooks and reference materials for health students are scarce at this level and the depth of coverage in the area of communicable diseases and control in the higher learning health institutions in Ethiopia. The author hopes that the material will, to some extent, solve this problem. Although, this lecture note is prepared and intended for use primarily for nursing students, other health science students and health professionals can use it. After using this material, students are expected to be able to:

- describe the epidemiology and scope of communicable diseases in Ethiopia and factors involved in the transmission of communicable diseases;
- identify the preventive and control measures of each of the communicable diseases;
- play an active role in the prevention and control of communicable diseases;
- organize and implement effective health education on communicable diseases, and;
- participate in teaching junior staff and significant others in health courses on managing patients with communicable diseases.
In order to accomplish the above objectives, efforts have been made to address all the topics mentioned in the communicable disease outline of nursing students, including: epidemiology of communicable diseases in Ethiopia; definition and descriptions of the transmission, prevention and control of communicable diseases, air-borne diseases, vector-borne diseases, sexually-transmitted diseases, zoonotic diseases, and food-borne diseases (food poisoning and infection). The last chapter has a brief description of nursing principles in the management of communicable diseases.

Specific learning objectives and review questions have been set for each chapter. Moreover, each disease has been discussed in terms of its definition, infectious agent, epidemiology, clinical manifestation, diagnostic criteria, treatment, nursing care (for some diseases) and prevention and control methods. Important words or phrases in the text have been defined in the glossary. References used have been also listed at the end.
Acknowledgments

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**Abbreviations and Acronyms**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>AFB</td>
<td>Acid Fast Bacilli</td>
</tr>
<tr>
<td>AIDS</td>
<td>Acquired Immuno-Deficiency Syndrome</td>
</tr>
<tr>
<td>BCG</td>
<td>Bacillus of Calmate-Guirein</td>
</tr>
<tr>
<td>Bid</td>
<td><em>Bies in dies</em> (two times a day)</td>
</tr>
<tr>
<td>B. Sc.</td>
<td>Bachelor of Science degree</td>
</tr>
<tr>
<td>C°</td>
<td>Degree Celsius</td>
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<tr>
<td>CNS</td>
<td>Central Nervous System</td>
</tr>
<tr>
<td>CSF</td>
<td>Cerebro-spinal fluid</td>
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<tr>
<td>CT</td>
<td>Computerized Tomography</td>
</tr>
<tr>
<td>DEC</td>
<td>Diethylcarbamazin Citrate</td>
</tr>
<tr>
<td>DOTS</td>
<td>Directly Observed Treatment Short course</td>
</tr>
<tr>
<td>GIT</td>
<td>Gastro-intestinal Tract</td>
</tr>
<tr>
<td>HIV</td>
<td>Human Immuno-deficiency Virus</td>
</tr>
<tr>
<td>IgM</td>
<td>Immunoglobulin M.</td>
</tr>
<tr>
<td>IM</td>
<td>Intramuscular</td>
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<tr>
<td>IU</td>
<td>International Unit</td>
</tr>
<tr>
<td>IV</td>
<td>Intravenous</td>
</tr>
<tr>
<td>Kg</td>
<td>Kilogram</td>
</tr>
<tr>
<td>MOH</td>
<td>Ministry of Health</td>
</tr>
<tr>
<td>MRI</td>
<td>Magnetic Resonance Imaging</td>
</tr>
<tr>
<td>OPV</td>
<td>Oral Polio Vaccine</td>
</tr>
<tr>
<td>PO</td>
<td><em>Per os</em> (per mouth)</td>
</tr>
<tr>
<td>PTB⁺</td>
<td>Smear Positive Pulmonary Tuberculosis</td>
</tr>
<tr>
<td>QID</td>
<td><em>Quadris in dies</em> (four times a day)</td>
</tr>
<tr>
<td>Acronym</td>
<td>Term</td>
</tr>
<tr>
<td>---------</td>
<td>--------------------------------</td>
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<tr>
<td>STD</td>
<td>Sexually Transmitted Disease</td>
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<tr>
<td>STI</td>
<td>Sexually Transmitted Illness</td>
</tr>
<tr>
<td>TB</td>
<td>Tuberculosis</td>
</tr>
<tr>
<td>Tid</td>
<td><em>Tries in dies</em> (three times a day)</td>
</tr>
<tr>
<td>URTI</td>
<td>Upper Respiratory Tract Infection</td>
</tr>
<tr>
<td>USA</td>
<td>United States of America</td>
</tr>
<tr>
<td>WHO</td>
<td>World Health Organization</td>
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</tbody>
</table>
CHAPTER ONE

INTRODUCTION

1.1 Learning Objectives

At the end of this chapter, the student will be able to:
- Describe the burden of communicable diseases in Ethiopia.
- Define epidemiology and epidemiological terminologies.
- Identify the major communicable diseases that pose health problems in Ethiopia.

Diseases can be classified according to two major dimensions, namely the time course and cause. According to the time course, they are further classified as acute (characterized by a rapid onset and a short duration), and chronic disease (characterized by prolonged duration).

Based on the cause, diseases can be broadly categorized as infectious, (i.e. caused by living parasitic organisms such as viruses, bacteria, parasitic worms, insects, etc.), or as non-infectious (which are caused by something other than a living parasitic organism).

However, most of the common diseases in Africa are environmental diseases (infectious) due to infection by living
organisms. These are called communicable diseases, because they spread from person to person, or sometimes from animals to people. They occur at all ages but are most serious in childhood and they are to a great extent preventable. In developed countries where they have been prevented, other health conditions such as accidents and degenerative diseases become the most common. Therefore, communicable diseases remain very important in developing countries because:

- Many of them are very common
- Some of them are serious and cause death and disability
- Some of them cause widespread outbreaks of disease or epidemics
- Most of them are preventable by fairly simple means.
- Poor socio-economic status of the individuals makes them vulnerable to a variety of diseases
- Low educational status
- Lack of access to modern health care service

1.2 Epidemiology and Scope of Communicable Diseases in Ethiopia

During the past 70 years, there has been a dramatic fall in the incidence of infectious diseases, particularly in developed countries. This is due to several factors including:

- Immunization
- Anti-microbial chemotherapy
- Improved nutrition
- Better sanitation and housing

In less developed countries, however, especially in the tropics, infectious diseases continue to be one of the commonest causes of death, particularly in children. Ethiopia, as part of the developing world, has two big health problems. These are:

- Infectious diseases (communicable diseases) 80% of these can be prevented by simple sanitary measures.
- Nutritional problems

The magnitude of infectious diseases in Ethiopia can be seen from the 1993 E.C. MOH report. Accordingly:

- The top leading causes of outpatient visits were:
  - All types of malaria (10.4%)
  - Helminthiasis (6.7%)
  - Acute upper respiratory infection (6.5%)
  - Bronchopneumonia (5.5%)
  - Infections of skin and subcutaneous tissue (4.6%)
  - Dysentery (3.5%)
  - Tuberculosis of respiratory system (2.2%)
  - Sexually transmitted infection (2.2%)
  - Bronchitis, chronic and unqualified (1.9%)

- The top leading causes of admission were:
  - All types of malaria (14.8%)
- Pneumonia (8.9%)
- Tuberculosis of respiratory system (7.8%)
- Bacillary dysentery (1.6%)
- Gastroenteritis and colitis (1.5%)
- Meningitis (0.9%)

The top leading causes of deaths were:
- Tuberculosis of the respiratory system (10.1%)
- Pneumonia (7.3%)
- All types of malaria (4.6%)
- Bacillary dysentery (2.2%)
- Meningitis (1.5%)
- Gastroenteritis and colitis (1.1%)
- AIDS (0.8%)
- Leishmaniasis (0.5%)

Others, like yellow fever, acute febrile illnesses, trachoma (commonest cause of blindness in Ethiopia), and trypanosomiasis, are the major public health problems in our country.

1.3 Epidemiological Terms and Definitions

**Epidemiology** - the study of the frequency, distribution and determinants of disease and other health related conditions in human populations, and the application of this study to the
promotion of health and to the prevention and control of health problems.

Some of the components in the definition of epidemiology are:

- **“Populations”** Epidemiology focuses on the effects of disease on populations
- **“Disease and health related conditions”** Epidemiology indicates that everything around us and everything we do affects our health.
- **“Frequency”** shows that “epidemiology” is a quantitative science (e.g. occurrence of illness is measured using morbidity rates).
- **“Distribution”** refers to the occurrence of disease by place, person and time.
- **“Determinants”** These are factors that determine whether or not a person will get a disease.

The causes of diseases are classified epidemiologically as:

**Primary causes** - Factors that are necessary for a disease to occur, and in whose absence the disease will not occur (e.g. infectious agents, vitamin deficiencies).

1. **Contributing, predisposing, or aggravating factors** - Risk factors whose presence is associated with an increased probability that disease will occur/develop later (e.g. Poverty is the most powerful environmental determinant in the disease occurrence, Habit of cigarette smoking).
smoking leads to lung cancer. Having multiple sexual partners results in STI).

Definition of other epidemiological terms:

1. **Epidemics** - the occurrence of any health related condition in a given population in excess of the usual frequency in that population.
2. **Endemic** - a disease that is usually present in a population or in an area at a more or less stable level.
3. **Sporadic** - a disease that does not occur in that population, except at occasional and irregular intervals.
4. **Pandemic** - an epidemic disease which occurs world-wide.
5. **Disease** - a state of physiological or psychological dysfunction.
6. **Infection** - the entry and development or multiplication of an infectious agent in the body of man or animal.
7. **Contamination** – presence of living infectious agent upon articles.
8. **Infestation** – presence of living infectious agent on the exterior surface of the body.
9. **Infectious** - caused by microbes and can be transmitted to other persons.
10. **Infectious agent** - an agent capable of causing infection.
Review Questions

1. How do you compare the impact of communicable disease in Ethiopia with that of the developed world?

2. What are some of communicable diseases that create major health problems in Ethiopia?

3. Define the following terms:
   - Epidemiology
   - Epidemics
   - Endemic
   - Pandemic
   - Sporadic
   - Infection and infectious agent

4. Why are communicable diseases very important in Ethiopia?
CHAPTER TWO

DEFINITION, DESCRIPTION OF THE TRANSMISSION, PREVENTION AND CONTROL OF COMMUNICABLE DISEASES

2.1 Learning Objectives

At the end of this chapter the student will be able to:
- Define communicable disease.
- Describe the factors involved in the chain of communicable disease transmission.
- Identify the different levels of disease prevention.
- Apply the different control methods of communicable diseases.

2.2 Communicable Diseases

These are illnesses due to specific infectious agents or its toxic products, which arise through transmission of that agent, or its toxic products from an infected person, animal or inanimate reservoir to a susceptible host, either directly or
indirectly, through an intermediate plant or animal host, vector or inanimate environment.

### 2.3 Chain of Disease Transmission

This refers to a logical sequence of factors or links of a chain that are essential to the development of the infectious agent and propagation of disease. The six factors involved in the chain of disease transmission are:

- **a. Infectious agent (etiology or causative agent)**
- **b. Reservoir**
- **c. Portal of exit**
- **d. Mode of transmission**
- **e. Portal of entry**
- **f. Susceptible host**

**a. Infectious agent:** An organism that is capable of producing infection or infectious disease. On the basis of their size, etiological agents are generally classified into:

- **Metazoa** (multicellular organisms), (e.g. Helminths).
- **Protozoa** (Unicellular organisms) (e.g. Ameobae)
- **Bacteria** (e.g. Treponema pallidum, Mycobacterium tuberculosis, etc.)
- **Fungus** (e.g. Candida albicans)
- **Virus** (e.g. Chickenpox, polio, etc.)
b. Reservoir of infection: Any person, animal, arthropod, plant, soil or substance (or combination of these) in which an infectious agent normally lives and multiplies, on which it depends primarily for survival and where it reproduces itself in such a manner that it can be transmitted to a susceptible host.

Types of reservoirs

1. Man: There are a number of important pathogens that are specifically adapted to man, such as: measles, smallpox, typhoid, meningococcal meningitis, gonorrhea and syphilis. The cycle of transmission is from human to human.

2. Animals: Some infective agents that affect man have their reservoir in animals. The term “zoonosis” is applied to disease transmission from animals to man under natural conditions. For example:
   - Bovine tuberculosis - cow to man
   - Brucellosis - Cows, pigs and goats to man
   - Anthrax - Cattle, sheep, goats, horses to man
   - Rabies - Dogs, foxes and other wild animals to man
   Man is not an essential part (usual reservoir) of the life cycle of the agent.
   Animal ........ Animal...........Animal
   ↓
   Human
3. **Non-living things as reservoir:** Many of the agents are basically saprophytes living in soil and fully adapted to live freely in nature. Biologically, they are usually equipped to withstand marked environmental changes in temperature and humidity.

E.g.  
- Clostridium botulinum etiologic agent of Botulism  
- Clostridium tetani etiologic agent of Tetanus  
- Clostridium welchi etiologic agent of gas gangrene

c. **Portal of exit (mode of escape from the reservoir):** This is the site through which the agent escapes from the reservoir. Examples include:
- GIT: typhoid fever, bacillary dysentery, amoebic dysentery, cholera, ascariasis, etc.
- Respiratory: tuberculosis, common cold, etc.
- Skin and mucus membranes: Syphilis

d. **Mode of transmission (mechanism of transmission of infection):** Refers to the mechanisms by which an infectious agent is transferred from one person to another or from a reservoir to a new host. Transmission may be direct or indirect.

1. **Direct transmission:** Consists of essentially immediate transfer of infectious agents from an infected host or reservoir to an appropriate portal of entry. This could be:
a. Direct Vertical
Such as: transplacental transmission of syphilis, HIV, etc.

b. Direct horizontal
Direct touching, biting, kissing, sexual intercourse, droplet spread onto the conjunctiva or onto mucus membrane of eye, nose or mouth during sneezing coughing, spitting or talking; Usually limited to a distance of about one meter or less.

2. Indirect transmission
a. Vehicle-borne transmission: Indirect contact through contaminated inanimate objects (fomites) like:
- Bedding, toys, handkerchiefs, soiled clothes, cooking or eating utensils, surgical instruments.
- Contaminated food and water
- Biological products like blood, serum, plasma or IV-fluids or any substance serving as intermediate means by which an infectious agent is transported and introduced into a susceptible host through a suitable portal of entry. The agent may or may not multiply or develop in the vehicle before it is introduced into man.

b. Vector-borne transmission: Occurs when the infectious agent is conveyed by an arthropod (insect) to a susceptible host.
1. **Mechanical transmission**: The arthropod transports the agent by soiling its feet or proboscis, in which case multiplication of the agent in the vector does not occur. (e.g. common house fly.)

2. **Biological transmission**: This is when the agent multiplies in the arthropod before it is transmitted, such as the transmission of malaria by mosquito.

C. **Air-borne transmission**: Dissemination of microbial agent by air to a suitable portal of entry, usually the respiratory tract. Two types of particles are implicated in this kind of spread: dusts and droplet nuclei.

**Dust**: small infectious particles of widely varying size that may arise from soil, clothes, bedding or contaminated floors and be resuspended by air currents.

**Droplet nuclei** : Small residues resulting from evaporation of fluid (droplets emitted by an infected host). They usually remain suspended in the air for long periods of time.

e. **Portal of entry**: The site in which the infectious agent enters to the susceptible host. For example:

- Mucus membrane
- Skin
- Respiratory tract
- GIT
- Blood

f. Susceptible host (host factors): A person or animal lacking sufficient resistance to a particular pathogenic agent to prevent disease if or when exposed. Occurrence of infection and its outcome are in part determined by host factors. The term “immunity” is used to describe the ability of the host to resist infection.

Resistance to infection is determined by non-specific and specific factors:

Non-specific factors
- Skin and mucus membrane
- Mucus, tears, gastric secretion
- Reflex responses such as coughing and sneezing.

Specific factors
- Genetic-hemoglobin resistant to Plasmodium falciparum

Naturally acquired or artificially induced immunity. Acquired immunity may be active or passive.

Active immunity- acquired following actual infection or immunization.
Passive immunity- pre-formed antibodies given to the host.
2.4 Carrier and Its Type

A carrier is an infected person or animal who does not have apparent clinical disease but is a potential source of infection to others.

a. **Healthy or asymptomatic carriers**: These are persons whose infection remains unapparent. For example, in poliovirus, meningococcus and hepatitis virus infections, there is a high carrier rate.

b. **Incubatory or precocious carriers**: These are individuals or persons who excrete the pathogen during the incubation period (i.e. before the onset of symptoms or before the characteristic features of the disease are manifested).

   *E.g.* Measles, mumps, chickenpox and hepatitis.

c. **Convalescent Carriers**: These are those who continue to harbor the infective agent after recovering from the illness. *E.g.* Diphtheria, Hepatitis B virus.

d. **Chronic Carriers**: The carrier state persists for a long period of time. *E.g.* Typhoid fever, Hepatitis B virus infection.

2.5 Time Course of Infectious Diseases

**Incubation period**: It is the interval of time between infection of the host and the first appearance of symptoms and signs of the disease.
**Prodormal period**: It is the interval between the onset of symptoms of an infectious disease and the appearance of characteristic manifestations. For example, in a measles patient, fever and coryza occur in the first three days and Koplick spots in the buccal mucosa and characteristic skin lesions appear on the fourth day.

**Period of communicability**: The period during which that particular communicable disease (infectious agent) is transmitted from the infected person to the susceptible host.

### 2.6 Levels of Prevention

The different points in the progression of a disease at which one can intervene can be classified according to three levels of prevention: primary, secondary, and tertiary.

**a. Primary prevention**: The objectives here are to promote health, prevent exposure, and prevent disease.

**Health promotion**: This consists of general non-specific interventions that enhance health and the body’s ability to resist disease, such as measures aimed at the improvement of socio-economic status through the provision of adequately-paid jobs, education and vocational training, affordable and adequate housing, clothing, and food, old-age pension benefits; emotional and social support, relief of stress, etc. In
short it is any intervention that promotes a healthier and happier life.

**Prevention of exposure:** This includes actions such as the provision of safe and adequate water, proper excreta disposal, vector control, safe environment at home (e.g., proper storage of insecticides and medicines, out of children’s reach), at school and at work (e.g., proper ventilation, monitoring of harmful substances in factories), and on the streets (e.g., driver licensing laws).

**Prevention of disease:** This occurs during the latency period between exposure and the biological onset of disease. An example for this is immunization. Immunization against an infectious organism does not prevent it from invading the immunized host, but prevents it from establishing an infection. Active immunization means exposing the host to a specific antigen against which it will manufacture its own protective antibodies after an interval of about three weeks (during which the immunized person remains susceptible to the disease). Passive immunization means providing the host with the antibodies necessary to fight against disease. Both forms of immunization act after exposure. However, for active immunization to be protective, the timing of its administration must be at least three weeks prior to exposure. Passive immunization, on the other hand,
is commonly given after exposure has occurred (as in the case of exposure to rabies or tetanus), or shortly before an exposure is expected, as in the administration of immune globulin to prevent viral hepatitis A).

Breastfeeding is an example of an intervention that acts at all three levels of primary prevention:

- **Health promotion**: by providing optimal nutrition for a young child, either as the sole diet up to four months of age, or as a supplement in later months.

- **Prevention of exposure**: by reducing exposure of the child to contaminated milk.

- **Prevention of disease after exposure**: by the provision of anti-infective factors, including antibodies, white blood cells, and others.

b. **Secondary prevention**: After the biological onset of disease, but before permanent damage sets in, we speak of secondary prevention. The objective here is to stop or slow the progression of disease so as to prevent or limit permanent damage, through the early detection and treatment of disease. (e.g. breast cancer (prevention of the invasive stage of the disease), trachoma (prevention of blindness), and syphilis (prevention of tertiary or congenital syphilis))
c. **Tertiary prevention**: After permanent damage has set in, the objective of tertiary prevention is to limit the impact of that damage. The impact can be physical, psychological, social (social stigma or avoidance by others), and financial. Rehabilitation refers to the retraining of remaining functions for maximum effectiveness, and should be seen in a very broad sense, not simply limited to the physical aspect. Thus the provision of special disability pensions would be a form of tertiary prevention.

### 2.7 Communicable Disease Control

This refers to the reduction of the incidence and prevalence of communicable disease to a level where it cannot be a major public health problem.

**Methods of Communicable Disease Control**

There are three main methods of controlling communicable diseases:

1. **Elimination of the Reservoir**
   a. **Man as reservoir**: When man is the reservoir, eradication of an infected host is not a viable option. Instead, the following options are considered:
• **Detection and adequate treatment of cases:** arrests the communicability of the disease (e.g. Treatment of active pulmonary tuberculosis).

• **Isolation:** separation of infected persons for a period of communicability of the disease. Isolation is indicated for infectious disease with the following features:
  - High morbidity and mortality
  - High infectivity

• **Quarantine:** limitation of the movement of apparently well person or animal who has been exposed to the infectious disease for a duration of the maximum incubation period of the disease.

b. **Animals as reservoir:** Action will be determined by the usefulness of the animals, how intimately they are associated to man and the feasibility of protecting susceptible animals. For example:

  • Plague: The rat is regarded as a pest and the objective would be to destroy the rat and exclude it from human habitation.
  • Rabies: Pet dogs can be protected by vaccination but stray dogs are destroyed.
  • Infected animals used for food are examined and destroyed.

c. **Reservoir in non-living things:** Possible to limit man’s exposure to the affected area (e.g. Soil, water, forest, etc.).
2. ** Interruption of transmission  
This involves the control of the modes of transmission from the reservoir to the potential new host through:  
- Improvement of environmental sanitation and personal hygiene  
- Control of vectors  
- Disinfections and sterilization  

3. **Protection of susceptible host:** This can be achieved through:  
- Immunization: Active or Passive  
- Chemo-prophylaxis- (e.g. Malaria, meningococcal meningitis, etc.)  
- Better nutrition  
- Personal protection. (e.g. wearing of shoes, use of mosquito bed net, insect repellents, etc.)
Review Questions

1. State the six important factors that involve the chain of communicable diseases transmission.
2. Describe the three levels of disease prevention.
3. What are the methods used to control communicable diseases?
CHAPTER THREE

ORAL-FECAL TRANSMITTED DISEASES

3.1 Learning Objectives

At the end of this chapter, students will be able to:

- Identify the five important “Fs” in oral-fecal disease transmission.
- State diseases transmitted mainly in water and in soil.
- List diseases commonly transmitted by having direct contact with feces.
- Participate in the diagnosis and treatment of cases.
- Implement preventive and control methods of oral-fecal transmitted diseases.

3.2 Introduction

What the diseases in this group have in common is that the causative organisms are excreted in the stools of infected persons (or, rarely, animals). The portal of entry for these diseases is the mouth.
Therefore, the causative organisms have to pass through the environment from the feces of an infected person to the gastro-intestinal tract of a susceptible person. This is known as the fece-oral transmission route. Oral-oral transmission occurs mostly through unapparent fecal contamination of food, water and hands.

As indicated in the schematic diagram below, food takes a central position; it can be directly or indirectly contaminated via polluted water, dirty hands, contaminated soil, or flies.

![Schematic diagram of fecal-oral disease transmission]

Fig. 3.1 The five “Fs” which play an important role in fecal oral diseases transmission (finger, flies, food, fomites and fluid). (From Eshuis, Manschot, 1978, Communicable Diseases: A Manual for Rural Health Workers, African Medical and Research Association, Nairobi, Kenya)

### 3.3 Feces Mainly in Water

The diseases in this group are mainly transmitted through fecally contaminated water rather than food.
3.3.1 Typhoid fever

Definition
A systemic infectious disease characterized by high continuous fever, malaise and involvement of lymphoid tissues.

Infectious agent
Salmonella typhi
Salmonella enteritidis (rare cause)

Epidemiology
Occurrence- It occurs worldwide, particularly in poor socio-economic areas. Annual incidence is estimated at about 17 million cases with approximately 600,000 deaths worldwide. In endemic areas the disease is most common in preschool and school aged children (5-19 years of age).

Reservoir- Humans

Mode of transmission- By water and food contaminated by feces and urine of patients and carriers. Flies may infect foods in which the organisms then multiply to achieve an infective dose.

Incubation period –1-3 weeks
Period of communicability - As long as the bacilli appear in excreta, usually from the first week throughout convalescence. About 10% of untreated patients will discharge bacilli for 3 months after onset of symptoms, and 2%-5% become chronic carriers.

Susceptibility and resistance - Susceptibility is general and increased in individuals with gastric achlorhydria or those who are HIV positive. Relative specific immunity follows recovery from clinical disease, unapparent infection and active immunization but inadequate to protect against subsequent ingestion of large numbers of organisms.

Clinical manifestation
First week - Mild illness characterized by fever rising stepwise (ladder type), anorexia, lethargy, malaise and general aches. Dull and continuous frontal headache is prominent. Nose bleeding, vague abdominal pain and constipation in 10% of patients.

Second week - Sustained temperature (fever). Severe illness with weakness, mental dullness or delirium, abdominal discomfort and distension. Diarrhea is more common than first week and feces may contain blood.
Third week- Patient continues to be febrile and increasingly exhausted. If no complications occur, patient begins to improve and temperature decreases gradually.

Clinical manifestations suggestive of typhoid fever

- **Fever**- Sustained fever (ladder fashion)
- **Rose spots**- Small pallor, blanching, slightly raised macules usually seen on chest and abdomen in the first week in 25% of white people.
- **Relative bradycardia**- Slower than would be expected from the level of temperature.
- **Leucopoenia**- White cell count is less than 4000/mm$^3$ of blood.

Diagnosis

- Based on clinical grounds but this is confused with wide variety of diseases.
- Widal reaction against somatic and flagellar antigens.
- Blood, feces or urine culture.

Treatment

1. Ampicillin or co-trimoxazole for carriers and mild cases.
2. Chloramphenicol or ciprofloxacin or ceftriaxone for seriously ill patients.
Nursing care
1. Maintain body temperature to normal.
2. Apply comfort measures.
3. Follow side effects of drugs.
4. Monitor vital signs.
5. Follow strictly enteric precautions:
   - wash hands
   - wear gloves
   - teach all persons about personal hygiene
6. Observe the patient closely for sign and symptoms of
   - bowel perforation
   - erosion of intestinal ulcers
   - sudden pain in the lower right side of the abdomen
   - abdominal rigidity
   - sudden fall of temperature and blood pressure
7. Accurately record intake and output.
8. Provide proper skin and mouth care.

Prevention and control
1. Treatment of patients and carriers
2. Education on handwashing, particularly food handlers, patients and childcare givers
4. Provision of safe and adequate water
5. Safe handling of food.
6. Exclusion of typhoid carriers and patients from handling of food and patients
7. Immunization for people at special risk (e.g. Travelers to endemic areas)

8. Regular check-up of food handlers in food and drinking establishments

3.3.2 Bacillary Dysentery (Shigellosis)

Definition
An acute bacterial disease involving the large and distal small intestine, caused by the bacteria of the genus shigella.

Infectious agent
Shigella is comprised of four species or serotypes.
- Group A= Shigella dysenterae (most common cause)
- Group B= Shigella flexneri
- Group C= Shigella boydii
- Group D= Shigella sonnei

Epidemiology
Occurrence- It occurs worldwide, and is endemic in both tropical and temperate climates. Outbreaks commonly occur under conditions of crowding and where personal hygiene is poor, such as in jails, institutions for children, day care
centers, mental hospitals and refugee camps. It is estimated that the disease causes 600,000 deaths per year in the world. Two-thirds of the cases, and most of the deaths, are in children under 10 years of age.

Reservoir- Humans

Mode of transmission- Mainly by direct or indirect fecal-oral transmission from a patient or carrier. Transmission through water and milk may occur as a result of direct fecal contamination. Flies can transfer organisms from latrines to a non-refrigerated food item in which organisms can survive and multiply.

Incubation period- 12 hours-4 days (usually 1-3 days)

Period of communicability- During acute infection and until the infectious agent is no longer present in feces, usually within four weeks after illness.

Susceptibility and resistance- Susceptibility is general. The disease is more severe in young children, the elderly and the malnourished. Breast-feeding is protective for infants and young children.
Clinical Manifestation
- Fever, rapid pulse, vomiting and abdominal cramp are prominent.
- Diarrhea usually appears after 48 hours with dysentery supervening two days later.
- Generalized abdominal tenderness.
- Tenesmus is present and feces are bloody, mucoid and of small quantity.
- Dehydration is common and dangerous - it may cause muscular cramp, oliguria and shock.

Diagnosis
- Based on clinical grounds
- Stool microscopy (presence of pus cells)
- Stool culture confirms the diagnosis

Treatment
1. Fluid and electrolyte replacement
2. Co-trimoxazole in severe cases or Nalidixic acid in the case of resistance.

Prevention and control
1. Detection of carriers and treatment of the sick will interrupt an epidemic.
2. Handwashing after toilet and before handling or eating food.
3. Proper excreta disposal especially from patients, convalescent and carriers.
4. Adequate and safe water supply.
5. Control of flies.
6. Cleanliness in food handling and preparation.

### 3.3.3 Amoebiasis (Amoebic Dysentery)

**Definition**
An infection due to a protozoan parasite that causes intestinal or extra-intestinal disease.

**Infectious agent**
Entamoeba histolytica

**Epidemiology**
**Occurrence** - worldwide but most common in the tropics and sub-tropics. Prevalent in areas with poor sanitation, in mental institutions and homosexuals. Invasive amoebiasis is mostly a disease of young people (adults). Rare below 5 years of age, especially below 2 years.

**Mode of transmission** – Fecal-oral transmission by ingestion of food or water contaminated by feces containing the cyst. Acute amoebic dysentery poses limited danger.
Incubation period- Variable from few days to several months or years; commonly 2-4 weeks.

Period of communicability- During the period of passing cysts of E. histolytica, which may continue for years.

Susceptibility and resistance- Susceptibility is general. Susceptibility to reinfection has been demonstrated but is apparently rare.

Life cycle

Fig. 3.2 Transmission and life cycle of Entamoeba histolytica. (From Monica Chesbrough, 1998, District Laboratory Practice in Tropical Countries, Part One, Cambridge University Press, London.)
Clinical Manifestation
- Starts with a prodromal episode of diarrhea, abdominal cramps, nausea, vomiting and tenesmus.
- With dysentery, feces are generally watery, containing mucus and blood.

Diagnosis
- Demonstration of etamoeba histolytica cyst or trophozoite in stool.

Treatment
1. Metronidazole or Tinidazole

Prevention and control
1. Adequate treatment of cases
2. Provision of safe drinking water
3. Proper disposal of human excreta (feces) and handwashing following defecation.
4. Cleaning and cooking of local foods (e.g. raw vegetables) to avoid eating food contaminated with feces.

3.3.4 Giardiasis

Definition
A protozoan infection principally of the upper small intestine associated with symptoms of chronic diarrhea, steatorrhea,
abdominal cramps, bloating, frequent loose and pale greasy stools, fatigue and weight loss.

**Infectious agent**
Giardia lamblia

**Epidemiology**

**Occurrence**- Worldwide distribution. Children are more affected than adults. The disease is highly prevalent in areas of poor sanitation.

**Reservoir**- Humans

**Mode of transmission**- Person to person transmission occurs by hand to mouth transfer of cysts from feces of an infected individual especially in institutions and day care centers.

**Period of communicability**- Entire period of infection, often months.

**Susceptibility and resistance**- Asymptomatic carrier rate is high. Infection is frequently self-limited. Persons with AIDS may have more serious and prolonged infection.
Life cycle

**TRANSMISSION**
1. Cysts ingested in food, water or from hands contaminated with feces.

**ENVIRONMENT**
6. Feces containing infective cysts contaminate the environment.

**HUMAN HOST**
2. Cysts excyst, forming trophozoites
3. Multiply in intestine
4. Trophozoites encyst.
5. Infective cysts passed in feces. *
* trophozoites passed in feces disintegrate.

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**Clinical Manifestation**
- Ranges from asymptomatic infection to severe failure to thrive and mal-absorption.
- Young children usually have diarrhea but abdominal distension and bloating are frequent.
• Adults have abdominal cramps, diarrhea, anorexia, nausea, malaise, bloating, many patients complain of sulphur testing (belching).

Diagnosis
• Demonstration of Giardia lamblia cyst or trophozoite in feces.

Treatment
1. Metronidazole or Tinidazole

Prevention and control
1. Good personal hygiene, and handwashing before food and following toilet use
2. Sanitary disposal of feces
3. Protection of public water supply from contamination of feces
4. Case treatment
5. Safe water supply

3.3.5 Cholera

Definition
An acute illness caused by an enterotoxin elaborated by vibrio cholerae.
Infectious agent
Vibrio cholerae

Epidemiology
Occurrence- has made periodic outbreaks in different parts of the world and given rise to pandemics. Endemic predominantly in children.

Reservoir- Humans

Mode of transmission- by ingestion of food or water directly or indirectly contaminated with feces or vomitus of infected person.

Incubation period- from a few hours to 5 days, usually 2-3 days.

Period of communicability- for the duration of the stool positive stage, usually only a few days after recovery. Antibiotics shorten the period of communicability.

Susceptibility and resistance- Variable. Gastric achlorhydria increases risk of illness. Breast-fed infants are protected.
Clinical Manifestation
- Abrupt painless watery diarrhea; the diarrhea looks like rice water.
- In severe cases, several liters of liquid may be lost in few hours leading to shock.
- Severely ill patients are cyanotic, have sunken eyes and cheeks, scaphoid abdomen, poor skin turgor, and thready or absent pulse.
- Loss of fluid continues for 1-7 days.

Diagnosis
- Based on clinical grounds
- Culture (stool) confirmation

Treatment
1. Prompt replacement of fluids and electrolytes
   - Rapid IV infusions of large amounts
   - Isotonic saline solutions alternating with isotonic sodium bicarbonate or sodium lactate.
2. Antibiotics like tetracycline dramatically reduce the duration and volume of diarrhea resulting in early eradication of vibrio cholerae.

Nursing care
1. Wear gown and glove.
2. Wash your hands.
3. Monitor output including stool output.
4. Protect the patient family by administering Tetracycline.
5. Health education.

**Prevention and control**
1. Case treatment
2. Safe disposal of human excreta and control of flies
3. Safe public water supply
4. Handwashing and sanitary handling of food
5. Control and management of contact cases

**3.3.6 Infectious hepatitis**
*(Viral hepatitis A, Epidemic hepatitis, type A hepatitis)*

**Definition**
An acute viral disease characterized by abrupt onset of fever, malaise, anorexia, nausea and abdominal discomfort followed within a few days by jaundice.

**Infectious agent**
Hepatitis A virus

**Epidemiology**
**Occurrence**- Worldwide distribution in sporadic and epidemic forms. In developing countries, adults are usually immune and
epidemics of HA are uncommon. Infection is common where environmental sanitation is poor and occurs at an early age.

Reservoir- Humans.

Mode of transmission- Person to person by fecal-oral route. Through contaminated water and food contaminated by infected food handlers.

Incubation period- 15-55 days, average 28-30 days.

Period of communicability- High during the later half of the incubation period and continuing for few days following onset of jaundice. Most cases are non-infectious following first week of jaundice.

Susceptibility and resistance- Susceptibility is general. Immunity following infection probably lasts for life.

Clinical manifestation
- Abrupt onset of fever, malaise, anorexia, nausea and abdominal discomfort, followed in few days by jaundice.
- Complete recovery without sequel or recurrence as a rule.

Diagnosis
- Based on clinical and epidemiological grounds
- Demonstration of IgM (IgM anti-HAV) in the serum of acutely or recently ill patients.

**Treatment**
Symptomatic: Rest, high carbohydrate diet with low fat and protein.

**Prevention and control**
1. Public education about good sanitation and personal hygiene, with special emphasis on careful handwashing and sanitary disposal of feces.
2. Proper water treatment and distribution systems and sewage disposal.
3. Proper management of day care centers to minimize possibility of fecal-oral transmission.
4. HA vaccine for all travelers to intermediate or highly endemic areas.
5. Protection of day care centers’ employees by vaccine.

### 3.4 Feces Mainly in Soil

The diseases in this category are mainly transmitted through fecal contamination of soil. These infections are acquired through man’s exposure to fecally contaminated soil.
3.4.1 Ascariasis

Definition
A helminthic infection of the small intestine generally associated with few or no symptoms.

Infectious agent
Ascaris lumbricoides.

Epidemiology
Occurrence- The most common parasite of humans where sanitation is poor. School children (5-10 years of age) are most affected. Highly prevalent in moist tropical countries.

Reservoir- Humans; ascarid eggs in soil.

Mode of transmission- Ingestion of infective eggs from soil contaminated with human feces or uncooked produce contaminated with soil containing infective eggs but not directly from person to person or from fresh feces.

Incubation period- 4-8 weeks.

Period of communicability- As long as mature fertilized female worms live in the intestine. Usual life span of the adult worm is 12 months.
Susceptibility and resistance- Susceptibility is general.

Life Cycle

TRANSMISSION
1. Infective eggs ingested in food or from contaminated hands

ENVIRONMENT
6. Eggs become infective (embryonated) in soil in 30-40 days.
7. Infective eggs contaminate the environment.

HUMAN HOST
2. Larvae hatch.
   Migrate through liver and lungs.
3. Pass up trachea and are swallowed
4. Become mature worms in small intestine
5. Eggs produced and passed in feces.

Fig. 3.4 Transmission and life cycle of Ascaris lumbricoides. (From Monica Chesbrough, 1998, District Laboratory Practice in Tropical Countries, Part One, Cambridge University Press, London.)

Clinical Manifestation

- Most infections go unnoticed until large worm is passed in feces and occasionally the mouth and nose.
- Migrant larvae may cause itching, wheezing and dyspnea, fever, cough productive of bloody sputum may occur.
- Abdominal pain may arise from intestinal or duct (biliary, pancreatic) obstruction.
- Serious complications include bowel obstruction due to knotted/intertwined worms.

**Diagnosis**
- Microscopic identification of eggs in a stool sample
- Adult worms passed from anus, mouth or nose.

**Treatment**
1. Albendazole or
2. Mebendazole or
3. Piperazine or
4. Levamisole

**Prevention and control**
1. Treatment of cases
2. Sanitary disposal of feces
3. Prevent soil contamination in areas where children play
4. Promote good personal hygiene (handwashing).

### 3.4.2 Trichuriasis

**Definition**
A nematode infection of the large intestine, usually asymptomatic in nature.
Infectious agent
Trichuris trichuria (whip worm)

Epidemiology


Reservoir- Humans

Mode of transmission- Indirect, particularly through pica or ingestion of contaminated vegetables. Not immediately transmissible from person to person.

Incubation period- Indefinite

Period of communicability- Several years in untreated carriers.

Susceptibility and resistance- Susceptibility is universal.
Life Cycle

**Fig. 3.5** Transmission and life cycle of *Trichuris trichiura.* (From Monica Chesbrough, 1998, District Laboratory Practice in Tropical Countries, Part One, Cambridge University Press, London.)

**Clinical manifestation**

- Severity is directly related to the number of infecting worms.
- Most infected people are asymptomatic.
- Abdominal pain, tiredness, nausea and vomiting, diarrhea or constipation are complaints by patients.
- Rectal prolapse may occur in heavily infected very young children.
**Diagnosis**
- Demonstration of eggs in feces.

**Treatment**
1. Albendazole or
2. Mebendazole

**Prevention and control**
1. Sanitary disposal of feces
2. Maintaining good personal hygiene (i.e. washing hands and vegetables and other soil contaminated foods)
3. Cutting nails especially in children
4. Treatment of cases.

**3.4.3 Entrobiasis**
* (Oxyuriasis, pinworm infection)

**Definition**
A common intestinal helminthic infection that is often asymptomatic.

**Infectious agent**
Entrobius vermicularis
Epidemiology

Occurrence- Worldwide, affecting all socio-economic classes with high rates in some areas. Prevalence is highest in school-aged children, followed by preschools and is lowest in adults except for mothers of infected children. Prevalence is often high in domiciliary institutions. Infection usually occurs in more than one family member.

Reservoir- Human

Mode of transmission- Direct transfer of infective eggs by hand from anus to mouth of the same or another person or indirectly through clothing, bedding, food or other articles contaminated with eggs of the parasite.

Incubation period- 2-6 weeks

Period of communicability- As long as gravid females are discharging eggs on perianal skin. Eggs remain infective in an indoor environment for about 2 weeks.

Susceptibility and resistance- Susceptibility is universal.
Life Cycle

**Fig. 3.6** Transmission and life cycle of *Entrobius vermicularis*. (From Hegazi M., 1994, *Applied Human Parasitology*, 1st edition, the Scientific Book Centers, Cairo.)

**Clinical manifestation**
- Perianal itching, disturbed sleep, irritability and sometimes secondary infection of the scratched skin.

**Diagnosis**
- Stool microscopy for eggs or female worms.

**Treatment**
1. Mebendazole.

**Prevention and control**
1. Educate the public about hygiene (i.e. handwashing before eating or preparing food, keeping nails short and discourage nail biting).
2. Treatment of cases
3. Reduce overcrowding in living accommodations.
4. Provide adequate toilets.
3.4.4. Strongyloidiasis

Definition
An often asymptomatic helminthic infection of the duodenum and upper jejunum.

Infectious agent
Strongyloides stercoralis

Epidemiology
Occurrence- In tropical and temperate areas. More common in warm and wet regions.

Reservoir- Human

Mode of transmission- Infective (filariform) larvae penetrate the skin and enter the venous circulation.

Incubation period- 2-4 weeks (from skin penetration up to when rhabditiform larvae appear in the feces).

Period of communicability- As long as living worms remain in the intestine; up to 35 years in cases of auto-infection.

Susceptibility and resistance- Susceptibility is universal. Patients with AIDS or on immuno-suppressive medication are at risk of dissemination.
Life Cycle

**TRANSMISSION**

1. Infective filariform larvae penetrate skin, e.g. feet. Autoinfection also occurs.

**ENVIRONMENT**

6. In soil larvae become free-living worms produce more rhabditiform larvae*  
   * Free-living cycle can be repeated several times  
7. Become infective filariform larvae in the soil

**HUMAN HOST**

2. Larvae migrate, pass up trachea and are swallowed.  
3. Become mature worms in small intestine  
5. Rhabditiform larvae:  
   - Passed in feces, or  
   - Become filariform larvae in intestine, causing autoinfection.

Fig. 3.7. Transmission and life cycle of Strongyloides stercoralis. (From Monica Chesbrough, 1998, District Laboratory Practice in Tropical Countries, Part One, Cambridge University Press, London.)

**Clinical Manifestation**

- Pneumonia occurs during heavy larval migration.  
- Mild peptic ulcer like epigastric discomfort to severe watery diarrhea.  
- Heavy infection may result in malabsorption syndrome.

**Diagnosis**

- Identification of larvae in stool specimen.
Treatment
1. Albendazole or
2. Thiabendazole

Prevention and control
1. Proper disposal of human excreta (feces)
2. Personal hygiene including use of footwear.

3.4.5 Hookworm disease
(Ancylostomiasis, Necatoriasis)

Definition
A common chronic parasitic infection with a variety of symptoms usually in proportion of the degree of anemia.

Infectious agent
Ancylostoma duodenale and Necator americanus

Epidemiology
Occurrence- Widely endemic in tropical and subtropical countries where sanitary disposal of human feces is not practiced and the soil moisture and temperature conditions favor development of infective larvae.

Reservoir- Humans
Mode of transmission- Through skin penetration by the infective larvae.

Incubation period- Symptoms may develop after a few weeks to many months depending on intensity of infection and iron intake of the host.

Period of communicability- Infected people can contaminate the soil for several years in the absence of treatment.

Susceptibility and resistance- Susceptibility is universal. No evidence that immunity develops with infection.

Life cycle

Fig. 3.8 Transmission and life cycle of Hookworms: Ancylostoma duodenale and Necator americanus. (From Monica Chesbrough, 1998, District Laboratory Practice in Tropical Countries, Part One, Cambridge University Press, London.)
Clinical Manifestation
The clinical manifestation is related to:
1. Larval migration of the skin
   - Produces transient, localized maculopapular rash associated with itching called ground itch.
2. Migration of larva to the lungs.
   - Produces cough, wheezing and transient pneumonitis.
3. Blood sucking
   - Light infection-no symptoms
   - Heavy infection-result in symptoms of peptic ulcer disease like epigastric pain and tenderness. Further loss of blood leads to anemia manifested by exertional dyspnea, weakness and light-headedness.

Diagnosis
- Demonstration of eggs in stool specimen.

Treatment
1. Mebendazole or
2. Albendazole or
3. Levamisole

Prevention and control
1. Sanitary disposal of feces
2. Wearing of shoes
3.5 Direct Contact with Feces

These are diseases transmitted mainly through direct contact with feces of the infected person.

3.5.1 Poliomyelitis

Definition
A viral infection most often recognized by the acute onset of flaccid paralysis.

Infectious agent
Polio viruses (type I, II and III)

Epidemiology
Occurrence – Worldwide prior to the advent of immunization. Cases of polio occur both sporadically and in epidemics. Primarily a disease of infants and young children, 70-80% of cases are less than three years of age. More than 90% of infections are unapparent. Flaccid paralysis occurs in less than 1% of infections.

Reservoir – humans, especially children

Mode of transmission- Primarily person-to-person, spread principally through the fecal-oral route. In rare instances, milk,
food stuffs and other materials contaminated with feces have been incriminated as vehicles.

**Incubation period** - commonly 7-14 days

**Period of communicability** – not precisely known, but transmission is possible as long as the virus is excreted.

**Susceptibility and resistance** - Susceptibility is common in children but paralysis rarely occurs. Infection confers permanent immunity.

**Clinical manifestation**
- Usually asymptomatic or non-specific fever is manifested in 90% of cases.
- If it progresses to major illness, severe muscle pain, stiff neck and back with or without flaccid paralysis may occur.
- Paralysis is asymptomatic and occurs within three to four days of illness.
- The legs are more affected than other part of the body.
- Paralysis of respiratory and swallowing muscles is life-threatening.

**Diagnosis**
- Based on clinical and epidemiological grounds
Treatment
Symptomatic

Prevention and control
1. Educate public about the advantage of immunization in early childhood.
2. Trivalent live attenuated vaccine (OPV) at birth.
3. Safe disposal of human excreta (feces)

3.5.2 Hydatid Disease (Echinococcosis)

Definition
The tapeworm Echinococcus granulosus is the most common species of Echinococcus and causes cystic hydatid disease.

Infectious agent
Echinococcus granulosus, a small tapeworm of dog

Epidemiology
Occurrence – occurs on all continents except Antarctica. Especially common in grazing countries where dogs consume viscera containing cysts.

Reservoir- Domestic dogs and other canids are definitive hosts; they may harbor thousands of adult tapeworms in their
intestines without signs of infection. Sheep act as intermediate hosts.

**Mode of transmission** – directly with hand to mouth transfer of eggs after association with infected dogs or indirectly through contaminated food, water, soil or fomites.

**Incubation period** – variable from 12 months to many years, depending on the number and location of cysts and how rapidly they grow.

**Period of communicability** – Infected dogs begin to pass eggs approximately 7 weeks after infection. Most canine infections resolve spontaneously by six months.

**Susceptibility and resistance** – Children are more likely to be exposed to infection because they are more likely to have close contact with infected dogs.

**Clinical manifestations**
- The signs and symptoms vary according to location of the cyst and number.
- Ruptured or leaking cysts can cause severe anaphylactic reactions.
- Cysts are typically spherical, thick walled and unilocular and are most frequently found in the liver and lungs.
Diagnosis
- History of residence in an endemic area along with association with canines
- Sonography and CT scan
- Serologic test

Treatment
1. Surgical resection of isolated cysts is the most common treatment.
2. Albendazol (mebendazol)
3. If cysts rupture, praziquantel

Prevention and control
1. Educate the public at risk to avoid exposure to dog feces. Handwashing should be emphasized.
2. Interrupt transmission from intermediate to definitive hosts by preventing dogs' access to uncooked viscera.
3. Safe disposal of infected viscera.
Review Questions

1. What does fecal-oral transmission mean?
2. Mention some of the diseases transmitted through unapparent fecal contamination of food, water and hands.
3. State some of the common preventive and control measures of oral-fecal transmitted diseases.
CHAPTER FOUR

AIR-BORNE DISEASES

4.1 Learning Objectives

At the end of this chapter, students will be able to:
- List common air-borne diseases.
- Identify the common modes of air-borne diseases transmission.
- Participate in diagnosis and treatment of common air-borne diseases.
- Apply preventive and control methods for air-borne diseases.

4.2 Introduction

The organisms causing the diseases in the air-borne group enter the body via the respiratory tract. When a patient or carrier of pathogens talks, coughs, laughs, or sneezes, he/she discharges fluid droplets. The smallest of these remain up in the air for some time and may be inhaled by a new host. Droplets with a size of 1-5 microns are quite easily drawn in to the lungs and retained there.
Droplets that are bigger in size will not remain air-borne for long but will fall to the ground. Here, however, they dry and mix with dust. When they contain pathogens that are able to survive drying, these may become air-borne again by wind or something stirring up the dust, and they can then be inhaled. Air-borne diseases, obviously, will spread more easily when there is overcrowding, as in overcrowded class rooms, public transport, canteens, dance halls, and cinemas. Good ventilation can do much to counteract the effects of overcrowding. Air-borne diseases are mostly acquired through the respiratory tract.

4.3 Common Cold (Acute Viral Rhinitis or Coryza)

Definition
An acute catarrhal infection of the upper respiratory tract.

Infectious agent
Rhino viruses (100 serotypes) are the major causes in adults. Parainfluenza viruses, respiratory syncytial viruses (RSV), Influenza, and Adeno viruses cause common cold-like illnesses in infants and children.
**Epidemiology**

**Occurrence**- Worldwide both in endemic and epidemic forms. Many people have one to six colds per year. Greater incidence in the highlands. Incidence is high in children under 5 years and gradually declines with increasing age.

**Reservoir**- Humans

**Mode of transmission**- by direct contact or inhalation of airborne droplets. Indirectly by hands and articles freshly soiled by discharges of nose and throat of an infected person.

**Incubation period**- between 12 hours and 5 days, usually 48 hours, varying with the agent.

**Period of communicability**- 24 hours before onset and for 5 days after onset.

**Susceptibility and resistance**- Susceptibility is universal. Repeated infections (attacks) are most likely due to multiplicity of agents.

**Clinical Manifestation**

- Coryza, sneezing, lacrimation, pharyngeal or nasal irritation, chills and malaise
- Dry or painful throat.
Diagnosis
- Based on clinical grounds

Treatment
1. No effective treatment but supportive measures like:
   - Bed rest
   - Steam inhalation
   - High fluid intake
   - Anti pain
   - Balanced diet intake

Prevention and Control
1. Educate the public about the importance of:
   - Handwashing
   - Covering the mouth when coughing and sneezing
   - Sanitary disposal of nasal and oral discharges
2. Avoid crowding in living and sleeping quarters especially in institutions
3. Provide adequate ventilation

4.4 Measles (Rubella)

Definition
An acute highly communicable viral disease

Infectious agent
Measles virus
**Epidemiology**

**Occurrence**- Prior to widespread immunization, measles was common in childhood so that more than 90% of people had been infected by age 20; few went through life without any attack.

**Reservoir**- Humans

**Mode of transmission**- Airborne by droplet spread, direct contact with nasal or throat secretions of infected persons and less commonly by articles freshly solid with nose and throat secretion. Greater than 94% herd immunity may be needed to interrupt community transmission.

**Incubation period**- 7-18 days from exposure to onset of fever.

**Period of communicability**- slightly before the prodromal period to four days after the appearance of the rash and minimal after the second day of rash.

**Susceptibility and resistance**- All those who are non-vaccinated or have not had the disease are susceptible. Permanent immunity is acquired after natural infection or immunization.
Clinical Manifestation
- Prodromal fever, conjunctivitis, coryza, cough and Koplik spots on the buccal mucosa
- A characteristic red blotchy rash appears on the third to seventh day, beginning on the face, gradually becoming generalized, lasting 4-7 days.
- Leucopenia is common.
- Complications like otitis media, pneumonia, diarrhea, encephalitis, croup (Laryngo tracheo bronchitis) may result from viral replication or bacterial super infection.

Diagnosis
- Based on clinical and epidemiological grounds

Treatment
1. No specific treatment
2. Treatment of complications
3. Vitamin A provision

Nursing care
1. Advise patient to have bed rest.
2. Relief of fever.
3. Provision of non-irritant small frequent diet.
4. Shorten the fingernails.

Prevention and control
1. Educate the public about measles immunization.
2. Immunization of all children (less than 5 years of age) who had contact with infected children.
3. Provision of measles vaccine at nine months of age.
4. Initiate measles vaccination at 6 months of age during epidemic and repeat at 9 months of age.

4.5 Influenza

Definition
An acute viral disease of the respiratory tract

Infectious agent
Three types of influenza virus (A, B and C)

Epidemiology
Occurrence- In pandemics, epidemics and localized outbreaks.
Reservoir- Humans are the primary reservoirs for human infection.
Mode of transmission- Airborne spread predominates among crowded populations in closed places such as school buses.
Incubation period- short, usually 1-3 days
Period of communicability - 3-5 days from clinical onset in adults; up to 7 days in young children.

Susceptibility and resistance - when a new sub-type appears, all children and adults are equally susceptible. Infection produces immunity to the specific infecting agent.

Clinical Manifestation
- Fever, headache, myalgia, prostration, sore throat and cough
- Cough is often severe and protracted, but other manifestations are self-limited with recovery in 2-7 days

Diagnosis
- Based on clinical ground

Treatment
1. Same as common cold, namely:
   - Anti-pain and antipyretic
   - High fluid intake
   - Bed rest
   - Balanced diet intake

Prevention and control
1. Educate the public in basic personal hygiene, especially the danger of unprotected coughs and sneezes and hand to mucus membrane transmission.
2. Immunization with available killed virus vaccines may provide 70-80% protection.
3. Amantadine hydrochloride is effective in the chemoprophylaxis of type A virus but not others.

4.6 Diphtheria

Definition
An acute bacterial disease involving primarily tonsils, pharynx, nose, occasionally other mucus membranes or skin and sometimes the conjunctiva or genitalia.

Infectious agent
Corynebacterium diphtheriae

Epidemiology
Occurrence- Disease of colder months in temperate zones, involving primarily non-immunized children under 15 years of age. It is often found among adult population groups whose immunization was neglected. Unapparent, cutaneous and wound diphtheria cases are much more common in the tropics.

Reservoir- Humans

Mode of transmission- contact with a patient of carrier. i.e. with oral or nasal secretions or infected skin.
Incubation period- usually 2-5 days

Period of communicability- variable, until virulent bacilli have disappeared from discharges and lesion; usually 2 weeks or less.

Susceptibility and resistance- Susceptibility is universal. Infants borne to immune mothers are relatively immune, but protection is passive and usually lost before 6 months. Recovery from clinical disease is not always followed by lasting immunity. Immunity is often acquired through unapparent infection. Prolonged active immunity can be induced by diphtheria toxoid.

Clinical Manifestation
- Characteristic lesion marked by a patch or patches of an adherent grayish membrane with a surrounding inflammation (pseudo membrane).
- Throat is moderately sore in pharyngo tonsillar diphtheria, with cervical lymph nodes somewhat enlarged and tender; in severe cases, there is marked swelling and edema of neck.
- Late effects of absorption of toxin appearing after 2-6 weeks, including cranial and peripheral, motor and sensory nerve palsies and myocarditis (which may occur early) and are often severe.
Diagnosis
- Based on clinical and epidemiological grounds
- Bacteriologic examination of discharges from lesions.

Treatment
1. Diphtheria antitoxin
2. Erythromycin for 2 weeks but 1 week for cutaneous form or
3. Procaine penicillin for 14 days or single dose of Benzathin penicillin

Primary goal of antibiotic therapy for patients or carriers is to eradicate C. diphtheriae and prevent transmission from the patient to susceptible contacts.

Prevention and control
1. Educate the public, and particularly the parents of young children, of the hazards of diphtheria and the necessity for active immunization.
2. Immunization of infants with diphtheria toxoid.
3. Concurrent and terminal disinfection of articles in contact with patient and soiled by discharges of patient.
4. Single dose of penicillin (IM) or 7-10 days course of Erythromycin (PO) is recommended for all persons exposed to diphtheria.
4.7 Pertusis (whooping cough)

Definition
An acute bacterial disease involving the respiratory tract.

Infectious agent
Bordetella pertussis

Epidemiology
Occurrence- An endemic disease common to children especially young children everywhere in the world. A marked decline has occurred in incidence and mortality rates during the past four decades. Outbreaks occur periodically. Endemic in developing world and 90% of attacks occur in children under 6 years of age.

Reservoir- Humans

Mode of transmission- Primarily by direct contact with discharges from respiratory mucus membranes of infected persons by airborne route, probably by droplets. Indirectly by handling objects freshly solid with nasopharyngeal secretions.

Incubation period- 1-3 weeks
Period of communicability - Highly communicable in early catarrhal stage before the paroxysmal cough stage. The most contagious disease with an attack rate of 75-90%. Gradually decreases and becomes negligible in about 3 weeks. When treated with erythromycin, infectiousness is usually 5 days or less after onset of therapy.

Susceptibility and resistance - Susceptibility to non-immunized individuals is universal. One attack usually confers prolonged immunity but may not be lifelong.

Clinical manifestation
The disease has insidious onset and 3 phases:
1. Catarrhal phase
   - Lasts 1-2 weeks
   - Cough and rhinorrhea
2. Paroxysmal phase
   - Explosive, repetitive and prolonged cough
   - Child usually vomits at the end of paroxysm
   - Expulsion of clear tenacious mucus often followed by vomiting
   - Whoop (inspiratory whoop against closed glottis) between paroxysms.
   - Child looks healthy between paroxysms
   - Paroxysm of cough interferes with nutrition and cough
   - Cyanosis and sub conjunctiva hemorrhage due to violent cough.
3. **Convalescent phase**
   - The cough may diminish slowly or may last long time.
   - After improvement the disease may recur.

**Diagnosis**
- Difficult to distinguish it from other URTI
- History and physical examination at phase two (paroxysmal phase) ensure the diagnosis.
- Marked lymphocytosis.

**Treatment**
1. Erythromycin- to treat the infection in phase one but to decrease transmission in phase two
2. Antibiotics for super infections like pneumonia because of bacterial invasion due to damage to cilia.

**Nursing care**
1. Proper feeding of the child.
2. Encourage breastfeeding immediately after an attack (each paroxysm).
3. Proper ventilation- continuous well humidified oxygen administration.
4. Reassurance of the mother (care giver),
Prevention and control
1. Educate the public about the dangers of whooping cough and the advantages of initiating immunization at 6 weeks of age.
2. Consider protection of health workers at high risk of exposure by using erythromycin for 14 days.

4.8 Pneumococcal pneumonia

Definition
An acute bacterial infection of the lung tissue and bronchi.

Infectious agent
Streptococcus pneumoniae (pneumococcus)

Epidemiology
Occurrence- Endemic particularly in infancy, old age and persons with underlying medical conditions. Epidemics can occur in institutions, barracks and on board ship where people are living and sleeping in close quarters. Common in lower socio-economic groups and developing countries.

Reservoir- Humans - pneumococci are usually found in the URT of healthy people throughout the world.
**Mode of transmission** - droplet spread, direct oral contact or indirectly through articles freshly soiled with respiratory discharges. Person to person transmission is common.

**Incubation period** - not well determined, may be as short as 1-3 days.

**Period of communicability** - Until discharges of mouth and nose no longer contain virulent pneumococci in significant number.

**Susceptibility and resistance** - Susceptibility is increased by influenza, pulmonary edema of any cause, aspiration following alcohol intoxication, chronic lung disease, exposure to irritants in the air, etc. Malnutrition and low birth weight are important risk factors in infants and young children in developing countries. Immunity following an attack may last for years.

**Clinical Manifestation**
- Sudden onset of chill, fever, pleural pain, dyspnea, tachypnea, a cough productive of rusty sputum,
- Chest indrawing, shallow and rapid respiration in infants and young children.
- Vomiting and convulsion may occur in infants and young children.
Communicable Disease Control

**Diagnosis**
- Based on clinical grounds
- Chest X-ray- reveals consolidation of the affected lung tissue but not in children.
- Sputum gram stain- reveals gram negative diplococci

**Treatment**
1. Antipyretic and antipain
2. Antibiotics like Ampicillin or procaine penicillin for adults but usually crystalline penicillin for children
3. Anticonvulsants for infants.

**Nursing care**
1. Monitor vital signs especially of children.
2. Maintain high body temperature to normal.
3. Intermittent administration of humidified oxygen if indicated especially for young children.
4. Timely administration of ordered medication.

**Prevention and control**
1. Treatment of cases
2. Treatment of other underlying medical conditions
3. Improved standard of living (adequate and ventilated housing and better nutrition)
4. Avoid overcrowding.
4.9 Meningococcal Meningitis

Definition
An acute bacterial disease that causes inflammation of the pia and arachnoid space.

Infectious agent
Neisseria meningitides (the meningococcus)

Epidemiology
Occurrence - Greatest incidence occurs during winter and spring. Epidemics occur irregularly. Common in children and young adults. It is also common in crowded living conditions.

Reservoir - Humans

Mode of transmission - Direct contact with respiratory droplets from nose and throat of infected person.

Incubation period - 2-10 day, commonly 3-4 days.

Period of communicability - as long as the bacteria is present in the discharge.

Susceptibility and resistance - Susceptibility is low and decreases with age.
Clinical Manifestation
- Sudden onset of fever, intense headache, nausea and often vomiting, neck stiffness and frequently, petechial rash with pink macules.
- Kernig’s sign may be positive (i.e. patient feels back pain when one of the lower limbs is flexed at the knee joint and extended forward in an elevated position).
- Brudinski’s sign may be positive (i.e. when the patient’s neck is flexed, the two lower extremities get flexed or raised up).
- Delirium and coma often appear.

Diagnosis
- Based on clinical and epidemiological grounds
- White blood cell count (neutrophils)
- Cerebrospinal fluid analysis (Gram stain, white cell count, etc.)

Treatment
1. Admit the patient and administer high dose of crystalline penicillin intravenously
2. Antipyretic

Nursing care
1. Maintain fluid balance (input and output)
2. Maintain body temperature to normal
3. Timely administration of antibiotics
4. Monitor vital signs.

**Prevention and control**
1. Educate the public on the need to reduce direct contact and exposure to droplet infection.
2. Reduce overcrowding in work places, schools, camps, etc.
3. Vaccines containing group A,C and Y strains.
4. Chemotherapy of cases.
5. Chemo prophylaxis (e.g. Rifampin for 2 days)
6. Report to the concerned health authorities.

### 4.10 Tuberculosis

**Definition**
A chronic and infectious mycobacterial disease important as a major cause of illness and death in many parts of the world.

**Infectious agent.**
Mycobacterium tuberculosis- human tubercle bacilli (commonest cause)
Mycobacterium bovis- cattle and man infection
Mycobacterium avium- infection in birds and man.
Epidemiology

Occurrence- Worldwide, however underdeveloped areas are more affected. Affects all ages and both sexes. Age groups between 15-45 years are mainly affected. According to the WHO 1995 report, 9 million cases and 3 million deaths have occurred. According to the Ministry of Health report in 1993 E.C, tuberculosis was a leading cause of outpatient morbidity (ranked 8th with 2.2%), leading cause of hospitalization (ranked 3rd with 7.8%) and leading cause of hospital death (ranked 1st with 10.1%). Tuberculosis has two major clinical forms. Pulmonary (80%) primarily occurs during childhood and secondarily 15-45 years or later. The other is extra pulmonary, which affects all parts of the body. Most common sites are lymph nodes, pleura, Genitourinary tract, bone and joints, meninges and peritoneum.

Mode of transmission- Through aerosolized droplets mainly from persons with active ulcerative lesion of lung expelled during talking, sneezing, singing, or coughing directly. Untreated pulmonary tuberculosis positive (PTB+) cases are the source of infection. Most important is the length of time of contact an individual shares volume of air with an infectious case. That is intimate, prolonged or frequent contact is required. Transmission through contaminated fomites (clothes, personal articles) is rare. Ingestion of unpasteurized
milk transmits bovine tuberculosis. Overcrowding and poor housing conditions favor the disease transmission.

**Incubation period** - 4-12 weeks

**Period of communicability** - as far as the bacilli is present in the sputum

**Susceptibility and resistance** - under 3 years old children, adolescents, young adults, the very old and the immuno-suppressed are susceptible. Everyone who is non-infected or non-vaccinated can be infected.

HIV is an important risk factor for the development of HIV-associated tuberculosis by facilitating:
- Reactivation or
- Progression of recent infection or
- Reinfection

**Clinical Manifestation**

**Pulmonary tuberculosis**
- Persistent cough for 3 weeks or more
- Productive cough with or without blood-stained sputum
- Shortness of breath and chest pain
- Intermittent fevers, night sweats, loss of weight, loss of appetite, fatigue and malaise.
TB lymph adenitis
- Slowly developing and painless enlargement of lymph nodes followed by matting and drainage of pus.

Tuberculosis pleurisy
- Pain while breathing in, dull lower chest pain, slight cough, breathlessness on exertion.

TB of bones and joints
- Localized pain and/or swelling, discharging of pus, muscle weakness, paralysis and stiffness of joints.

Intestinal TB
- Loss of weight and appetite
- Abdominal pain, diarrhea and constipation
- Mass in the abdomen
- Fluid in the abdominal cavity (ascites)

Tuberculos meningitis
- Headache, fever, vomiting, neck stiffness and mental confusion of insidious onset.

Diagnosis
1. Clinical manifestations
2. Sputum smears for acid-fast bacilli (AFB), which is the Golden standard. However, one positive result does not
justify starting anti TB treatment since errors can never be excluded.

3. Acid-fast stain for AFB can be done for extra pulmonary tuberculosis having pus-y discharge.

4. Radiological examination: This is unreliable because it can be caused by a variety of conditions or previous TB patients who are healed may have chest x-ray giving the appearance of active TB, which requires treatment.

5. Histopathological examination: Biopsies for extrapulmonary TB (e.g. Tuberculos lymphadenitis)

6. Tuberculin test (mantoux): Helpful in non-BCG vaccinated children under 6 years of age

7. Culture: Complex and sophisticated tool, which takes several weeks to yield results. Not a primary diagnostic tool in our country.

Treatment
The following drugs are being used for treatment of TB in Ethiopia.

- Streptomycin (s) daily IM injection
- Ethambutol (E)
- Rifampin (R)
- Thiacetazone (T)
- Isoniazid (H)
- Pyrazinamide (Z)
All drugs, except streptomycin, which is administered daily through in route) are to be taken orally as a single daily dose preferably on an empty stomach.

**Drug regimens (prescribed course of therapy)**

1) Short course chemotherapy regimen
   - (DOTS) intensive phase- S(RH)Z for two months
   - Continuation phase- TH (EH) for the next 6 months.
2) Long course chemotherapy regimen.
   - Intensive phase- S(TH) or S(EH) for 2 months
   - Continuation phase- TH or EH for the next 10 months

**Nursing care**
1. Educate the patient how and when to take the prescribed medication.
2. Tell the patient not to stop the medication unless he/she is told to do so.
3. Tell the patient to come to the health institution if he/she develops drug side effects.
4. Advice the patient on the importance of taking adequate and balanced diet and to eat what is available at home.

**Prevention and control**
1. Chemotherapy of cases
2. Chemoprophylaxis for contacts
4. INH (Isoniazid) for adults and children who have close contact with the source of infection

3. Immunization of infants with BCG

4. Educate patients with TB about the mode of disease transmission and how to dispose their sputum and cover their mouth while coughing, sneezing, etc.

5. Public health education about the modes of disease transmission and methods of control
   - Improved standard of living
   - Adequate nutrition
   - Health housing
     - Environmental sanitation
   - Personal hygiene; etc.
   - Active case finding and treatment

4.11 Leprosy (Hansen’s disease)

Definition
A chronic bacterial disease of the skin, peripheral nerves and, in lepromatous patients, the upper airway

Infectious agent
Mycobacterium leprae
Epidemiology

Occurrence- Although common in rural tropics and subtropics, socio-economic conditions may be more important than climate itself. Endemic in south and southeast Asia, tropical Africa and Latin America.

Reservoir- Humans

Mode of transmission- Not clearly established. Household and prolonged close contact appear to be important. Millions of bacilli are liberated daily in the nasal discharges of untreated lepromatous patients. Cutaneous ulcers in lepromatous patients may shed large number of bacilli. Organisms probably gain access (entrance) through the URT and possibly through broken skin. In children less than one year of age, transmission is presumed to be transplacental.

Incubation period- 9 months to 20 years.

Period of communicability- Infectiousness is lost in most instances within 3 months of continuous and regular treatment with dapsone or clofazamin or within 3 days of rifampicin treatment.
**Susceptibility and resistance**- The presence and form of leprosy depend on the ability to develop effective cell-mediated immunity.

**Clinical Manifestation**
Clinical manifestations vary between two polar forms: **lepromatous** and **tuberculoid** leprosy.

**Lepromatous (Multibacillary form)**
Nodules, papules, macules and diffused infiltration are bilaterally symmetrical and usually numerous and extensive. Involvement of the nasal mucosa may lead to crusting, obstructed breathing and epistaxis. Occular involvement leads to iritis and keratitis.

**Tuberculoid (Paucibacillary form)**
Skin lesions are single or few, sharply demarcated, anesthetic or hyperesthetic and bilaterally symmetrical. Peripheral nerve involvement tends to be severe.

**Borderline**
Has features of both polar forms and is more liable to shift toward the lepromatous form in untreated patients and toward the tuberculoid form in treated patients.
Diagnosis
- Complete skin examination (hyperesthesia, anesthesia, paralysis, muscle wasting or trophic ulcer which are signs of peripheral nerve involvement) with bilateral palpation of peripheral nerves (ulnar nerve at the elbow, peroneal nerve at head of fibula and the great auricular nerve) for enlargement and tenderness.
- Skin lesion are tested for sensation (light touch, pink prick, temperature discrimination).
- Demonstration of AFB in skin smears made by scraped incision method.
- Skin biopsy confined to the affected area should be sent to the experienced pathologists in leprosy diagnosis.

Treatment
1. Dapsone  three drugs for 12 months and then
2. Rifampicin  dapsone alone for the next 12 months.
3. Clfazamin
4. Aspirin for mild reactions and inflammation
5. Severe reaction can be treated with corticosteroids
Review Questions

1. What do you understand by air-borne disease transmission?

2. Which airborne disease occurrence should be reported immediately to the concerned health authorities for their prompt action?
   a. Pneumonia
   b. Tuberculosis
   c. Leprosy
   d. Meningococcal meningitis

3. Select diseases which cause chronic illness:
   a. Tuberculosis
   b. Leprosy
   c. Measles
   d. Infection hepatitis

4. State some of the preventive and control methods for tuberculosis.
CHAPTER FIVE

ARTHROPOD OR INTERMEDIATE
VECTOR-BORNE DISEASES

5.1 Learning Objectives

At the end of this chapter, the student will be able to:

- Describe what arthropod or intermediate vector-borne disease means.
- Identify the common vectors which transmit disease to man.
- List the common vector-borne diseases.
- Participate in diagnosis and treatment of vector-borne diseases.
- Implement the common preventive and control methods of vector-borne diseases.

5.2 Introduction

Generally speaking a vector is any carrier of disease, but in the case of the ‘vector-borne diseases’ we restrict the word to those invertebrate hosts (insects or snails), which are an
essential part of the life cycle of the disease organism. A housefly just carrying bacteria or amoebic cysts on its feet to food is not regarded as a vector: this would be simple mechanical spread.

Insect vectors usually acquire the disease organism by sucking blood from infected persons, and pass it on, later, by the same route. There are other routes, however; infection may enter skin cracks or abrasions either from infected feces deposited when feeding, or from body fluid when an insect is crushed.

By definition the disease organism undergoes a period of development inside the vector, and the time taken for this is called the extrinsic incubation period.

5.3 Mosquito-Borne Diseases

5.3.1 Malaria

Definition
An acute infection of the blood caused by protozoa of the genus plasmodium.
Infectious agent.
- *Plasmodium falciparum/malignant tertian*: Invades all ages of red blood cells. Red blood cell cycle is 48 hours.
- *Plasmodium vivax/benign tertian*: Invades reticulocytes only. Red blood cell cycle is 48 hours.
- *Plasmodium ovale/tertian*: Invades reticulocytes only. Red blood cell cycle is 48 hours.
- *Plasmodium Malariae/Quartan malaria*: Invades reticulocytes only. Red blood cell cycle is 72 hours.

Epidemiology
**Occurrence**- Endemic in tropical and sub-tropical countries of the world. Affects 40% of the world population. Children less 5 years of age, pregnant women and travelers to endemic areas are risk groups. *Plasmodium falciparum* 60% and *vivax* 40% are common in Ethiopia.

Predisposing factors are:
- Environment- physical environment for the propagation
- Patient source
- Susceptible recipients
- Anopheles capable to transmit the parasite
- Socio-economic factors like immigration, war, poverty, ignorance, agricultural irrigation farms, etc.
Reservoir- Humans

Mode of transmission- By the bite of an infective female anopheles mosquito, which sucks blood for egg maturation. Blood transfusion, hypodermic needles, organ transplantation and mother to fetus transmission is possible. Since there is no pre-erythrocytic (tissue) cycle, the incubation period is short. Anopheles gambiae and funestus are common vectors in Ethiopia.

Incubation period- Varies with species
- Plasmodium falciparum 7-14 days
- Plasmodium vivax 8-14 days
- Plasmodium ovale 8-14 days
- Plasmodium malariae 7-30 days

Period of communicability- Mosquitoes are infective as long as infective gametocytes are present in the blood of patients. Once infected, mosquito remains infective for life.

Susceptibility and resistance- Susceptibility is universal except in some host-resistance factors:

Non specific factors
- Increased splenic clearance reaction
- Hyperpyrexia- which is said to be schizontcidal
- Sickle cell traits are resistant to plasmodium falciparum
- Duffy blood group deficiency (Duffy antigen negative red blood cells) lack receptor for plasmodium vivax.
- Because of passive immunity infants are resistant in early life.

**Specific factors**
This is a humoral and cell mediated immunity that is species and strain specific, and hard-won after repeated infection.

**Life cycle**

**Transmission**
1. Sporozoites inoculated when Anopheles mosquito takes a blood meal.

**Human Host**
2. Sporozoites infect liver cells. Multiply by schizogony.
   - Note: some sporozoites of P.vivax and P.ovale become dormant hypnozoites in liver. Become active after several months.
3. Liver schizonts rupture. Merozoites enter red cells, become trophozoites. Multiply by schizogony.*
   - * with P. falciparum, schizogony occurs in capillaries of body organs.
5. Some merozoites develop into male and female gametocytes.

**Mosquito**

*Fig. 5.1 Transmission and life cycle of Malaria parasites. (From Monica Chesbrough, 1998, District Laboratory Practice in Tropical Countries, Part One, Cambridge University Press, London.)*
Clinical Manifestation
Chills, rigor, fever, head ache, diarrhea, hallucinations, abdominal pain, aches, renal or respiratory symptoms, jaundice, etc.

Diagnosis
- Clinical manifestation and epidemiological grounds
- Blood film for hemoparasite
- White blood cell count
- Blood culture to rule out sepsis
- Chest X-ray to rule out pneumonia.

Treatment
1. Plasmodium vivax, ovale and sensitive plasmodium falciparum
   - Chloroquine or
   - Fansidar
2. Chloroquine resistant falciparum and when sensitivity pattern is not known.
   - Quinine or
   - Fansidar

Nursing care
1. Advise patient to come back if the illness gets severe.
2. Advise on personal protection (bed nets, etc).
3. Reduce fever and maintain comfort.
Prevention and control
1. Chemoprophylaxis- for those who go to endemic areas but not for those who live in the endemic area (travelers and newcomers); for under-five children and pregnant mothers who have not enough immunity.

2. Vector control
   - Avoiding mosquito breeding sites
   - Residual DDT spray or other chemicals
   - Personal protection against mosquito bite (use of bed nets, etc.)

3. Chemotherapy of cases

5.3.2 Bancroftian filariasis

Definition
A disease caused by the reaction of the body to the presence of worms in the lymphatic system.

Infectious agent
Wuchereria bancrofti (vectors are culex, Anopheles and Aedes species)
Brugia malayi and (vector is mansonia species)
Brugia timori (vector is Anopheles)
Epidemiology

Occurrence- Widely prevalent in tropical and subtropical areas of Africa, Asia, Pacific Region, Central and South America. Found in Gambella region (western Ethiopia).

Reservoir- Humans are definitive hosts.

Mode of transmission- by bite of mosquito harboring infective larvae

Incubation period- one month, while allergic inflammatory manifestations may appear.

Period of communicability- Humans may infect mosquitoes when microfilariae are present in the peripheral blood. Microfilaremia may persists for 5-10 years or longer. The mosquito becomes infective about 12-14 days after an infective blood meal.

Susceptibility and resistance- Universal. Susceptibility to infection is probable.
Life cycle

Fig. 5.2 Transmission and life cycle of W. bancrofti and Brugia species (From Monica Chesbrough, 1998, District Laboratory Practice in Tropical Countries, Part One, Cambridge University Press, London.)

Clinical Manifestation

The presence of worms in the lymph vessels gives rise to a foreign-body reaction. After the death of the worm, more proteins are released; the reaction then is even more severe. Three phases may be distinguished.

Acute phase:
- Starts within a few months after infection
- Lymphadenopathy
- Fever
- Eosinophilia
  - In this stage microfilariae are not demonstrable in the peripheral blood because the worms are not yet mature. The acute phase is mainly due to a hypersensitivity reaction.

**Subacute phase:**
- This occurs after about one year following acute phases. In this phase worms have matured and microfilariae are present in the peripheral blood.
- Reactions to the adult worms cause attacks of fever with lymphangitis, funiculitis or Epididymitis. Recurrent attacks will sooner or later lead to hydrocele.
- Lesions caused by microfilariae are less common and are associated with hypereosinophilia and lung symptoms (tropical pulmonary eosinophilia syndrome).

**Chronic phase:**
- After many years of repeated attacks, lymph glands and lymph vessels become obstructed; as a result lymph edema develops. Lymph edema most commonly seen in the legs or scrotum (elephantiasis) but may also be present in vulva, breasts, or arms.
- Since the adult worms have usually died, microfilariae are not seen in the blood.
Studies showed that elephantiasis of the lower legs is not encountered in Ethiopia. But there is elephantiasis of the foot called the big foot disease (elephantiasis of lower leg) as a result of accumulation of silica and other minerals in the leg (lymphatics) mostly occurring in bare-footed individuals. This big foot disease is named podoconiosis, which is common in the eastern high lands of Ethiopia (Wolayita, Gojjam, Gondar, Gedeo, Sidamo, etc.).

**Diagnosis**

- Clinical and epidemiological grounds
- Obstructive signs with history and travel to and residence in endemic areas.
- Best established by identifying microfilariae in the peripheral blood (blood film).
- Before taking blood sample one should know the periodicity of microfilariae. That is, microfilariae appear in the peripheral blood during the night (nocturnal) in most parts of the world and during day (diurnal) in the South Pacific region.
- Single dose of Diethylcarbamazin Citrate (DEC) causes the sequestered microfilariae to emerge to blood 45-60 minutes later. This test is said to be the mazoti test, which is used in nocturnal periodicity.
Treatment
1. Diethyl carbamazin Citrate (DEC) results in rapid disappearance of most microfilariae from blood but may not destroy the adult worm. Because of this, we need to repeat DEC annually for some years.
2. Refer the patient for surgical treatment of hydrocele.

Prevention and control
1. Reducing the vector population
2. Mass and selective treatment
3. Personal protection against mosquito bite.

5.3.3 Yellow fever

Definition
An acute infectious viral disease of short duration and varying severity.

Infectious agent
Yellow fever virus

Epidemiology
Occurrence- The disease exists in two transmission cycles. Namely, the sylvatic or Jungle cycle, which occurs between mosquitoes and non-human primates, and an urban cycle,
involving Aedes aegypti mosquitoes and humans. Found in southwest Ethiopia (Gambella region).

**Reservoir**- Urban areas- humans and Aedes aegypti mosquitoes. Forest areas- Vertebrates other than humans (mainly monkeys) and forest mosquitoes.

**Mode of transmission**- By the bite of infective Aedes aegypti mosquitoes.

**Incubation period**- 3-6 days

**Period of communicability**- Blood of patients is infective for mosquitoes shortly before onset of fever and for the first 3-5 days of illness. Not communicable by contact or common vehicles. The disease is highly communicable where many susceptible people and abundant vector mosquitoes co-exist.

**Susceptibility and resistance**- Recovery from yellow fever is followed by lasting immunity; second attacks are unknown. Transient passive immunity in infants born to immune mothers may persist for up to 6 months. In natural infections, antibodies appear in the blood within the first week.
**Clinical Manifestation**

- Typical attacks are characterized by sudden onset of fever, chills, headache, backache, generalized pain, prostration, nausea and vomiting.
- Slow and weak pulse.
- Bleeding tendency is common resulting in epistaxis, bleeding of gums, hematemesis, melaena.
- Jaundice occurs due to liver cell necrosis and this may result in liver failure and death.
- Albuminuria occurs due to nephrosis and this may result in kidney failure and anuria.
- Patients surviving the seventh day of the disease usually recover.

**Diagnosis**

- History of residence and/or travel to endemic area
- Clinical manifestation

**Treatment**

- No specific treatment.

**Nursing care**

1. Monitor vital signs regularly.
2. Maintain body temperature to normal.
4. Keep patient in screened rooms or under mosquito nets to avoid further infection.
Prevention and control
1. Active immunization of all people greater than 9 months of age necessarily exposed to infection because of residence, occupation or travel.
2. Eradication or control of Aedes aegypti mosquitoes in urban areas.
3. Sylvatic/Jungle yellow fever immunization to all people in rural communities whose occupation brings them into forests in yellow fever areas and for people who visit those areas.
4. Notification of the disease to the concerned health authorities.

5.4 Flea-Borne Diseases

5.4.1. Plague

Definition
A highly infectious bacterial disease which can kill many people within a short time.

Infectious agent
Yersinia pestis, the plague bacillus.

Epidemiology
Occurrence- Endemic in wild rodents living in forests in the highlands. Wild rodent plague exists in western USA, large
areas of South America, North, Central, Eastern and Southern Africa, Central and Southeast Asia. However, urban plague is controlled in most of the world.

**Reservoir**- Wild rodents (especially ground squirrels) are the natural vertebrate reservoir of plague. Wild carnivores and domestic cats may also be a source of infection to people.

**Mode of transmission**- Through the bite of infected fleas. Handling of tissues of infected animals.

**Incubation period**- 1-7 days.

**Period of communicability**- Fleas may remain infective for months under suitable conditions of temperature and humidity. Bubonic plague is not usually transmitted directly from person to person unless there is contact with pus from suppurating buboes. Pneumonic plague may be highly communicable under appropriate climatic conditions. Overcrowding facilitates transmission.

**Susceptibility and resistance**- Susceptibility is general. Immunity after recovery is relative; it may not protect against a large inoculums.
Clinical Manifestation

Bubonic plague- Characterized by swelling of lymph glands (bubos); mostly the glands of the groins, sometimes arm pit or other places. Swelling may be the size of an egg, tender or non-tender. Other symptoms are:
- Sudden high fever
- Shock
- Prostration
- Coma
- Death within 3-5 days

Pneumonic plague
- Acute onset
- Severe prostration
- Watery sputum quickly followed by blood-stained sputum.
- Pleural effusion
- Death within 1-2 days

Diagnosis
- Gram stain of sputum or pus-gram negative bacilli.

Treatment
1. Early treatment with antibiotics like streptomycin or tetracycline or sulfa groups.
Prevention and Control
1. Chemotherapy of patient
2. Chemoprophylaxis of all contacts with Sulfa drugs
3. The area where disease occurs must be quarantined (isolated from outer world)
4. Insecticides to kill fleas
5. Encourage people to kill rats
6. Notify the disease to the concerned health authority.

5.4.2 Endemic Typhus (Flea-borne typhus)

Definition
A rickettsial disease whose course resembles that of louse-borne typhus, but is milder.

Infectious agent
Rickettsia typhi (Rickettsia mooseri)

Epidemiology
Occurrence- Worldwide, found in areas where people and rats occupy the same buildings and where large numbers of mice live. Occurs sporadically.

Reservoir-Rats, mice and possibly other small animals. Infection is maintained in nature by a rat-flea-rat cycle where rats are reservoirs (Commonly rattus and rattus novergicus).
Mode of Transmission- Infective rat fleas defecate rickettsia while sucking blood, contaminating the bite site and other fresh skin wounds. An occasional case may follow inhalation of dried infective flea feces.

Incubation period- from 1 to 2 weeks; commonly 12 days

Period of communicability- Not directly transmitted from person to person. Once infected, fleas remain so for life.

Susceptibility and resistance- Susceptibility is general. One attack confers immunity.

Clinical Manifestation
- Prodromal symptoms of headache, myalgia, arthralgia, nausea, and malaise developing 1 to 3 days before the abrupt onset of chills and fever. Nearly all patients experience nausea and vomiting early in the illness.
- The duration of untreated illness averages 12 days.
- Rash is present in only 13% of patients
- Pulmonary involvement: non-productive cough and pneumonia.

Diagnosis
- Epidemiological ground
- Weilfelix agglutination test (Serology)
Treatment
1. Doxycyclin or
2. Chloramphenicol

Prevention and control
1. Destroy rats from burrows and harborages.
2. Use insecticides to abolish flea from livingquarters.
3. Treatment of patients.

5.5 Louse-Borne Diseases

5.5.1 Epidemic Typhus

Definition
An acute rickettsial disease often with sudden onset.

Infectious agent
Rickettsia Prowazeki

Epidemiology
Occurrence- In colder areas where people may live under unhygienic conditions and are louse-infected. Occurs sporadically or in major epidemics, for example during wars or famine, when personal hygiene deteriorates and body lice flourish.
Reservoir- Humans. Infected lice die and don’t serve as a reservoir.

Mode of transmission- The body louse and head louse are infected by feeding on the blood of a patient with acute typhus fever. Infected lice excrete rickettsiae in their feces and usually defecate at the time of feeding. People are infected by rubbing feces or crushed lice into the bite or into superficial abrasions (scratch inoculation).

Incubation period- From 1 to 2 weeks, commonly 12 days

Period of communicability- Patients are infective for lice during febrile illness and possibly for 2-3 days after the temperature returns to normal. Infected lice pass rickettsiae in their feces within 2-6 days after the blood meal; it is infective earlier if crushed. The louse die within 2 weeks after infection. Rickettsiae may remain viable in the dead louse for weeks.

Susceptibility and resistance- Susceptibility is general. One attack usually confers long-lasting immunity.

Clinical Manifestation
- Early symptoms of fever, headache, myalgia, macular eruption appear on the body.
- Patient may have pneumonia, renal or CNS involvement, gastrointestinal disease, skin rash singly or in combination.
- Disease usually terminates by rapid lysis after 2 weeks of fever.

**Diagnosis**
- Based on clinical and epidemiologic grounds
- Serologic test (weil-felix agglutination test)

**Treatment**
1. Chloramphenicol or Tetracycline

**Prevention and control**
1. Delousing of clothes by insecticides or dipping into boiling water
2. Public education on personal hygiene
3. Treatment of cases

**5.5.2 Relapsing Fever**

**Definition**
An acute infectious bacterial disease characterized by alternating febrile periods (recurrent pyrexial attacks).
Infectious agent
Borrelia recurrentis- cause of louse-borne relapsing fever
Borrelia duttoni-cause of tick-borne relapsing fever

Epidemiology
Occurrence- Occurs in Asia, eastern Africa (Ethiopia and Sudan), the highland areas of central Africa and South America. It occurs in epidemic form when it is spread by lice and in endemic form when spread by ticks.

Reservoir- Humans for Borrelia recurrentis; , wild rodents and soft ticks through transovarian transmission. for tick borne relapsing fever

Mode of transmission- vector-borne. Acquired by crushing an infected louse so that it contaminates the bite wound or an abrasion of the skin.

Incubation period- 5-10 days usually 8 days.

Period of communicability- Louse becomes infective 4-5 days after ingestion of blood from an infected person and remains so for life (20-40 days)
Susceptibility and resistance- Susceptibility is general. Duration and degree of immunity after clinical attack are unknown; repeated infection may occur.

Clinical Manifestation

- Sudden onset of illness with chills, fever and prostration, headache, myalgia and arthralgia.
- There may be nausea and vomiting, jaundice and liver swelling.

After 4-5 days the temperature comes down, the patient stays free for 8-12 days and then a relapse follows with the same signs but less intense.

In untreated cases there may be up to ten relapses.

Diagnosis

- Clinical and epidemiological grounds
- Giemsa or Wright stain (blood film)
- Dark field microscopy of fresh blood.

Treatment

1. Admit the patient.
2. Open vein (i.e. start iv-line) before administering penicillin.
3. Administer 400,000-600,000 IU procaine penicillin IM stat
4. Tetracycline during discharge for 3 days
5. Chloramphenicol in infants and children can be used in place of tetracycline.
Nursing care
1. Maintain body temperature to normal.
2. Close vital sign monitoring for 3 hours after medication.
3. Check whether there is reaction or not and report.
4. Comfort the patient by providing antipain.
5. Shaving of hair, and delousing of clothes.

Prevention and control
1. Control of vectors (louse)
2. Personal hygiene
3. Health education about hygiene and modes of disease transmission
4. Delousing of patient’s clothes and his/her family
5. Chemotherapy of cases and Chemoprophylaxis for contacts.

5.6 Snail-Borne Diseases

5.6.1 Schistosomiasis

Definition
It is a blood fluke (trematode) infection with adult worms living within mesenteric or vesicle veins of the host over a life span of many years.
**Infectious agent**

The major schistoma species that cause schistosomiasis of humans are:

- Schistosoma mansoni
- Schistosoma Japonicum
- Schistosoma Hematobium

Others in limited areas are S. mekongi, S. intercalatum, S. malayesis, S. mattheei.

Most prevalent species in Africa are S. mansoni and S. hematobium.

Snail vectors are:
- Bulinus-S. hematobium
- Biomphalaria-S. mansoni
- Onchomelania-S. japonicum

**Epidemiology**

**Occurrence** - S. mansoni is found in South America, Caribbean Islands, Africa and the Middle East. S. hematobium is found in Africa and the Middle East. S. Japonicum is found in the Far East. The disease occurs worldwide and 2 million people are expected to be infected; however, most infected individuals show few or no signs and symptoms, and only a small minority develop significant disease.
Reservoir-The principal reservoir for S. mansoni, S. hematobium and S. intercalatum is man. Other animals, like dog, cat, pig, cattle, water buffalo, horse and wild rodents, are hosts for S. japonicum.

Mode of transmission-Infection is acquired from water containing free-swimming larval forms (cercariae) that have developed in snails.

Incubation period-Acute systemic manifestations (katayama fever) may occur in primary infections 2-6 weeks after exposure, immediately before and during initial egg deposition. The infection in humans can persist up to 10 years. Snails release cercariae as long as they live (from several weeks to 3 months).

Susceptibility and resistance-Susceptibility is universal. Resistance is poorly defined.
**Communicable Disease Control**

**Life cycle**

**TRANSMISSION**
1. Cercariae penetrate skin when person in contact with contaminated water

**FRESH WATER**
7. Eggs reach water. Miracidia hatch

**Snail host**

**HUMAN HOST**
2. Cercariae \( \rightarrow \) Schistosomula. Migrate through lungs and liver.
4. Migrate to veins of lower large intestine (S. haematobium to veins of bladder)
5. Eggs laid in venules. Burrow through into intestine (eggs of S. haematobium into bladder)
7. *S. japonicum also infects animals.*

**Clinical Manifestation**

The stages of schistosomiasis are:

- A. invasion
- B. maturation
- C. established infection and
- D. late stage.

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*Fig. 5.3 Transmission and life cycle of Schistosoma species. (From Monica Chesbrough, 1998, District Laboratory Practice in Tropical Countries, Part One, Cambridge University Press, London.)*
A. Invasion stage
- Cercariae penetrate skin
- Cercarial dermatitis with itching papules and local edema
- Cercariae remain in skin for 5 days before they enter the lymphatic system and reach the liver.

B. Maturation
- Schistosoma mature in the liver.
- Fever, eosinophilia, abdominal pain and transient generalized urticaria (known as katayama syndrome)
- Worms descend the portal vein. S. manson; migrates to mesenteric veins in the intestinal wall and S. haematobium to bladder plexus.
- This stage may be diagnosed as clinical malaria or may pass unnoticed.

C. Established infection
- This is a stage of egg production and eggs reach to the lumen of bladder and bowel.
- Some eggs penetrate the tissue, reach the bladder and intestinal wall are discharged with urine and feces.
- Eggs that could not penetrate the tissue are carried with blood to the liver and lungs.
- Other eggs that fail to reach the lumen of the bladder or bowel provoke an inflammatory reaction.
- The inflammatory reaction, resulting in fibrosis, causes signs and symptoms of schistosomiasis.
- Sign of colitis with bloody diarrhea and cramps in *S. mansoni* infection. Terminal haematuria and dysuria in *S. haematobium* infection.

**D. Late stage**
- This is the stage of fibrosis, which occurs where there are eggs in the tissues. Around the bladder this may result in:
  - Stricture of urethra leading to urine retention or fistula.
  - Dilatation of ureters (hydroureter) and kidney (hydronephrosis) possibly leading to kidney failure
  - Calcification of bladder.
- In the liver portal hypertension leads to hypersplenism and anemia, erosophageal varices and bleeding.
- In the lungs fibrosis results in pulmonary hypertension, which leads to congestive cardiac failure.

**Diagnosis**
- Demonstration of ova in urine or feces,
- Biopsy of urine and feces are repeatedly negative (rectal snip, liver biopsy, bladder biopsy).

**Treatment**
Praziquantel and oxamniquine are the drugs of choice but in Africa praziquantel is best because of resistance strain of oxamniquine.

**Prevention and control**

1. Treatment of cases
2. Intermittent irrigation
3. Drainage of water bodies
4. Clearing of vegetation in water bodies to deprive snails of food and resting place
5. Flooding
6. Straightening and deepening margins of water bodies
7. Educating the public about the mode of transmission and ways of prevention
8. Proper disposal of human feces and urine
9. Avoid swimming in water bodies known to have the infection
10. Use rubber boots to prevent exposure to contaminated water.

### 5.6.2 Guinea Worm Infection

**Definition**

An infection of the subcutaneous and deeper tissues by large nematode.
Infections agent
Dracunculus medinensis, a nematode

Epidemiology

Occurrence- In Africa (16 countries south of the Sahara) and in Asia (India and Yemen) especially in regions with dry climates. Local prevalence varies greatly. In some locales, nearly all inhabitants are infected, in others, few, mainly young adults.

Reservoir- Humans

Mode of transmission- Larvae discharged by the female worm into stagnant fresh water are ingested by minute crustacean copepods (Cyclops species). In about 2 weeks, the larvae develop into the infective stage. People swallow the infected copepods in drinking water from infested step-wells and ponds. The larvae are liberated in the stomach, cross the duodenal wall, migrate through the viscera and become adults. The female, after mating, grows and develops to full maturity, then migrates to the subcutaneous tissues (most frequently of the legs).

Incubation period- About 12 months
**Period of communicability** - From rupture of vesicle until larvae have been completely evacuated from the uterus of the gravid worm, usually 2-3 weeks. In water, the larvae are infective for the copepods for about 5 days. After ingestion by copepods, the larvae become infective for people after 12-14 days at temperatures >25°C and remain infective in the copepods for about 3 weeks.

**Susceptibility and resistance** - Susceptibility is universal. No acquired immunity; multiple and repeated infections may occur in the same person.

**Clinical Manifestation**
- Few or no clinical manifestations are evident until just before the blister forms.
- Fever and generalized allergic symptoms, including periorbital edema, wheezing, and urticaria.
- The emergence of the worm is associated with local pain and swelling.
- When the blister ruptures, the adult worm releases larva-rich fluid and this is associated with a relief of symptoms.
- The shallow ulcer surrounding the emerging adult worm heals over weeks to months.

**Diagnosis**
- Based on clinical and epidemiological grounds
Treatment
1. Gradual extraction of the worm by winding of a few centimeters on a stick each day remains the common and effective practice. Worms may be excised surgically.
2. Administration of thiabendazole or metronidazol may relieve symptoms but has no proven activity against the worm.

Prevention and control
1. Provide health education programs in endemic communities to convey three messages:
   - The guinea-worm infection comes from their drinking water
   - Villagers with blisters or ulcers should not enter any source of drinking water and
   - That drinking water should be filtered through fine mesh cloth to remove copepods
2. Provision of safe drinking water
Review Questions

1. What do you understand by vector-borne disease transmission?

2. Which of the vector-borne diseases pose major health problems in Ethiopia?

3. Except one, others do not require notification to the health authorities
   a) Malaria
   b) Yellow fever
   c) Plague
   d) B and C
   e) Schistosomiasis

4. What are the preventive and control methods for malaria and schistosomiasis?
CHAPTER SIX

SEXUALLY TRANSMITTED DISEASES

6.1 Learning Objectives

At the end of this chapter, the student will be able to:

- List the common sexually transmitted diseases (STDs).
- Identify the diagnostic symptoms of sexually transmitted diseases.
- Identify sexually transmitted diseases that are transmitted through vertical route.
- Apply the management of sexually transmitted diseases.
- State the preventive and control measures for sexually transmitted diseases.

6.2. Introduction

The diseases belonging to this group are usually transmitted during sexual intercourse; hence the name sexually transmitted diseases or STDs. During sexual intercourse there is close body contact, which is an ideal situation for
transmission. The causative organisms of the STDs are very easily killed by drying or by cooling to below body temperature. Therefore transmission of these agents from one person to another can only occur under very special circumstances, mostly during sexual intercourse. STDs are very common in adults, but they are often hidden for fear of the opinion of others. Single young men are a high-risk group for STDs, as they satisfy their sexual needs with women who have many sexual partners (promiscuity). They may be professional prostitutes, barmaids, or persons who in other ways gain from casual sexual relationships. This group is called the promiscuous women pool (PWP). They are the reservoir of STDs.

Risk factors are:

1. Age: 15 years and older
2. Marital status: unmarried people who often change their sexual partners are more frequently exposed. Most of the women in the PWP are unmarried or divorced.
3. Occupation: soldiers, policemen, students, seasonal laborers, and other people who are temporarily away from home tend to expose themselves more easily.
4. Residence: Due to industrialization and consequent urbanization there is usually a large group of single young men in towns. Women in towns may have more difficulty
in earning their daily living than women in rural areas and may take up prostitution for money.

5. Promiscuity

6.3 Syphilis (Hard chancre)

**Definition**
A disease characterized by a primary lesion, a later secondary eruption on the skin and mucus membranes, then a long period of latency, and finally late lesions of skin, bones, viscera, CNS and cardiovascular systems.

**Infectious agent**
Treponema pallidum, a spirochete.

**Epidemiology**
**Occurrence:** Worldwide spread. Primarily involving sexually active young people between 20 and 29 years. More common in urban than rural areas.

**Reservoir** - Humans

**Mode of transmission:** by direct contact with lesion mainly during sexual intercourse. Accidentally by touching infective tissues. Or via blood transfusion. Or congenitally, which may occur before birth, in the case of an infected mother.
**Incubation period** – 10 days to 3 months, usually 3 weeks.

**Period of communicability** – variable and indefinite, during primary and secondary stages and also in mucocutaneous recurrences that may occur during the first 4 years of latency. Extent of communicability through sexual activity during this latent period is not established. Adequate penicillin treatment usually ends infectivity within 24 – 48 hours.

**Susceptibility and resistance** – Susceptibility is universal, although only approximately 30% of exposures result in infection. Infection leads to developing immunity against T. pallidum gradually and to some extent, but immunity usually fails to develop because of early treatment in the primary and secondary stages.

**Clinical Manifestation**

The clinical presentation is divided into three groups:

a) **Primary syphilis** – consists of hard chancre, the primary lesion of syphilis, together with regional lymphadenitis. The hard chancre is a single, painless ulcer on the genitalia or elsewhere (lips, tongue, breasts) and heals spontaneously in a few weeks without treatment.

The lymph glands are bilaterally enlarged and not painful. There will not be suppuration.
b) **Secondary syphilis** - After 4 – 6 weeks of the primary infection, a generalized secondary eruption appears, often accompanied by mild constitutional symptoms. These early rashes tend to be symmetrical, quickly passing, and do not itch. These early skin lesions are highly infective and many spirochetes are demonstrated in them.

c) **Tertiary syphilis** - This stage is characterized by destructive, non-infectious lesions of the skin, bones, viscera, and mucosal surfaces. Other disabling manifestations occur in the cardiovascular system (aortic incompetence, aneurysms) or central nervous system (dementia paralytica, tabes dorsalis).

d) **Syphilis in pregnancy** - According to the severity, congenital syphilis can result in congenital abnormalities, still birth, or repeated spontaneous abortions.

**Diagnosis**
- Serological test – will be positive 6 to 8 weeks after infection
- Dark field microscopy of smears from primary lesion (hard chancre) or from skin lesions in the early secondary stage will show the spirochaetes.
Treatment

1. Primary and secondary syphilis
   - Benzathin penicillin 2.4 M IU Im stat or
   - Tetracycline or Erythromycin 500mg PO Qid for 2 weeks for penicillin sensitive people

2. Tertiary syphilis
   - Benzathin penicillin 2.4 M IU Im single dose every week for 3 consecutive weeks or
   - Tetracycline or Erythromycin for one month for penicillin sensitive individuals.

3. Early congenital syphilis
   - Crystalline penicillin 50,000 IU/ Kg per dose IV or Im bid in the first 7 days of life and Tid then after for 10-14 days.

Prevention and control

1. Treatment of cases
2. Treatment of contacts and source of infection
3. Health education on safe sex
4. Controlling STDs among commercial sex workers
   - Monthly check up and treatment of cases
   - Provision of condom
5. Screening of pregnant women and early treatment to prevent congenital syphilis
6. Screening of blood before transfusion.
6.4 Chancroid (soft chancre)

Definition
An acute bacteria infection localized in the genital area and characterized clinically by single or multiple painful narcotizing ulcers at the site of infection.

Infectious agent.
Haemophilus ducreyi, the Ducrey bacillus

Epidemiology
Occurrence – endemic in many developing countries. The commonest cause of genital ulcer in many developing counties. Most frequently diagnosed in men, especially those who frequently prostitutes.

Reservoir – Humans

Mode of transmission – by direct sexual contact with discharges from open lesion and pus from buboes. Infected males don’t pass the infection farther because of the painful ulcer.

Incubation period – from 3 to 5 days, up to 14 days after sexual contact.

Period of communicability – until healed and as long as the infectious agent persists in the original lesion or discharging
regional lymph nodes, which lasts for several weeks or months without antibiotic treatment. Antibiotic therapy eradicates H. ducreyi, and lesions heal in 1 – 2 weeks.

**Susceptibility and resistance** – Susceptibility is general. The uncircumcised are at higher risk than the circumcised. No evidence of natural resistance.

**Clinical manifestation**
- Classic Chancroid ulcer begins as a tender papule that ulcerates within 24 hours.
- The ulcer is painful, irregular and sharply demarcated from the nearby skin.
- About 50% of men will have single ulcer.

**Diagnosis**
- Clinical, but always rule out syphilis
- Gram stain of smear from ulcer shows typical rods in chain
- Culture

**Treatment**
1. Co-trimorazele or
2. Erythromycin or
3. Tetracycline can be used

**N.B.** Do not incise lymph nodes even with fluctuation because they will completely heal with treatment.
Prevention and control
1. Case treatment
2. Investigation of contacts, source of infection and treatment
3. Thorough washing of genitalia with soap and water promptly after intercourse is very effective.
4. Controlling STDs among commercial sex workers
5. Sex education for high risk groups

6.5 Lymphogranuloma venereum.

Definition
A venereal disease caused by chlamydia microorganisms, most commonly manifested by acute inguinal lymph adenitis.

Infectious agent
Chlamydia trachomatis (L_1, L_2, and L_3)

Epidemiology
Occurrence – Common in most parts of the world but very common in tropical and subtropical regions of Africa and Asia. Its incidence is more common in males than females, and is lower than Gonorrhea and Chancroid.

Reservoir- Humans often asymptomatic (particularly in females)
**Mode of transmission** - Direct contact with open lesions of infected people, usually during sexual intercourse.

**Incubation period** – variable, with a range of 3 – 30 days for a primary lesion.

**Period of communicability** – variable, from weeks to years, during presence of active lesions.

**Susceptibility and resistance** – Susceptibility is general. Status of natural or acquired resistance is unclear.

**Clinical manifestation**
- Lymph adenopathy with non-specific symptoms of fever, chills, head ache, malaise, anorexia and weight loss.
- Regional lymph nodes undergo suppuration followed by extension of inflammatory process to the adjacent tissues.
- In the female, inguinal nodes are less frequently affected and involvement is mainly of the pelvic nodes with extension to the rectum and recto vaginal septum, resulting in proctitis, stricture of the rectum and fistula.
- Elephantiasis of genitalia, scrotum and vulva occur in either sex.

**Diagnosis**
- Clinical presentation (i.e. presence of bubo.)
- Culture of bubo aspirate.
**Treatment**
1. Tetracycline or
2. Erythromycin or
3. Co-trimoxazole can be used
4. Aspiration of fluctuating bubo and wound care

**Prevention and control**
1. Early diagnosis and treatment of cases
2. Investigation of contacts, source of infection and treatment
3. Control STDs among commercial sex workers
4. Sex education for high risk groups

### 6.6 Herpes Genitalia

**Definition**
A viral infection characterized by a localized primary lesion, latency and a tendency to localized recurrence.

**Infectious agent**
Herpes simplex virus (HSV) type 2

**Epidemiology**

**Occurrence** – worldwide. HSV 2 infection usually begins with sexual activity and is rare before adolescence, except in sexually abused children. Prevalence is greater (up to 60%) in
lower socio-economic groups and persons with multiple sexual partners.

**Reservoir** – Humans.

**Mode of transmission** - Usually by sexual contact. Transmission to the neonate usually occurs via the infected birth canal, but less commonly occurs intrauterine or postpartum

**Incubation period** – 2 – 12 Days

**Period of communicability** – Patients with primary genital lesions are infective for about 7 –12 days, with recurrent disease for 4 days to a week. Reactivation of genital herpes may occur repeatedly in > 50% of women.

**Susceptibility and resistance** – Humans are universally susceptible.

**Clinical manifestation**
- First – episode primary genital herpes is characterized by fever, head ache, malaise and myalgias
- Pain, itching, dysuria, vaginal and urethral discharge, and tender inguinal lymph adenopothy are the predominant local symptoms.
Widely-spaced bilateral lesions of the external genitalia are characteristic: lesions may be vesicles, pustules, or painful erythematous ulcers.

Cervix and urethra are involved in more than 80% of women with first episode infection.

A clear mucoid discharge and dysuria are characteristics/symptoms of urethritis.

Occasionally, HSV genital tract disease is manifested by endometritis and salpingitis in women and by prostatitis in men.

**Treatment**

1. Oral acyclovir is effective

**Prevention and control**

1. Consistent use of condom is an effective means of reducing the risk of genital HSV – 2 transmission.

### 6.7 Candidiasis

**Definition**

A mycosis usually confined to the superficial layers of skin or mucus membranes, presenting clinically as oral thrush or vulvovaginitis.

**Infectious agent**

Candida albicans (most common cause)

Candida tropicalis (rare cause)
Epidemiology

Occurrence – Worldwide. Candida albicans is often part of the normal human flora.

Reservoirs – Humans

Mode of transmission – contact with secretions or excretions of mouth, skin, vagina and feces, from patients or carriers. Passage from mother to neonate during childbirth.

Incubation period – variable.

Period of communicability - presumably while lesions are present.

Susceptibility and resistance – Susceptibility is very low except in low host defense. It is common in diabetes, HIV-infected; women are prone to vulvovaginitis in the third trimester of pregnancy. Oral contraceptive users, individuals with prolonged steroid therapy are susceptible.

Clinical manifestation

- Severe vulvar pruritis (prominent feature)
- Vaginal discharge (scanty, whitish, yellow, thick to form curds, non-offensive)
- Sore vulva due to itching
- Speculum examination – thick whitish plugs attached to vaginal wall
vaginal epithium bleeds when the plug is removed but the cervix is normal

**Diagnosis**
- Based on clinical grounds
- Microscopic demonstration of pseudohyphae or yeast cells in infected tissue or body fluids (vaginal discharge)
- Culture (vaginal discharge)

**Treatment**
1. Nystatine vaginal pessary or
2. Miconazole or clotrimazole creams or
3. Ketoconazole or
4. Fluconazole in recurrent cases

**Prevention and control**
1. Case treatment
2. Treatment of underlying medical conditions or predisposing factors

### 6.8 Gonorrhea

**Definition**
An acute or chronic purulent infection of the urogenital tract.

**Infectious agent**
*Neisseria gonorrhoea*, the gonococcus
Communicable Disease Control

Epidemiology

Occurrence – worldwide, affecting both genders, especially sexually active adolescents and young adults. Common in rural areas. Prevalent in communities of lower socio-economic status. In most industrialized countries, the incidence has decreased during the past two decades.

Reservoir - Strictly a human disease

Mode of transmission - almost always as a result of sexual activity

Incubation period - usually 2-7 days

Period of communicability - may extend for months in untreated individuals. Effective therapy ends communicability within hours.

Susceptibility and resistance - Susceptibility is general. No immunity following infection and reinfection is common.

Clinical manifestations

Males- Usually involves the urethra resulting in purulent discharge, dysurea and frequency.

Females - Females are usually asymptomatic. Vaginal discharge is common. Most common site of infection is cervix, followed by urethra, anal canal and pharynx. Bartholinitis occurs unilaterally. Salpingitis as a complication
occurs in 20% of women. Neonates borne to infected mothers
develop a purulent discharge which exudes from between
eyelids which are edematous and erythematous 2-3 days
postpartum.

**Diagnosis**
- Gram stain of discharge (urethral, cervical, conjunctival
discharge)
- Culture on selective media

**Treatment**
1. Co-trimoxazole or
2. Erythromycin or
3. Ceftriaxone can be used

**Prevention and control**
1. The same as syphilis
2. Application of 1% tetracycline in both eyes of newborn as
   soon as delivered.

### 6.9 Trichomoniasis

**Definition**
A common and persistent protozoal disease of the genito-
urinary tract.

**Infectious agent**
Trichomonas vaginalis, a flagellate protozoan
Epidemiology

Occurrence - worldwide spread, a frequent disease of all continents and all races, primarily of adults, with the highest incidence among females 16 - 35 years. Overall, about 20% of females may become infected during their reproductive years.

Reservoir - Humans.

Mode of transmission - by contact with vaginal and urethral discharges of infected people during sexual intercourse. Indirectly through contact with contaminated articles and clothes.

Incubation period - 4 - 20 days, average 7 days. Many are symptom-free carriers for years.

Period of communicability - the duration of the persistent infection, which may last for years.

Susceptibility and resistance - Infection is general, but clinical disease is seen mainly in females.

Clinical manifestation

- Most men remain asymptomatic although some develop arthritis, and a few have epididymitis or prostatitis.
Infection in women is usually symptomatic and manifests with malodorous vaginal discharge often yellow, vulvar erythema and itching dysuria or urinary frequency (in 30 - 50% of cases) and dyspareunia. These manifestations don't clearly distinguish trichomoniasis from other types of infections/vaginitis.

**Diagnosis**
- Detection of motile trichomonads by microscopy of wet mounts of vaginal or prostatic secretions remains the conventional means of diagnosis.
- Culture (most effective) takes 3 - 7 days.

**Treatment.**
1. Metronidazole or
2. Clotrimazole vaginal suppository for pregnant women cures up to 50%.

**Prevention and control**
1. case detection and treatment
2. condom use
3. Educate public to seek medical help whenever there is an abnormal discharge from the genitalia and to refrain from sexual intercourse until investigation and treatment of self and partners are completed.
6.10 HIV/AIDS

Definition
A severe, life-threatening clinical condition, first recognized as a distinct syndrome in 1981. This syndrome represents the late clinical stage of infection with the human immuno-deficiency virus (HIV), which most often results in progressive damage to the immune and other organ systems, including the CNS.

Infections agent
Human immuno-deficiency virus (HIV) (HIV-1 and HIV-2)

Epidemiology
Occurrence - worldwide spread pandemic. HIV-1 infections are now distributed worldwide, but are most prevalent in Sub-Saharan Africa, the Americas, western Europe and southern and Southeast Asia. HIV-2 has been found primarily in West Africa, with some cases in the western hemisphere and other African countries that are linked epidemiologically to West Africa.

The MOH 2002 report depicts the following about the HIV/AIDS situation in Ethiopia:
- The HIV prevalence rate for the country as a whole is estimated at 6.66 percent.
- The estimated HIV prevalence rate for urban areas is 13.7 percent
- Prevalence rates for some urban centers other than Addis Ababa are much higher than the rate for Addis Ababa.
- The estimated rural prevalence rate is 3.7 percent, which is 25 percent of Addis Ababa’s rate.
- HIV seems to be driving the TB epidemic in Ethiopia.
- The highest prevalence of HIV is seen in the age group 15 to 24.

The figure is worrying as it represents “recent” infections. Among the top ten leading causes of deaths, AIDS ranked 9th with 0.8% in 1993 E.C.

**Reservoir** - Humans

**Mode of transmission** – Mainly through sexual exposure and exposure to blood or tissues. Moreover, transplacental transmission from an infected mother to the fetus.

**Incubation period**- variable. Although the time from infection to the development of detectable antibodies is generally 1-3 months, the time from HIV infection to diagnosis of AIDS has an observed range of less than 1 year to 10 years or longer. About half of infected adults will have developed AIDS within 10 years after infection.
**Period of communicability** - unknown. Presumed to begin early after onset of HIV infection and extend throughout life.

**Susceptibility and resistance** - unknown, but susceptibility presumed to be general. Susceptibility is increased in the presence of other STDs, especially those with ulcerations.

**Clinical manifestations**

**Acute HIV syndrome.** Occurs 3 - 6 weeks after primary infection. Clinical findings in the acute syndrome are: fever, pharyngitis, lymphadenopathy, headache, retro-orbital pain, arthralgias, myalgias, lethargy or malaise, anorexia, weight loss, nausea or vomiting or diarrhea. Meningitis, Encephalitis, peripheral neuropathy, myopathy, erythematous maculopapular rash, mucocutaneous ulceration.

**Late complications of HIV infection**
These result from opportunistic infections like pneumocystis carinii pneumonia, Tuberculosis, cryptococcal meningitis, etc.

**Diagnosis**
- Based on clinical ground in the late stage
- Based on serologic test in the early and late stage
Treatment
1. No specific treatment.
2. Treatment of opportunistic infections.
3. Use of anti-HIV drug to reduce transmission of the virus to the fetus of pregnant mothers reduces fetal infection.

Prevention and control
1. prevention and control methods for other STDs
Review Questions

1. What are the common sexually transmitted infections?
2. What is the basic difference in the clinical manifestation of syphilis, Chancroid and Herpes genitalia?
3. What are the common preventive and control methods applicable to all STIs?
CHAPTER SEVEN

ZOOBOOSTIC DISEASES

7.1 Learning Objectives

At the end of this chapter, the student will be able to:

- Define what zoonotic disease means.
- Identify common modes of transmission for each disease.
- Describe the clinical manifestations of each disease.
- Participate in diagnosis and treatment of these diseases.
- Implement preventive and control methods.

7.2 Introduction

Infectious diseases transmitted under natural conditions between vertebrate animals and man are called zoonosis. For most of these diseases, man is a dead-end of the transmission cycle. This means under normal conditions, man will not infect other human beings.
7.3 Food of Animals

7.3.1 Taeniasis

Definition
Taeniasis is an intestinal infection with the adult stage of large tapeworms. Cysticercosis is a tissue infection with the larval stage.

Infectious agent
Taenia saginata (beef tapeworm)
Taenia solium (pork tapeworm)

Epidemiology
Occurrence- Worldwide; frequent where beef or pork is eaten raw or insufficiently cooked and where sanitary conditions permit pigs and cattle to have access to human feces. Prevalent in Latin America, Africa, South East Asia and Eastern Europe.

Reservoir- Humans are definitive hosts of both species of Taenia; cattle are the intermediate hosts for Taenia saginata and pigs for Taenia solium.

Mode of transmission- Eggs of Taenia saginata passed in the stool of an infected person are infectious only to cattle in the flesh of which the parasites develop into “cysticercus
bovis”; the larva stage of Taenia saginata. In humans, infection follows after ingestion of raw or under-cooked beef containing cysticerci; the adult worm develops in the intestine. Taenia Solium eggs to mouth of oneself or to another person or ingestion of food or water infected with eggs-embryos escape from the shells-penetrates the intestinal wall lymphatics or blood vessels and are carried to the various tissues where they develop to produce the human disease of cysticercosis.

**Incubation period**- 8-14 weeks, eggs appear in stool in both species.

**Period of communicability**- T. saginata is not directly transmitted from person to person but T. solium may be. Eggs of both species are disseminated into the environment as long as the worm remains in the intestine, sometimes more than 30 years. Eggs may remain viable in the environment for months.

**Susceptibility and resistance**- Susceptibility is general. No apparent resistance follows infection but more than one tapeworm in a person has rarely been reported.
Life cycle

**COMMUNICABLE DISEASE CONTROL**

**Fig. 7.1 Transmission and life cycle of Taenia solium and Taenia saginata.**
(From Monica Chesbrough, 1998, District laboratory practice in tropical countries, part one, Cambridge University press, London.)

**Cysticercosis:** Infection with T. solium larvae can occur by ingesting eggs in food or from hands contaminated with feces. Eggs develop into cysticerci causing cysticercosis and neurocysticercosis.
Clinical manifestation (for both species)
- Symptoms of cysticercosis may appear after some days and stay for 10 years after infection.
- Passage of proglottid (segmented adult worms) in the feces and perianal discomfort when proglottidis are discharged.
- Minimal or mild abdominal pain or discomfort, nausea, change in appetite, weakness and weight loss.
- Usually asymptomatic.
- Epigastric discomfort, nausea, a sensation of hunger, weight loss, nervousness, and anorexia.
- Passage of proglottidis.

Diagnosis
- Identification of proglottid (segments)
- Eggs in feces or anal swab
- Cysticercus – palpable subcutaneous cysticercus and microscopic examination of an excised cysticercus confirms the diagnosis.
- Intracerebral and other tissues- CT scan, MRI or by x-ray when the cysticerci are calcified.

Treatment
1. Single dose of praziqantel is highly effective or
2. Niclosamide or
3. Dechlorophil or
4. Mebendazole or 
5. Albendazole

**T. Solium**

- Treatment is the same as to T. saginata but praziqantel can evoke an inflammatory response in the CNS if cryptic cysticercosis is present.
- Cysticercosis management
  - Chemotherapy
  - Surgery and supportive medical treatment
- For symptomatic patients with neurocysticercosis, admission is required. Combination of Praziquantel and Albendazole can be used. Besides, high dose of glucocorticoids can be used to decrease inflammation.

**Prevention and control**

1. Educate the public to:
   - Prevent fecal contamination of soil, water, human & animal foods
   - Cook beef and pork thoroughly.
   - Use latrines.
2. Identification and immediate treatment of cases.
3. Freezing of pork/beef below –5°C for more than 4 days kills the cysticerci effectively or cooking to a temperature of 56°C for 5 minutes destroys cysticerci.
4. Deny swine access to latrines and human feces.
7.3.2 Brucellosis

**Definition**
A systemic bacterial disease with acute or insidious onset transmitted to humans from infected animals.

**Infectious agent**
Brucella melitensis (most common worldwide), acquired primarily from goats, sheep and camels.
B. abortus from cattle
B. suis from pigs
B. canis from dogs
These are small aerobic gram-negative bacilli, intracellular parasites.

**Epidemiology**
**Occurrence** - Worldwide. Predominantly an occupational disease of those working with infected animals or their tissues especially farm workers, veterinarians and abattoir workers, which is more frequent among males. Outbreaks can occur among consumers of raw milk and milk products, especially unpasteurized soft cheese from cows, sheep and goats.

**Reservoir** - cattle, swine, goats and sheep, pet dogs.
**Mode of transmission** - by contact with tissues, blood, urine, vaginal discharges, aborted fetuses and especially placentas (through breaks in the skin). Most commonly through ingestion of raw milk and dairy products from infected animals (raw meat or bone marrow). Airborne infection occurs in humans in laboratories and abattoirs.

**Incubation period** - may last about 1-3 weeks but may be as long as several months.

**Period of communicability** - no evidence of communicability from person to person.

**Susceptibility and resistance** - Severity and duration of clinical illness are subject to wide variation. Duration of acquired immunity is uncertain.

**Clinical manifestation**
- Abrupt onset of symptoms
- Most common symptoms are: Fever, chills, diaphoresis, headache, myalgia, fatigue, anorexia, joint and low back pain, weight loss, constipation, sore throat, and dry cough.
- Physical examination reveals
  - Often no abnormalities and patient looks well
- Some are acutely ill, with pallor, lymphadenopathy, hepatosplenomegaly, arthritis, spinal tenderness, epididymoorchitis, skin rash, meningitis, cardiac murmurs, or pneumonia
- Reactive asymmetric polyarthritis (knees, hips, shoulders, sacroiliac and sternoclavicular joints)

**Diagnosis**
- Exposure and consistent clinical features
- Serology- raised levels of B. agglutinin
- Blood or bone marrow culture

**Treatments**
- Doxycycline + aminoglycoside for 2 weeks followed by Doxycycline + Rifampcin for 4-8 weeks is the most effective regimen.
- In pregnancy and in children less than 7 years, Bacterium and Rifapcin for 8-12 weeks

**N:B** 4-14 days after the initiation of therapy, patients become afebrile and constitutional symptoms disappear but enlarged liver and spleen return to normal size within 2-4 weeks.

**Prevention and Control**
1. Control depends on elimination of the disease among domestic animals.
2. Educate people not to drink untreated milk or eat products made from untreated milk.

3. Educate farmers and slaughterhouse workers and those in meat processing plants and butcher shops as to the nature of the disease and the risk in the handling of carcasses and products of potentially infected animals.

4. Educate hunters to use barrier precaution (gloves and clothing).

5. Eliminate infected animals.

6. Pasteurize milk; cook meat and bone well.

7. Proper disposal of placenta, discharges or fetus from an aborted animal. Disinfect contaminated areas.

7.3.3 Trichinellosis or Trichinosis

Definition
A disease caused by an intestinal round worm whose larvae (trichinae) migrate to and become encapsulated in the muscles.

Infectious agent
Trichinella spiralis, an intestinal nematode

Epidemiology
Occurrence - Worldwide, but variable incidence, depending in part on practices of eating and preparing pork or wild animal meat.
Reservoir - swine, dogs, cats, horses, rats and many wild animals, including fox, wolf, etc.

Mode of transmission - By eating raw or insufficiently cooked flesh of animals containing viable encysted larvae, chiefly pork and pork products and "beef products" such as hamburger adulterated either intentionally or inadvertently with raw pork.

Incubation period - Systemic symptoms usually appear about 8 - 15 days after ingestion of infected meat.

Susceptibility and resistance - Susceptibility is universal. Infection results in partial immunity.

Clinical manifestation
- Symptoms result from invasion of the body by larvae produced by the adult female worm in the intestine and from their encystment in striated muscles.
- Infection ranges from symptomatic to mild febrile illness to a severe progressive illness with multiple system involvement.
- Fever (low - high grade)
- Muscle pain mainly upon movement
- Edema, and spasm (periorbital and facial)
- Photophobia and conjunctivitis
- Weakness or prostration
- Pain on swallowing
- Dyspnea, coughing and hoarseness
- Subconjunctival, retinal and nail splinter hemorrhage and rashes
- Diarrhea
- Abdominal cramps
- Nausea and vomiting

Inflammatory reactions around larvae that reach tissues other than muscles may result in:
- Meningitis
- Encephalitis
- Myocarditis
- Broncho-pneumonia
- Nephritis
- Peripheral and cranial nerve disorders

**Diagnosis**
- History of ingestion of raw or inadequately cooked pork
- Larvae in muscle biopsy
- Positive serologic test
- Eosinophilia

**Treatment**
1. Hospitalization of the patient
2. Mebendazole or
3. Albendazole or
4. Thiabendazole
5. High doses of corticosteroids for 1-2 days followed by lower doses for several days or weeks. But not for intestinal stage.

**Prevention and control**
1. Educate the public on the need to cook all fresh pork and pork products and meat from wild animals.
2. Freezing of pork and its products inactivates trichinae.

### 7.3.4 Toxoplasmosis

**Definition**
Toxoplasmosis is a systemic protozoal disease that can be either acute or chronic type with intracellular parasite.

Toxoplasma gondii in which the parasite is responsible for the development of clinically evident disease, including lymphadenopathy, myocarditis and encephalitis.

**Infectious agent**
Toxoplasma gondii
Epidemiology

Occurrence- Worldwide in mammals and birds. Infection in man is common. In the United States and most European countries, the prevalence of sero-conversion increases with age and exposure. In Central America, France, Turkey and Brazil, sero-prevalence is much higher, approaching 90% by age of 40.

Reservoir- The definitive hosts are cats and other felines, which acquire the infection mainly from eating infected mammals (especially rodents) or birds and rarely from feces of infected cats. Only felines harbor the parasite in the intestinal tract where the sexual stages of its life cycle takes place, which result in the excretion of the oocyst in feces for 10-20 days or rarely longer. The intermediate hosts of T. gondii include sheep, goats, rodents, cattle, chicken and birds. Intermediate hosts are man and other animals.

The life cycle can be either heteroxenous (requiring two hosts) or monoxenous (one host). Both sexual and asexual reproduction occur in man.

There are five main developmental forms in the life cycle, but only trophozoites and cyst stages are found in human. All stages occur in the felines (cats).
Toxoplasma has two forms
1. Tachyzoites- occur in the early acute stage of infection.
2. Bradyzoites-occur in the chronic stage of infection, develop slowly and multiply in the tissue to form a true cyst.

Mood of Transmission
1. Ingestion of cysts in raw or under-cooked meat
2. Ingestion of oocysts in food, drink or from hands contaminated with feces of an infected cat.
3. Transplacental/congenital
4. Blood transfusion
5. Organ transplantation

Incubation period- from 10-23 days. One common source outbreak from ingestion of under-cooked meat is possible.

Period of communicability- Not directly transmitted from person to person, except in utero. Oocysts shed by cats sporulate and become infective 1-5 days later and may remain infective in water or moist soil for about a year.

Cysts in the flesh of an infected animal remain infective as long as the meat is edible and uncooked.
Susceptibility and resistance- Susceptibility to infection is general, but immunity is readily acquired and most infections are asymptomatic. Duration and degree of immunity are unknown, but are assumed to be long-lasting or permanent. Antibodies persist for a year, and probably for life. Patient undergoing cytotoxic or immuno-suppressive therapy or patients with AIDS are at risk of developing the disease.

Clinical manifestation

General symptoms: Although severe symptoms may be noted, Toxoplasmosis gondii symptoms are mild and mimic those seen in cases of infectious mononucleosis. The acute form of this disease is characterized by fatigue, lymphadenitis, chills, fever, headache and myalgia. In addition to chronic disease, the patient may develop maculopapular rash, encephalomyelitis and hepatitis; retinochoriditis with subsequent blindness has been known to occur on rare occasions.

Congenital Toxoplasmosis: The typical symptoms in an infected child include hydrocephaly, microcephaly, choreoretinitis, convulsion and psychomotor disturbance. Most of these infections ultimately result in mental retardation, severe visual impairment or blindness.
Diagnosis

- Clinical sign and symptom
- Serological test
- Demonstration of the agent in body fluid or tissue biopsy
- Cell culture

Treatment

1. Treatment is not routinely indicated for a healthy immunocompetent host, except in an initial infection during pregnancy or the presence of active choreoretinitis and myocarditis or other organ involvement.

2. The preferred treatment for those with severe symptomatic disease is: Pyrimethamine combined with sulfadiazine and folinic acid for four weeks.

3. For pregnant women, Spirimycin is commonly used to prevent placental infection. If ultrasound or other studies indicate that fetal infection has occurred, Pyrimethamine and sulfadiazine should be considered.

Treatment for infants

1. Pyrimethamine
2. Sulfadiazine
3. Folinic acid
Prevention and control

1) The cause of primary infection with Toxoplasma can be reduced by avoiding eating under-cooked or raw meat and avoiding cyst-contaminated materials (i.e. cat’s litter box).

2) Meat should be heated to 60°C or frozen to kill cysts.

3) Hands should be washed thoroughly after work in the garden and all fruits and vegetables should be washed.

4) Discourage cats from hunting.

5) Dispose cats’ feces daily.

6) Control stray cats and prevent them from gaining access to sand boxes and sand piles.

7) Educate pregnant women.
   - To avoid cleaning litter pans or contact with cats.
   - Dietary meat; to heat to 60°C or freeze it.
   - To wear gloves during gardening.

8) Blood intended for transfusion into Toxoplasma sero-negative immuno-compromised individuals should be screened for antibody to toxoplasma gondii.

9) Patients with HIV/AIDS who have severe symptomatic toxoplasmosis should receive prophylactic treatment (Prymethamine, sulfadizine, folinic acid) throughout their life span.
7.4 Animal Bite Diseases

7.4.1 Rabies

Definition
It is almost invariably fatal: acute viral encephalomyelitis (attacking brain and meninges).

Infectious agent
Rabies virus

Epidemiology
Occurrence- Worldwide in wildlife particularly in developing countries. It is primarily a disease of animals (zoonotic). It is primarily an infection of carnivores transmitted through bite.

Reservoir- Dog is common in urban areas; in the wild, wild carnivores and bats are reservoirs.

Mode of transmission- Transmitted with saliva of rabid animal introduced by a bite or scratch. Transmission from man to man is dead-ended.

Incubation period- Usually 3-8 weeks

Period of communicability - Usually 3-7 days before the onset of the disease and throughout the course of the disease.
**Susceptibility and resistance**- All mammals are susceptible to varying degrees. Humans are more resistant to infection than several animal species.

**Clinical Manifestation**

The clinical manifestation, which is the same in all species including humans, has 3 phases:

- Prodromal phase
- Excitatory phase
- Paralytic phase

**Prodromal phase:** Onset is heralded by a sense of apprehension, headache, fever and nausea, abnormal sensations at the site of inoculation (bite) is most significant, (i.e. paraesthesia, tingling sensations at the bite site).

**Excitatory phase or Aerophobia:** Slightest sound/wind excites the victim, irritability, restless, nervousness, tendency to bite, are some of the symptoms.

**Paralytic phase:** Spasm of swallowing muscles leads to drooling of saliva and fear of water (hydrophobia). Delirium and convulsions form and death is often due to respiratory muscle paralysis.
Diagnosis

- History of bite by known rabid animal and the bitten person show typical symptoms leading to clinical diagnosis.

Treatment

1. Wound Care
   - Wash the wound with soap and water thoroughly to decrease the viral load.
   - If there is bleeding cover the wound.
   - Never suture the wound as this will spread the virus.

2. Start anti-rabies vaccine immediately if it is proved to be rabid animal bite.

Prevention and control

1) Immunize all dogs and cats.

2) Detain and clinically observe for 10 days any healthy appearing dog or cat known to have bitten a person.

3) Post exposure prophylaxis
   - Treatment of bite wounds
   - Specific immunologic protection

4) Keep dogs and cats at home.

5) Destroy stray animals where rabies is endemic.
7.5 Direct Contact Diseases

7.5.1 Anthrax

**Definition**
An acute bacterial disease usually affecting the skin, but which may very rarely involve the oropharynx, lower respiratory tract, mediastinum or intestinal tract.

**Infectious agent**
Bacillus anthracis, spore forming bacteria.

**Epidemiology:**

**Occurrence**- Worldwide. Primarily a disease of herbivores. Humans and carnivores are incidental hosts. Primarily an occupational hazard of workers who process hides, hair (especially from goats), bone and bone products and wool: and of veterinarians and agriculture and wildlife workers who handle infected animals. Human anthrax is common (endemic) in those agricultural regions of the world where anthrax in animals is common, including countries in South and Central America, southern and eastern Europe, Asia and Africa.

**Reservoir**- Animals, normally herbivores, both livestock and wildlife, shed the bacilli in terminal hemorrhages or spilt blood
at death. On exposure to air, the vegetative forms sporulate, and the spores of B. anthracis, which are very resistant to adverse environmental conditions and disinfections, may remain viable in contaminated soil for many years after the source animal infection has terminated. Dried or processed skins and hides of infected animals may harbor the spores for years and are the fomites by which the disease is spread worldwide.

Mode of transmission-

- Cutaneous anthrax: Contact with tissues of animals (Cattle, sheep, goats, horses, pigs and others) dying of the disease. Bite of flies that had partially fed on such animals, contaminated hair, wool, hides, or products made from them such as drums or brushes or contact with soil associated with infected animals.
- Inhalation anthrax: inhalation of spores in risky industrial processes such as tanning of hides, or wool or bone processing, where aerosols of B. antracis spores may be produced.
- Intestinal and oropharyngeal anthrax: ingestion of contaminated meat; but there is no evidence that milk from infected animals transmits anthrax.

N:B. The disease is transmitted among grazing animals through:
- contaminated soil and feed, and among omnivorous bone meal or other feeds and among wildlife from feeding on anthrax carcasses.
- Vultures have been reported to spread the organism from one area to another.

**Incubation period**- A few hours to seven days; most cases occur within 48 hours of exposure.

**Period of communicability**- transmission from person to person is very rare. Articles and soil contaminated with spores may remain infective for decades.

**Susceptibility and resistance**- uncertain

**Clinical manifestation**

**Cutaneous Anthrax**
- Approximately 95% of human cases of anthrax are cutaneous form and about 5% are the inhalation form.
- Found on exposed areas of skin (head, neck, face and hands).
- Small red macules appear.
- Lesion- progress to papule, vesicle or pustule during the next week and formation of an ulcer with blackened necrotic eschar surrounded by a highly characteristic, expanding zone of brawny edema.
• The early lesion may be pruritic but painless.
• Small satellite vesicle may surround the original lesion and painful non-specific regional lymphadenitis is common.
• Most patients are afebrile with mild or no constitutional symptoms; in severe cases, however, edema may be extensive and associated with shock.
• Spontaneous healing occurs in 80-90% of untreated cases but edema may persist for weeks.
• In 10-20% of cases, infection progresses, bacteria develops and is often associated with high fever and rapid death.

**Inhalation anthrax**
• Presentation of symptoms of severe viral respiratory diseases makes early diagnosis difficult.
• Acute phase supervenes after 1-3 days. With increasing fever, dyspnea, stridor, hypoxia, and hypotension usually leading to death within 24 hours.

**Gastrointestinal Anthrax**- Symptoms are variable and include:
• Fever, nausea and vomiting, abdominal pain, blood, diarrhea, and sometimes rapidly developing ascites.
• Diarrhea is occasional and massive in volume.
Oropharyngeal anthrax
- Fever, sore throat, dysphagia, painful regional lymphadenopathy toxemia, respiratory distress may be evident.
- The primary lesion is most often on the tonsils.

Diagnosis
- Clinical data
- Gram stain of wound discharge
- Culture from the wound discharge or blood

Treatment
For Cutaneous anthrax
1. Penicillin-G IV until edema subsides and with subsequent oral penicillin to complete the course (adults). For Penicillin-sensitive adults, Ciprofloxacin, erythromycin, Tetracycline, Chloramphenicol can be substituted.
2. Clean and cover the cutaneous lesions.

For Inhalation anthrax, Gastrointestinal and Anthrax meningitis
- High dose of penicillin is recommended.

Prevention and control
1. Decontaminate wool and goat’s hair and improvement of working condition for handlers of animal products.
2. Vaccination of susceptible groups and domestic herbivores.
3. Carcasses of animals should be buried intact.
4. Butchering of infected animals should be avoided.
5. Education in mode of transmission and in care of skin abrasions for employees handling potentially contaminated articles.
6. Dust control and proper ventilation in hazardous industries.
7. Treat all animals exposed to anthrax with Tetracycline or penicillin.

7.6 Animal Reservoir Diseases

7.6.1 Leishmaniasis

Definition
A polymorphic protozoan disease of the skin and mucous membrane or a chronic systemic disease caused by a number of species of the genus leishmania.

Infectious agents
For cutaneous and mucosal Leishmaniasis
- Leishmania tropica  Leishmania donovani *
- Leishmania major and Leishmania infantum *
- Leishmania aethiopica*
For visceral Leishmaniasis
- Leishmania donovani. *
- Leishmania infantum. *
- Leishmania tropica. * and
- Leishmania chagasi. *
*Common agents in Ethiopia.

Epidemiology

Occurrence- It occurs in Pakistan, India and recently China, the Middle East including Iran and Afghanistan, southern regions of the former Soviet Union, sub-Saharan Africa, Sudan, the highlands of Ethiopia, Kenya and Namibia. Urban populations including children may be at risk. In the developed world, the disease is restricted to occupational groups, such as those involved in work in forest areas; to those whose homes are in or next to a forest and to visitors to such areas from non-endemic countries. It is common where dog populations are high, generally more common in rural than urban areas.

Reservoirs- locally variable; include human beings, wild carnivores and domestic dogs.

Mode of transmission- Transmission is through the bite of the female phlebotomine (sand flies). From person to person, by blood transfusion, and sexual contact has been reported, but rare.

Incubation period- at least a week; up to many months.
**Period of communicability** - Infectious to sand flies as long as parasites remain in lesion, in untreated cases, usually a few months to 2 years. Eventual spontaneous healing occurs in most cases.

**Susceptibility and resistance** - Susceptibility is probably general. Life-long immunity may be present after lesion due to *L. tropica* or *L. major* but may not protect against other leishmanial species.

**Life cycle**

![Diagram of Leishmania Parasites Life Cycle]

Fig. 7.2 Transmission and life cycle of Leishmania parasites VL: Visceral leishmaniasis, CL: Cutaneous leishmaniasis MCL: Mucocutaneous leishmaniasis. (From Monica Chesbrough, 1998, District Laboratory Practice in Tropical Countries, Part One, Cambridge University Press, London.)
Clinical Manifestation
- There are papules that further develop to ulcers. The disease is characterized by fever, hepatosplenomegaly, lymphadenopathy, anaemia, leucopenia, thrombocytopenia, and progressive emaciation and weakness.

Diagnosis
- Demonstration of the parasite (blood or tissue)
- By culture of the motile promastigote
- Using serologic test

Treatment
Pentalvalent antimonial agents
- Pentamidine or
- Amphotericin or
- Aminoglycoside aminosidine or
- Cytokine immunotherapy

Prevention and control
1. The avoidance of outdoor activities.
2. The use of mechanical barriers such as screens and bed nets.
3. Wearing of protective clothing.
5. Treatment of cases.
6. Vector control and elimination of reservoir host (e.g. domestic dogs).
7.6.2 African Trypanosomiasis

Definition
A systemic disease caused by protozoa characterized by fever followed by general weakness and cerebral involvement leading to death.

Infectious agent
The commonest agents are:
- T. Brucei rhodesiense
- T. Brucei gambiense
Other species which are less important are:
- T. Cruzi, which causes American Trypanosomiasis

Vectors for all species are tsetse flies of Genus Glossina.

Epidemiology
Occurrence-The trypanosomes that cause sleeping sickness are found only in Africa. 20,000 new cases are reported each year. This number surely under-estimates the true incidence. T. brucei gambiense occurs and is widely distributed in the tropical rainforests of Central and West Africa. Gambiense trypanosomes are primarily a problem in rural population; tourists rarely become infected. The principal reservoir of T.B rhodesiense in savanna and woodland areas of Central and East Africa are Trypotolerant antelope species. Humans acquire T.B. rhodesiense infection only incidentally while
working in areas where infected vectors are present. T. ganebie has no animal reservoir. However, T. rhodesiense causes the more severe trypanosomiasis without sleeping sickness. In Ethiopia, the distribution of Trypanosomiasis is mostly found in Jinca, Afar, Setitu Humera, Konso, Moyale, Woito, and Dilla.

**Reservoir**- for T. brucie gambiense it is only humans. For T. brucie rhodensiense the reservoir is dog, cattle, fox, wolf and human beings.

**Mode of transmission**- by the bite of infective Glossina Tsetse fly during blood meal. Congenital transmission can occur in humans. Direct mechanical transmission is possible by blood on the proboscis of Glossina and other man-biting insects, such as houseflies or in laboratory accidents.

**Incubation period**- T. brucei rhodensiense: 3 days to few weeks. T. brucei gambiense: several months up to one year.

**Period of communicability**- The disease is transmitted as long as the parasite is present in the blood of infected person or animal and infected Tsetse fly.

**Susceptibility and resistance**- All persons are equally susceptible for the disease.
Life cycle

**Fig. 7.3 Transmission and life cycle of T.b. rhodesiense and T.b. gambiense.**
(From Monica Chesbrough, 1998, District Laboratory Practice in Tropical Countries, Part One, Cambridge University Press, London.)

**Clinical Manifestation**

**Stage I (Signs & symptoms)**
1. Painful trypanosoma chancre
2. Hematogenous and lymphatic dissemination
3. High body temperature
4. Lymphadenopathy discrete
5. Winter bottom’s sign (classic), painless enlargement of lymph node
6. Malaise
7. Headache
8. Weight loss
9. Edema
10. Hepatomegally and
11. Tachycardia

Stage II
1. Abnormality in CSF
2. Day time somnolence
3. Tremors
4. Parkinson's disease may appear
5. Hypertonia
6. Congestive heart failure
7. CNS disease develops
8. Coma and death

Diagnosis
- Wet blood smear
- Thick blood smear
- Serological test
- CSF analysis
- Blood film
- Bone marrow biopsy
**Treatment**

1. Pentamidine or
2. Eflornithine or
3. Helarsupron or
4. Trypansamide

These are drugs to be used for treatment of different stages.

**For stage I** (Normal CSF) – *T. b. gambie* treated with
- Suramin or
- Eflornithine or
- Pentamidine

**For stage II**
Trypansamide

**Prevention and control**

1. Public education on personal measures to protect against insect bite.
2. Eradication of vectors.
3. Drug treatment of infected humans.
4. Avoiding areas to be known by harboring infected insects.
5. By wearing protective cloth and by using insect repellents.
6. Reducing tsetse fly number by
   - Identifying and studying the breeding habits of local vector
• Selectively clearing the bush and wooden areas especially around game reservoirs, water holes, bridges and along rivers bank
• Using and maintaining insecticide impregnated tsetse fly traps.

7. Spraying vehicles with insecticide as they enter and leave tsetse fly infested areas
8. Prohibit blood donation from those who have visited or lived in endemic areas.
Review Questions

1. List the common zoonotic diseases and their main mode of transmission.

2. Which of the Taenia species are most common in Ethiopia?
   a. Taenia solium
   b. Taenia saginata
   c. Trypanosomiasis
   d. Echinococcus granulosis

3. What are the preventive and control methods for zoonotic diseases?
CHAPTER EIGHT

FOOD-BORNE DISEASES (FOOD POISONING, FOOD-BORNE INTOXICATIONS, FOOD-BORNE INFECTION)

8.1 Learning Objectives

At the end of this chapter, the student will be able to:

- List the common food-borne diseases.
- Identify the difference between food poisoning and food infection.
- Describe the clinical manifestations and possible sources of infection.
- Participate in the diagnosis and management of food-borne diseases.
- Implement the preventive and control methods.

8.2 Introduction

Food-borne diseases, including food-borne intoxications and food-borne infections, are terms applied to illnesses acquired
by consumption of contaminated food. They are frequently and inaccurately referred to as food poisoning. While these terms would include illnesses caused by chemical contaminants (heavy metals and organic compounds), this chapter will cover illnesses caused by toxins elaborated by bacterial growth in the food before consumption (staphylococcus aureus and botulism) and a food-borne infection (salmonellosis).

8.3 Staphylococcal Food Poisoning (Intoxication)

Definition
An intoxication (not infection) of abrupt and sometimes violent onset.

Infectious agent (Toxic agent)
Several enterotoxins of staphylococcus aureus, stable at boiling temperature. Staphylococci multiply in food and produce the toxins.

Epidemiology
Occurrence- Widespread and relatively frequent

Reservoir- Humans in most instances; occasionally cows with infected udders.
Mode of transmission- By ingestion of a food product containing staphylococcal enterotoxin. Foods involved are particularly those that come in contact with food handlers’ hands, either without subsequent cooking or with inadequate heating or refrigeration, (e.g. salad, sandwiches, sliced meat and meat products, pastries, etc.). When these foods remain at room temperature for several hours before being eaten, toxin-producing staphylococci multiply and elaborate the heat-stable toxin. The organisms may be of human origin, from purulent discharges of an infected finger or eye, abscesses, nasopharyngeal secretions.

Incubation period- 30 minutes to 8 hours, usually 2-4 hours.

Period of communicability- not applicable

Susceptibility and resistance- Most people are susceptible.

Clinical Manifestation
- Sudden onset of vomiting and watery diarrhea
- Fever and abdominal cramp
- The intensity of illness may require hospitalization.

Diagnosis
- Group of cases with characteristic acute predominantly upper gastrointestinal symptoms and the short interval
between eating a common food item and the onset of symptoms.

- Culture – staphylococcal recovery ($\geq 10^5$ organisms per gram of food) or detection of enterotoxin from an epidemiologically implicated food item confirms the diagnosis.

**Treatment**
1. Fluid and electrolyte replacement if fluid loss is significant particularly in severe cases.

**Prevention and Control**
1. Educate food handlers in strict food hygiene, sanitation and cleanliness of kitchens, proper temperature control, handwashing, cleaning of finger nails, need to cover wounds on the skin, etc.
2. Reduce food-handling time (initial preparation to service) to an absolute minimum, with no more than 4 hours at ambient temperature. Keep perishable food hot ($>60^\circ C$) or cold (below $10^\circ C$).
3. Temporarily exclude people with boils, abscesses and other purulent lesions of hands, face or nose from food handling.
8.4 Botulism

Definition
A paralytic disease that begins with cranial nerve involvement and progresses caudally to involve the extremities.

Infectious agent (Toxic agent)
Toxin produced by Clostridium botulinum (Neurotoxin)

Epidemiology
Occurrence- Worldwide occurrence. Home-canned foods, particularly vegetables, fruits and less commonly with meat and fish. Outbreaks have occurred from contamination through cans damaged after processing. Commercial products occasionally cause outbreaks but some of these outbreaks have resulted from improper handling after purchase. Food-borne botulism can occur when a food to be preserved is contaminated with spores.

Reservoir- The bacteria is found in the soil and in the intestine of animals.

Mode of transmission- Food ingestion in which preformed toxin is found.
Incubation period- Neurological symptoms of food-borne botulism usually appear within 12-36 hours, sometimes several days, after eating contaminated food.

Period of communicability- not communicable

Susceptibility and resistance- Susceptibility is general.

Clinical Manifestations
- Illness varies from a mild condition to very severe disease that can result in death within 24 hours.
- Symmetric descending paralysis is characteristic and can lead to respiratory failure and death.
- Cranial nerve involvement marks the onset of symptoms; usually produces diplopia, dysphagia. Weakness progresses, often rapidly, from the head to involve the neck, arms, thorax and legs; the weakness is occasionally asymmetric.
- Nausea, vomiting, abdominal pain may proceed or follow the onset of paralysis.
- Dizziness, blurred vision, dry mouth, and occasionally sore throat are common.
- No fever
- Ptosis is frequent.
- Papillary reflexes may be depressed: fixed or dilated pupils are noted in half of patients.
The gag reflex may be suppressed; deep tendon reflexes may be normal or decreased.

- Paralytic illness, severe constipation and urinary retention are common.

**Diagnosis**

- Clinical- afebrile, mentally intact patients who have symmetric descending paralysis without sensory findings.

- Appropriate History.

- Demonstration of organisms or its toxin in vomitus, gastric fluid or stool is strongly suggestive of the diagnosis.

- Wound culture

**Treatment**

1. Hospitalize the patient and monitor closely.

3. Intubation and mechanical ventilation may be needed.

4. Antitoxin administration after hypersensitivity test to horse serum.

5. Emesis and lavage if short time after ingestion of food to decrease the toxin.

**Prevention and control**

1. Ensure effective control of processing and preparation of commercially canned and preserved foods.

2. Education about home canning and other food preservation techniques regarding the proper time,
pressure and temperature required to destroy spores, the need for adequate refrigeration, storage, boiling with stirring home-canned vegetables for at least 10 minutes to destroy botulinal toxin.

3. Canned foods in bulging containers should not be used, eaten or tasted.

8.5 Salmonellosis

Definition
A bacterial disease commonly manifested by an acute enterocolitis.

Infectious agent
Salmonella typhimurium and Salmonella enteritidis are the two most commonly reported.

Epidemiology:
Occurrence- Worldwide

Reservoir- Domestic and wild animals including poultry, swine, cattle, rodents and pets (tortoises, dogs, cats and humans) and patients or convalescents are carriers, especially of mild and unrecognized cases.

Mode of transmission:- ingestion of organisms in food derived from infected food animals or contaminated by feces
of an infected animal or person. Raw and under-cooked eggs and egg products, raw milk and its products, contaminated water, meat and its products, poultry and its products. Consumption of raw fruits and vegetables contaminated during slicing.

**Incubation period** – from 6 – 72 hours, usually about 12-36 hours

**Period of communicability** - extremely variable through the course of infection; usually several days to several weeks.

**Susceptibility and resistance** - Susceptibility is general and increased by achlorhydria, antacid therapy, gastrointestinal surgery, prior or current broad spectrum antibiotic treatment, neoplastic disease, immunosuppressive treatment and malnutrition.

**Clinical manifestation**
- Self limited fever and diarrhea (bloody or dysenteric when colon is involved)
- Nausea, vomiting and abdominal cramp
- Microscopic leukocytosis.

**Diagnosis**
- Blood culture initially
- Stool, culture
Treatment
1. Symptomatic
2. If there is an underlying immunosuppressive disease (conditions like AIDS, lymphoma, immunosuppressive treatment), treat the underlying cause.

Prevention and control
1. Improved animal rearing and animal marketing
2. Quality testing of the known and commonly contaminated foods
3. Avoid consuming raw or partially cooked eggs
4. Wear gowns and gloves when handling stool and urine and handwashing after patient contact.
Review Questions

1. What is the basic difference between food poisoning and food infection?
2. What is the common cause of food infection?
3. How do you prevent and control food poisoning?
CHAPTER NINE

NURSING RESPONSIBILITIES IN
THE MANAGEMENT OF
COMMUNICABLE DISEASES

9.1 Learning Objectives

At the end of this chapter, the student will be able to:

- List the different prevention and control methods for common diseases in Ethiopia.
- Implement the preventive and control measures for each disease category at any level of health care.

The proper nursing management of communicable diseases involves both trying to stop people getting diseases (prevention) and looking after those who have them (treatment and care). The two are frequently close related and doing one without the other is only half the job. The measures based on each disease category are described as follows:
For Oral-fecal transmitted diseases:

- Control of diarrheal diseases including dysentery is only possible when the problem of stool disposal is solved (deep pit latrines in rural areas).
- Providing handwashing facilities at toilets: wash hands after going to toilet, wash hands before cooking or eating.
- Fly control by proper refuse disposal and proper disposal of feces.
  - Screen toilets, cover latrines
  - Screen kitchens and food stores
  - Store left-over food where flies cannot reach it
  - Spray with residual insecticides
- Food should always be properly cooked.
- Raw vegetables and fresh fruits without intact skins should be avoided.
- Milk should be boiled or pasteurized.
- Protection, purification and chlorination of public water.
- Health education based on dangers of bottle-feeding; encourage cup/spoon feeding methods and encourage prolonged breastfeeding.
- Demonstrate prevention of dehydration by homemade soup or salt solution.
- Appropriate treatment of cases
**For air-borne diseases**
- Ventilation removes used air and replaces it with clean air.
- Having too many people in the same room should be avoided. This is especially important in prisons, dormitories, boarding schools, and in urban housing where many people may be forced to live in a single room.
- Health education about personal hygiene
  - To cover the mouth when coughing and sneezing
  - To use a hand kerchief or tissue paper for disposal of nasal secretions and sputum
  - Not to spit on the ground in or outside the house.

**For vector-borne diseases**
- Draining water or ditches, and any accumulation of water around the village or filling in holes and ditches so that water will not accumulate.
- Clearing bush and grass along water banks and in the village.
- All containers likely to hold water are to be collected and disposed.
- No water container should have water in it longer than one week.
- Snails can be controlled by disturbing their habitant through changes in water level, filling or damming habitant, and clearing habitat.
- Mosquito net should be used at night.
- Appropriate treatment of cases and provision of chemoprophylaxis.

For sexually transmitted diseases
- Early diagnosis and treatment: This is the most important measure and not difficult to achieve.
- Patients should be encouraged to bring their contacts (including their husband or wives) for treatment. Failure to do this will result in high numbers of infection.
- Elimination of reservoirs: The reservoir is exclusively human; it includes untreated patients and especially unsuspected infectious persons in the promiscuous women pool (PWP).
- Regular examination and treatment of known prostitutes and other promiscuous women will reduce the reservoir, but will not completely eliminate the risk of infection (contact must be treated).
- Sex education

This is a form of health education and should be directed at the groups at risk, especially: students, soldiers, laborers, etc.
Here it is important to stress:
- Dangers of sexual promiscuity
- The early signs and symptoms of STD
- The possibilities for individual prophylaxis.
- Normal sexual behavior
- The dangers of antibiotic chemoprophylaxis

Suggest periodical check ups for STDs for bar ladies and other women at risk.

For Zoonotic diseases

- Appropriate treatment of cases
- Educate the public to:
  - Prevent fecal contamination of soil, water, human and animal foods
  - Cook beef and pork
  - Use latrines
  - Avoid drinking untreated milk or eating products made from untreated milk
  - Eliminate infected animals
  - Use barrier precaution (gloves and clothing in the handling of carcasses and products of potentially infected animals)
  - Keep dogs and cats at home and immunize them
  - Destroy stray animals where rabies is endemic
  - Bury carcasses of animals intact
  - Vector control and elimination of reservoir host

- Education of workers to control dust by ventilating rooms of hazardous industries where wool and goat’s hair is processed
For food-borne diseases

- Appropriate treatment of cases
- Educate food handlers in strict food hygiene, sanitation and cleanliness of kitchens, proper temperature control, handwashing, cleaning of finger nails, need to cover wounds on the skin, etc.
- Temporary exclusion of people with boils, abscesses and other purulent lesions of hands, face or nose for food handling
- Education about home canning and other food preservation techniques.
- Educate public to avoid eating canned foods in bulging or damaged containers
- Avoid consuming raw eggs or partially cooked ones
- Wearing gowns and gloves when handling stool and urine and handwashing after patient contact
Review Questions

1. State the major modes of disease transmission in Ethiopia.
2. What are the main nursing responsibilities in managing communicable diseases?
# GLOSSARY

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
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<tbody>
<tr>
<td><strong>Albuminuria</strong></td>
<td>Urine containing protein</td>
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<td><strong>Anuria</strong></td>
<td>Cessation of the production of urine</td>
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<td><strong>Biopsy</strong></td>
<td>The removal and examination of tissue from somebody who is ill, in order to find out more about his/her disease.</td>
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<td><strong>Bloating</strong></td>
<td>Full of liquid or gas and therefore abdomen is felt larger than normal in a way that is unpleasant.</td>
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<td><strong>Case</strong></td>
<td>An infected or diseased person or animal having specific clinical, laboratory and epidemiological characteristics.</td>
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<td><strong>Cercariae</strong></td>
<td>The stage of the fluke life cycle that develops from germ cells in a daughter sporocyst. This is the final developmental stage in the snail host, consisting of a body and a tail that aids in swimming.</td>
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<td><strong>Chemoprophylaxis</strong></td>
<td>The administration of a chemical, including antibiotics, to prevent the development of an infection or the progression of an infection to clinical disease.</td>
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<td><strong>Chemotherapy</strong></td>
<td>The treatment of diseases with the use of chemical substances.</td>
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<td><strong>Chronic diarrhea</strong></td>
<td>Diarrhea which persists for more than two weeks.</td>
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<td><strong>Contact</strong></td>
<td>A person or animal that has been in such association with an infected person or animal, or a contaminated environment as to have an opportunity to acquire the etiologic agent.</td>
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<td><strong>Cyst</strong></td>
<td>The immotile stage protected by a cyst wall. In this stage the protozoan is readily transmitted to a new host.</td>
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<td><strong>Epididymoorchitis</strong></td>
<td>Inflammation of testis and epididmis.</td>
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<td><strong>Fomites</strong></td>
<td>A subclass of vehicles including inanimate objects such as articles of clothing which can become contaminated and transmit agents.</td>
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<td><strong>Health</strong></td>
<td>A state of physical, mental and social wellbeing of an individual, not merely the absence of disease or infirmity.</td>
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<td><strong>Health education</strong></td>
<td>The process by which individuals and groups of people learn to behave in a manner conducive to the promotion, maintenance or restoration of health.</td>
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<td><strong>Hematemesis</strong></td>
<td>Vomitus consisting blood.</td>
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<td><strong>Host</strong></td>
<td>A person or other living animal including birds that affords subsistence or lodgment to an infectious agent under natural (as opposed to experimental) conditions. Hosts in which the parasite attains maturity</td>
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</tbody>
</table>
or passes its sexual stage are primary or definitive hosts; those in which the parasite is in a larval or asexual stage are secondary or intermediate hosts.

**Hydrocele**
Accumulation of serous fluid in the scrotum

**Immune individual**
A person or an animal that has specific protective antibodies and/or cellular immunity as a result of previous infection or immunization, or is so conditioned by such previous specific experience as to respond in such a way that prevents the development of infection and/or clinical illness following re-exposure to the specific infectious agent.

**Immunity**
That resistance usually associated with the presence of antibodies or cells having a specific action on the microorganism concerned with a particular infectious disease or its toxin.

**Unapparent infection**
The presence of infection in a host without recognizable clinical signs or symptoms. Unapparent infections are identifiable only by laboratory means such as blood test or by the development of positive reactivity to specific skin tests. (Synonymous:
asymptomatic, subclinical, occult infection).

**Incidence**  
The number of instances of illness commencing or, of persons falling ill during a given period in a specified population. More generally the number of new events (e.g. New cases of a disease in a defined population within a specified period.)

**Infected individual**  
A person or animal that harbors an infectious agent and who has either manifest disease (patient or sick personal) or unapparent infection (see carrier).

**Intermediate host**  
A host for only the larval or sexually immature stages of parasite development.

**Jaundice**  
A syndrome characterized by an increased level of bile pigments in the blood and tissue fluid.

**Lymphadenopathy**  
Enlargement of lymph glands in more than one centimeter for a variety of disease conditions.

**Lymphadenitis**  
Inflammation of the lymphatic vessels.

**Melaena**  
Feces containing blood.

**Merozoite**  
One of the trophozoite released from human red blood cells or liver cells at maturation of the asexual cycle of malaria.
<table>
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<tr>
<td>Microfilaria</td>
<td>A term used for the embryo of a filaria, usually in the blood or tissues of human beings ingested by the arthropod intermediate host.</td>
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<tr>
<td>Miracidium</td>
<td>Ciliated first swimming larva of a trematode, which emerges from the egg and must penetrate the appropriate species of snail in order to continue its life cycle development.</td>
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<tr>
<td>Oocyst</td>
<td>The encysted form of the ookinete, which occurs on the stomach wall of anopheles mosquito species infected with malaria.</td>
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<tr>
<td>Ookinete</td>
<td>The motile zygote of plasmodium species formed microgamete (male) fertilization of a macrogamete (female).</td>
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<tr>
<td>Resistance</td>
<td>The sum total of body mechanisms that interpose barriers to the invasion or multiplication of infectious agents, or to damage by their toxic products.</td>
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<tr>
<td>Source of infection</td>
<td>The person, animal, object or substance from which an infectious agent passes to a host.</td>
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</tbody>
</table>
References

3. Donowitz, 1996, Infection Control in the Child Care Center and Preschool, 3rd edition, Williams Wilkins, USA.

